

Medial Collateral Ligament Reconstruction for Anteromedial Instability of the Knee

A Biomechanical Study In Vitro

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Investigation performed at Imperial College London, London, UK

Background: Although a medial collateral ligament (MCL) injury is associated with anteromedial rotatory instability (AMRI) and often with an anterior cruciate ligament (ACL) injury, there has been little work to develop anteromedial (AM) reconstruction to address this laxity.

Purpose: To measure the ability of a novel “anatomic” AM reconstruction technique to restore native knee laxity for isolated AM insufficiency and combined AM plus posteromedial insufficiency.

Study Design: Controlled laboratory study.

Methods: A total of 12 cadaveric knees were mounted in a kinematic testing rig that allowed the tibia to be loaded while the knee flexed-extended 0° to 100° with 88-N anteroposterior translation, 5-N·m internal rotation–external rotation (ER), 8-N·m valgus, and combined anterior translation plus ER to simulate AMRI. Joint motion was measured using optical trackers with the knee intact, after superficial MCL (sMCL) and deep MCL (dMCL) transection, and after AM reconstruction of the sMCL and dMCL with semitendinosus autografts. The posteromedial capsule (PMC)/posterior oblique ligament (POL) was then transected to induce a grade 3 medial injury, and kinematic measurements were repeated afterward and again after removing the grafts. Laxity changes were examined using repeated-measures analysis of variance and post-testing.

Results: sMCL and dMCL deficiency increased valgus, ER, and AMRI laxities. These laxities did not differ from native values after AM reconstruction. Additional PMC/POL deficiency did not increase these laxities significantly but did increase internal rotation laxity near knee extension; this was not controlled by AM reconstruction.

Conclusion: AM reconstruction eliminated AMRI after transection of the dMCL and sMCL, and also eliminated AMRI after additional PMC/POL transection.

Clinical Relevance: Many MCL injuries occur in combination with ACL injuries, causing AMRI. These injuries may rupture the AM capsule and dMCL. Unaddressed MCL deficiency leads to an increased ACL reconstruction failure rate. A dMCL construct oriented anterodistally across the medial joint line, along with an sMCL graft, can restore native knee ER laxity. PMC/POL lesions did not contribute to AMRI.

Keywords: medial collateral ligament reconstruction; biomechanics; anteromedial rotatory instability

Although medial collateral ligament (MCL) and combined MCL plus anterior cruciate ligament (ACL) injuries are relatively common,^{4,9,42} the medial aspect of the knee has received little attention recently, influenced by a common assumption that nonsurgical treatment allows the MCL complex to heal adequately in many cases. However, a nonsurgically treated MCL lesion at the time of ACL reconstruction is associated with an increased rate of ACL graft failure,³⁷ and MCL deficiency increases tension in the ACL.³¹ In the presence of unaddressed MCL laxity, the

rate of failure of ACL grafts can increase by 13 times after primary ACL reconstruction¹ and by 17 times after revision ACL reconstruction.² A review of what had been classified and treated as clinically “isolated” ACL injuries discovered that 67% of cases had MCL injuries detected on imaging.⁴⁰

The pioneering works on anteromedial (AM) rotatory instability (AMRI) of Slocum and Larson³³ and Kennedy and Fowler²⁰ reported that, in response to external rotation (ER), the first essential lesion on the medial aspect was a rupture of the deep (capsular) MCL (dMCL), followed by a rupture of the superficial MCL (sMCL) and only then by an ACL rupture. The dMCL passes anterodistally across the joint line toward an AM attachment below the rim of the tibial plateau,^{5,26} and it is stretched directly by ER.^{26,41} The dMCL is a primary restraint to ER⁶ and



resists ER laxity,¹² particularly near knee extension. Although the MCL usually heals with nonsurgical treatment, the injured dMCL may cause chronic symptoms when the knee is loaded in ER in sports participation.²⁷

Despite this evidence, most MCL reconstruction procedures have not included an AM component of the dMCL. However, techniques leaving the hamstring tendon attached to the tibia distally and taken up to the femoral medial epicondyle provide a slanting orientation of the graft, which may thereby resist AMRI.²³ MCL reconstruction described as “anatomic” involves 2 graft strands to mimic the sMCL and posterior oblique ligament (POL), and biomechanical studies have reported that this combination restores native knee laxity.^{13,24} However, it seems that the dMCL and AM capsule were left intact in these studies, so they did not simulate a full AM injury. Noting that the majority of medial-sided knee injuries associated with ACL ruptures relate to AM laxity, it may be beneficial to use a novel AM reconstruction technique that includes dMCL and sMCL grafts to restrain AMRI. A similar method, with both grafts attached at the medial epicondyle, has been described,²¹ but there are no data on its ability to stabilize the knee. It has been shown that 3-strand (sMCL + dMCL + POL) reconstruction can restore native knee stability.²⁵

The aim of the present work was to design and test a reconstruction method, based on the evidence cited in the preceding paragraphs, that would include dMCL and sMCL grafts. It was hypothesized that this novel anatomic AM reconstruction technique would restore AM laxity (anterior tibial translation [ATT], ER, valgus, and AMRI [ATT plus ER combined]) to match native knee laxity.

