Original Article

Neuromechanical characteristics in the knees of patients who had primary conservative treatment for a torn cruciate ligament and reconstruction afterward

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Background/Purpose: To compare the neuromechanical characteristics and subjective outcomes for knees of patients with a cruciate ligament tear and reconstruction with those for knees of controls at three time intervals, and to determine correlations between the characteristics and subjective outcomes.

Methods: Ten participants with a cruciate ligament tear and at least a 12-week conservative treatment prior to ligament reconstruction were prospectively measured prior to and 3 months and 6 months after surgery. Ten healthy individuals were recruited as controls. Questionnaire
Introduction

Bilateral quadriceps arthrogenic muscle inhibition (AMI) is often observed in knee joints with unilateral cruciate ligament lesions or reconstruction.1–3 Quadriiceps AMI on the ipsilateral side of knee injuries involves changes in afferent discharge due to pain, joint swelling, laxity, inflammation, and mechanoreceptor damage,4–7 which may also inhibit the contralateral quadriceps musculature.8 Quadriiceps AMI is synomimic to failure of voluntary muscle activation and is demonstrated by strength impairments, including maximal strength,9 fast force capacity,10 and fatigue resistance of the knee extension.3 Impairments in maximal knee extension strength in anterior cruciate ligament (ACL) reconstruction are explained by voluntary activation defects in the quadriceps muscles by an interpolation twitch technique11 or an increased hamstring coactivation.12 Nevertheless, the interpolation twitch technique is not capable of analyzing activation contributions of individual quadriceps to knee extension forces.13,14 Decreases in the absolute rate of force development (RFD) of knee extension in the ACL reconstruction implied impairments in fast force output.10,15 However, neural factors in quadriiceps AMI representing the ability to rapidly activate muscles were not assessed. Furthermore, decreases in electromyography (EMG) median frequency in the vastus medialis (VM) muscle were observed in patients with cruciate ligament reconstruction.7 This finding suggests that there were changes in neural strategies and endurance in the VM muscle 5 weeks postoperatively.3,16,17 Nevertheless, correlations between the median frequency and the endurance of the knee, such as total work,18 were not assessed. Collectively, previous studies provide preliminary information regarding the neural strategies of quadriiceps and hamstring muscles interacting with extension force in a knee with a cruciate ligament defect or reconstruction. This information implies a neuromechanical model involving the neural control and the production of joint motion in relation to biomechanical principles19–21 is helpful to design an optimal rehabilitation program for quadriiceps AMI.

Research shows that almost two-thirds of the primarily conservatively treated ACL ruptures need operative reconstruction in the long term,22 although there were equal functional scores in conservatively treated patients and ACL reconstruction.23 There are clinical needs to assess the characteristics of the neural contribution to force capacities at different pre- and postsurgery intervals in a group who had conservative treatments prior to a reconstruction surgery for a cruciate ligament lesion. The hypotheses of this prospective study, which recruited participants who had antecedent conservative treatment for their unilateral torn cruciate ligaments, were: (1) there are changes in the neuromechanical characteristics (neural strategies of thigh muscles and force capacities of knee extension), or patients’ opinions about their knees at different time intervals close to their surgeries (prior to surgery and 3 months and 6 months after surgery); (2) there are correlations between the neural strategies, force capacities, or subjective outcomes; and (3) there are differences in the neural strategies and force capacities between the knees of the patients and those of the controls.

