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Neuromuscular Control During Stair Descent and Artificial Tibial Translation After Acute ACL Rupture

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Background: Anterior cruciate ligament (ACL) rupture has direct effect on passive and active knee stability and, specifically, stretch-reflex excitability.

Purpose/Hypothesis: The purpose of this study was to investigate neuromuscular activity in patients with an acute ACL deficit (ACL-D group) compared with a matched control group with an intact ACL (ACL-I group) during stair descent and artificially induced anterior tibial translation. It was hypothesized that neuromuscular control would be impaired in the ACL-D group.

Study Design: Cross-sectional study; Level of evidence, 3.

Methods: Surface electromyographic (EMG) activity of the vastus medialis (VM), vastus lateralis (VL), biceps femoris (BF), and semitendinosus (ST) muscles was recorded bilaterally in 15 patients with ACL-D (mean, 13.8 days [range, 7-21 days] since injury) and 15 controls with ACL-I during stair descent and artificially induced anterior tibial translation. The movements of stair descent were divided into preactivity, weight acceptance, and push-off phases. Reflex activity during anterior tibial translation was split into preactivity and short, medium, and late latency responses. Walking on a treadmill was used for submaximal EMG normalization. Kruskal-Wallis test and post hoc analyses with Dunn-Bonferroni correction were used to compare normalized root mean square values for each muscle, limb, movement, and reflex phase between the ACL-D and ACL-I groups.

Results: During the preactivity phase of stair descent, the hamstrings of the involved leg of the ACL-D group showed 33% to 51% less activity compared with the matched leg and contralateral leg of the ACL-I group ($P < .05$). During the weight acceptance and push-off phases, the VL revealed a significant reduction (approximately 40%) in the involved leg of the ACL-D group compared with the ACL-I group. At short latency, the BF and ST of the involved leg of the ACL-D group showed a significant increase in EMG activity compared with the uninvolved leg of the ACL-I group, by a factor of 2.2 to 4.6.

Conclusion: In the acute phase after an ACL rupture, neuromuscular alterations were found mainly in the hamstrings of both limbs during stair descent and reflex activity. The potential role of prehabilitation needs to be further studied.

Keywords: anterior cruciate ligament; rupture; acute; neuromuscular control; tibial translation; stairs

Injuries of the anterior cruciate ligament (ACL) happen frequently and concern elite (0.15 injuries per 1000 athlete-exposures) but also recreational athletes.[27,30] Most ACL injuries occur during cutting and pivoting sports and are caused by a noncontact multiplane mechanism because of high knee valgus stress and foot abduction position.[26,32,57] The injury often leads to surgical reconstruction or repair. This applies especially to younger people with high demands on their knees at work or in sports or experiencing persistent giving-way episodes. It is known that patients with an ACL injury show neuromuscular adaptations due to altered sensorimotor control.[20,55] These changes in sensorimotor control are caused by altered afferent inputs to the central nervous system due to the loss of the mechanoreceptors of the native (original) ACL.[46,60] In recent years, studies have reported findings of neuromuscular control in patients after ACL reconstruction by investigating activities of daily living such as stair ambulation[10,23] or sport-specific tasks such as hop landing,[52] both of which demand high levels of neuromuscular control. For example, increased neuromuscular activity amplitude was found in the gluteus maximus of participants (1-18 years postoperatively) with ACL reconstruction compared with healthy controls during stair ascent and descent.[23] A pilot study with participants 1 year after ACL reconstruction reported a significantly increased activity of the semitendinosus (ST) in the surgically treated leg and of the vastus lateralis (VL) in

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the uninvolved leg compared with healthy controls during stair descent.[10] The same authors found a decreased activity in vastus medialis (VM) of the reconstructed knee compared with the uninvolved leg and matched limb of controls.[10]

Neuromuscular alterations in the hamstrings—in terms of increased hamstring activation in the reconstructed leg compared with the contralateral leg—have been found not only in the active population but also in athletes returning to sport after ACL reconstruction.[52] These alterations during landing persisted even 6 months after clearance for return to sport.[52] Moreover, different environmental conditions (cognitive dual-task, unpredictable perturbation of supporting surface) during a step-down task affected athletes' neuromuscular response of thigh muscles and adaptation to perturbed tasks in a negative way.[51]

In contrast, there have been few studies investigating neuromuscular alterations in patients with ACL deficiency with nonoperative treatment only.[43-45,54] To date, only 3 studies have measured ACL deficiency in patients in the acute stage before any rehabilitative intervention.[11,16,24] Women with an ACL rupture (2-11 months after accident) showed altered neuromuscular activation, and in general a lower quadriceps/hamstring ratio, in the injured leg compared with the uninvolved leg during single- and double-leg squats.[54] In that study, surface electromyographic (EMG) signals were collected, and ratios of average EMG amplitude between injured and uninjured sides, as well as between antagonistic muscles on the same side, were compared without prior normalization.[54] A pilot study with male and female participants with an acute ACL deficiency (1-3 weeks after accident) found an activation of only 35% to 50% in the thigh muscles of the injured leg compared with the uninvolved leg (with 100% of activation as reference) during stair ascent.[11] The authors used surface EMG signals during walking on a treadmill at a speed of 5 km/h (1.39 m/s) for submaximal EMG normalization, with stair ascent activity normalized and expressed as a percentage (EMG %) of respective level walking activity.[11] Furthermore, a systematic review reported an increased muscular activity of the hamstrings in patients with acute ACL deficiency during walking, but also a decreased quadriceps activation in the acute stage. A minority of included studies found prolonged duration of activity in patients with ACL deficiency compared with a control group.[48] Normalization procedures varied and included maximum isometric voluntary contraction, peak EMG activity during a gait cycle, mean EMG activity over the entire stride, or deviation from obtained standard muscle activity.[48]

Active joint stability is determined not only by neuromuscular activity (reflex-induced and voluntary activation), but also by muscular reaction times.[28] So far, mainly nonspecific tests, such as hop tests, have been used to test active joint stability. However, testing paradigms that assess neuromuscular control of the knee joint–stabilizing muscles in isolated situations can resemble much more precisely the physiological function of the involved muscle. During anterior tibial translation, the hamstrings, working synergistically with the ACL, respond to a sudden perturbation with a corresponding reflex activation (stretch reflex).[9] Only a few studies have shown that the extent of protective reflex activation of the hamstrings after immediate tibial translation results in increased active joint stiffness.[6,13] To date, neuromuscular control has mainly been assessed in patients with ACL reconstruction during functional or sport-specific tasks. Some authors have also investigated patients with ACL deficiency, but in the chronic stage, usually after nonoperative treatment; some authors have assessed reflex activity in healthy participants. However, literature investigating neuromuscular control and reflex activity in patients with an acute ACL deficiency is limited.