METHODS

Specimen Preparation

This study used fresh-frozen knees (MedCure) after Wales Research Ethics Committee approval (12/WA/0196; license ICHTB 12275; application R15092-1A). After work to develop the surgical procedure, 12 unpaired knees were used: 8 male and 4 female with a mean age of 52 ± 8 years. Each knee was thawed at room temperature overnight before use. Visual and manual examinations by an orthopaedic surgeon (N.M.) confirmed the absence of

abnormalities such as ligament laxity. The skin and subcutaneous fat were removed, leaving the deeper soft tissue intact. The femur and tibia were cut at 170 mm above and below the joint line. The femur was drilled out, and an intramedullary rod was cemented into it using polymethylmethacrylate to allow mounting in a kinematic testing rig.¹⁹ The distal 60 mm of the tibia had all soft tissue removed and was cemented into a cylindrical pot that had a rod extending 0.5 m from its distal end. The proximal fibula was transected at 100 mm long and secured to the tibia in its anatomic position by a bone screw. The knee was mounted into the testing rig using the femoral intramedullary rod. It was aligned such that the tibia hung vertically when the knee was extended and when flexed to 90°, with the transepicondylar axis in line with the flexion-extension axis of the kinematic testing rig (Figure 1). The knee was flexed-extended by moving the femur above the vertical free-hanging tibia.

Specimen Loading

A Steinmann pin was drilled mediolaterally across the tibia at 40 mm below the joint line for the mounting of 2 semicircular steel hoops with a pulley mounted on each of them. This allowed anteroposterior translation forces to be applied using cords attached to 88-N hanging weights while not inhibiting internal rotation (IR)–ER. A 250-mm pulley disc was fixed on the end of the tibial extension rod, allowing 5-N·m IR-ER torque to be applied (Figure 1). Similarly, 8-N·m valgus-varus moments could be applied. The tibial extension rod had a clamp mechanism that allowed a repeatable position of native neutral rotation at 30° of flexion when tensioning the reconstruction later. This fixture also controlled IR-ER instability that followed medial tissue transection when the knee was loaded in valgus.

Kinematic Measurements

Tibiofemoral kinematics was measured using an optical tracking system (Polaris Vega; Northern Digital) with a mean translational accuracy of ± 0.12 mm (Northern Digital specification), with passive reflective marker triads (Brainlab) on the femur and tibia. Joint motion was defined according to the Grood and Suntay¹⁶ system. To define the coordinate system, small digitization screws

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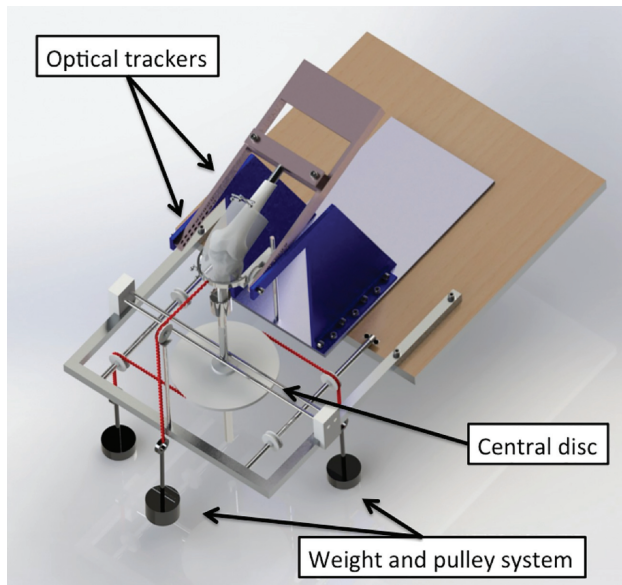


Figure 1. The knee was mounted in a 6 degrees of freedom kinematic testing rig with optical trackers rigidly mounted to the femur and tibia. The femur was flexed-extended above the vertical tibia. A weight and pulley system was used to apply external loads to the tibia. Rotation was imposed by a central disc mounted on the tibial rod. Reproduced from Inderhaug et al.¹⁹

were inserted into the femur at 10 mm proximal to the medial and lateral epicondyles (allowing a graft tunnel at the medial epicondyle) and at its proximal end as well as into the medial and lateral rims of the tibial plateau and the distal tibia. The knee was defined as being at 0° of flexion when the femoral and tibial rods were parallel when viewed in the sagittal plane. Kinematic data were collected at 60 Hz during 3 cycles of flexion-extension from 0° to 100°, and then the mean value was calculated. Before data collection, the knee was flexed-extended 10 times to loosen (precondition) the previously frozen tissue.

For the native knee, the femur was flexed-extended without displacing loads on the tibia to define the neutral path of motion. This was repeated with 88-N ATT–posterior tibial translation forces, 5-N·m IR-ER torques, 8-N·m valgus-varus moments, and combined ATT plus ER (AMRI) loads imposed. Data were calculated as changes from the neutral path of motion.