Methods

Patients

This study’s protocol was approved by the Institutional Review Board of the National Taiwan University Hospital, Taipei, Taiwan (Reference No. 201007065R). All patients provided written informed consent, and the rights of the patients were protected. The authors recruited participants who were: (1) aged 20–45 years and had unilateral torn cruciate ligaments [ACL or posterior cruciate ligament (PCL)] in the knee joint; and (2) candidates for nonoperative ACL or PCL deficiency management.24,25 The primary criteria for nonoperative management include no concomitant knee ligament or meniscal damage, and that the patients must have completed at least 12 weeks of primary conservative treatment for their ACL24 or PCL25 ruptures.
and decided to have reconstructive surgery afterward. This nonoperative rehabilitation consists of exercises in range of motion, muscle strengthening, endurance, and agility. The exclusion criteria include that participants, prior to the reconstruction, showed evidence of: (1) concurrent injury to the lateral collateral ligament or a Grade II or III medial collateral ligament tear; (2) osteoarthritis on radiographic evaluation; (3) articular cartilage lesions with exposed bone observed during arthroscopy; (4) meniscal tears that required repair; or (5) lack of compliance to a 16-week postsurgery rehabilitation program. One surgical group was invited to join the study and the surgeons primarily used the arthroscopy technique with an ipsilateral semitendinosus and gracilis tendon autograft to reconstruct a ruptured knee cruciate ligament and recommended a 16-week criteria-based rehabilitation protocol after the reconstruction. The control participants were matched with the physical characteristics of the study participants with ligament reconstruction but had no history of knee pain or injury that would cause them to seek medical treatment or surgery. They were recruited from charity organizations that volunteered at the same hospital. Ten controls and 10 patients with unilateral ligament reconstruction who met the inclusion criteria were recruited (Table 1). The nonoperative ACL or PCL participants decided to have the ligament reconstruction due to ipsilateral knee instability. Arthroscopic assisted transportal double-bundle ACL reconstruction and transtibial double femoral-single tibial PCL reconstruction techniques with semitendinosus and gracilis tendon autografts were performed, respectively, on the ACL and PCL deficient participants. No participants were excluded from the study. The participants were prospectively tested in three sessions, with the first occurring 1 day prior to the operation and the second and third occurring 3 months and 6 months postsurgery, respectively. The controls were assessed within 1 week of recruitment. Data for both sides in the control group were averaged to represent the results of the outcomes.

Study setting

Prior to the measurements, participants filled the knee injury and osteoarthritis outcome score (KOOS) questionnaire in Mandarin to depict their knee pain, symptoms, and quality of daily life activities and then warmed up on a stationary bike for 10 minutes. During the assessment, participants first lay on the examination table, and active EMG recording electrode pads (TSD150B; Biopac, Santa Barbara, CA, USA) were placed to record myoelectrical signal amplitudes from the surface of the VM and vastus lateralis (VL) muscles. Then, the participants lay prone (face down), and electrode pads were placed on the semitendinosus muscle medially placed halfway between the gluteal fold and the knee joint. A reference electrode was placed over the patella. The skin was prepared and a portable machine (Sierra II; Cadwell Laboratories Inc, Kennewick, WA, USA) was used to ensure that interelectrode resistance was < 5 kΩ. These active recording electrodes were connected to an interface (HLT-100C) of the MP100 system (Biopac), and the MP100 system was connected to a computer on which AcqKnowledge 3.8 acquisition software (Biopac) was installed (Fig. 1). Signals were amplified from the surface electrodes (stainless steel disk diameter = 11.4 mm, disc spacing = 20 mm, impedance = 100 MΩ, gain = 350), band-pass filtered from 20 Hz to 500 Hz, and sampled at 1500 Hz with a common rejection ratio of 95 dB. A 60 Hz notch filter was used to eliminate background noise from electrical power sources. After an electrode placement, each participant sat on an isokinetic dynamometer chair (Biodex Multi-Joint System 4; Biodex Medical System Inc., Shirley, NY, USA) and was then secured with straps to stabilize the trunk and was positioned at 90° of flexion at the hip. The dynamometer’s mechanical axis of rotation was aligned with the lateral condyle of the knee to be evaluated.

To record the fast force capacity and maximal isometric strength of the quadriceps muscles, the tested knees were evaluated in the above sitting position with a 60° knee flexion. The participants were instructed to contract their quadriceps muscles as fast and as forcefully as possible and to maintain their maximal voluntary isometric contraction (MVIC) for 5 seconds, followed by a 2 minute rest between each trial. Trials with an identified visible drop in the force signal were discarded and replaced by additional trials. After 10 minutes of rest, 30 repetitions involving knee isokinetic extension were performed to record the total work of the knee extension. The tested knees were evaluated with a motion ranged from a 90° knee flexion to full extension at an angular velocity of 240 °/s. The participants received standardized verbal encouragement: “Exert your maximal efforts in repetitive knee extension,” for all contractions. To ascertain the antagonist coactivation from the semitendinosus co-contraction during knee extension, each patient also performed 5 seconds isometric contractions of knee flexion and relaxation.