The objective of this cross-sectional study was to compare neuromuscular activity during (1) stair descent and (2) artificially induced anterior tibial translation while standing in patients with acute ACL rupture compared with a matched healthy control group. Based on the literature,[10,11,22,23] we hypothesized that neuromuscular control would be impaired in participants with acute ACL deficiency, meaning that they would show (1) a reduced quadriceps and enhanced hamstring activation and (2) a decreased and prolonged reflex activity of the hamstrings during artificially induced anterior tibial translation compared with healthy controls.

METHODS

The protocol for this cross-sectional study received ethics committee approval, and the study was conducted in accordance with the Declaration of Helsinki.[58] All included patients provided written informed consent.

Sample Size Calculation

The a priori analysis of sample size was performed using the software G*Power (Version 3.1; University of Kiel).[15]

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The effect size (ES) was calculated with data from a study that investigated the effect of maximal fatigue on reflex responses of the ST during the time interval 20 to 40 ms after tibial translation.[4] The difference was 20% with small variability and resulted in a large ES (1.098).[4] Based on this ES and the assumptions made, a total sample size of 30 patients (n = 15 per group) and a power of 0.82 was obtained. The following assumptions were used: protocol of sample size calculation: n = 12; test family = *t* tests; statistical test = *t* test; differences between 2 independent means (2 groups); 2-tailed; type of power analysis = a priori; ES = 0.8; alpha-error probability = .05; power (1 – beta-error probability) = 0.8; number of groups = 2; number of measurements = 1.

Participants

Two groups were recruited for this study. One group consisted of patients with an acute (7-21 days after accident), isolated, complete ACL rupture, ACL deficiency, respectively (ACL-D) without any concomitant injuries, confirmed by clinical examination and magnetic resonance imaging scan. Furthermore, patients were included if they had no acute inflammatory signs, limited swelling (<2 cm difference in the circumferential measurement compared with the contralateral side), no acute pain on visual analog scale (VAS; score, <5 of 10),[50] and an approximately free range of motion in flexion and extension of <20° difference from the contralateral side and were able to walk on even ground and climb stairs without any walking aids such as crutches and orthoses. The patients were recruited in collaboration with Philipp Henle MD, Sonnenhof Orthopaedic Center, Lindenhof Group, Bern, Switzerland, between February 2016 and February 2020. From the initial 19 patients recruited, 4 could not be measured for time reasons (not enough time before surgery or hospital stay), leaving 15 patients in this group.

The control group consisted of 15 healthy matched individuals with an intact ACL (ACL-I). The participants were recruited from local sport clubs and students and collaborators of the Bern University of Applied Sciences. Matching was based on sex, age, body height, body mass, and dominant leg (defined as the preferred leg for kicking a ball).

Inclusion criteria for all participants were age range between 18 and 60 years, being physically active for at least 45 minutes twice per week, and having a minimum Tegner activity score of 5, in which 0 represents disabled and 10 represents professional-level sports.[8,40] Exclusion criteria for all participants were cardiac problems, neurological diseases, peripheral vascular diseases, musculoskeletal complaints, acute infections, alcohol abuse, use of analgesic drugs, knee surgery beforehand, concomitant knee injuries (regarding menisci, medial or lateral collateral ligaments), other concomitant injuries of the lower limb (foot, hip), back pain, trunk injury, thrombosis, and pregnancy.

Measurements

All measurements were performed at the Bern Movement Laboratory. The same setup as for a pilot study was used,[10,11] including a retrospective analysis of pilot data of healthy matched controls.

For all participants, anthropometric data (age, body height, body mass, etc), limb dominance, and data regarding physical activity (type of sports, number of hours per week, Tegner activity score[53]) were collected with the aid of a standardized case report form. Moreover, the Knee injury and Osteoarthritis Outcome Score (KOOS)[41] was completed by every participant. Afterward, the skin was prepared (shaved, smoothed, and cleaned with alcohol) for bipolar, self-adhesive electrodes (Blue Sensor Type P-00-S; Ambu; interelectrode distance, 20 mm), which were applied to the VM, VL, ST, and biceps femoris (BF) muscle of both limbs according to Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles standards.[25] The reference electrode was always placed on the right patella. Then, the interelectrode impedance was controlled (impedance meter D175; Digitimer) and accepted at $\leq$2 kΩ.

After the preparation and before the measurements started, the actual health status (general well-being) and actual pain level of the participants were assessed by using a 100-mm VAS.[47] This was done to test for inconvenience and pain from any measurements or the presence of the participant in our laboratory.

Each participant started with a warm-up on an instrumented treadmill (h/p/cosmos; sports & medical GmbH) for 10 minutes at 5 km/h (1.39 m/s). The initial contact of each gait cycle was detected by 2 force transducers (series KMB52, 10 kN; Megatron Elektronik) that had been mounted under the treadmill. Signals of electromyography of all 4 muscles on both limbs were recorded during walking at a speed of 5 km/h (1.39 m/s) for 2 minutes and were used for submaximal EMG normalization of stretch reflexes and root mean square (RMS) values of the gait phases during stair climbing.[2,3] After this warm-up, each participant completed 2 experimental situations in the same order: a functional task consisting of stair descent (Figure 1) and a physiological experimental task, stretch-reflex measurements induced by artificial tibial translation (Figure 2).

Stair Descent

Participants were asked to descend a 6-step stairway 10 times at a self-selected speed without using the handrails. The custom-made wooden staircase (Figure 1) was composed of 2 integrated multicomponent force plates (type 9286BA; Kistler), which were embedded in the third and fourth step to identify gait cycles during stair climbing. The inclination was 30.6°, with a step height of 17.1 cm and a step depth of 29.0 cm. The configurations for vertical ground-reaction forces had been previously described and had shown adequate reliability.[34]

The cycle of stair descent was divided into the 3 movement phases: preactivity (PRE), weight acceptance (WA), and push-off (PO). The PRE phase was defined from 150 ms before initial foot-floor contact until initial contact with the force platform. The WA phase covered the period from initial contact until the lowest applied vertical ground-reaction force ("braking phase" until anterior-posterior

Figure 1. Experimental setup for stair descent.

force crosses the zero line). The PO phase followed the WA phase until the vertical ground-reaction forces declined to zero ("propulsion phase").