Kinematic data were measured in the following states: native, sMCL + dMCL transected/excised (AM injury), and dMCL + sMCL reconstructed (AM reconstruction). For the AM injury, the sMCL was detached from its tibial attachment, elevated proximally, and then detached from the femur; the dMCL was transected at the proximal edge of the meniscus. Because this study was about AM reconstruction to address AMRI, the posteromedial capsule (PMC)/POL was left intact initially because the PMC/POL is slackened by ATT and ER.⁴¹ The knee was then further injured by transection of the PMC/POL at the proximal edge of the meniscus, and thus, there was complete

transection/excision of the ligaments across the medial aspect of the knee (a grade 3 medial injury). Tests were then repeated to show the ability of AM reconstruction to stabilize a grade 3 injury. The grafts were then removed, and stability of the knee with a grade 3 injury was measured.

Surgical Procedure

The semitendinosus tendon was harvested and divided to form sMCL and dMCL grafts. Each single-strand graft had both ends whipstitched using No. 2 sutures (Ultra-braid; Smith & Nephew). Grafts were preconditioned by hanging an 88-N weight on them for 20 minutes. Femoral graft tunnels were placed at the centers of the anatomic attachments: for the sMCL, 1 mm proximal to the medial epicondyle, and for the dMCL, 5 mm posterior and 6 mm distal to the epicondyle.⁵ The sMCL tibial tunnel was placed at 60 mm distal to the plateau at the midpoint of the width of the ligament attachment. The dMCL tibial tunnel was placed at 10 mm below the plateau at the midpoint of the width of the attachment so that the graft was aligned 30° anterodistally at 0° of knee flexion (Figure 2). Tunnels were drilled to the lateral surface using a 2.4-mm eyelet pin, and sutures between these pins showed isometry during flexion-extension. The medial tunnel apertures were redrilled 7 mm in diameter with a 25-mm depth. The graft was secured in the femoral tunnel using a 7 × 25-mm interference screw (RCI; Smith & Nephew), and then lead sutures were tied over a cortical button (Endobutton; Smith & Nephew) at the lateral cortex. Grafts were pulled into their tibial tunnels and tensioned manually while the knee was flexed-extended 15 times. The sMCL graft was tensioned using a tensiometer in line with the tibial tunnel to 60 N at 30° of flexion and neutral rotation, with a 2-N·m varus moment to close the medial joint space, and secured with a 7 × 25-mm interference screw at the medial tunnel entrance. Lead sutures were tied to lateral cortical screw posts as backup. The dMCL graft was passed deep to the sMCL graft and fixed similarly using 20-N tension at 30° of flexion.

Statistical Analysis

A power analysis with G*Power Version 3.1.9.7 using published data¹³ found that a change of 2° in ER could be identified with 88% power and 95% confidence with 7 specimens. We used 12 knees in case we could not obtain such consistent data as those of Coobs et al.¹³

The data were confirmed to be compatible with normal distributions using the Shapiro-Wilk test. Differences between testing states were examined for each of the laxity measures by 2-way analysis of variance with repeated contrasts, with the primary variables being the testing state of the knee and knee flexion angle. The dependent variables were knee laxity changes. If significant effects were found, post-testing used repeated-measures *t* tests every 10° of knee flexion, in which *P* < .05 indicated significance, with the Bonferroni correction for multiple contrasts.

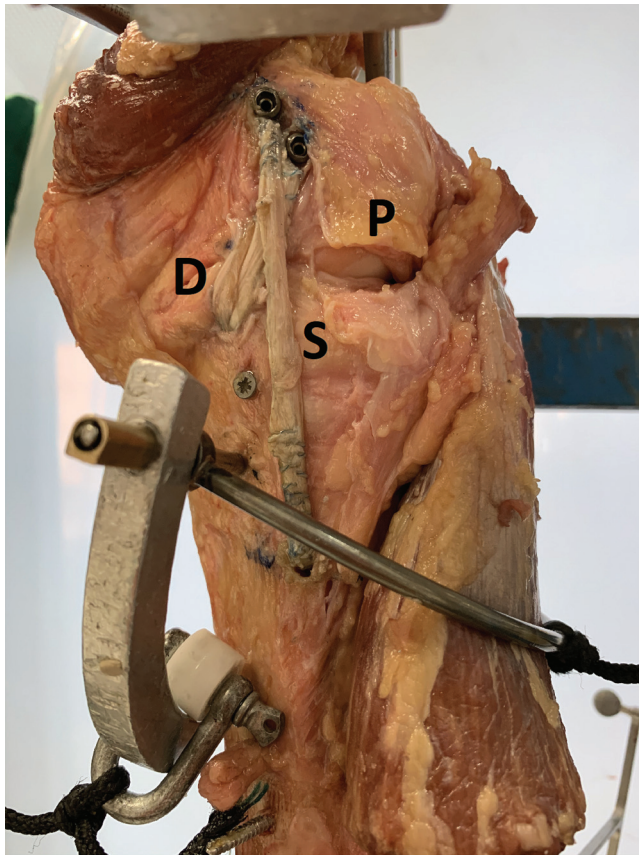


Figure 2. Medial aspect of a right knee with anteromedial reconstruction at 0° of flexion and neutral rotation. The superficial medial collateral ligament (MCL) graft (S), the deep MCL graft (D) passing beneath the superficial MCL graft, and the transected posteromedial capsule/posterior oblique ligament (P) are labeled. Metal hoops were used when applying anteroposterior translation forces.

