Original Article

Individual and Combined Effects of Anterior Cruciate Ligament and Lateral Meniscus Injuries and Surgical Interventions on Tibiofemoral Peak Pressure Magnitude Vary by Flexion Angle

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Purpose: To quantify changes in peak tibiofemoral pressure magnitude and location following anterior cruciate ligament (ACL) and meniscus injuries and surgical interventions after cyclic motion. **Methods:** Six matched pairs of cadaveric knees underwent ACL or lateral meniscus injury and surgical intervention; specimens later underwent the other injury and surgical intervention. A servohydraulic testing system flexed and extended specimens 100 times before and following each intervention. Pressure magnitudes and locations were measured via submeniscal sensors on the medial and lateral tibial plateaus under 4 conditions (each at 0°, 15°, 30°, 45°, and 60° of knee flexion): native anatomy, first injury (InjPost1), first surgical intervention (SurgPost1), and second surgical intervention (SurgPost2). Linear mixed models compared interactions between group, side, and condition through SurgPost1 and interactions between side and condition for native and SurgPost2 for the magnitude and location of peak pressures. **Results:** Peak pressure was greater at 0°, 15°, and 30° in the medial compartment than the lateral compartment, regardless of injury condition. For the ACL group, at 0°, peak pressure in the lateral compartment was more posterior than the medial compartment, and at 15°, the meniscus group displayed more posterior peak pressure in the medial compartment than the lateral compartment. Greater anterior peak pressure was observed at 15° in SurgPost1 than native anatomy. Peak pressure was greater in SurgPost2 than native in the lateral compartment at 30°. For native and SurgPost2, peak pressure was greater in the medial compartment than the lateral compartment at 15° and greater in the lateral compartment than the medial compartment at 60°. **Conclusions:** ACL and meniscus injuries and surgical interventions result in similar anterior peak pressure translation. Sustaining both injuries and surgical interventions increases peak pressure magnitude. **Clinical Relevance:** Understanding how peak tibiofemoral contact pressures change in magnitude and location following an ACL or lateral meniscus injury and treatment may help guide treatment decisions and rehabilitation strategies that best prevent knee joint degeneration.

Osteoarthritis is one of the foremost debilitating conditions in the United States.[1,2] Post-traumatic arthritis accounts for approximately 12% of osteoarthritis cases in the United States.[3,4] Individuals with a knee injury are 4 to 7 times more likely to develop post-traumatic arthritis than individuals without previous knee injury.[3] Anterior cruciate ligament (ACL) and meniscus injuries are known osteoarthritis risk

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factors.[4,5] Highly active individuals and individuals with physically demanding occupations are particularly vulnerable to these injuries, including military service members.[6-8] With the rising incidence of ACL and meniscus injuries and limited therapeutic options for osteoarthritis, better understanding risk factors for knee joint degeneration after initial injury is essential to reduce the long-term burden of these injuries.

Increased osteoarthritis risk postinjury is likely the result of aberrant joint-loading patterns in addition to intra-articular biochemical changes.[9-11] Cadaveric studies have identified greater loading in the posteromedial knee in ACL-deficient specimens as compared to ACL-intact knees[12,13]; however, the changes in magnitude and pressure location have not been consistently reported.[14-16] Similarly, meniscus injury increases contact pressure and decreases contact pressure area within the joint.[14,16,17] Beyond the ACL and meniscus injuries themselves, ACL reconstruction and meniscus repair may also impact osteoarthritis risks.[5,18] These interventions improve functional outcomes but do not completely restore preinjury joint biomechanics, nor do they fully prevent osteoarthritis progression.[19-22] Understanding the roles of both injury and surgical intervention on post-traumatic osteoarthritis progression is necessary to guide treatment decisions and produce optimal postsurgical outcomes. To date, cadaveric studies examining the effects of ACL or meniscus injuries and their respective surgical interventions have utilized a static model.[13,17,23-25] These static models do not mimic the cyclic nature of the knee joint during human locomotion. Thus, more dynamic models that include cycling of the knee joint before and following both the injury and surgical interventions are essential for understanding how these injuries and interventions affect knee joint loading.

The purpose of this study was to quantify changes in peak tibiofemoral pressure magnitudes and locations following ACL and lateral meniscus injuries and surgical interventions after cyclic motion. We hypothesized ACL injury and reconstruction would increase peak contact pressure, ACL injury would shift the location of peak pressure posteriorly, and lateral meniscus injury would increase peak pressure in the lateral compartment with no effect on peak pressure location and that greater peak pressure magnitude and posterior translation of peak pressure location would be observed after the combined effects of both injuries and surgical interventions.

Methods

This was an in vitro human cadaveric study. Before commencement, the study received an institutional review board exempt determination, as the work was determined to be nonhuman subjects research.

Specimen Preparation

Six matched pairs of adult human knee specimens (12 total knee specimens) were obtained from 6 fresh-frozen cadavers (female = 16.7%, age = 68.7 ± 11.9 years). All specimens were free of observable deformities, previous injuries, osteoarthritis, fractures, and previous surgeries. No other inclusion or exclusion criteria were used. Specimens were fresh-frozen at −30 °C before study initiation, and specimens were thawed at room temperature before testing. Soft tissues beyond 20 cm distal and proximal to the knee joint were removed from the specimens while meniscocapsular attachments and capsular ligamentous structures were maintained. The proximal and distal ends of the specimens were potted in 1:1 Bondo and fiberglass resin (Bondo; 3M Company). Pressure sensors (Medical Sensor 4000 [1,500 psi]; Tekscan) were inserted submeniscally by detaching the meniscotibial ligaments. The periphery of the sensor was then sutured to the remaining soft tissues on the anterior and posterior tibia. Sensors covered the total area under each meniscus and the tibial plateau. Sensors were secured via sutures in the surrounding capsular tissue.