Artificial Tibial Translation

The participants were assessed in a relaxed, upright, bipedal stance with hands on hips and knees in 30° of flexion (Figure 2). To guarantee a standardized standing position, participants stood with each foot on a force plate (type 9286BA; Kistler) and, to ensure equal distribution of the body weight, received visual feedback provided by a computer screen at eye level. In addition, the participants had to wear headphones with music and an attenuator to avoid any kind of acoustic anticipation. An online tool (www.randomization.com) was used to create a list for testing both legs in a randomized order. To evoke standardized tibial translation in the posterior-anterior direction, a reliable rope-and-pulley system[9] was used (Figure 2). A standardized impulse was applied to the tibial shank and monitored by a force transducer (type KM1506, 2 kN; Megatron Elektronik), which was used as a trigger signal for the measurements of onset of tibial translation. As it has been shown that the neuromuscular hamstring reflex response is greatest to a high magnitude of impulse and a high rate of force development,[9] a barbell weight of 2.5 kg and a drop height of barbell weight of 1.0 m were applied.

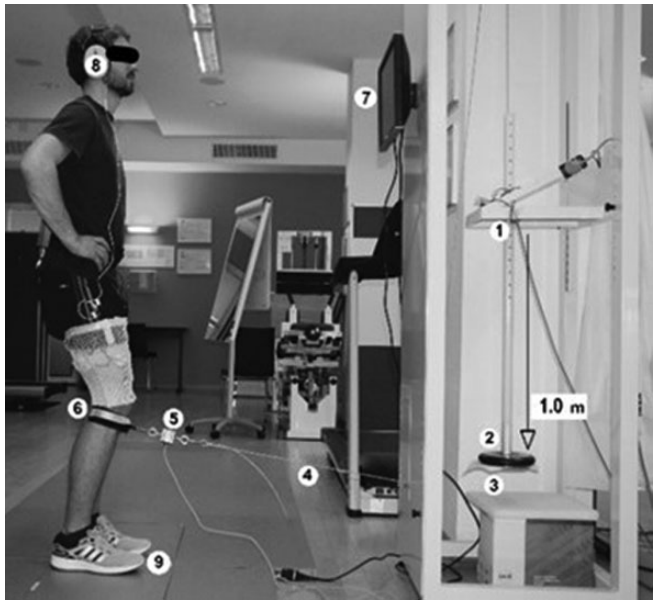

Figure 2. Experimental setup for stretch-reflex measurements (anterior tibial translation): (1) electromagnet, (2) falling barbell weight, (3) stopper, (4) wire rope, (5) force transducer, (6) brace, (7) computer screen for visual control of equal body weight distribution, (8) headphones and attenuator, and (9) 2 force plates.

Artificial tibial translation was elicited 30 times per lower extremity, with a short break after 15 repetitions to avoid excessive co-contraction or muscular fatigue. Between sets, participants were allowed to walk around and relax during the resting intervals (5 minutes). Between each stimulus, a break of about 30 seconds was taken to avoid impulse anticipation and to allow participants to adjust their standing position (interstimulus interval, 20-30 seconds). Basically, the participants were unaware of the point in time of the tibial translation.

After the measurements were completed, the actual health status (general well-being) and actual pain level of the participants were assessed once more by VAS.

Study Outcomes

As primary outcomes, the RMS values of the EMG signals after the activation of the stretch reflex (after onset of tibial translation) were calculated for 4 predefined time intervals: –50 to 0 ms background activity and preactivity (PRE_50); 20 to 40 ms, short latency response (SLR); 40 to 60 ms, medium latency response (MLR); and 60 to 95 ms, late latency response (LLR).[4,5,9] The RMS values of stair descent in the PRE, WA, and PO phases served as secondary outcomes.[2,3]

Signal Transmission, Data Processing, and Normalization

Transmission of the EMG signal occurred across a differential preamplifier (gain, 500; input impedance,

TABLE 1
Comparison of Characteristics Between the ACL-D and ACL-I Groups[a]

Characteristic	ACL-D (n = 15)	ACL-I (n = 15)	*P*
Age, y	32.67 ± 9.53	32.20 ± 6.78	.953
Body height, cm	174.0 ± 7.83	177.73 ± 7.27	.187
Body mass, kg	77.98 ± 12.14	73.55 ± 11.74	.512
Female sex, %	33	33	–
Leg dominance, right/left, n	14/1	14/1	–
Time since injury, d	13.8 ± 5.6	–	–
Physical activity, min/wk	340.67 ± 277.83	265.67 ± 126.36	.775
Preinjury Tegner score (10 points max)	6.20 ± 1.86	5.27 ± 1.10	.202
KOOS total score (168 points max)	102.53 ± 23.30	165.67 ± 1.92	**<.0001**
Pain (36 points max)	24.40 ± 5.73	35.80 ± 0.41	**<.0001**
Symptoms (28 points max)	18.07 ± 4.27	26.13 ± 1.60	**<.0001**
ADL (68 points max)	49.73 ± 10.41	68.0 ± 0.00	**<.0001**
Sport and Recreation (20 points max)	5.20 ± 4.40	19.87 ± 0.35	**<.0001**
HRQoL (16 points max)	5.13 ± 2.50	15.87 ± 0.52	**<.0001**
VAS pain			
Premeasurement	9.40 ± 11.84	0.67 ± 1.63	**.045**
Postmeasurement	17.40 ± 21.20	1.87 ± 3.14	**.016**
VAS well-being			
Premeasurement	14.07 ± 14.52	6.20 ± 8.76	**.037**
Postmeasurement	15.20 ± 15.17	5.73 ± 7.31	**.001**

[a]Data are reported as mean ± SD unless otherwise indicated. Boldface *P* values indicate a statistically significant difference between groups ($P < .05$). Dashes indicate not applicable. ACL-D, anterior cruciate ligament deficient; ACL-I, anterior cruciate ligament intact; ADL, Activities of Daily Living; HRQoL, health-related Quality of Life; KOOS, Knee injury and Osteoarthritis Outcome Score; max, maximum; VAS, visual analog scale.

4000 MΩ; common mode rejection, 90 dB at 60 Hz) to a telemetric main amplifier (PowerPack; Pfitec; band-pass filter, 10 Hz to 1 kHz; gain, 5.0; resultant overall gain, 2500), in which it was recorded at 2000 Hz for walking or stair descent and at 4000 Hz for the stretch-reflex measurement.[35] Then, an analog-digital conversion was conducted (NI PCI 6255; 1.25 ms/s, 16 bit; National Instruments), and finally, LabVIEW-based software (Imago Record; Pfitec) was used to register the signals.

EMG data processing was performed using the same LabVIEW-based software. All corresponding raw EMG signals were full wave rectified. The raw EMG signals from the treadmill and stair descent were additionally band-pass filtered at 10 to 500 Hz (second-order Butterworth).