RESULTS

Anteroposterior Translation

Significant changes in ATT were not found, with the largest mean effect being 1 mm (Figure 3). Significant changes in posterior tibial translation also were not found, with the largest mean effect being 1 mm.

Internal Rotation

The mean IR of the intact knee varied from 9° to 19° at 0° to 100° of knee flexion (Figure 4). Transection of the sMCL and dMCL (AM injury) did not cause a significant increase in IR (see Appendix Table A1, available in the online version of this article). AM reconstruction did not change IR significantly; it remained not significantly different from native laxity.

After the PMC/POL had also been transected (grade 3 medial deficiency), IR of the unreconstructed knee increased significantly above that of the AM injury state at 0° to 40° of flexion (Figure 4; Appendix Table A1,

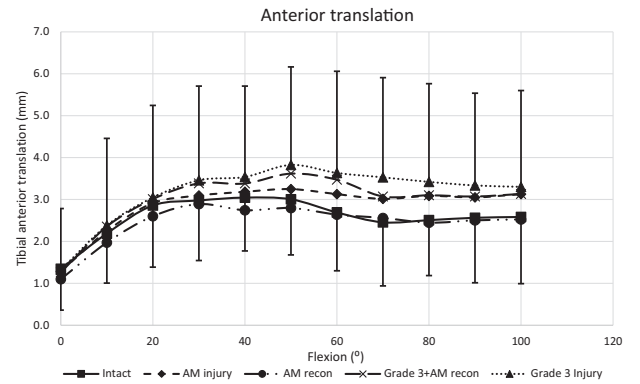


Figure 3. Anterior translation (mm) in response to 88-N force versus knee flexion (deg). Values are shown as mean ± SD (n = 12). AM injury, after transection of the superficial and deep medial collateral ligaments (sMCL and dMCL, respectively); AM reconstruction (recon), after recon of the sMCL and dMCL; Grade 3 + AM recon, after additional transection of the posteromedial capsule (PMC)/posterior oblique ligament (POL) with AM recon; Grade 3 injury, with the sMCL, dMCL, and PMC/POL transected and the AM recon grafts removed.

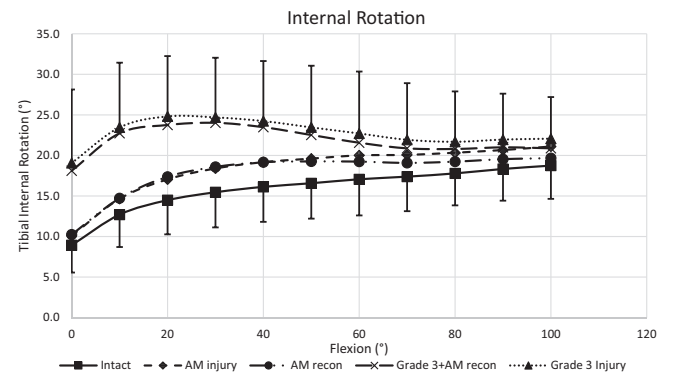


Figure 4. Internal rotation (deg) in response to 5-N·m torque versus knee flexion (deg). Values are shown as mean ± SD (n = 12). Anteromedial (AM) injury, after transection of the superficial and deep medial collateral ligaments (sMCL and dMCL, respectively); AM reconstruction (recon), after recon of the sMCL and dMCL; Grade 3 + AM recon, after additional transection of the posteromedial capsule (PMC)/posterior oblique ligament (POL) with AM recon; Grade 3 injury, with the sMCL, dMCL, and PMC/POL transected and the AM recon grafts removed.

available online). Additional PMC/POL transection led to an increase in IR that was not reduced significantly after AM reconstruction, and it remained significantly (6°-10°) more lax than the native knee at 10° to 50° of flexion.

External Rotation

The mean ER of the intact knee varied from 11° to 19° at 0° to 100° of flexion (Figure 5). This increased by up to 13° after the AM injury, which was significant at all angles of flexion tested (see Appendix Table A2, available online).

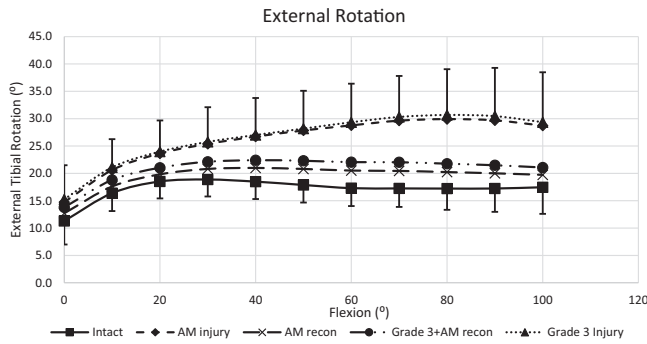


Figure 5. External rotation (deg) in response to 5-N-m torque versus knee flexion (deg). Values are shown as mean \pm SD (n = 12). Anteromedial (AM) injury, after transection of the superficial and deep medial collateral ligaments (sMCL and dMCL, respectively); AM reconstruction (recon), after recon of the sMCL and dMCL; Grade 3 + AM recon, after additional transection of the posteromedial capsule (PMC)/posterior oblique ligament (POL) with AM recon; Grade 3 injury, with the sMCL, dMCL, and PMC/POL transected and the AM recon grafts removed.

