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ACL Deficient Patients With Passive Knee Joint Instability Overcompensate During Active Movements

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Summary:
By developing a novel technique to allow assessment of tibio-femoral kinematics, this study aimed to define the role of passive joint stability on active tibio-femoral kinematics during walking.

Abstract:
Introduction:
It is well known that rupture of the ACL does not only modify movement patterns of the lower limb, but also influences muscle activation, and hence results in a movement strategy that is thought to compensate for pain and joint instability by actively contracting and co-contracting local muscles during dynamic activities. The amount of additional joint stability provided by muscle activation remains unclear, but the additional muscular forces are thought to raise joint loading in a process that could play a key role in the subsequent clinically observed degenerative changes to the joint, including osteoarthritis (OA). By developing a novel technique to allow assessment of tibio-femoral kinematics, this study aimed to define the role of passive joint stability on active tibio-femoral kinematics during walking.

Methods:
Using motion capture, together with combinations of advanced techniques for assessing skeletal kinematics (SARA [1], SCoRE [2], OCST [3]), a novel non-invasive approach to evaluate dynamic tibio-femoral motion was tested in 8 healthy subjects. Here, the coefficient of variation (CV) was used in order to test the variability in A-P translation between multiple repetitions of walking trials. The intra-class correlation was calculated using ICC(3,1), to assess the intra-subject repeatability of A-P translation as well as the range of tibio-femoral A-P translation (difference between the minimum and maximum A-P movement over an entire gait cycle) between repetitions. The passive (KT1000) and active tibio-femoral joint stability was then examined in 13 patients with ACL rupture and compared to their healthy contralateral limbs. After verification using multi-factorial ANOVA analyses that ACL deficiency has an effect on A-P translation, a post-hoc test was applied to determine the magnitude of the difference in A-P translation between ACL ruptured and healthy (contralateral) knees.

Results:
The mean CV across all 16 healthy knees showed 5.2% variation (SD 1.2%) in A-P translation, demonstrating the high repeatability of the novel approach for assessing tibio-femoral kinematics between trials. Furthermore, excellent reliabilities were observed for both A-P translation over an entire gait cycle (mean ICC(3,1) = 0.92) as well as the range of A-P translation (mean ICC(3,1) = 0.98) between repetitions in individuals.
The femora of the ACL ruptured knees generally remained more posterior relative to the tibia over the walking cycles compared to the healthy contralateral limbs. Surprisingly, the mean active range of tibio-femoral anterior-posterior

translation over an entire gait cycle was significantly lower in ACL ruptured knees than in healthy joints ($p<0.05$). However, the passive tibial anterior translation was significantly greater in the ACL ruptured knees than in the healthy controls. A positive correlation was detected for both the ACL ruptured ($R^2=0.34$) and the healthy contralateral knee joints ($R^2=0.47$) between active and passive instability, but with a consistent offset of approximately 4mm between the healthy and ACL deficient knees.

Discussion:
Joints with pathological knee instability displayed reduced tibio-femoral A-P translation during active movements compared to healthy, passively stable knees. These results indicate that active stabilization of tibio-femoral kinematics appears to not only compensate for any passive joint instability, but actually overcompensate, and produce a situation that is kinematically more stable than in knees that are physiologically stable.
One key finding of this study was that a positive relationship between passive and active instability exists for both physiologically healthy as well as for unstable knees. Additional stabilization of the joint in unstable knees, presumably through muscular co-contraction, led to a significant and almost constant 4 mm reduction of anterior motion of the tibia compared to healthy joints during the same activity. It seems that stabilization of the kinematics of the joint beyond these levels may either not be required or not be feasible, possibly since the anatomical lines of action of the stabilizing muscles are limited in their ability to restrain anterior tibial motion.
An additional important observation was that the femora of the ACL ruptured knees were consistently located more posteriorly relative to the tibia during active movements. This phenomenon is unlikely to be an artefact of the assessment approach since the relative tibio-femoral motion was determined in a global manner (the approach imposes no artificial reference or “zero” position on the bones). It therefore seems reasonable that the additional femoral posterior translation occurred due to ACL deficiency [4]. The consequence of such altered kinematics would be joint loading in regions that may not be adapted to such loading conditions and therefore an increased risk for possible early joint degeneration [5].

Significance:
Subjects after ACL rupture appear to over-compensate for passive knee joint instability by actively reducing tibio-femoral A-P translation, suggesting overloading of the joint, and thereby providing a plausible mechanism for explaining post-traumatic degeneration of cartilage in the joint.

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