Comparison of Laxity in Cruciate Retaining Total Knee Arthroplasty Patients and Healthy Subjects Using a Novel Multiplanar Arthrometer

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INTRODUCTION: Knee instability in midflexion is a leading cause of patient dissatisfaction and revision surgery following total knee arthroplasty (TKA) [1]. Although surgeons typically focus on intra- and peri-operative frontal pane laxity intraoperatively, midflexion instability can also involve excessive laxity in the anterior-posterior (AP) direction [2]. Moreover, quantitative intraoperative targets for both coronal laxity and ligament tensioning to achieve clinically acceptable sagittal plane (i.e., anterior) laxity in midflexion remain ill-defined. Despite growing awareness of midflexion instability as a contributor to poor outcomes after TKA, intraoperative strategies remain largely focused on coronal plane balancing, with less emphasis on sagittal plane laxity. This is particularly concerning given that the ACL, a key stabilizer of anterior tibial translation, is typically resected during TKA. In its absence, the medial collateral ligament (MCL), primarily a valgus stabilizer, may be required to compensate for anterior stability, a role for which it is not biomechanically optimized. While cadaveric studies have suggested that MCL tensioning can influence anterior-posterior (AP) laxity in posterior-stabilized TKA, in vivo evidence is lacking [3]. Further, these studies are specific to PS TKA, and it is unknown if these findings will translate to other cruciate retaining (CR) systems. Moreover, it remains unclear how AP and varus-valgus (VV) laxity interact in midflexion, and how these patterns differ from those of the native knee. To address these gaps, we used a novel knee arthrometer to quantify midflexion laxity in both TKA and healthy knees. We posed two questions: 1) How does AP and VV laxity following CR TKA compare to that of young, healthy subjects with no history of previous injury? And 2) Are AP translations and VV rotations related?

METHODS: To quantify midflexion laxity, we utilized a novel knee arthrometer to measure knee laxity in multiple planes. To conduct a laxity test, the patient sat reclined in the chair of the arthrometer, their leg was then aligned in the linkage mechanism of the arthrometer at 30° of flexion, the thigh was then fixed to the chair, and, finally, the shank was fixed to the linkage mechanism, both with controlled compressive forces. With IRB approval and informed consent, we tested 32 patients (20 females/12 males; mean age: 64 ± 6 years; mean BMI: 29 ± 4; mean time after surgery: 383 ± 30 days) who underwent robotic-assisted TKA with a single design of PCL-retaining implant one year after surgery. The examiner then performed four cycles each of the uniplanar assessments: 1) AP: 30 N posterior to 50 N Anterior and 2) VV: ± 6 Nm. Laxity was calculated as the translation or rotation from the position of no applied load to the peak applied load. To achieve our first objective, a cohort of consecutively tested healthy volunteers (10 females/10 males; mean age: 28± 6 years; mean BMI: 24 ± 4) with no history of previous knee injuries underwent laxity testing following the same protocol. Outcomes in each direction from the TKA cohort were compared to the healthy cohort via unpaired t-test (α=0.05). Second, AP and VV laxity from the TKA knee were related via simple linear regression ($\alpha < 0.05$).

RESULTS: First, translations in the anterior and posterior direction in the TKA knee exceeded that of the healthy knee by an average of 3.4 ± 0.9 and 4.0 ± 0.4 mm, respectively ($p < 0.001$)(Fig. 1). However, differences in varus or valgus rotation were not detected ($p \geq 0.5$) (Fig. 2). Second, we identified a positive relationship between AP and VV laxity ($\beta = 0.3 \pm 0.04$, $p <0.001$, $r^2 = 0.6$) (Fig. 3).

DISCUSSION: These findings demonstrate that anterior-posterior laxity in midflexion is greater in TKA patients compared to healthy individuals, supporting the hypothesis that the MCL assumes a compensatory stabilizing role in the absence of the ACL. However, the MCL is not biomechanically oriented to resist AP loads effectively, which may contribute to the increased laxity and potential for progressive stretching observed clinically over time. This in vivo evidence corroborates prior in vitro studies and underscores the importance of considering MCL tensioning and alignment during TKA to mitigate midflexion instability. Interestingly, posterior laxity was elevated in this cohort as well, despite the PCL remaining intact. Furthermore, AP laxity and VV laxity were related to one another, which suggests that the MCL may be contributing to stability in both directions. This reinforces the idea that tensioning or alignment of the MCL during TKA affects not just coronal balance but also sagittal stability. These findings provide experimental evidence for key intraoperative coronal laxity and MCL tensioning targets for achieving clinically acceptable anterior laxity in midflexion.

SIGNIFICANCE/CLINICAL RELEVANCE: This study provides in vivo evidence that anterior-posterior laxity in midflexion is elevated in CR TKA patients compared to healthy individuals, highlighting the compensatory role of the MCL in the absence of the ACL. These findings underscore the clinical importance of optimizing MCL tensioning and alignment intraoperatively to reduce midflexion instability and improve long-term outcomes following TKA.

REFERENCES: [1] Paxton. JBJS 2010. [2] Abdel. BJJ 2014. [3] Berube. JBJS 2024

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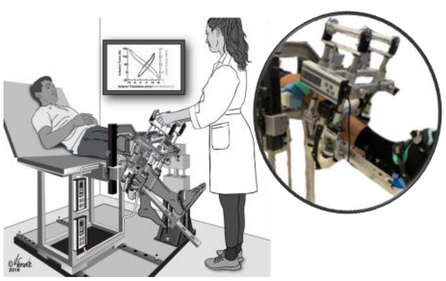

Figure 1: Knee Arthrometer

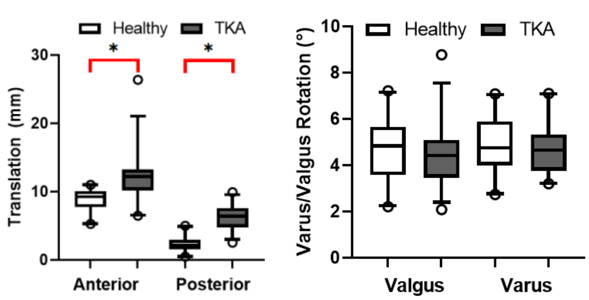

Figure 2: Left: Anterior and posterior translations of the healthy (white) and CR-TKA (gray) subjects. Right: varus and valgus rotations of the healthy and CR-TKA knee. Boxes indicate standard deviation, horizontal line indicates the mean, whiskers indicate 5-95%, circles indicate outliers. Red brackets with * indicate statistically significant differences.

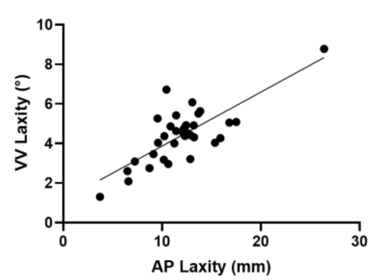

Figure 3: Simple linear regression between AP laxity and VV laxity in CR-TKA. $P < 0.001$. $\beta = 0.3 \pm 0.04$.