For interparticipant comparability, RMS values were normalized according to the corresponding time intervals retrieved during level walking. Level walking was expressed as 100% of neuromuscular activity, and reflex responses during tibial translation and stair descent were normalized and expressed as a percentage (EMG %) of respective level walking activity. This kind of evaluation has been used for related research.[2,3]

The RMS values were then exported into Excel spreadsheets (Windows 10; Microsoft Corp), in which individual means of 10 strides, 10 steps (stair descent), and 30 tibial translations per extremity for each muscle in each time interval were calculated and checked for plausibility. Individual values >2 standard deviations (SDs) for stair descent and >3 SDs for reflex measurement were traced back to the original data set and corrected if possible.

Statistical Analysis

Data were transferred from the case report forms to Excel spreadsheets and later processed with SPSS software (Version 27; IBM Corp) and RStudio software (Version 4.1; PBC). Testing of normal distribution of the data was performed with the Shapiro-Wilk test. Most of the variables were not normally distributed. However, to allow comparison with other studies, means and SDs are reported. The Mann-Whitney *U* test was used to compare participant characteristics. To test for significant ($\alpha = .05$) differences in nonparametric data between the ACL-D and ACL-I groups, a Kruskal-Wallis analysis of variance (ANOVA) (rank-based ANOVA, post hoc analyses with Dunn-Bonferroni correction) was conducted for between-group and within-group comparisons for the 4 recorded muscles (VM, VL, BF, and ST), the 3 different movement phases (PRE, WA, and PO) during stair descent, and the 4 reflex time windows (PRE_50, SLR, MLR, and LLR) during artificial tibial translation. The ES was calculated with the rank epsilon-square test, where 0.2 = small effect, 0.5 = medium effect, and 0.8 = large effect.[12]

RESULTS

Characteristics of patients and healthy controls are displayed in Table 1. There were no significant differences in age, body height, body mass, physical activity, and Tegner score between the ACL-D and ACL-I groups. However, the

TABLE 2
Between-Group and Within-Group Comparisons of Normalized RMS Values (%) for Stair Descent During the PRE, WA, and PO Phases[a]

	Stair Descent: PRE										
	ACL-D		ACL-I (Control)		*P*						
Muscle	(1) Involved	(2) Uninvolved	(3) Involved	(4) Uninvolved	ANOVA	(1) vs (2)	(1) vs (3)	(1) vs (4)	(2) vs (3)	(2) vs (4)	(3) vs (4)
VM	122.9 ± 50.8	144.6 ± 62.9	121 ± 41.4	113 ± 45.2	.389	—	—	—	—	—	—
VL	124.1 ± 55.6	155.5 ± 67.7	113.9 ± 33.5	108 ± 38.4	.059	—	—	—	—	—	—
BF	68.3 ± 32.8	81.8 ± 25.2	122.1 ± 34.7	140.8 ± 45.3	**<.0001**	>0.99	**.0009** ES = 1.5	**<.0001** ES = 1.8	**.02** ES = 1.3	**.0002** ES = 1.6	.96
ST	87.1 ± 34.4	71.5 ± 22.9	134.7 ± 32.7	130.8 ± 35.5	**<.0001**	>0.99	**.001** ES = 1.4	**.003** ES = 1.2	**<.0001** ES = 2.2	**<.0001** ES = 1.9	>.99
	Stair Descent: WA										
	ACL-D		ACL-I (Control)		*P*						
Muscle	(1) Involved	(2) Uninvolved	(3) Involved	(4) Uninvolved	ANOVA	(1) vs (2)	(1) vs (3)	(1) vs (4)	(2) vs (3)	(2) vs (4)	(3) vs (4)
VM	181.7 ± 70.6	193.4 ± 107.7	261.8 ± 97	222.2 ± 104.6	.149	—	—	—	—	—	—
VL	154.4 ± 61.4	160.4 ± 54.9	254.5 ± 114.9	200.6 ± 69.2	**.010**	>.99	**.019** ES = 0.9	.736	**.034** ES = 0.9	>.99	.765
BF	60.9 ± 40.4	67.1 ± 35.9	98.7 ± 55.6	92.4 ± 44.6	.119	—	—	—	—	—	—
ST	56.9 ± 27.2	42.1 ± 27.1	56.2 ± 47.2	75.6 ± 44	.139	—	—	—	—	—	—
	Stair Descent: PO										
	ACL-D		ACL-I (Control)		*P*						
Muscle	(1) Involved	(2) Uninvolved	(3) Involved	(4) Uninvolved	ANOVA	(1) vs (2)	(1) vs (3)	(1) vs (4)	(2) vs (3)	(2) vs (4)	(3) vs (4)
VM	193.6 ± 110.8	293.8 ± 126.5	231 ± 127.2	268.9 ± 125.3	.206	—	—	—	—	—	—
VL	149 ± 87.4	247 ± 54.5	177.6 ± 97.7	206.4 ± 83.2	**.018**	**.017** ES = 0.1	>.99	.401	.168	>.99	>.99
BF	53.4 ± 23.5	78.1 ± 40	83.6 ± 35.5	73.9 ± 51	.1	—	—	—	—	—	—
ST	71.8 ± 30	51.9 ± 20.2	64.3 ± 35.3	54.5 ± 30.2	.229	—	—	—	—	—	—

[a]Stair descent data are reported as mean ± SD. Boldface *P* values indicate a statistically significant difference between and within groups as indicated ($P < .05$). Dashes indicate not applicable. ACL-D, anterior cruciate ligament deficient; ACL-I, anterior cruciate ligament intact; ANOVA, analysis of variance; BF, biceps femoris; ES, effect size; involved, injured leg and respective matched leg of controls; PO, push-off; PRE, preactivity; RMS, root mean square; ST, semitendinosus; uninvolved, uninjured leg and respective matched leg of controls; VL, vastus lateralis; VM, vastus medialis; WA, weight acceptance.

2 groups differed significantly in KOOS total scores and on each of the KOOS subscales ($P < .0001$ for all).

Stair Descent

During stair descent, post hoc analysis comparing neuromuscular activity revealed significant differences in the VL and the hamstrings in some of the movement phases. All data regarding comparisons are displayed in Table 2 and are graphically shown in Figure 3. During PRE, significantly less neuromuscular activity was found in the hamstrings of the ACL-D compared with the ACL-I group: the BF of the ACL-D involved leg had 44% less activity than the ACL-I involved leg ($P = .0009$; ES = 1.5) and 51% less activity than the ACL-I uninvolved leg ($P < .0001$; ES = 1.8). The ST of the ACL-D involved leg showed 35% and 33% less neuromuscular activity versus the ACL-I involved leg ($P = .001$; ES = 1.4) and ACL-I uninvolved leg ($P = .003$; ES = 1.2), respectively. During the WA phase, VL activity showed a 39% reduction in the ACL-D involved leg versus the ACL-I involved leg ($P = .019$; ES = 0.9). And during PO, there was 40% less activity in the VL of the involved leg compared with the uninvolved leg of ACL-D participants ($P = .017$; ES = 0.1).

