Cerebral Control of Jump Landing in Anterior Cruciate Ligament Reconstructed Individuals

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Context: The anterior cruciate ligament (ACL) is vital to mechanical knee stability and maintaining neuromuscular control. ACL injury may cause cortical remodeling due to disrupted mechanoreceptors and/or compensations in neuromuscular control. These alterations are not completely understood and may not be addressed in rehabilitation. Assessing brain function more directly after ACL injury using functional magnetic resonance imaging (fMRI) may help address this knowledge gap, but requires patients to remain still, limiting measurement of complex actions (walking; jumping). To assess brain activation during jump landing we developed a novel, action observation paradigm involving mirror neurons and mental imagery to provide an assay of jump landing neuromotor control. **Objective:** To investigate brain activation differences during an action observation jump landing task in those with ACL reconstruction (ACLR) versus matched healthy controls. **Design:** Descriptive laboratory study. **Setting:** Academic medical neuroimaging center. **Participants:** Participants were matched on height, mass, extremity dominance, and physical activity level. Four left ACLR participants (23.7±3.20 years, 1.80±0.08 m, 83.0±19.8 kg, Tegner activity level 6.75±2.0, 42±36 months post-surgery) and four matched healthy controls (24.6±2.08 years, 1.74±0.06 m, 80.5±12.55 kg, Tegner activity level 6.75±2.0). **Interventions:** fMRI was collected while participants watched a video of a 1st person perspective of jump landing while lying supine in a Siemens 3T MRI scanner. Data were collected in 30 second epochs with 90 whole brain gradient-echo scans at a 3 second temporal resolution and voxel dimension of 2.5 mm for 55 transverse slices. **Main Outcome Measures:** Subject specific analysis was completed on the contrast of landing – baseline (1st person video of standing) to get the brain activation pattern of landing for each subject. The two groups were compared with a general linear model second-level fixed-effects paired analysis *a priori* threshold at $p < .01$ corrected. **Results**: Results are reported as z-score (relative activation to matched control) for the peak voxel in the respective brain regions that demonstrated significantly higher or lower activation in the ACLR cohort. Patients with ACLR exhibited increased activation in the superior parietal region ($z=9.32\pm2.7$ $p<.001$) and dorsal visual processing regions ($z=13.5\pm3.7$ $p<.001$) and diminished activation in fusiform ventral visual processing areas ($z=12.65\pm2.9$, $p<.001$) compared to matched controls. **Conclusions:** ACLR and rehabilitation may alter brain activation to control jump landing. The higher parietal activation indicates greater sensory integration, possibly due to the altered afferent input from the ACLR. The increased dorsal visual processing may indicate greater reliance on vision to guide the motor imagery and internally program motion. The decrease in ventral activation may be associated with greater recognition of object details by the control subjects. Alternatively, the ACLR individuals increase in dorsal visual motor programming may relatively suppress the ventral system, inhibiting visual integration of environmental cues.

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