

# The Effects of Visual Disruption on Muscle Activation Strategies During Walking Following Anterior Cruciate Ligament Reconstruction

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**INTRODUCTION:** The anterior cruciate ligament (ACL) is the most commonly injured ligament in the knee, and even after reconstruction (ACLR), many individuals demonstrate abnormal gait biomechanics that increase the risk of post-traumatic osteoarthritis. Many ACLR patients exhibit persistent neuromuscular impairments, including increased quadriceps-hamstring co-contraction during gait. While this strategy may help stabilize the joint by more evenly distributing loads across joint surfaces and resisting external forces, it also elevates joint compression and cartilage degradation risk. Heightened co-activation during midstance is commonly observed in ACLR patients and is associated with reduced peak knee extension moments, which are harmful to long-term joint health. While co-contraction indices (CCI) quantify the relative balance between agonist and antagonist muscle activity, analyzing average EMG amplitudes also provides critical information about the absolute level of neuromuscular activation, offering a more complete understanding of compensatory strategies following ACLR. ACL injury also disrupts somatosensory input via damaged mechanoreceptors, leading to sensory reweighting, often toward visual reliance. Stroboscopic goggles can perturb visual input and reveal deficits in sensory compensation. Therefore, the objective of this study was to determine the effect of a visual disruptive task on muscle activation strategies in those with ACLR. We hypothesized that those with ACLR would show increased CCI relative to controls, and visual disruption would further amplify CCI and increase EMG amplitudes due to sensory reweighting.

**METHODS:** This study was approved by the Institutional Review Board. Thirty-six individuals with unilateral ACL reconstruction (ACLR) and thirty-six matched controls (age  $\pm 2$  years, sex, BMI  $\pm 4$  kg/m<sup>2</sup>, Tegner score  $\pm 1$ ) consented to participate in this study. EMG (Delsys) and force plate data (Bertec) were recorded at 1200 Hz. Overground walking trials were completed at both preferred and fixed (1.3 m/s ( $\pm 5\%$ )) speeds, monitored via infrared timing gates (Dashr). Muscle activity was assessed under three gait conditions: control (CON; goggles worn but inactive), LOW (100 ms opaque/100 ms transparent), and HIGH (250 ms opaque/100 ms transparent). To ensure visual engagement, participants focused on four light modules at the end of a 6m walkway and waved at a randomly illuminated light triggered by an infrared beam during walking. EMG data were bandpass filtered (20–350 Hz), notch filtered (59.5–60.5 Hz), rectified, and low-pass filtered (10 Hz) to create a linear envelope. Outcomes included mean EMG amplitude and co-contraction index (CCI) during three intervals: (a) preparatory (100 ms pre-heel strike), (b) heel strike (200 ms centered on heel strike), and (c) load acceptance (first 50% of stance). Stance phase was defined from heel strike to toe-off (vGRF  $> 20$ N). Mean EMG amplitude signals were normalized to amplitude during maximum voluntary isometric contraction. Quadriceps (rectus femoris, vastus lateralis) and hamstring (medial/lateral) signals were combined and normalized to peak stance-phase amplitude. CCI was our primary outcome of interest, as CCI is associated with a smaller peak knee extension moment. Prior to calculation, EMG waveforms for the rectus femoris and vastus lateralis, and for the medial and lateral hamstrings, were combined to create a composite quadriceps and hamstrings signals. The composite signals were then normalized to the peak value during the stance phase. CCI was then calculated for the composite signals during each phase noted above using the formula below where lesser and greater EMGi represent the relative magnitudes of the composite hamstring and quadriceps amplitudes at each sample iteration and n represents the total number of samples for each interval:

$$CCI = \frac{\sum_{i=1}^n \frac{\text{lesser EMGi}}{\text{greater EMGi}} (\text{lesser EMGi} + \text{greater EMGi})}{n}$$

Statistical analysis was performed in SPSS v28.0 ( $\alpha = 0.05$ ) using a 2 (Group: ACLR, control)  $\times$  3 (Condition: CON, LOW, HIGH) mixed-model repeated-measures ANOVA. Post hoc Bonferroni comparisons assessed significant interaction effects across and within groups.

**RESULTS:** At fixed speed, co-contraction index (CCI) during the preparatory phase was significantly higher in healthy controls compared to the ACLR group ( $p = 0.026$ ). Similar trends were observed during heel strike ( $p = 0.061$ ) and load acceptance ( $p = 0.064$ ). A significant condition effect was observed during load acceptance ( $p < 0.001$ ), with lower CCI in the HIGH visual disruption condition compared to CONT ( $p < 0.001$ ) and LOW ( $p < 0.004$ ) conditions. No condition  $\times$  group interactions were significant. At preferred speed, no significant effects of group or condition were observed for CCI across any gait phase ( $p > 0.05$ ). For EMG amplitudes, vastus lateralis activation was significantly higher in controls than ACLR participants across all gait phases at fixed speed ( $p \leq 0.021$ ), with greatest reductions in the HIGH condition. The main effect of condition was also significant at the fixed speed ( $p = 0.018$ ), with post-hoc comparisons revealing higher activation in the CONT condition compared to HIGH condition ( $p = 0.004$ ). Vastus medialis activity was unaffected by group or condition. Medial hamstring activity was higher in the CONT versus HIGH condition during heel strike at the fixed speed ( $p = 0.006$ ) and preferred speed ( $p = 0.002$ ), independent of group. Lateral hamstring activation showed a significant group  $\times$  condition interaction during the preparatory phase ( $p = 0.006$ ), with higher activation in the ACLR group under CONT ( $p = 0.008$ ) and LOW conditions ( $p = 0.023$ ), though neither met the Bonferroni-adjusted threshold ( $p < 0.006$ ), and higher activation in the ACLR group during heel strike ( $p = 0.037$ ).

**DISCUSSION:** Contrary to our hypothesis, individuals with ACLR exhibited lower co-contraction and reduced quadriceps activity relative to healthy controls during fixed-speed gait, particularly during the preparatory and early stance phases, when knee stabilization is most critical. This suggests a failure to appropriately engage dynamic stabilizing mechanisms, which may compromise joint integrity over time despite successful reconstruction. In addition, rather than provoking maladaptive co-contraction, visual perturbation suppressed muscle activity in both groups, especially under the high-occlusion condition. This may reflect increased cognitive load or reduced confidence in motor execution under sensory uncertainty. Notably, ACLR individuals failed to increase co-contraction or EMG amplitude in response, suggesting a blunted adaptive response. These findings align with previous evidence of impaired feedforward control and proprioceptive reweighting after ACLR and suggest that residual deficits in sensorimotor integration may limit functional joint stability under variable conditions. The disproportionate reduction in vastus lateralis activity and altered lateral hamstring activation further imply the use of compensatory, and possibly maladaptive, neuromuscular strategies, highlighting the need for targeted rehabilitation approaches that emphasize anticipatory control, proprioceptive acuity, and joint-specific muscular coordination.

**SIGNIFICANCE/CLINICAL RELEVANCE:** Co-activation may reflect a flexible, stabilizing response rather than a pathological stiffness pattern, that is insufficiently accessed by some individuals with ACLR under sensory challenge. This challenges the notion that reducing co-activation should always be a rehabilitation goal, and future research should aim to determine whether identifying "non-adapters" through visual-disruptive or perturbation-based assessments could inform clinicians which patients may need additional somatosensory retraining to restore functional joint control and potentially mitigate long-term cartilage degeneration after ACLR.

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