Artificial Tibial Translation

All data regarding comparisons of reflex activity during anterior tibial translation are displayed in Table 3 and are graphically shown in Figure 4. At PRE_50, ANOVA revealed overall significant differences for the VM and VL ($P = .043$ and .002, respectively) but not for within- and between-group comparisons (Table 3). During SLR, the hamstrings revealed significant differences ($P = .009$ for BF and $P = .002$ for ST) with ES values of 0.2 (for BF) and 0.4 (for ST) between the ACL-D involved leg and ACL-I

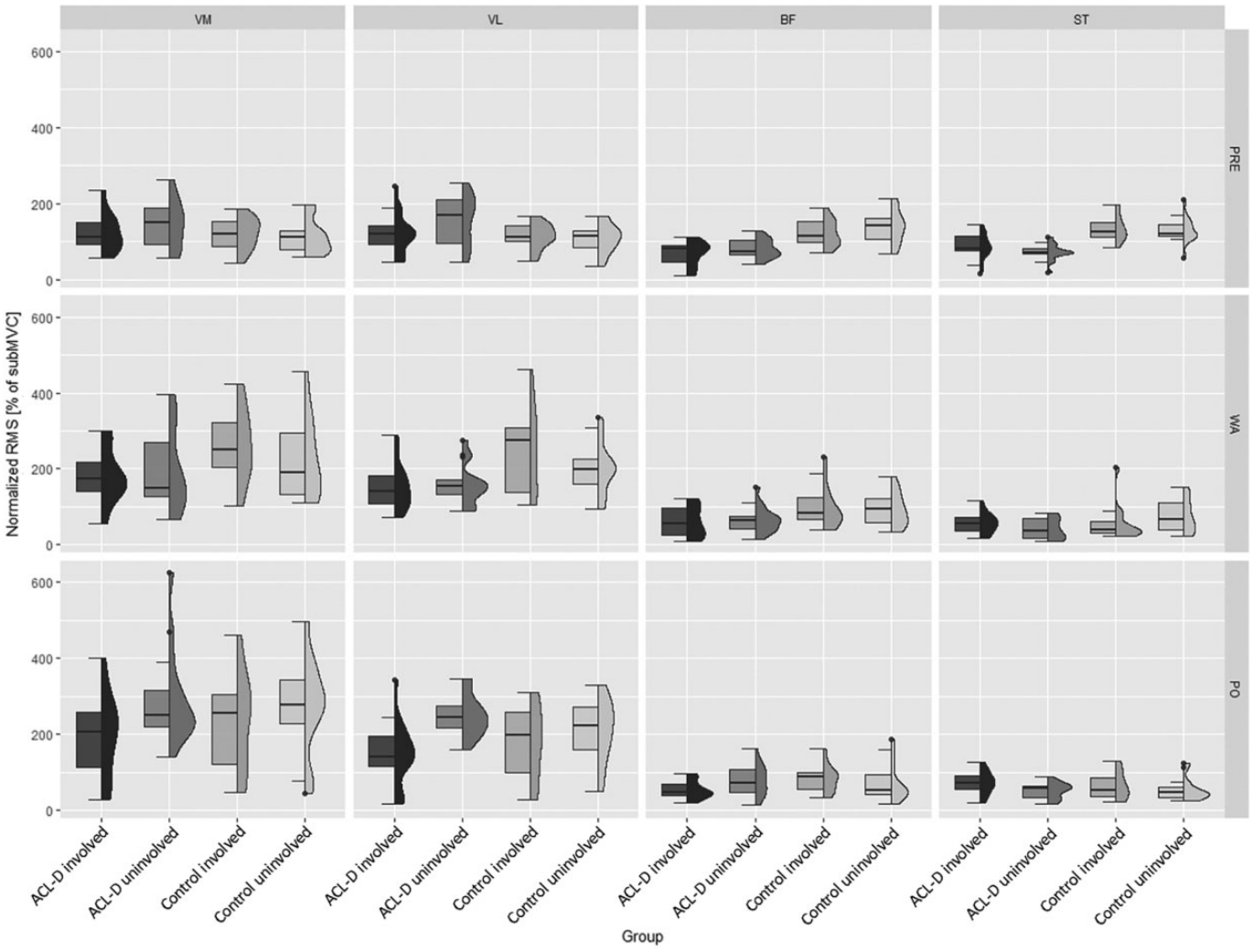

Figure 3. Violin plots of normalized root mean square (RMS) values for the involved and uninvolved legs of the participants in the anterior cruciate ligament–deficient (ACL-D) group and the matched respective legs of the control participants (ACL-I) in the 3 phases of stair descent: preactivity (PRE), weight acceptance (WA), and push-off (PO). BF, biceps femoris; involved, injured leg and respective matched leg of controls; ST, semitendinosus; %subMVC, percentage of submaximal values of maximum voluntary contraction; uninvolved, uninjured leg and respective matched leg of controls; VL, vastus lateralis; VM, vastus medialis.

uninvolved leg. Post hoc analysis of neuromuscular activity revealed that the BF of the ACL-D involved leg had an increase of 168% compared with the ACL-I uninvolved leg ($P = .006$; ES = 0.3), and the ST of the ACL-D involved leg showed an increase in activity of 221% compared with the ACL-I involved leg ($P = .04$; ES = 0.2) and an increase of 460% compared with the ACL-I uninvolved leg ($P = .005$; ES = 0.4). There were no significant between-group or within-group differences in reflex activity for any muscle during the MLR and LLR time windows.

DISCUSSION

So far, neuromuscular control has mainly been assessed in patients with ACL reconstruction during functional or sport-specific tasks. Only a few publications have reported neuromuscular control of patients with an ACL deficiency, but in the chronic stage, and usually after nonoperative treatment. In addition, some researchers assessed reflex activity in healthy participants. However, literature investigating neuromuscular control and reflex activity in patients with an acute ACL deficiency is limited. Therefore, this cross-sectional study investigated neuromuscular activity in patients with an acute ACL rupture during stair descent as a functional task and an artificially induced tibial translation while standing in comparison with a healthy matched control group. Beforehand, it was hypothesized that neuromuscular control would be impaired in the acute ACL-D group, meaning that the participants with acute ACL injury would show a reduced quadriceps and hamstring activation and a decreased and prolonged reflex activity of the hamstrings during tibial translation compared with healthy matched controls.