AM reconstruction restored ER so that it did not differ significantly from native laxity.

Additional transection of the PMC/POL (grade 3 medial injury) did not cause significant increases in ER (Figure 5; Appendix Table A2, available online). AM reconstruction restored ER so that it was not significantly more lax than the native knee in the presence of a grade 3 injury. After the removal of AM reconstruction grafts, ER with a grade 3 medial injury was not significantly different than that with an AM injury.

Varus-Valgus Laxity

Varus laxity varied from 2° to 3° at 0° to 100° of flexion and was not changed significantly (mean changes $<0.5^\circ$) by the medial procedures. The mean valgus laxity of intact knees varied from 1° to 3° at 0° to 100° of flexion (Figure 6). This increased by 4° to 6° after an AM injury to 5° to 9° of laxity, which was significant above 20° of flexion (see Appendix Table A3, available online). After AM reconstruction, valgus laxity did not differ significantly from native laxity at any angle of flexion tested.

In the MCL-deficient knee, transecting the PMC/POL, thus creating a grade 3 medial injury, did not increase valgus laxity significantly at any angle of flexion (Figure 6; Appendix Table A3, available online). However, it reduced the ability of AM reconstruction to stabilize the knee, with a 2° mean increase in residual valgus after AM reconstruction. With a grade 3 medial injury, AM reconstruction reduced valgus laxity significantly, but it was also significantly more lax than the native knee, above 10° of flexion.

Combined Anterior Translation Plus ER Loading

Combined (AMRI-type) loading caused a mean ATT laxity of the native knee of 0 to 4 mm at 0° to 100° of flexion (Figure

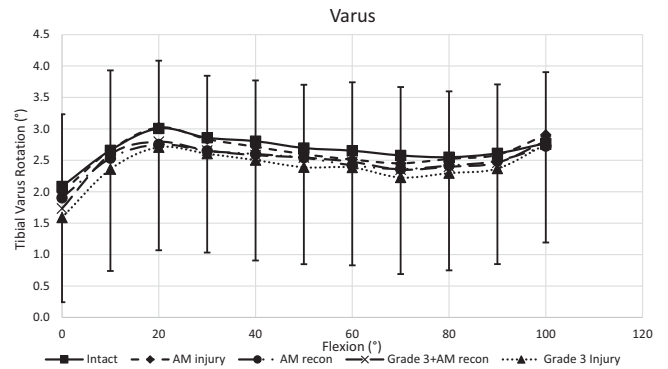


Figure 6. Valgus (deg) in response to 8-N-m moment versus knee flexion (deg). Values are shown as mean \pm SD (n = 12). Anteromedial (AM) injury, after transection of the superficial and deep medial collateral ligaments (sMCL and dMCL, respectively); AM reconstruction (recon), after recon of the sMCL and dMCL; Grade 3 + AM recon, after additional transection of the posteromedial capsule (PMC)/posterior oblique ligament (POL) with AM recon; Grade 3 injury, with the sMCL, dMCL, and PMC/POL transected and the AM recon grafts removed.

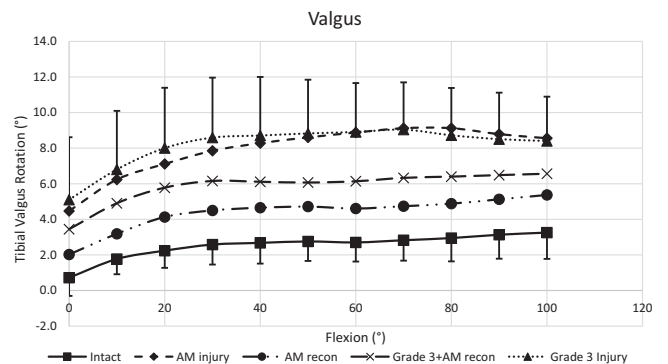


Figure 7. Anterior tibial translation (mm) in response to combined 5-N-m external rotation torque plus 88-N anterior tibial translation force versus knee flexion (deg). Values are shown as mean \pm SD (n = 12). Anteromedial (AM) injury, after transection of the superficial and deep medial collateral ligaments (sMCL and dMCL, respectively); AM reconstruction (recon), after recon of the sMCL and dMCL; Grade 3 + AM recon, after additional transection of the posteromedial capsule (PMC)/posterior oblique ligament (POL) with AM recon; Grade 3 injury, with the sMCL, dMCL, and PMC/POL transected and the AM recon grafts removed.