During the offline analysis, the EMG signals were digitally high-pass filtered using a fourth-order, zero-lag Butterworth filter (cutoff frequency of 20 Hz) and then a moving root-mean-square filter with a time constant of 50 ms using the MATLAB 7.1 software (MathWorks, Natick, MA, USA). In this study, raw EMG data were used instead of normalized EMG data because normalization could conceal effects of neuromuscular systematic adaptations. The onset of EMG integration was set 70 ms prior to the onset of torque. The mean rate of EMG rise (RER: the slope of ΔEMG/Δtime) at 0–30 ms and 0–75 ms of the VM muscle were defined from the onset of EMG integration. The mean root mean square (RMS) EMG amplitudes of the VM muscle were quantified for 5.0-second epochs.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Patient physical characteristics.</th>
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</thead>
<tbody>
<tr>
<td>Ligament reconstruction</td>
<td>Healthy control</td>
</tr>
<tr>
<td>Sex</td>
<td>8 male/2</td>
</tr>
<tr>
<td>Female</td>
<td>24.6 (4.0)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>170.9 (9.2)</td>
</tr>
<tr>
<td>Body height (cm)</td>
<td>68.2 (12.9)</td>
</tr>
<tr>
<td>Body weight (kg)</td>
<td>Data are presented as means (standard deviation).</td>
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</table>
corresponding to the plateau level of knee extension MVIC. The median frequency of the VM muscle at the 5-second MVIC was determined through a fast Fourier transformation of 512 points (Hamming window processing), which was used to analyze the power density spectrum. The antagonist coactivation of the semitendinosus muscle was calculated as the ratio of RMS EMG obtained during maximal knee extension divided by RMS EMG of the muscle performing a maximal knee flexion contraction.

The torque signal was smoothed by using a technique described in a previous study and onset of voluntary contraction was determined when torque exceeded 7.5 Nm. The absolute RFD was derived as the average slope of the torque time curve at 0–30 ms and 0–100 ms. A normalized RFD was determined as the RFD relative to the maximal torque. Total work was defined as the sum total of areas under all the torque curves in the 30 repetitions at 240/s.

Statistical analysis

All data were averaged over at least three trials, except the KOOS survey. All data are presented as means with standard deviation. The Friedman test was used to determine the significance between three time intervals (prior to surgery and 3 months and 6 months after surgery) in the injured and uninjured knees. The Mann–Whitney U test was used to analyze the values between the injured and uninjured or control knees. Correlations between the neural variables and performance were assessed using the Spearman rank correlation test. Data were analyzed using SPSS 16.0 software (SPSS Inc, Chicago, IL, USA), with the a level set at 0.05.

Results

Tables 1 and 2 summarize the physical characteristics and injury history including the side, the ligament reconstructed, the interval between ligament ruptures and reconstruction, and the participation period in primary nonoperative and postsurgery rehabilitation in these 10 participants and controls. There was no significant finding regarding the neural strategies of the thigh muscles or force capacities of the knee extension between the three time intervals (prior to surgery and 3 months and 6 months after surgery) in the injured and uninjured knees. The Mann–Whitney U test was used to analyze the values between the injured and uninjured or control knees. Correlations between the neural variables and performance were assessed using the Spearman rank correlation test. Data were analyzed using SPSS 16.0 software (SPSS Inc, Chicago, IL, USA), with the a level set at 0.05.