Surgical Intervention

One specimen from each matched pair was randomly assigned to either initial ACL injury (i.e., "ACL-first") or initial meniscus injury (i.e., "meniscus-first"); the other specimen from the cadaver was assigned to the opposite initial injury group (Fig 1). A medial arthrotomy was created to expose the intra-articular structures. ACL injuries were created by sharply transecting the ACL with a scalpel. After testing the injured condition, the specimen underwent single-bundle bone−patellar tendon−bone autograft ACL reconstruction. A 10-mm × 30-mm tibial bone plug and a 10-mm × 25-mm patellar bone plug were harvested with an oscillating saw to obtain a 10-mm patellar tendon graft. Next, a medial parapatellar arthrotomy was created to identify the anatomic femoral and tibial footprint of the ACL. Femoral and tibial tunnels were drilled to a 10-mm diameter and a 30-mm depth using an inside-out retrograde reamer (FlipCutter II; Arthrex). The graft was pretensioned at 20 N for 10 minutes. The graft was then passed through the tibial and femoral tunnels. Bone plugs were secured with 2 No. 2 high-tensile strength sutures (FiberWire; Arthrex) and secured with suspensory fixation via a screw and washer on the tibia and a suspensory button on the femur (Attachable Button System; Arthrex). A horizontal meniscus tear was created in the posterior horn with a scalpel via the medial arthrotomy. An No. 11 blade was inserted in line with the meniscal fibers to create a 1-cm-wide horizontal tear in the posterior horn.[22,23] After testing the injured condition, the meniscus was repaired in an outside-in fashion using a No. 2 high-tensile strength

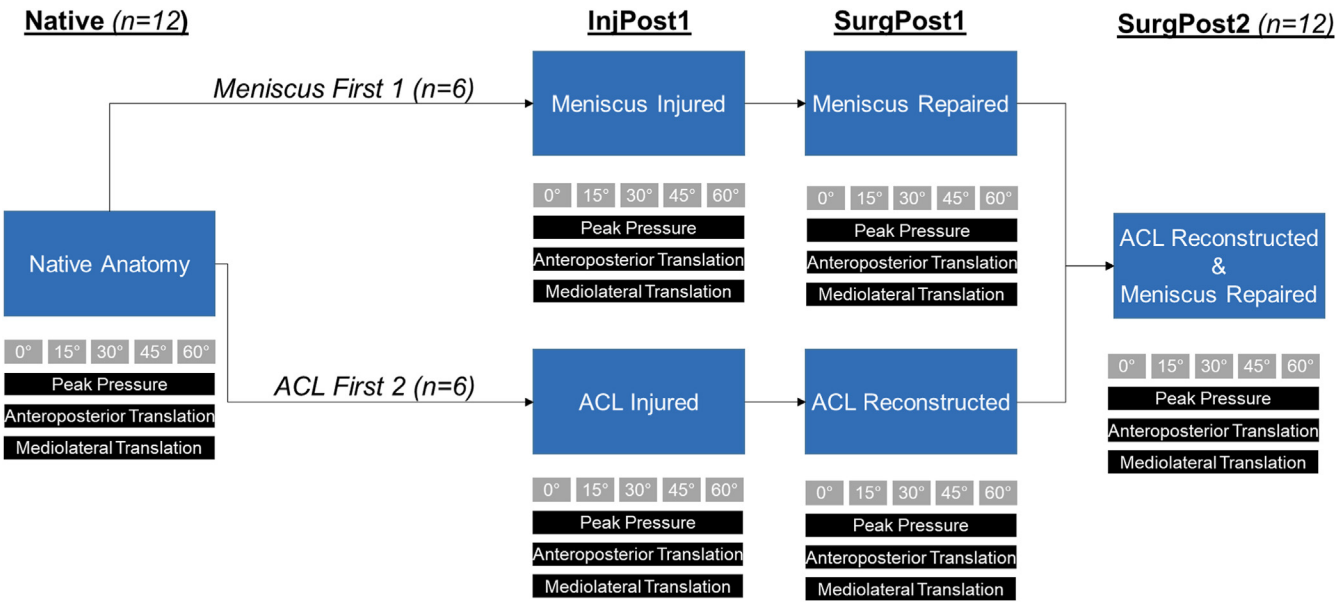

Fig 1. Experimental design. Matched pairs from each cadaver were assigned to either the ACL-first or meniscus-first group. (ACL, anterior cruciate ligament.)

suture with 2 evenly spaced mattress stitches. After the initial injury and surgical interventions, all specimens received the alternate injury and surgical intervention, so that all specimens received both injuries and surgical interventions.