7). The AM injury caused a significant 4-mm increase above 60° of flexion (Figure 7; Appendix Table A4, available online). ATT after AM reconstruction was not significantly different from the native knee. After the PMC/POL had also been transected, the grade 3 medial deficiency did not lead to a significant increase in ATT either after AM reconstruction or after the removal of AM reconstruction grafts (Figure 7; Appendix Table A4, available online).

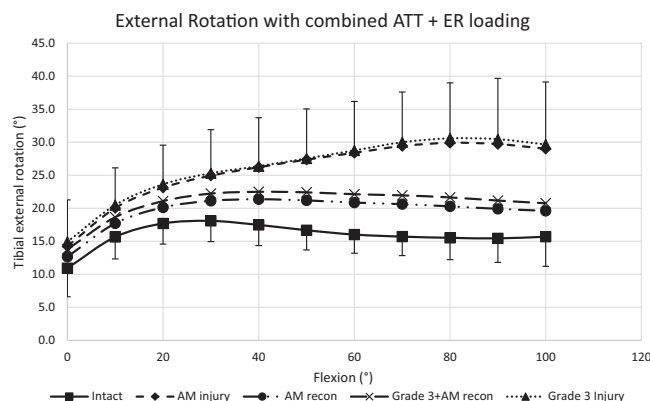


Figure 8. External rotation (ER) (deg) in response to combined 5-N·m ER torque plus 88-N anterior tibial translation (ATT) force versus knee flexion (deg). Values are shown as mean \pm SD ($n = 12$). Anteromedial (AM) injury, after transection of the superficial and deep medial collateral ligaments (sMCL and dMCL, respectively); AM recon, after reconstruction (recon) of the sMCL and dMCL; Grade 3 + AM recon, after additional transection of the posteromedial capsule (PMC)/posterior oblique ligament (POL) with AM reconstruction; Grade 3 injury, with the sMCL, dMCL, and PMC/POL transected and the AM reconstruction grafts removed.

Combined (AMRI-type) loading caused a mean ER laxity in the intact knee of 11° to 18° at 0° to 100° of flexion (Figure 8). This increased to 14° to 31° after an AM injury, which was significant above 10° of flexion (see Appendix Table A5, available online). ER after AM reconstruction was not significantly different from the intact knee. The grade 3 medial deficiency did not cause a significant increase in ER either after AM reconstruction or after the grafts were removed (Figure 8; Appendix Table A5, available online).

DISCUSSION

In knees with combined sMCL plus dMCL transection, a novel 2-strand AM reconstruction technique was able to restore native laxity in ER, ATT, and valgus at the tested range of 0° to 100° of flexion, as hypothesized. It also restored native laxity under combined ATT plus ER loading, simulating AMRI, which may occur in conjunction with ACL injuries. This AM stabilizing action was unaffected by a soft tissue injury also including the PMC/POL, that is, a grade 3 medial injury. This demonstrates the role of the dMCL graft, which crosses the joint anterodistally toward the tibial attachment on the AM rim of the plateau,⁵ in controlling AM joint laxity; the dMCL is an important restraint of ER near knee extension.^{6,12} AM stability was not affected significantly by the PMC/POL; thus, the common clinical practice of adding a POL procedure in many cases of an MCL injury combined with an ACL rupture is not supported by this study. Conversely, AM reconstruction did not affect IR after a grade 3 medial injury with PMC/POL deficiency, when a POL procedure may be indicated.

The changes in stability after transecting the medial ligaments are supported by other studies. Anteroposterior translation did not change significantly after MCL transection, as reported previously,^{25,36} because the ACL and posterior cruciate ligament, the primary restraints,^{6,11,28} remained intact. ER in the native knee increased with flexion and was increased significantly at all angles of flexion after a dMCL plus sMCL injury. These structures are primary restraints of ER,⁶ with a dMCL rupture being what Kennedy and Fowler described as the “essential lesion,” meaning that ER would not increase significantly unless it was ruptured.²⁰ ER did not change significantly after transection of the PMC/POL, which slackens with ER and so has no effect on ER laxity limits.^{30,41} The present work relates to the static stabilizing function of the medial ligaments, and not to dynamic loading from semimembranosus muscle tension, which limits ER²² and may cause traction injuries such as meniscocapsular and POL ruptures.³² The greatest change in IR occurred after PMC/POL transection; this structure is tight and aligned to resist IR near knee extension.^{29,30,41} AM reconstruction could not control IR, being oriented to resist ER, although the sMCL is a secondary restraint to IR.^{6,29} MCL transection led to significantly increased valgus laxity beyond 20° of flexion, when the posterior structures slackened, as reported previously.¹⁵ The sMCL is the primary restraint of valgus laxity^{6,15} and was addressed by AM reconstruction; native laxity was restored at the whole range of flexion angles examined. To attain this, the graft tunnel was placed anatomically at the medial epicondyle,⁵ which controls isometric behavior.⁴¹ By reproducing both the sMCL and the dMCL, AM reconstruction restored laxities to normal during simulated AMRI loading.