<table>
<thead>
<tr>
<th>Patient</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ligament reconstruction</td>
<td>left ACL</td>
<td>right ACL</td>
<td>left ACL</td>
<td>left ACL</td>
<td>right ACL</td>
<td>right PCL</td>
<td>right ACL</td>
<td>right ACL</td>
<td>right PCL</td>
<td>right PCL</td>
</tr>
<tr>
<td>Interval from rupture to reconstruction (mo)</td>
<td>16</td>
<td>8</td>
<td>3</td>
<td>5</td>
<td>48</td>
<td>7</td>
<td>39</td>
<td>9</td>
<td>5</td>
<td>21</td>
</tr>
<tr>
<td>Participation in primary nonoperative treatment (wk)</td>
<td>14</td>
<td>16</td>
<td>12</td>
<td>16</td>
<td>12</td>
<td>18</td>
<td>18</td>
<td>14</td>
<td>13</td>
<td>19</td>
</tr>
<tr>
<td>Participation in postoperative treatment (wk)</td>
<td>16</td>
<td>18</td>
<td>12</td>
<td>18</td>
<td>16</td>
<td>24</td>
<td>24</td>
<td>16</td>
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</table>
MVIC knee extension results in the knee with an ACL reconstruction were greater than the results at 3 months postsurgery (152.4 ± 46.4 Nm vs. 132.5 ± 47.5 Nm, p = 0.012); and (2) that the KOOS questionnaire score results at 6 months postsurgery regarding knee quality of life (59.4 ± 22.4 vs. 43.8 ± 18.0, p = 0.005) and function in sport and recreation (78.8 ± 17.9 vs. 48.8 ± 34.5, p = 0.003) were greater when compared, respectively, to the results at the presurgery and 3 months postsurgery timepoints. In addition, the results indicate that there were significant differences in the neural strategies of the thigh muscles and the force capacities between injured and uninjured knees, between injured and control knees, and between uninjured and control knees (Figs. 2 and 3). The correlation tests show that the neural strategies related to muscle activation (RMS EMG and RER) of the VM muscle and antagonist coactivation with the semitendinosus muscle are associated with the force capacities of the knee extension, including maximal force exertion (MVIC torque), fast force capacity (RFD), and total work (R ranged from 0.317 to 0.653), in the participants with unilateral cruciate ligament tear/reconstruction (Table 2). In addition, in the knees with cruciate ligament tear/reconstruction, the KOOS scores regarding function in sport and recreation were correlated to the fast force capacities (RFD; Table 3). The pain, other symptoms, function in daily living, and function in sport and recreation were correlated to total works of knee extension (R ranged from 0.454 to 0.600). Finally, the median frequency of the VM muscle was correlated to the knee related quality of life by the KOOS score (Table 4).

**Discussion**

This prospective study shows that participants who had nonoperative ACL or PCL deficiency management had, from the time prior to surgery to 6 months after surgery, lower force capacities of the knee extension and altered neural strategies in activating the VM and semitendinosus muscles in the injured knees when compared with those in the uninjured knees or controls. Furthermore, our results show correlations between neural strategies and force capacities in two knees of participants with unilateral cruciate ligament tear/reconstruction. These neuromechanical characteristics (neural strategies and force capacities) were associated with the symptoms and function in the knees with cruciate ligament lesion and reconstruction. These findings partially support our hypotheses and provide important information on nonoperative ACL or PCL deficiency management and on the treatment after reconstruction surgery.

The first part of our findings regarding neural strategies in the VM and semitendinosus muscles demonstrates: (1) lower activation (RMS EMG and RER) during maximal contraction and explosive contraction of knee extension in the VM muscle; (2) greater and less antagonist coactivation...
in the semitendinosus muscle in the injured and uninjured knees, respectively; and (3) changes in the muscle fiber type or recruitment of motor units (median frequency) in the VM muscle.34 A previous study with a unilateral ACL deficit using the interpolation twitch technique observed bilateral activation defects in both injured and uninjured knees.11,17 Although both studies recruited patients who undertook primary conservative treatment after a cruciate ligament lesion, our study has further observed defects of maximal voluntary activation in the VM muscle. Our RER results concerning the VM muscle in our participants additionally imply that during explosive contractions, there were bilateral AMI or decreases in the motor unit firing frequency or delays in the recruitment of the motoneurons.31 These bilateral neuromechanical defects may be related to bilateral proprioceptive defects, increases of the effectiveness of tonic descending inhibition and central mechanism adjustments to the bilateral fusimotor-muscle-spindle system.1–3 However, similar findings were not found for the VL muscle (data not shown). Correlation tests showed that the RMS EMG and the RER of the VM muscle were associated with the peak isometric torque and the RFD, respectively. These are consistent with previous studies.6,15 A decrease in maximal or fast muscle activation in the VM muscle may be caused by knee pain, swelling, laxity, inflammation etc.,4,7 and may be related to the anterior knee pain after the ACL or PCL injuries or reconstruction surgery.35

Table 3  Correlation analyses between force capacity and neural control in patients with cruciate ligament reconstruction.