Biomechanical Testing

The potted ends of the specimens were secured to a servohydraulic system (MTS 858 Mini Bionix II; MTS Systems) (Fig 2). A soft tissue clamp was affixed to the proximal quadriceps tendon and attached to a spring

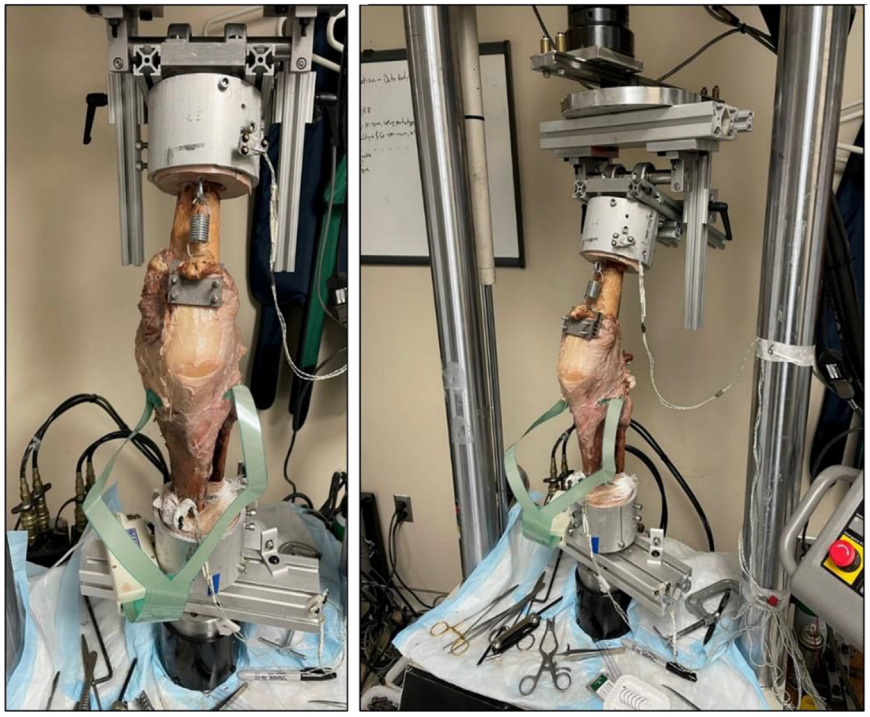

Fig 2. Experimental setup of the MTS 858 Mini Bionix II servohydraulic testing system. Specimens were cycled unloaded from 0° to 60° for 100 cycles between injury/surgical conditions. Peak pressures were recorded at 0°, 15°, 30°, 45°, and 60° of knee flexion after cycling with 500 N axial load.

affixed to the MTS fixture to maintain tension on the tendon throughout range of motion. Specimens were situated such that there were no anterior-posterior and no torsional forces acting on the femur and tibia-fibula segments at the resting position. However, free anterior-posterior, medial-lateral movement and free valgus-varus angulation of the knee joint were allowed during cycling and biomechanical testing. Peak intra-articular pressure magnitudes and locations were assessed in the medial and lateral compartments of the knee at 5 joint flexion angles (0°, 15°, 30°, 45°, 60°) for the following conditions: native, initial injury post-cycling (InjPost1), initial surgical intervention post-cycling (SurgPost1), and second surgical intervention postcycling (SurgPost2). These conditions were selected to assess both the effects of each injury and surgical intervention, as well as potential changes due to cyclic loading. In each condition, excluding native anatomy, specimens were cycled without axial load from 0° to 60° of knee flexion at 0.05 Hz for 100 cycles. Before and following cycling for each of the above-described conditions, the knee was locked in the joint angle of interest and loaded to 500 N; pressure data were recorded at 100 Hz for both the medial and lateral compartments.

Table 1. Postcalibrated Peak Contact Pressure (MPa)—Single Injuries

ACL	Native	InjPost1	SurgPost1
Lateral compartment			
0	1.38 ± 1.06	0.37 ± 0.65	0.85 ± 0.68
15	1.04 ± 0.90	0.41 ± 0.44	0.95 ± 0.77
30	1.10 ± 0.74	1.13 ± 0.60	1.26 ± 0.66
45	2.44 ± 2.25	1.82 ± 1.38	2.35 ± 2.05
60	2.35 ± 1.48	1.93 ± 1.46	1.64 ± 1.08
Medial compartment			
0	2.82 ± 1.03	2.85 ± 1.37	2.44 ± 0.83
15	2.93 ± 1.31	2.75 ± 1.26	2.50 ± 1.10
30	2.17 ± 0.85	2.55 ± 1.21	2.09 ± 1.17
45	1.28 ± 1.81	2.04 ± 1.61	1.74 ± 0.93
60	1.19 ± 1.67	1.60 ± 1.66	1.52 ± 1.43
Meniscus			
Lateral compartment			
0	0.98 ± 0.77	1.19 ± 0.87	1.89 ± 1.98
15	1.04 ± 0.88	1.04 ± 0.88	1.38 ± 0.60
30	1.24 ± 0.83	1.63 ± 0.53	1.51 ± 0.94
45	1.47 ± 0.61	1.79 ± 0.76	1.69 ± 0.99
60	1.80 ± 0.74	1.85 ± 0.73	1.77 ± 1.08
Medial compartment			
0	1.99 ± 0.50	2.39 ± 0.51	2.41 ± 1.16
15	1.99 ± 0.66	2.33 ± 1.04	1.79 ± 0.74
30	2.28 ± 1.45	1.87 ± 0.94	2.10 ± 1.45
45	2.07 ± 1.04	1.57 ± 1.1	2.21 ± 1.22
60	1.5 ± 1.07	1.32 ± 0.99	1.79 ± 1.05

NOTE. Descriptive statistics presented as mean ± standard deviation; positive values indicate anterior translation, while negative values indicate posterior translation.

ACL, anterior cruciate ligament; InjPost1, first injury postcycling; Native, before injury; SurgPost1, first surgical intervention postcycling.