Among many articles describing MCL surgery, only 3 describe a dMCL reconstruction component. Black et al⁸ repaired the torn meniscotibial part of the dMCL. Their aim was to restabilize the meniscus, preventing lift-off and extrusion, rather than knee stability. Biomechanical studies of anatomic 2-strand MCL reconstruction^{13,38} included sMCL and POL grafts, but not dMCL grafts, and thus may be described as posteromedial reconstruction. AM reconstruction with sMCL and dMCL grafts has been described,²¹ noting that the dMCL limits ER during knee arthroplasty.¹² Indeed, in arthroplasty, dMCL release is often undertaken to allow anterior subluxation of the medial tibial plateau. The technique of Kim et al²¹ simplified the femoral attachments, taking the dMCL graft to the sMCL tunnel at the medial epicondyle. Although that simplified the reconstruction procedure, its biomechanical and clinical performance remains unreported. There may be a dMCL injury while the sMCL remains intact. In that case, valgus stability will be normal or nearly normal because the sMCL is the primary restraint, while ER may be increased and/or there may be pain localized over the dMCL with ER loading. This injury can be treated by dMCL suturing²⁷ and would not require AM reconstruction that includes an sMCL graft. A dMCL graft has also been reported as part of anatomic 3-strand reconstruction, which was found to restore native ER.²⁵

A limitation of the present study is that it concerns only reconstruction of the passive medial ligamentous

restraints. Further work could add simulated injuries of the peripheral meniscocapsular and semimembranosus tendon attachments to the posteromedial structures. Kittl et al²² found that semimembranosus tension reduced ER in vitro, particularly in the flexed knee. Sims and Jacobson³² reported that the most common abnormality found during surgery to address AMRI was a rupture of the semimembranosus tendon slips attaching to the PMC, which includes the POL, that provide dynamic restraint. Hughston and Eilers¹⁸ described an array of fiber bundles in the PMC, among them the POL. Other studies have not always found a distinct POL (reviewed by Robinson et al³⁰), but the semimembranosus tendon splits into slips that attach to the area of the POL and proximal tibia, providing a dynamic restraint of AMRI.³² However, transection of the PMC/POL was not found to increase ER at any angle of knee flexion, as in the present study.²⁹ Hughston and Barrett¹⁷ emphasized the repair of the semimembranosus-PMC complex during acute surgery of MCL, ACL, and medial meniscal injuries. Posterior deficiency of the medial meniscus and posteromedial meniscocapsular lesions enhance knee laxity, causing significant increases in ATT and ER beyond the laxity caused by ACL deficiency.^{33,35} Although AM lesions related to AMRI often occur with ACL ruptures, the ACL was intact in this experiment. Thus, it simulated an isolated AM injury and also a combined ACL plus AM injury after an ACL reconstruction that completely restored native ACL behavior. This allowed the experiment to avoid variability in the ACL procedure blurring the effects of AM reconstruction that are the focus of this study. Given the clinical importance and prevalence of combined ACL plus AM injuries, and having demonstrated the biomechanical effect of an AM injury and performance of AM reconstruction, a further study of combined ACL plus AM injuries and reconstruction is now appropriate.

There has recently been an increased interest in addressing secondary damage around the knee at the time of ACL reconstruction, hoping to reduce ACL graft stresses.³ Recent studies have concentrated on anterolateral rotatory instability because of increased mobility of the lateral compartment in pivot-shift instability.^{7,10} Knees with severe instability may be restabilized more completely by adding an extra-articular procedure to intra-articular ACL reconstruction.¹⁹ The ACL graft failure rate is much reduced by the addition of an anterolateral extra-articular procedure in knees with combined ACL plus anterolateral injuries.^{14,34} In contrast, evidence is only starting to appear to show that an MCL lesion treated nonsurgically at the time of ACL reconstruction is associated with a markedly increased rate of ACL graft failure,^{1,2,37} and there may be a high incidence of unrecognized MCL lesions with isolated ACL injuries.⁴⁰ This suggests that nonsurgical treatment may not be best for all MCL injuries and that it may be timely to reconsider surgical procedures, and their indications for use, to control AMRI.³⁹ Although it is inappropriate to recommend the clinical use of the AM procedure based solely on a biomechanical study in vitro, this work does suggest questions for clinical research, such as the following: In which cases

should AM reconstruction be used? Should it be used for an isolated MCL injury with an intact ACL? What should be done with a complete medial-sided (grade 3) injury? The present study provides some scientific data supporting such clinical studies.

