BIOMECHANICAL CONSIDERATIONS FOR NEUROMUSCULAR CONTROL OF KNEE ARTHROPLASTY PATIENTS AND PROPRIOCEPTION OF HEALTHY PARTICIPANTS

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To whom is this dissertation dedicated? I have often thought about that question. I did not pursue this degree for any one person, nor myself really, but rather for the idea of lifelong learning. Our lives are not defined by the letters behind our names or degrees that we hold, but by the people we have challenged to grow, inspired to learn, and to who we have brought laughter. To that end, this dissertation is dedicated to the innumerable people that have influenced my life and encouraged me to pursue a meaningful education, while having fun along the way. It is also dedicated to the countless individuals suffering with knee osteoarthritis, whom the findings in this collection of studies are intended to benefit.

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ABSTRACT

Knee osteoarthritis (OA) patients often experience significant limitations in walking and stair negotiation abilities. Total knee arthroplasty (TKA) and unicompartmental knee arthroplasty (UKA) patients are frequently unable to ascend and/or descend stairs due to pain, limited pre- and post-operative range of motion, or poor limb coordination associated with diminished quadriceps performance. Three-dimensional (3D) biomechanical gait analysis has served as a measurement technique to assess these pre- and post-operative functional deficits. Through the use of 3D biomechanical analysis, various indicators of knee OA progression and development, as well as risk of revision, have been established with external knee adduction moment (KAM) and knee flexion moment (KFM), among others. Though these risk factors are adequate in predicting medial loading and willingness to load the limb, respectively, they are not sufficient in determining pre- and post-operative neuromuscular control deficiencies. Longitudinal analyses of time-derivatives for frontal and sagittal plane measures of knee joint loading, as well as aggregate measures of leg stiffness are examined in this dissertation. These areas have been less well studied in this population and may further explain neuromuscular deficits from a biomechanical perspective. To further examine how biomechanical variables may explain neuromuscular control, this dissertation also includes a cross sectional analysis of knee joint proprioception, specifically joint position sense (JPS), in several testing positions for a healthy cohort. Several analyses were conducted to examine any differences in JPS between limbs, and associations between biomechanical measures of loading and knee joint proprioception in healthy individuals. Given the lack of evidence examining these associations, these analyses were largely exploratory; however, hypothesis driven analyses were conducted. The novel findings of this dissertation inform on these associations (or lack thereof) and provide insight into longitudinal gait changes between TKA and UKA patients. The results also inform on neuromuscular considerations for knee biomechanics in these patients during stair negotiation and level walking.

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PART I

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CHAPTER 1:

CROSS-SECTIONAL ANALYSIS OF LEG STIFFNESS AND KNEE JOINT POSITION SENSE RELATIONSHIPS BETWEEN LIMBS OF HEALTHY ADULTS DURING STAIR DESCENT

Abstract

Background: Abnormal loading patterns and proprioception during dynamic tasks are linked to overuse conditions, including but not limited to osteoarthritis (OA) development and progression. Stair descent is often difficult for OA patients and allows modeling limbs as spring-mass systems to assess load-bearing characteristics via three-dimensional leg stiffness (K_{leg}) calculations. Knee joint position sense (JPS) may provide insight into lower extremity responses to loading and quantifies proprioception. Currently, no evidence exists examining relationships between K_{leg} and knee JPS. The objective was to examine relationships between three leg stiffness calculations and knee JPS measures in healthy adults during stair descent, and differences between limbs of healthy adults. **Methods:** Fifty-seven subjects with no history of injury or surgery within the last 6 months were included. Ten familiarization trials to 30° were completed prior to JPS determination. Open-kinetic chain (OKC) knee extension and closed-kinetic chain (CKC) double-leg and single-leg squats were performed for JPS testing. Subjects completed JPS testing followed by biomechanical assessment of stair descent. Sagittal plane mean angular errors during knee JPS testing were calculated. All trials of stair descent and JPS were measured via 3D motion capture. Pearson's correlations were calculated to determine relationships between three K_{leg} calculations and angular error measures. **Findings:** No significant correlations were observed between K_{leg} and JPS angular error. No differences between limbs were observed for angular error measures or leg stiffness, though a small effect (d=0.325, p=0.091) was detected for JPS in a single-leg stance test, with the non-dominant limb demonstrating greater absolute error by approximately two degrees. **Interpretation:** Joint position sense was not correlated with leg stiffness, perhaps indicating that JPS alone is unable to explain loading mechanisms during stair descent. These results provide baseline which can be compared to populations with knee OA and other pathological conditions.