Data Reduction

Pressure data were postcalibrated to ensure consistent comparisons between samples, and peak contact pressures were identified for both the medial and lateral compartments via custom MATLAB scripts (vR2020a; MathWorks). Peak pressure magnitude was determined by identifying the sensel of the sensor with the highest pressure and calculating the mean pressure of the sensel with the highest pressure and the 8 surrounding sensels. The sensel with the greatest peak pressure was identified at 0° of knee flexion in the native condition; the location of this sensel was defined as 0 for each specimen. Changes in peak pressure location were then measured relative to the peak pressure location at 0° of knee flexion for the native anatomy condition, such that the peak pressure location was subtracted from the peak pressure location at 0° of knee flexion for the native anatomy condition. Anterior and medial deviations were defined as positive values, and lateral and posterior deviations were defined as negative values. Peak pressure magnitudes and peak pressure locations were identified for both the medial and lateral compartments.

Statistical Analysis

Specimens were grouped by initial injury type, injury or surgical condition, and knee flexion angle. Separate analyses were performed for the effects of single injuries (where specimens were grouped by initial injury type) and combined injuries (where native anatomy was compared to all specimens that had undergone both injuries and surgical interventions). Outliers for peak pressure, anteroposterior translation (apTrans), and mediolateral translation (mlTrans) within each flexion angle were identified by calculating data-informed cutoff values (less than 3 times the interquartile range below the first quartile and greater than 3 times the interquartile range above the third quartile) for peak pressure, anteroposterior translation, and mediolateral translation. Values that fell outside the identified range were removed for analyses (missing counts and percentage for each outcome: single injury analysis peak pressure: n = 11, 3.1%; single injury analysis apTrans: n = 12, 3.3%; single injury analysis mlTrans: n = 16, 4.4%; combined injury analysis peak pressure: n = 11, 4.6%; combined injury analysis apTrans: n = 0, 0.0%; combined injury analysis mlTrans: n = 1, 0.4%). Following removal of the outliers, descriptive statistics were computed as means ± standard deviations. For the single injury analysis, linear mixed models were used to test the interaction of group (meniscus, ACL), side (lateral and medial compartment), and condition (native, InjPost1, SurgPost1) (fixed effects) in cadaveric knees (random effects) for peak pressure, mlTrans, and apTrans, separately for each knee flexion angle (0°, 15°, 30°, 45°, 60°) (n = 6 per group). For the combined

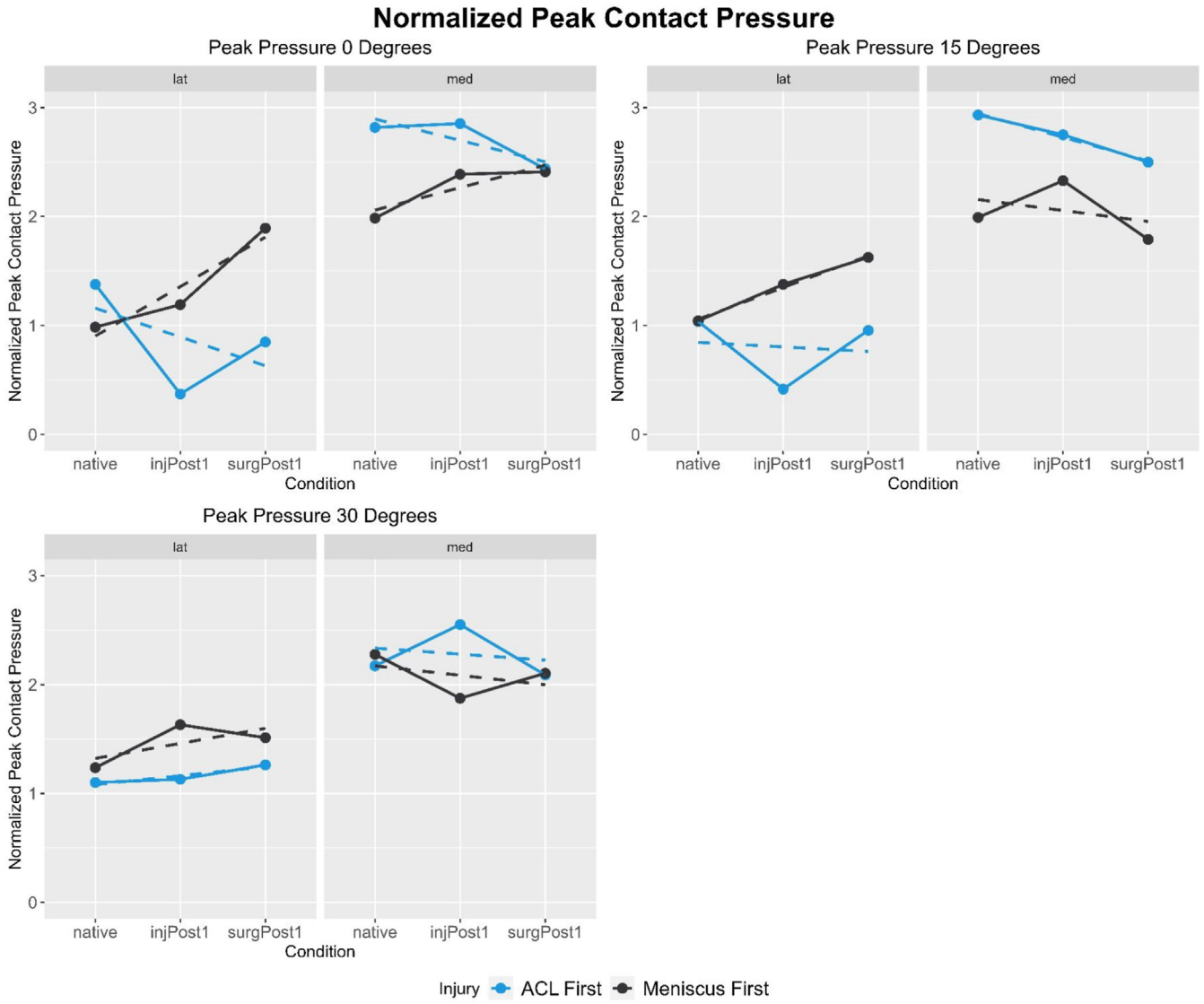

Fig 3. Normalized peak pressure magnitude in different knee flexion angles for anterior cruciate ligament and lateral meniscus injury and corresponding surgical intervention in both lateral and medial compartments. Dashed lines indicate trend lines for each group.

