EXPERIMENTAL ANTERIOR KNEE PAIN AFFECTS ACTIVATION OF CERTAIN MUSCLES DIFFERENTLY DURING LANDING AND JUMPING

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INTRODUCTION

Anterior knee pain (AKP) is a common problem that is associated with abnormal lower-extremity movement neuromechanics [1]. The independent effects of AKP are difficult to study due to other simultaneous effects of knee pathology (e.g., knee effusion or degradation). The purpose of this study was to evaluate the independent effects of AKP during landing and jumping on activation of lower-extremity muscles during two land and jump tasks performed at two different intensities.

METHODS

Thirteen healthy subjects (7 males, 6 females; mass = 70 ± 15 kg; height = 1.74 ± 0.11 m; age = 22 ± 2 yrs) gave informed consent and completed a control and pain data collection session, 48 hours apart. For each session, subjects performed two land and jump tasks at a low and high intensity: a forward double-leg land and jump task (FDJ) and lateral single-leg land and jump task (LSJ). The FDJ task required subjects to jump off of two legs, forward over an obstacle, and land on two legs, on a force platform; then, immediately jump forward off of two legs over a second obstacle. Low and high intensities for this task involved obstacle heights that were standardized to each subject. The LSJ task required subjects to jump off of the left leg, laterally, and land on the right leg on a force platform; then, immediately jump back to the beginning location, landing on the left leg. Low and high intensities for this task involved lateral jump distances that were standardized to each subject. Surface electromyography (EMG; 1000 Hz) was recorded for the right gastrocnemius (GA), vastus medialis (VM), medial hamstrings (MH), gluteus medius (GMD), and gluteus maximus (GMX) while subjects contacted the force platform during each trial. First and second halves of the time that subjects contacted the force platform defined landing and jumping.

For both sessions, subjects first completed three trials of both tasks, at both intensities, to provide reference EMG values. Next, for the pain session, 1 ml of hypertonic saline (5.0% NaCl) was injected into the right-leg infrapatellar fat pad, to induce experimental AKP, and subjects performed three more trials of both tasks at both intensities; these were the experimental trials. The control session was the same as the pain session, except the injection was omitted. Subject perceived AKP was measured every two minutes of both sessions using a 10-cm visual analog scale.

Functional linear models were used to evaluate the effect of session and intensity on normalized EMG amplitude throughout the time that the subjects contacted the force platform for each trial. Also, mixed-model repeated measures ANOVA and Tukey-Kramer post hoc tests were used to compare subject perceived AKP between sessions. All alpha levels were set to 0.01.

RESULTS AND DISCUSSION

Subject perceived AKP was greater for the pain-session trials, relative to the control-session trials (Figure 1; $p < 0.01$). The AKP significantly altered EMG amplitude for all of the observed muscles, at varying parts of both tasks, except for the VM during the FDJ ($p < 0.01$). Effects of the experimental AKP on EMG amplitude were admittedly varied, in direction and timing, and did not reveal a consistent pattern, and the space required to describe all of these effects is not
presently available. Three muscles (GA, MH, and GMD), however, did display a similar interesting pattern, for both tasks, that we wish to emphasize here. This pattern is demonstrated by MH EMG amplitude during the LSJ (Figure 2): as a result of AKP, muscle activation decreased during landing and increased during jumping. This pattern was also observed for the GA and GMD, however, we do not now have space enough to present these data.

The cause of this inhibition and subsequent facilitation is unclear and will require additional research to elucidate. We now speculate, however, that the decreased muscle activation during landing could be due to greater reliance on force contributions from passive muscle components [2] or the skeletal system (i.e., a more upright landing position). We further speculate that the increased muscle activation during jumping could reflect an attempt to compensate for the preceding inhibition during landing and/or increased neural drive, via the group Ib pathway, resulting from the high forces associated with landing [3]. Regardless of the cause(s) of the observed inhibition and facilitation, these EMG alterations are important because they likely result in altered tibiofemoral and patellofemoral joint loads.

REFERENCES


**Figure 1.** Mean subject perceived AKP for both sessions. Asterisks indicate between-session differences ($p < 0.01$).

**Figure 2.** Subplots A and B: mean right MH EMG during ground contact of the LSJ. Subplots C and D: mean between-session differences for right LSJ MH EMG during ground contact of the LSJ. 95% confidence intervals (vertical bars) that do not overlap the zero lines indicate a significant effect of AKP on MH EMG amplitude. Negative mean differences indicate MH EMG that was greater for the control session, while positive mean differences indicate MH EMG that was greater for the pain session.