TABLE 3
Between-Group and Within-Group Comparisons of Normalized RMS Values (%) for Reflex Activity During the PRE_50, SLR, MLR, and LLR Reflex Time Windows[a]

	Reflex Activity: PRE_50										
	ACL-D		ACL-I (Control)		*P*						
Muscle	(1) Involved	(2) Uninvolved	(3) Involved	(4) Uninvolved	ANOVA	(1) vs (2)	(1) vs (3)	(1) vs (4)	(2) vs (3)	(2) vs (4)	(3) vs (4)
VM	124.1 ± 52.1	113.8 ± 46.1	172.5 ± 84.2	165.6 ± 77.1	**.043**	>.99	.32	.57	.12	.23	>.99
VL	140.5 ± 28.6	108.4 ± 29.7	174.9 ± 73.3	154.6 ± 31.6	**.002**	.455	.317	>.99	**.001** ES = 1.1	.062	>.99
BF	76.5 ± 48	71.6 ± 45.2	78.4 ± 40.4	73.1 ± 50.6	.938	—	—	—	—	—	—
ST	44.5 ± 26.8	43.9 ± 31.2	34.7 ± 27.1	44.7 ± 43.9	.648	—	—	—	—	—	—
	Reflex Activity: SLR										
	ACL-D		ACL-I (Control)		*P*						
Muscle	(1) Involved	(2) Uninvolved	(3) Involved	(4) Uninvolved	ANOVA	(1) vs (2)	(1) vs (3)	(1) vs (4)	(2) vs (3)	(2) vs (4)	(3) vs (4)
VM	171.9 ± 102.3	202.1 ± 116.7	157.6 ± 74.4	148.6 ± 52.1	.655	—	—	—	—	—	—
VL	126.9 ± 40	167.3 ± 97.6	156.9 ± 60.6	167.5 ± 88.2	.572	—	—	—	—	—	—
BF	439 ± 321.6	239.1 ± 136.5	204.9 ± 139.1	163.7 ± 105	**.009**	.893	.128	**.006** ES = 0.3	>.99	.42	>.99
ST	456.5 ± 419.6	318.1 ± 253.2	141.9 ± 173.6	81.4 ± 637	**.002**	>.99	**.04** ES = 0.2	**.005** ES = 0.4	.349	.068	>.99
	Reflex Activity: MLR										
	ACL-D		ACL-I (Control)		*P*						
Muscle	(1) Involved	(2) Uninvolved	(3) Involved	(4) Uninvolved	ANOVA	(1) vs (2)	(1) vs (3)	(1) vs (4)	(2) vs (3)	(2) vs (4)	(3) vs (4)
VM	410.3 ± 223.8	395.6 ± 266.8	367.8 ± 196.3	326.8 ± 168.9	.865	—	—	—	—	—	—
VL	414.2 ± 195.2	322.3 ± 108.1	311.9 ± 168.3	289.7 ± 161.1	.276	—	—	—	—	—	—
BF	473.5 ± 210.9	354.4 ± 176.4	376.6 ± 299.8	348.8 ± 350.8	.103	—	—	—	—	—	—
ST	432 ± 226.2	231 ± 211	443.3 ± 396.8	273.2 ± 226.7	.124	—	—	—	—	—	—
	Reflex Activity: LLR										
	ACL-D		ACL-I (Control)		*P*						
Muscle	(1) Involved	(2) Uninvolved	(3) Involved	(4) Uninvolved	ANOVA	(1) vs (2)	(1) vs (3)	(1) vs (4)	(2) vs (3)	(2) vs (4)	(3) vs (4)
VM	334.2 ± 263.1	277.9 ± 233.9	242.3 ± 120.9	272.8 ± 183	.909	—	—	—	—	—	—
VL	245.9 ± 107.5	231.7 ± 167.7	208.1 ± 94.5	259.3 ± 154.9	.69	—	—	—	—	—	—
BF	233.9 ± 146.6	229.4 ± 132.3	241.7 ± 134.4	258 ± 154.1	.968	—	—	—	—	—	—
ST	229 ± 150.2	133.7 ± 87.9	203.7 ± 173.4	178.5 ± 116.1	.471	—	—	—	—	—	—

[a]Reflex activity data are reported as mean ± SD. Boldface *P* values indicate a statistically significant difference between and within groups as indicated ($P < .05$). Dashes indicate not applicable. ACL-D, anterior cruciate ligament deficient; ACL-I, anterior cruciate ligament intact; ANOVA, analysis of variance; BF, biceps femoris; ES, effect size; involved, injured leg and respective matched leg of controls; LLR, late latency response; MLR, medium latency response; PRE_50, preactivity; RMS, root mean square; SLR, short latency response; ST, semitendinosus; uninvolved, uninjured leg and respective matched leg of controls; VL, vastus lateralis; VM, vastus medialis.

In general, we found a huge range of differences in activity patterns within and between groups in the investigated time frames and movement phases, indicating a large intraindividual variability, especially during artificial tibial translation. The hypothesis that neuromuscular control would be negatively affected in the ACL-D group was confirmed. This was the case on one hand by a significant decrease of hamstring activity during all 3 phases of stair descent and a decreased VL activity in the WA and PO phases and on the other hand by a larger neuromuscular activity of the hamstrings at SLR during artificial tibial translation.

The findings implicate bilateral consequences in the early, acute stage after ACL rupture. Therefore, it is questionable to use limb symmetry indices as objective outcomes throughout rehabilitation or even as return-to-sport criteria, even though they are widely used. A study with participants with ACL-D (acute injury; mean, 23 days after ACL rupture) revealed a significantly reduced quadriceps