injury analysis, linear mixed models were used to test the interaction of side (lateral and medial compartment) and condition (native, SurgPost2) (fixed effects) in cadaveric knees (random effects) for peak pressure, mlTtrans, and apTrans, separately for each knee flexion angle for all specimens (n = 12). For both analyses, Bonferroni-adjusted pairwise comparisons were performed following each linear mixed-model analysis when appropriate. Statistical significance was set a priori at $\alpha < .05$.

Results

Single Injury Peak Contact Pressure

Descriptive statistics are shown in Table 1. A group-by-side interaction was observed at 15° ($P = .008$), specifically: peak pressure in the medial compartment was greater in the ACL-first group (2.73 ± 1.17 MPa) than the meniscus-first group (2.06 ± 0.82 mPa; $P = .040$; Fig 3), peak pressure in the medial compartment of the ACL group was greater than peak pressure in the lateral compartment of the ACL group ($P < .001$; Fig 3), and peak pressure in the medial compartment of the meniscus group (2.06 ± 0.82 MPa) was greater than peak pressure of the lateral compartment of the meniscus group (1.35 ± 0.84 mPa; $P = .004$; Fig 3). A main effect of side was observed at 0° ($P < .001$), in which peak pressure in the medial compartment (2.48 ± 0.91 MPa) was greater than peak pressure in the lateral compartment (1.13 ± 1.13 MPa; Fig 3). A main effect of side was also observed at 30° ($P < .001$), in which peak

Table 2. Peak Pressure Translation Relative to the Native Anatomy Condition (mm)—Single Injuries

	Native		InjPost1		SurgPost1	
	Anteroposterior	Mediolateral	Anteroposterior	Mediolateral	Anteroposterior	Mediolateral
ACL						
Lateral compartment						
0	—	—	−3.17 ± 4.49	0.95 ± 1.91	−4.57 ± 3.31	1.59 ± 3.50
15	−8.47 ± 8.49	−0.85 ± 8.56	−5.72 ± 13.93	−4.66 ± 10.74	1.48 ± 6.50	−4.02 ± 8.59
30	−9.74 ± 9.11	−0.21 ± 8.40	−5.29 ± 13.94	−0.64 ± 11.15	−0.63 ± 3.66	−3.81 ± 9.05
45	−3.39 ± 9.49	−0.42 ± 10.31	−7.41 ± 12.32	−1.06 ± 12.40	−5.93 ± 9.59	−2.96 ± 13.31
60	−4.87 ± 8.78	−2.96 ± 8.82	−7.62 ± 11.83	−1.27 ± 13.00	−7.20 ± 9.39	−3.60 ± 13.74
Medial compartment						
0	—	—	−0.85 ± 1.94	0.42 ± 1.47	0.00 ± 4.17	0.00 ± 1.04
15	1.27 ± 6.22	0.21 ± 4.98	−5.93 ± 10.18	4.66 ± 10.24	−0.42 ± 5.72	1.27 ± 7.09
30	−7.83 ± 7.51	3.17 ± 4.53	−4.87 ± 7.16	2.33 ± 11.05	−3.39 ± 8.56	4.23 ± 7.21
45	−4.87 ± 7.16	4.23 ± 10.28	−6.98 ± 5.37	2.75 ± 11.14	−6.14 ± 8.92	6.98 ± 8.06
60	−4.44 ± 14.36	4.87 ± 8.70	−8.68 ± 7.51	4.44 ± 11.09	−11.22 ± 7.89	2.96 ± 9.22
Meniscus						
Lateral compartment						
0	—	—	2.54 ± 4.97	0.85 ± 1.47	2.79 ± 2.75	0.64 ± 0.73
15	−0.64 ± 2.51	0.42 ± 2.97	−0.21 ± 12.63	6.35 ± 9.57	3.39 ± 7.52	4.44 ± 7.57
30	−5.29 ± 14.11	6.35 ± 14.28	−1.69 ± 12.56	4.44 ± 12.72	−1.69 ± 12.97	1.48 ± 10.70
45	−5.93 ± 13.50	2.96 ± 11.43	−2.33 ± 12.35	6.35 ± 13.17	−2.75 ± 14.08	2.75 ± 11.37
60	−5.72 ± 12.97	−0.21 ± 7.07	−2.33 ± 12.58	3.17 ± 12.79	−3.81 ± 15.60	1.06 ± 9.28
Medial compartment						
0	—	—	−1.27 ± 4.31	−1.02 ± 1.66	1.06 ± 4.78	−0.25 ± 3.41
15	−6.77 ± 4.15	2.12 ± 5.88	−6.56 ± 5.53	2.12 ± 4.30	−4.23 ± 5.31	4.87 ± 7.16
30	−8.68 ± 4.91	5.29 ± 10.05	−7.41 ± 9.62	3.81 ± 6.43	−5.72 ± 5.89	4.23 ± 8.56
45	−9.95 ± 7.89	4.23 ± 9.08	−7.62 ± 11.44	2.33 ± 10.05	−3.18 ± 10.28	2.96 ± 8.30
60	−9.74 ± 8.6	4.23 ± 7.90	−5.50 ± 10.37	1.48 ± 13.04	−2.54 ± 9.80	1.69 ± 10.80

NOTE. Descriptive statistics presented as mean ± standard deviation; positive values indicate anterior translation, while negative values indicate posterior translation.