<table>
<thead>
<tr>
<th>Force capacity</th>
<th>Neural strategy</th>
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<tbody>
<tr>
<td>Maximal knee extension torque (Nm)</td>
<td>RMS EMG VM (R = 0.518, p &lt; 0.001)</td>
</tr>
<tr>
<td>Total work (J)</td>
<td>Antagonist coactivation (R = −0.317, p = 0.006)</td>
</tr>
<tr>
<td>Absolute RFD 0–30 ms (Nm/s)</td>
<td>MF VM (R = 0.274, p = 0.046)</td>
</tr>
<tr>
<td>Absolute RFD 0–100 ms (Nm/s)</td>
<td>RER 0–30 VM (R = 0.514, p &lt; 0.001)</td>
</tr>
<tr>
<td>Normalized RFD 0–30 ms (%MVC/s)</td>
<td>RER 0–30 VM (R = 0.653, p &lt; 0.001)</td>
</tr>
<tr>
<td>Normalized RFD 0–100 ms (%MVC/s)</td>
<td>RER 0–75 VM (R = 0.484, p &lt; 0.001)</td>
</tr>
</tbody>
</table>

Data pools include both the injured and uninjured knees.

MF = median frequency; RER = rate of electromyography rise; RFD = rate of force development; RMS EMG = root mean square electromyography; VM = vastus medialis.
We also found that antagonist coactivation seemed to be or was greater in the injured knees compared with that in the uninjured or control knees at three time intervals. The coactivation in the uninjured knees was lower than that in the control knees prior to surgery. These demonstrated that there were bilateral changes in the central common drive mechanism controlling the motoneuron pools of an agonist-antagonist muscle pair. These findings may be related to bilateral proprioceptive defects in patients with a unilateral cruciate ligament reconstruction that result in alterations in the control of agonist/antagonist coactivation. Vairo et al recruited participants undertaking an ipsilateral semitendinosus and gracilis autograft for ACL reconstruction for 21.4 ± 10.7 months and observed elicited significant increases in the reactive muscle activation of the medial hamstrings when landing upon the involved lower extremity compared with the matched control. Their study and our findings suggested augmented coactivation in the medial hamstrings. Our results regarding the increased semitendinosus coactivation in the injured knees may be caused by: (1) compensatory mechanisms of the nerve system to maintain knee stability; and (2) protective responses to transaction on semitendinosus and gracilis tendon for a graft of ligament reconstruction. We also observed that hamstring coactivation attenuates about 10% of the peak torque of isometric knee extension. This suggests that maximal isometric strength defects are the consequence of an enhanced knee joint stability through hamstring coactivation after knee injuries. In addition, we found a significantly lower muscle coactivation in the uninjured knees prior to surgery, when compared to the controls. Therefore, instructions to patients regarding nonoperative ACL or PCL deficiency management should include a notification of a potential risk of knee ligament damage in the uninjured contralateral knee.

We observed a bilateral decrease in median frequency in the VM muscle in participants with knee ligament tear/reconstruction. This indicates muscle atrophy in the type II muscle fibers or changes in the orderly recruitment of motor units in the VM muscle bilaterally. Our results are consistent with the findings of a previous study that showed reduction in EMG median frequency in the VM muscle in patients with ACL reconstruction. Furthermore, the median frequency of the VM muscle was correlated to the total work and the quality of life in KOOS (Tables 3 and 4). However, familiar correlations were not found in the case
of the VL muscle (data not shown). These results indicate that augmenting the endurance of the VM muscle may be the key treatment to improve a low quality of life for people with knee injuries. Collectively, our results regarding activation impairments in the VM muscle demonstrated both present conservative therapies for a torn ligament, and post surgery treatments should additionally include: (1) bilaterally maximal electrical stimulations at a high frequency (∼50–100 Hz) to the VM muscle to preserve the muscle activation during a bed rest period shortly after the ligament injury or reconstruction; (2) exercises involving maximal, fast, and repetitive contractions in the bilateral VM muscle when the pain subsides; and (3) dynamic joint control training using unstable boards to reestablish agonist–antagonist coordination in the knee. 41 We also suggest that future studies regarding cruciate ligament lesions should be conducted to investigate whether the reduction activation in the VM muscle would recover after conservative treatments with these above programs.