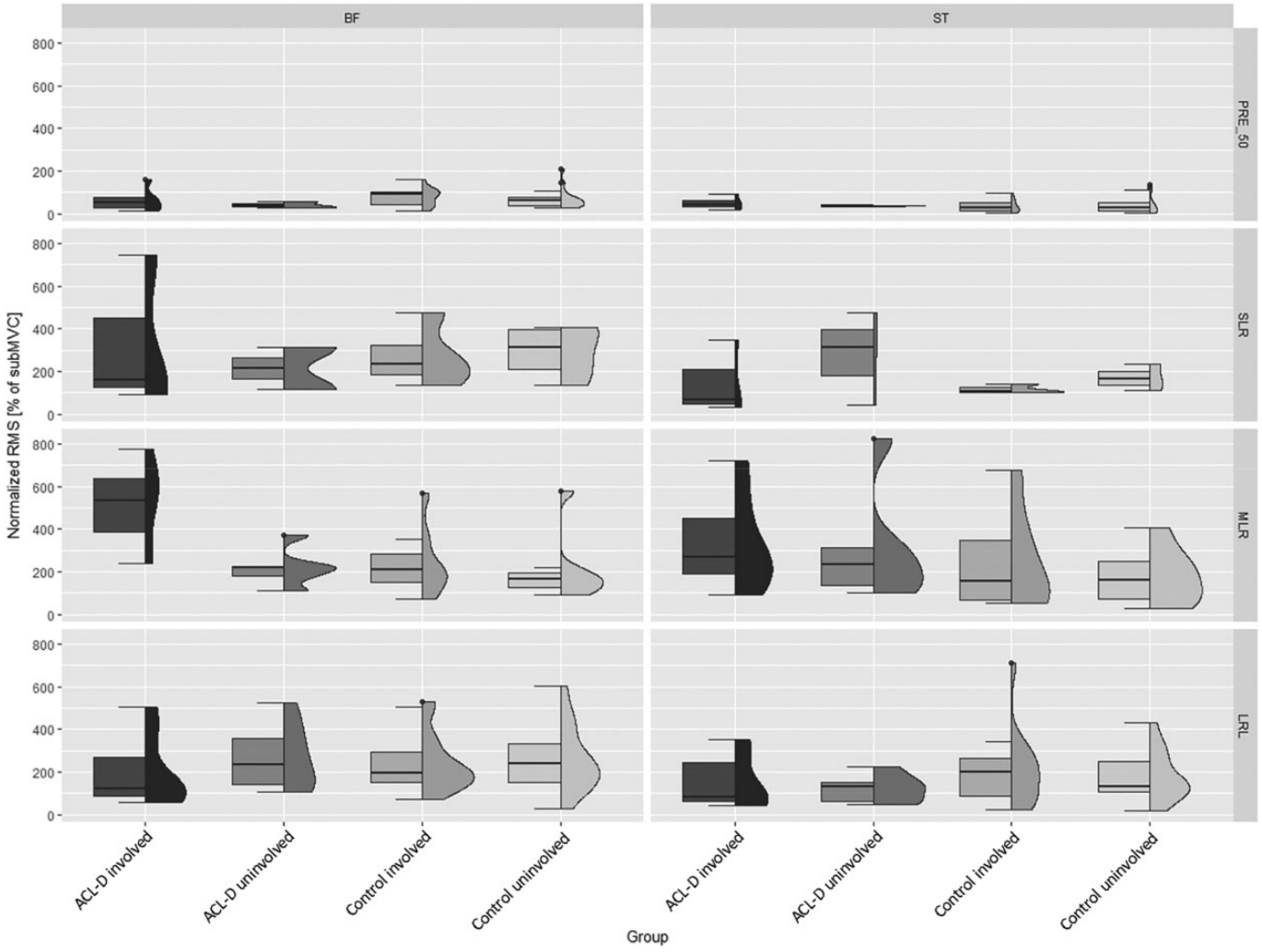

Figure 4. Violin plots of normalized root mean square (RMS) values of the hamstring muscles for the involved and uninvolved legs of the participants in the anterior cruciate ligament–deficient (ACL-D) group and the matched respective legs of the control participants (ACL-I) in the 4 reflex time windows: preactivity in time window from 50 ms before tibial translation (PRE_50), short latency response (SLR), medium latency response (MLR), and late latency response (LLR). BF, biceps femoris; involved, injured leg and respective matched leg of controls; ST, semitendinosus; %subMVC, percentage of submaximal values of maximum voluntary contraction; uninvolved, uninjured leg and respective matched leg of controls.

strength capacity of the uninvolved limb compared with the dominant leg of a healthy control group during a concentric contraction.[24] These results support the findings in the present study. Consequently, the uninvolved limb might not represent the condition of a healthy person, leading to an overestimation of knee function after ACL injury by using limb symmetry indices.[56] However, more research is needed to further develop and test evidence-based return-to-sport and competition criteria, as well as associated guidelines.[1,56] This aspect of bilateral alterations might be also used to conduct prehabilitation before surgery. After reconstruction, early rehabilitation with training interventions for the uninjured leg should be used to achieve an effect in that leg as well.[11] In addition, bilateral learning strategies and cross-educational effects should be further considered.[36]

Stair Descent

For stair ambulation, a high level of neuromuscular control is needed. The quadriceps is essential to decelerate body weight and to actively control flexion in the knee joint when descending stairs. In addition, the hamstrings are considered the most important muscle group to support the ACL in its knee-stabilizing function by limiting anterior tibial translation.[37,49] Tibial translation is controlled by hamstrings group 1 afferents in healthy persons, affirming that early onset of reflex activity of the hamstrings is crucial for a stable knee joint.[18] Furthermore, ACL load is significantly reduced with higher hamstring activity, which is far more effective than reduced VL activity.[49] Consequently, the activation of hamstrings can potentially reduce shear forces in the knee joint of a patient with ACL-D.[49] However,

these findings are from computer modeling and cadaveric studies but have been supported by studies evaluating strength, reflexes, and activity of hamstrings in healthy humans.[18,21]

The decreased neuromuscular activity of all recorded muscles—with only the hamstrings showing significant changes—of the ACL-D compared with the ACL-I group during PRE can be interpreted that either the ACL rupture itself leads to an immediate change of the individual motor program or the short period of $\leq$3 weeks between rupture and measurements might have altered the preprogrammed movement pattern. This reduced neuromuscular activity may be explained by the sensorimotor control loop and result in a "loss of neurosensory information eventually leading to an altered central movement program."[11] Several research groups who investigated brain activity after ACL reconstruction found altered cortical activation, indicating changes in the central nervous system, even years after surgery.[20,59] This neuroplasticity of the central nervous system provides further possible explanations for changed neuromuscular activity, which could also have affected our findings. However, to date and to our knowledge, there are no such investigations of brain activity with participants after an ACL rupture in the acute stage. Only 1 study was found with patients $\geq$6 months after ACL rupture[31] who showed reorganization of the central nervous system, suggesting that an ACL rupture should be seen as a "neurophysiologic dysfunction, not a simple peripheral musculoskeletal injury."[31] Hence, reduced neuromuscular activity before injury might be a risk factor for ACL rupture and cannot be ruled out by our data. Therefore, future studies should aim to confirm the findings from this study and, furthermore, consider subsequent changes in brain activity after an ACL rupture in the acute stage. Moreover, the adaptive possibilities of the central nervous system could be used during rehabilitation to prevent secondary injuries after an ACL rupture or reinjuries after ACL reconstruction.[23,31]