ACL, anterior cruciate ligament; InjPost1, first injury postcycling; Native, before injury; SurgPost1, first surgical intervention postcycling.

pressure in the medial compartment (2.18 ± 1.14 MPa) was greater than peak pressure in the lateral compartment (1.32 ± 0.70 MPa; Fig 3).

Single Injury Peak Pressure Translation

Descriptive statistics for apTrans and mlTrans are shown in Table 2. A group-by-side interaction for

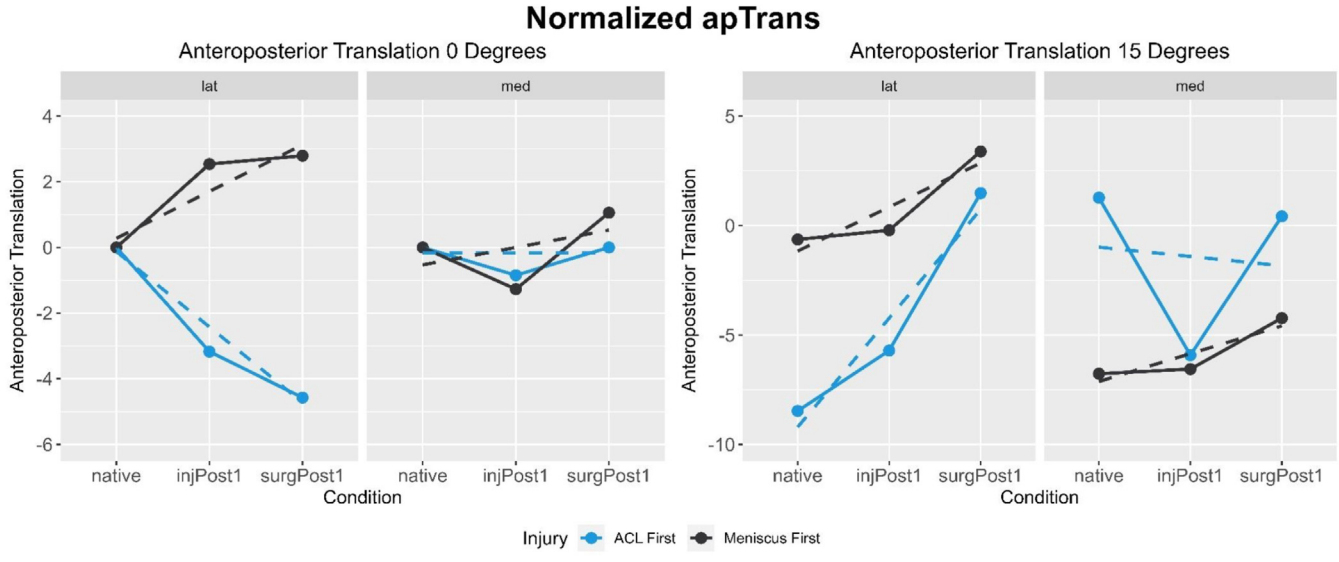

Fig 4. Normalized anteroposterior translation of peak pressure in different knee flexion angles for anterior cruciate ligament and lateral meniscus injury and corresponding surgical intervention in both lateral and medial compartments. Dashed lines indicate trend lines for each group.

Table 3. Postcalibrated Peak Contact Pressure (MPa)—Combined Injuries

	Native	SurgPost2
Lateral compartment		
0	1.18 ± 0.91	1.33 ± 1.25
15	1.04 ± 0.85	1.66 ± 1.38
30	1.18 ± 0.76	2.09 ± 0.85
45	1.54 ± 0.82-	2.13 ± 1.10
60	2.05 ± 1.11	2.83 ± 1.68
Medial compartment		
0	16.81 ± 48.04	2.45 ± 1.79
15	−2.50 ± 1.13	1.98 ± 0.98
30	2.23 ± 1.16	1.53 ± 0.74
45	1.68 ± 1.47	1.57 ± 0.83
60	1.36 ± 1.31	1.11 ± 0.68

NOTE. Descriptive statistics presented as mean ± standard deviation. Native, before injury; SurgPost2, after second injury and surgical intervention, postcycling.

apTrans was observed at 0° (P = .008), specifically: apTrans was less (more posterior) within the lateral compartment in the ACL group (−2.25 ± 3.20 mm) than the medial compartment of the ACL group (−0.17 ± 2.62 mm; P = .048; Fig 4), and apTrans was less (more posterior) in the lateral compartment of the ACL group than the lateral compartment of the meniscus group (1.61 ± 3.05; P = .003; Fig 4). Another group-by-side interaction was observed for apTrans at 15° (P = .002), specifically: apTrans was lower (more posterior) in the medial compartment of the meniscus group (−5.86 ± 4.87 mm) than the lateral compartment of the meniscus group (0.85 ± 8.30 mm; P = .002; Fig 4). A main effect of general condition was observed at 15°, in which apTrans was greater (more anterior) in SurgPost1 (0.26 ± 6.55 mm) than in the native condition (−3.65 ± 6.82 mm; P = .030; Fig 4). No significant findings were observed for mlTrans.

