The mechanism of quadriceps femoris weakness in patients with ruptured and reconstructed ACL

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Introduction

There is ample evidence that the anterior cruciate ligament (ACL) has a sensory function. Previous studies reported that afferent feedback from ACL contributed to increased tone of the muscles around the knee. However, it is unlikely that afferent feedback from ACL directly alter a motor neuron (MN); afferents from ACL could influence a motor neuron (MN) via the ? loop. Therefore, the loss of afferents from ACL could be the cause of reduced MVC because normal ? loop is necessary to maximal voluntary contraction (MVC) because normal ? loop function is necessary to MVC. We hypothesized that the lack of joint afferents from ACL to ? loop could be one of the mechanisms underlying the substantial weakness of quadriceps femoris (QF), which is could be the mechanisms underlying QF weakness, which is often observed in patients with ACL injury. The purpose of this study was to investigate the presence of any neurophysiological abnormality in ? loop of QF in patients who had ACL reconstruction and with ruptured ACL. We compared the effects of prolonged vibration on the MVC and integrated electromyogram (I-EMG) of the QF in patients with ruptured ACL, in patients who undergone ACL reconstruction, and uninjured subjects. In subjects with intact ? loop, prolonged vibration is expected to result in reduction of MVC and IEMG because Ia afferent, which is part of the ? loop, is attenuated as a result of continuous activation of the muscle spindle caused by the prolonged vibration. Therefore, if ? loop is intact in the QF of patients with ruptured ACL and patients with ACL reconstruction, the response should be similar to that of the normal subjects.

Methods

The subjects were 8 patients who had ACL repair using a semitendinosus tendon (Reconstruction Group; RG), 8 patients with ruptured ACL (Lesion Group; LG), and 8 volunteers, who had no knee injury (CG). Subjects performed knee extension to get MVC value. The I-EMG of the VM, VL, and RF was measured during MVC measurement. Afterward, 20min of vibration stimulation was applied to infrapatellar tendon. The same measurements were repeated again after vibration. The relative change after vibration was calculated by [(pre-vibration value – post-vibration value) / pre-vibration value × 100]. One-way ANOVA was used to determine differences among groups. Scheffe’s F test was used as post-hoc test

Results

Table 1. Relative change of MVC

<table>
<thead>
<tr>
<th>Group</th>
<th>Relative change (%)</th>
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<tbody>
<tr>
<td>LG</td>
<td>2±3.3&quot; **</td>
</tr>
<tr>
<td>RG</td>
<td>5±6.3&quot; **</td>
</tr>
<tr>
<td>CG</td>
<td>-9±5.6</td>
</tr>
</tbody>
</table>

" p<0.01, compared to the control group.

** Relative change= [(pre-vibration value – post-vibration value) / pre-vibration value × 100].

Discussion

The significant reductions of MVC and I-EMG after prolonged vibration in the CG represent normal responses to prolonged vibration. Our results indicated, however, that the relative changes of patients with ACL rupture and patients with ACL reconstruction in response to prolonged vibration were different from those of normal subjects. These results could be viewed as evidence for dysfunctional ? loop. Since the MVC could not be executed without normal ? loop (1, 2), the presence of dysfunctional ? loop could be the mechanism of QF weakness in these patients. We conclude that loss of afferents from ACL could be underlying mechanism of QF weakness in patients with ACL rupture and QF weakness still exist in patients who have undergone ACL repair.