Artificial Tibial Translation

Reflex activity of the hamstrings after tibial translation is considered to contribute to knee joint stiffness. The direct reflex arc from the ACL to the hamstrings with a latency of 70 ms is not sufficiently fast to produce a substantial protective force for the ACL.[14] However, it is assumed that this ligament-muscle reflex is involved in the coordination of muscle activity and alteration of movement patterns.[14] Thus, after an ACL rupture, there might possibly be differences in proprioceptive information of the knee joint with subsequent alterations of muscle activity of the knee's surrounding muscles because of the lack of information from ACL afferents.[9] However, the biphasic reflex measured by our setting in the standing position seems to arise from the direct stretch of the hamstrings and may be sufficient to compensate for ACL deficiency.[17] After muscle fatigue, reduced hamstring SLR in association with increased tibial translation has been reported in a similar setting,[5] indicating a fatigue-related reflex reduction. This reduction might imply that a patient experiencing increased anterior tibial translation caused by ACL rupture needs more neuromuscular activity to stabilize the knee joint. Therefore, the increased reflex activity of the hamstrings as SLR and MLR (the latter not significant) might reflect an active compensation pattern of the hamstrings for higher, acute injury–related stability demands. In the case of ACL deficiency, a reduction of MLR was found to be related to "giving way" symptoms in the first few weeks after injury.[38] Therefore, sufficient reflex response in the MLR time frame is essential and seems to be the case for BF and ST when compared with the uninvolved limb and the limbs of healthy controls in our study.

During artificial tibial translation in the standing position, equal distribution of body mass between right and left was monitored, and the knees were kept at 30° of flexion. However, the anterior and posterior directions of the knee joint were uncontrolled, and besides "standing upright with hands on hips," no further instruction for the trunk position was given. If the body mass is shifted more onto the heel or forefoot, the center of mass changes and results in a more extended or flexed spine, pelvis ante-/retroversion, or trunk inclination/reclination, respectively.[7,33,42] This altered, individual upright starting position could influence the activity of the hamstrings and could have led to a different neuromuscular strategy. Furthermore, the band-sling was placed at the proximal part of the shank over the triceps surae muscle, which could influence the activation of the hamstring muscles due to mechanical stimuli in triceps surae 1a and 2 afferent pathways.[9]

Strengths and Limitations

This study is one of the few publications reporting neuromuscular activity of thigh muscles in patients with an acutely injured ACL shortly after the accident. Moreover, to our current knowledge, it is the first study that has investigated reflex response after artificially induced tibial perturbation in this patient group. Another strength of this study is the relatively homogeneous patient sample, considering the diagnosis of an isolated complete ACL rupture without concomitant injuries. This strict diagnosis was chosen because, for example, patients with the frequent additional diagnosis of a bone bruise or subchondral fracture of the lateral femoral condyle show clinically relevant limitations[29]: they have longer-lasting and greater swelling, take longer to walk pain-free without crutches and achieve full range of motion in the knee joint, and report higher pain scores 1, 2, 3, and 4 weeks after injury compared with patients with an isolated ACL rupture.[29] Therefore, concomitant injuries were an exclusion criterion as these limitations would have hindered or even precluded the measurements. Furthermore, the matching was well-balanced, showing that no significant between-group differences regarding sex, age, body mass, body height, physical activity per week, Tegner score, and leg dominance were found. The fact that the patient group and the control group differed significantly in the overall KOOS and in each of its subscales is not surprising. Since the KOOS is used to assess self-reported pain, other symptoms, impairments in activities of daily living, sports and leisure, and

health-related quality of life[41] in patients with knee problems, it could be assumed that the healthy control group would have no or hardly any problems and would score higher in all subscales.

Several study limitations should be considered. First, the overall sample size of 30 participants was rather small and limits the generalizability of the findings. In addition, only 15 patients after an ACL rupture in the acute stage could be included, despite the long recruitment phase of 4 years. This was mainly because of the strict inclusion and exclusion criteria, especially regarding the diagnosis (no concomitant injuries; isolated, complete rupture), functional status (able to descend stairs without using handrails or crutches), and short time frame between injury, recruitment, and medical appointment, as well as the date of surgery. Therefore, comparisons with other studies should be made with caution. Nevertheless, the results expand new fields of research in an interdisciplinary setting to better understand the short- and long-term consequences of an ACL rupture, especially for neuromuscular control.[11] Second, sex-specific differences might have influenced the results.[5] In this study, subgroup analysis for sex was not applicable with this small number of men and women. Third, the participants were allowed to descend the stairs at self-selected speed. This method was also used in another study with patients who had experienced an acute ACL rupture, but it has implications for the recorded EMG activities during exercise.[19] In addition, a prescribed, standardized stride frequency changes the normal gait pattern decisively.[39] Fourth, an effusion of up to a 2-cm circumference difference compared with the uninjured leg was accepted for inclusion. However, effusion might diminish quadriceps activation and could therefore influence the neuromuscular activity, especially in this early stage after injury. Fifth, electromyography during normal walking was recorded for normalization of EMG data of stair descent and tibial translation. It has been shown that electromyography during walking is enhanced in patients with ACL injury.[48] Thus, the normalized EMG data might be influenced by the effects of ACL rupture on stair descent and tibial translation as well as the effects of ACL rupture on walking.

CONCLUSION

The present study, investigating neuromuscular activity in participants with acute ACL deficiency and healthy matched controls, revealed that already in the first 3 weeks after an ACL rupture, neuromuscular alterations can be found mainly in the hamstrings during stair descent and reflex activity. These findings indicate an alteration in the preprogrammed activity and in the underlying voluntary movement control pattern. Furthermore, neuromuscular control seems to be impaired in both extremities after unilateral ACL rupture.

This is clinically relevant, as alterations of neuromuscular control in both legs in the acute phase of an ACL rupture emphasize the need for prehabilitation to possibly achieve better results in the long term after reconstruction or nonoperative treatment. Therefore, conservative therapy with a health professional specialized in sport physical therapy with an early focus on neuromuscular control and treatment of the injured but also the uninjured leg before surgery is strongly recommended. In addition, prehabilitation could also contribute to the decision-making process of whether to reconstruct the ACL or consider a nonoperative treatment pathway. However, recommendations mainly concern rehabilitation standards after reconstruction and do not focus on early treatment before surgery.

Future researchers should focus especially on the aspect of bilateral deficits after ACL rupture. In addition, research in the future should investigate whether the contralateral leg can be an adequate reference for functional evaluation of the injured extremity. As the limb symmetry index is still widely used but probably underestimates the functional deficits of both lower extremities, other outcome measures for neuromuscular function should be considered when deciding for clearance for a safe return to sport.

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