Combined Injury Peak Pressure

Descriptive statistics are shown in Table 3. A condition-by-side interaction was observed at 30° (P = .020), specifically: peak pressure was greater in the lateral compartment in SurgPost2 (2.09 ± 0.85 MPa) than the lateral compartment in the native condition (1.18 ± 0.76 MPa; Fig 5). A main effect of side was observed at 15°, in which peak pressure was greater in the medial (2.24 ± 1.07 MPa) than lateral compartment (1.35 ± 1.17 MPa; P = .009; Fig 5). A main effect was also observed at 60°, in which peak pressure was greater in the lateral compartment (2.44 ± 1.45 MPa) than the medial compartment (1.24 ± 1.03 MPa; P = .003; Fig 5).

Combined Injury Peak Pressure Translation

Descriptive statistics for apTrans and mlTrans are shown in Table 4. No significant findings were observed for either apTrans or mlTrans.

Discussion

Our findings show limited effects of a single injury or surgical intervention on peak pressure magnitude and agree with prior work regarding changes in pressure location resulting from ACL and meniscus injuries.[12-17] Our results also highlight the importance of interpreting effects of ACL and meniscus injury or surgical intervention within the context of specific joint angles, as these effects are not consistent throughout ranges of knee flexion. Our observed peak pressures are comparable with other studies that used comparable axial loading, which we felt was appropriate given our serial testing and cycling procedures.[19] But, contrary to our hypothesis, we did not observe any effect of single injury or surgical condition on peak pressure. The reasons for this are unclear but may relate to the relatively light axial load used (500 N) at all flexion angles. We did observe greater pressure in the medial compartment than the lateral compartment at 0°, 15°, and 30°, with no mediating effect of injury status, in the single injury analysis. The greater peak pressures observed in the medial compartment agree with previous work regarding differences in loading between compartments. It is well established that meniscus injury increases peak pressure in the compartment in which the injury was sustained—our data suggest the effects of ACL and lateral meniscus injuries on peak pressure magnitude are comparable between injuries, but differences may be more pronounced at 15° of knee flexion, where peak pressure in the lateral compartment was similar to the medial compartment only in the meniscus group. We observed an anterior translation of peak pressure location after both surgical interventions with no differences between groups at 15° of knee flexion with no mediating effect of injury type. Reasons for this are unclear and caution is warranted when interpreting translation data, as we observed wide variability in our peak pressure translation data between specimens. While caution is warranted, the observed effect on apTrans indicates there are similar effects of surgical interventions for ACL and meniscus injuries on peak pressure location distinct from the injuries themselves, which warrants further investigation into the effects of surgical techniques on intra-articular joint loading.

In agreement with our hypothesis, the ACL group showed posterior translation of peak pressure location, particularly in the lateral compartment, which aligns with previous work.[12-16] Meniscus injury results in less total contact pressure area, and it has been suggested that changes in intra-articular loading are dependent on the location and severity of the injury.[17,23,26] To our knowledge, a consistent alteration in peak contact pressure location after meniscus injury has not been shown in the literature.[14,17,26] Our findings are in