The second part of our results shows that in relation to the force capacity in the injured knees, there were impairments in the maximal torque and endurance of the knee extension in the injured knees when compared with the uninjured knees; the fast force values in either the injured or uninjured knees were significantly lower than those in the control knees prior to and 3 months after surgery. These findings confirm that AMI involves a bilateral phenomenon of force impairment. 11 Angelozzi et al 10 found that the average MVIC and absolute RFD (0–30 ms) 6 months after the ACL reconstruction were 97% and 80%, respectively, of the preinjury value by an isometric leg press strength test. The amount of time between the injury and the ACL reconstruction surgery in their study was 4.3 months (range, 2–9 months). 10 However, our results demonstrate that defects of fast force capacity (absolute and normalized RFDs) were not observed 6 months after a ligament reconstruction in participants who underwent at least 3 months of conservative treatments for their torn ligament. Differences between this and previous studies may be caused by differences in the histories of intervals between injury and surgery (15.6 months vs. 4.3 months) or methodological variation in measuring forces. Risberg and Holm 42 observed that the total work of knee extension 6 months after an ACL reconstruction at a speed of 240°/s was 87.6 ± 18.4% in the contralateral knee. Their studies regarding differences in total works between injured and uninjured knees were similar to ours. Furthermore, the correlation tests in our study showed that: (1) the absolute RFD 0–100 ms was correlated to function in sports and recreation; and (2) total work was correlated to knee pain, symptoms, and function in daily living and in sport and recreation (Table 3). These novel findings showed that fast force capacity and endurance were associated with the subjective outcome of the knee joint with a tear/reconstructed cruciate ligament. Our findings imply that: (1) improvement in both knee extension strength and endurance after cruciate ligament ruptures is clinically required for the injured knees of patients who meet the criteria for a nonoperative ACL or PCL deficiency management; and (2) the RFD parameter is recommended for future studies when comparisons of fast force capacity are made between injured and control knees.

This study shows no significant changes in the neuromechanical profiles or KOOS between the three time intervals in the injured or uninjured knees in the participants who had at least 12 weeks of conservative treatment prior to a cruciate ligament reconstruction. These results showed that, despite the presurgery nonoperative management, the injured knee was not restored fully at the time of surgery for the patients who had an unstable knee. Therefore, studies with a 2-year follow-up (i.e., a follow-up of >6 months) are suggested in order to find neuromechanical improvements in the group undertaking primary conservative treatment prior to the ligament reconstruction. However, our results for the seven ACL injured patients showed that there were improvements in the MVIC torque and patient-relevant outcomes of knee quality of life and function in sport and recreation as of 6 months postsurgery. By contrast, these findings were not observed when the data for those participants were pooled with the data for the PCL injured patients. These differences in results between the ACL-alone and ACL/PCL groups imply that there are differences in the neuromechanical characteristics of ACL and PCL patients. Future studies are recommended, then, to verify these hypothesized differences in the neuromechanical presentations of ACL and PCL patients.

Our findings should stress again to surgeons that surgery is only part of the treatment of an ACL or PCL injury. Moreover, the return of mechanical knee stability and maximal thigh muscle strength per se (force/power output) after an ACL or PCL reconstruction is not conclusive proof of a successful rehabilitation; rather, other factors should be considered. For example, neuromuscular facilitation as reflected by the time for onset of knee muscle contraction sufficient to control pivoting during weight bearing is a vital issue to consider, but is one capacity that is generally not measured in routine knee scores. The clinical observation that patients with an ACL tear sometimes return with a contralateral injury 1 year later may partly be due to nonrestored neuromuscular balance and function of the lower extremity kinetic chain. Further research is required to evaluate the clinical validity of our findings.

The limitations of this study include a small sample size, and the specific population that was studied—young participants with unilateral cruciate ligament reconstruction. Therefore, the results may not be applicable to participants who have their ligament reconstruction shortly after the tear or older than middle age. This study demonstrated that changes in the neural control can alter the biomechanics of the task and coupling to the rehabilitation outcomes. It is necessary to document neuromechanical interactions to design a supplementary rehabilitation program for quadriceps AMI. We conclude that there were bilateral neuromechanical defects in the knees that have undergone conservative treatment as well as reconstruction after a unilateral cruciate ligament tear. Muscle activation in the VM and semitendinosus muscles affects maximal, fast, and endurable force capacities in patients with quadriceps AMI. In addition, these neuromechanical characteristics are associated with the knee pain, symptoms, and quality of daily life activities. It is recommended that neuromechanical parameters be considered for studies on AMI.
Acknowledgments

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References