On the basis of improving knowledge, 2 of the surgeon authors (S.V.B. and A.W.) perform MCL reconstruction according to the following criteria. In all cases, soft tissue suturing is undertaken: anatomic repair in acute cases, and retensioning "capsular shift" procedures to restore natural tension in chronic cases. Then, reconstruction is performed to protect these soft tissue repair constructs and allow unrestricted range of motion exercises. For isolated MCL tears, surgery is undertaken acutely in cases of excess valgus in full knee extension, tibial MCL avulsions in which the sMCL lies superficial to the pes anserine tendons or is folded into the joint cavity, and grade 3 lesions in athletes. Chronic isolated MCL lesions require surgery for medial instability and occasionally persistent pain. When in combination with ACL reconstruction, MCL repair plus reconstruction is performed for (1) any of the the previously mentioned indications for surgery of isolated MCL lesions, (2) concomitant grade 2 and 3 valgus laxity at 30° of flexion, (3) a positive Slocum test³³ finding in which ER fails to eliminate a positive anterior drawer test result, and (4) a positive dial test finding caused by anterior translation of the medial tibia (rather than posterior translation of the lateral tibia from posterolateral rotatory instability). The surgeon authors rarely reconstruct the POL, as simple suturing is usually adequate. The healing potential is so good because the POL is only a thickening of the capsule. The only indications for additional POL reconstruction are a posteromedial injury associated with posterior cruciate ligament laxity and, hence, posteromedial rotatory instability, which is very uncommon, or hyperextension with failure of posterior/posteromedial soft tissues. It is difficult to understand how POL reconstruction can be justified in any other scenario. It will not resist anterior translation of the medial tibia in AMRI, as it is oriented in the wrong direction. Despite this, there is a popular belief that POL reconstruction is important in such cases; our impression is that the POL is less important than has been previously thought.³⁹

In relation to clinical tests and decision making, the present work shows that an AM injury approximately doubled ER laxity and tripled valgus laxity. It is tempting, therefore, to speculate that clinical data from work performed on patients in a controlled and preferably randomized study may identify indications for AM reconstruction. With developing knowledge of functional anatomy, there should be better understanding of the clinical evaluation of MCL injuries. Excess valgus is primarily related to sMCL insufficiency, but larger laxities indicate disruption of the dMCL as well. dMCL insufficiency manifests in 2 ways. First, a positive dial test finding in which the medial tibial condyle subluxes anteriorly, causing excess ER (AMRI), indicates dMCL failure at 30°. At 90°, sMCL failure may also contribute to a positive test result.⁶ Second, the Slocum test³³ is useful. A positive anterior draw test finding in neutral rotation indicates ACL insufficiency. If

the test is repeated in ER and laxity is eliminated, the MCL is intact. Failure of the elimination of increased anterior translation in ER at 90° of flexion is probably caused by a combination of failure of the dMCL and possibly the sMCL. The use of clinical assessments is in decline because of excessive faith in magnetic resonance imaging, but with improved understanding of the subtleties of ligament function, more elegant and thorough examinations of the knee will allow an improved assessment of the components to an injury and thereby permit better fine-tuning of surgical solutions. Magnetic resonance imaging shows what is injured but rarely how significant that injury is.

Although this study provides clear findings on the biomechanical ability of AM reconstruction to stabilize the knee, it suffers limitations inherent in works on cadaveric tissues. In particular, MCL surgery may be used to protect concomitant soft tissue repair constructs, maintaining length while healing, but that cannot occur in vitro. An autograft construct may be appropriate for chronic tissue deficiency, but a more acute setting may only require a suture tape construct to support a repair procedure in the short term. Although 2 of the senior surgeon authors of this study use suture tapes for MCL surgery, their use in a novel AM reconstruction method can only be recommended after an appropriate clinical trial. The study was limited to the loads imposed during clinical stability testing and not the loads involved in daily activities because of concerns about slippage of graft fixation in the specimens from relatively old donor available. There is always a concern, when working on cadaveric specimens, relating to effects such as the order of cutting and reconstruction of tissues and whether there may be stretching out secondary to slippage of graft fixation and the deterioration of tissues. Slippage of graft fixation was minimized by using doubled fixation with secondary backup lateral fixation. It was not possible to change the order of tissue cutting because of the increased risk of tissue damage. The lack of slippage/stretching out is attested by the ER data (Figure 5), which show no measurable change in ER between the first and final cuts. Although the tibia hung freely when the knee was unloaded, restraining soft tissue tension ensured that the knee was compressed when the testing loads were applied. It would be desirable to add a more “dynamic” restraint from the semimembranosus tendon. A strength of this study was that it allowed native knee stability to be measured and used as a specimen-specific variable to assess the effects of ligament transection and reconstruction, by repeated-measures statistical analysis, under carefully controlled loads and with accurate measurements of bone-bone displacement.

Given that it is always desirable to simplify surgical procedures, it will be appropriate to study the AM reconstruction technique of Wang et al,³⁸ which places both the sMCL and the dMCL grafts into a single femoral tunnel; it is not known how this may affect the resulting stability. A further step could then be to combine these grafts into 1, with an intermediate orientation, on the basis that it should act as a short-term “splint” while the native tissues heal; then, only 1 fixation point is required on each bone. The present study of anatomic 2-strand AM

reconstruction showed that it can restore native laxity in an AM-injured knee when tested in vitro and questions the need for PMC/POL surgery for AMRI.

CONCLUSION

AM reconstruction of the superficial and deep bands of the MCL was able to restore native knee laxity measures related to AMRI in the face of complete transection of the MCL and PMC/POL in a cadaveric model.

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