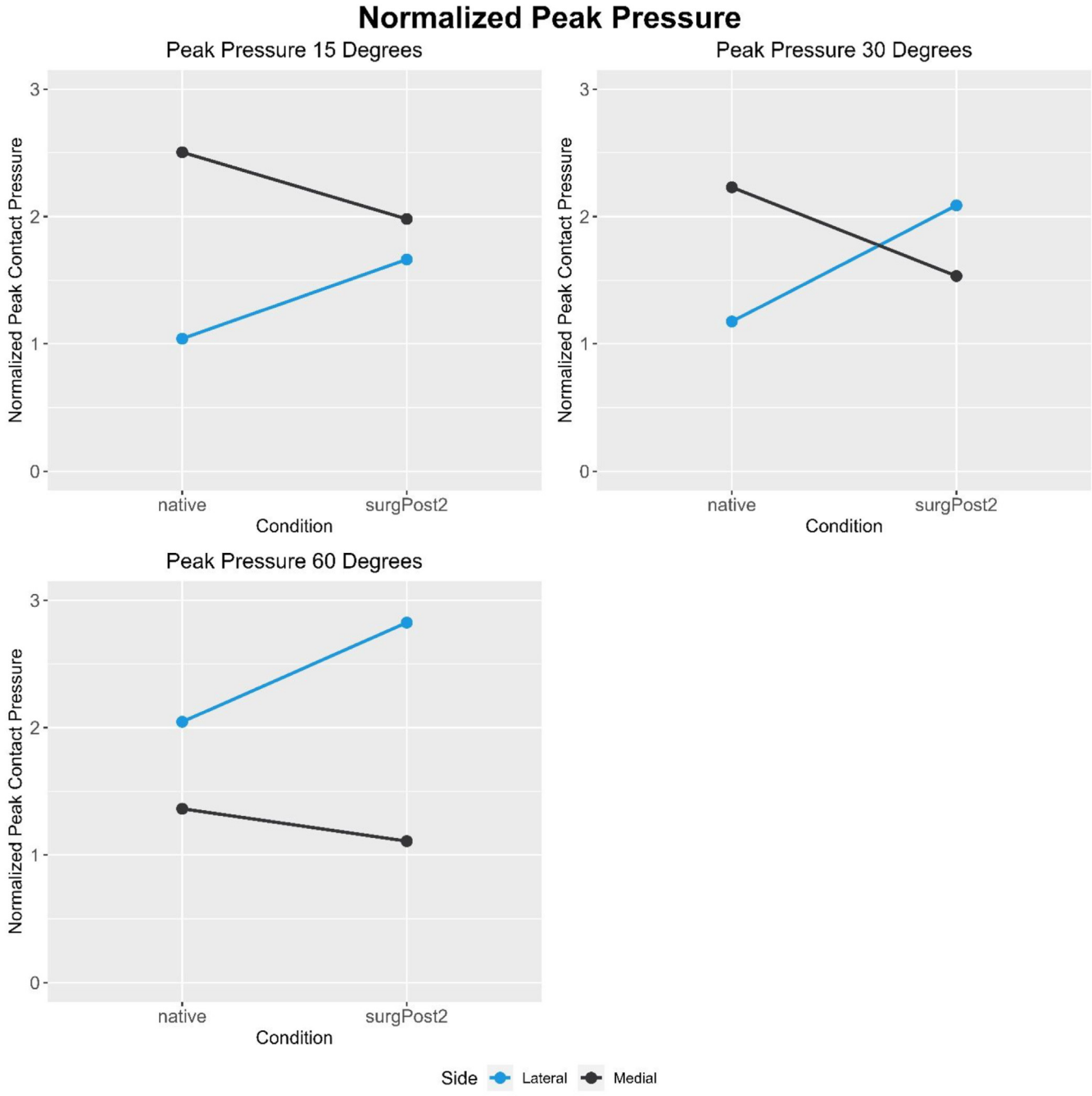

Fig 5. Normalized peak pressure in different knee flexion angles before and after both anterior cruciate ligament and lateral meniscus injury and respective surgical interventions in both the lateral and medial compartments.

alignment with these previous studies, as we administered identical injuries and repair techniques in our study, and we observed wide variability in both anteroposterior translation and mediolateral translation. Our results provide preliminary evidence that anteroposterior translation of peak pressure location resulting from meniscal injury and repair is variable and potentially less pronounced than the more predictable posterior shift in contact pressure in the lateral compartment after ACL injury, particularly at 0° and 15° of knee flexion.

Our findings partially support our second hypothesis. Specifically, peak pressure magnitude increased after both surgical interventions, although there was wide variability and no predictable pattern in translation of peak pressure location. This indicates there are additive effects of additional injury and surgery on peak pressure magnitude. While effects of injury order on intra-

Table 4. Peak Pressure Translation Relative to the Native Anatomy Condition (mm)—Combined Injuries

	Native		SurgPost2	
	Anteroposterior	Mediolateral	Anteroposterior	Mediolateral
Lateral compartment				
0	—	—	0.11 ± 9.57	0.95 ± 10.64
15	−4.55 ± 7.23	−0.21 ± 6.15	−0.95 ± 9.85	0.35 ± 7.05
30	−7.51 ± 11.56	3.07 ± 11.68	−2.75 ± 10.22	−1.27 ± 11.33
45	−4.66 ± 11.21	1.27 ± 10.53	−5.72 ± 10.49	−1.91 ± 11.60
60	−5.29 ± 10.57	−1.59 ± 7.76	−4.02 ± 11.21	−1.59 ± 12.23
Medial compartment				
0	—	—	0.85 ± 5.84	1.80 ± 5.59
15	−2.75 ± 6.56	1.16 ± 5.29	−1.27 ± 8.10	2.65 ± 7.86
30	−8.26 ± 6.07	4.23 ± 7.51	−5.82 ± 10.24	3.49 ± 9.46
45	−7.41 ± 12.43	4.23 ± 9.24	−8.57 ± 9.19	−4.34 ± 12.59
60	−7.09 ± 11.62	−4.55 ± 7.93	−9.63 ± 12.21	−2.01 ± 11.54

NOTE. Descriptive statistics presented as mean ± standard deviation; positive values indicate anterior translation, while negative values indicate posterior translation.

Native, before injury; SurgPost2, after second injury and surgical intervention, postcycling.

articular loading warrant investigation, injury order is impossible to control, and ACL and meniscus injuries often occur concurrently.[27,28] Sustaining both injuries simultaneously and/or undergoing both surgical interventions concurrently may result in synergistic effects on joint loading not seen when injuries and surgical interventions occurred separately. Alternatively, in vivo tibiofemoral loading may be further altered through individual compensatory patterns after injury, and the relationship between in vitro pressure alterations and in vivo pressures and compensatory strategies warrants further investigation. Regardless, it is noteworthy that we saw significant variability in both apTrans and mlTrans after both injuries, while a positive apTrans was observed for both ACL and meniscus groups. This suggests uniquely tailored surgical and rehabilitation interventions may be warranted for individuals with multiple injuries to avoid aberrant joint loading.

Limitations

Our study is not without limitations. First, our sample size for the comparison of native anatomy to multiple injuries and surgical interventions was twice that of our single injury analysis, as we included all specimens in the comparison regardless of injury order. Despite the use of cyclic motion between conditions, dynamic loading throughout the range of motion was not possible, and the 500 N load was not adjusted for each joint angle, which may differ from the loading scenario during common daily activities. Our specimens were also much older than the average age of a person who undergoes an ACL or meniscus injury and subsequent surgical interventions. While our fixation methodology of the femur and tibia-fibula segments to the servohydraulic system allowed for 6-degree of freedom knee motions, our experimental setup did not account for additional factors (e.g., muscles acting across the knee joint) that may alter the observed changes in peak pressure magnitude and location resulting from ACL and meniscus injuries and their respective corrective surgeries in vivo. Lastly, the high variability and relatively small magnitudes of change in peak pressure magnitude and location may indicate a lack of power and thus a need for similar studies to use additional specimens when assessing effects of ACL or meniscus injury on tibiofemoral peak pressure magnitude or location.

Conclusions

ACL and meniscus injuries and surgical interventions similarly result in anterior peak pressure translation. Sustaining both injuries and surgical interventions increases peak pressure magnitude.

Disclosures

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