1. Introduction

The sensorimotor system is vast and highly integrated. Sensory afferent pathways relay joint and limb position information to the central nervous system to produce an efferent response. Muscles controlling the limb respond via efferent motor responses collectively and in the case of the lower extremity, muscles will accommodate for load-bearing activity.[1] Proprioception and joint kinesthesia, or joint position sense (JPS), are therefore important to measure to identify afferent input that dictates neuromuscular control of the limb during loading. Various methods for evaluating proprioception have been validated and have been determined to be reliable,[2, 3] while limb compression has also been investigated via biomechanical assessment of leg stiffness. Stiffness has been suggested to be an indirect measure of lower extremity neuromuscular control by modeling the deformation of the leg with uniplanar, multiplanar, and triplanar methods.[4-7]

Analysis of leg stiffness and limb compression behavior has been well established during activities of daily living (ADLs) such as walking and running.[4, 8-12] The lower extremity behaves synonymously to a spring under a load. The behavior of limbs while load bearing during more functional activities such as descending stairs has been less documented. Furthermore, methods of determining leg stiffness have largely been uniplanar or biplanar,[5, 7] or have determined leg stiffness as an aggregate of whole-body compression, such as vertical stiffness.[9, 13, 14] While these approaches have been able to successfully model the lower extremity as a spring, they do not consider the lower extremity as a 3-dimensional unit. More recent methods employed by Liew et al[10, 15] that model both a 3D ground reaction force vector and 3D leg vector have attempted to do so. Given the paucity of evidence examining any of these methods during stair negotiation, it is prudent to examine which may give insight into leg compression during this activity.

Although JPS ability has been widely examined in injured populations,[16-23] the degree to which knee JPS and these methods of leg stiffness are related are unknown and have not been examined in either healthy or injured populations. The knee joint is a critical component of the lower kinetic chain, particularly during load bearing activities. Quadriceps and hamstring muscle spindles, as well as joint mechanoreceptor afferents during functional load bearing tasks are necessary for proper eccentric control.[1, 24, 25] Measures of knee JPS may give insight into one's joint kinesthetic ability and may also give evidence of the importance of the knee joint and surrounding muscles within the entire limb system. It would be advantageous to model the limb as a spring; however, it is unclear which model is associated with knee joint position sense, if at all. Therefore, the purpose of this analysis was to examine relationships between various measures of open-kinetic chain (OKC) and closed-kinetic chain (CKC) knee JPS and various methods of leg stiffness in healthy adults during stair descent. Differences between dominant and non-dominant limbs will also be analyzed to determine how self-reported limb dominance will change these relationships. We hypothesized that dominant limbs would demonstrate less angular error (better proprioception) for all JPS testing and less leg stiffness compared to non-dominant limbs, and that angular error would be positively correlated with leg stiffness on both limbs.

1.1 Methods

1.1.1 Subjects

Fifty-seven healthy volunteers (28 male, 29 female; age: 22.26 ± 3.23 years; mass: 71.21 ± 16.63 kg; height: 1.69 ± 0.09 m; BMI: 24.64 ± 4.21) were recruited from the community. Fifty-eight subjects were recruited; however, one participant was removed from the analysis due to inadequate left limb thigh and shank segment resolution. Subjects were screened using a standard, Office of Human Studies approved, health-history questionnaire. Subjects were recruited via convenience sampling in University classroom settings and they were excluded if pregnant, had known neurologic conditions, had a previous history of a hip, knee, or ankle ligament reconstruction, or had a history of recent lower extremity musculoskeletal injury (within 6 months) that hindered the ability to extend the knee, squat, or negotiate stairs. Subjects were asked to self-report limb dominance. Prior to enrollment in the study, all patients signed informed consent and the study was approved by the Institution’s Committee on Human Studies.

1.1.2 Procedures

All biomechanical analyses were conducted in the University Human Performance and Biomechanics Laboratory. Motion capture data was collected using 29 retroreflective markers placed on the thorax, pelvis, and lower extremities and four marker-based arrays placed on the thigh and shank segments (Appendix A). Stair descent motion capture procedures were similar to Vallabhajosula et al.[26] Five successful trails were collected, indicated by successful foot strike without the use of the handrail for assistance. All kinetic data were smoothed using a Butterworth filter with a 10 Hz cutoff frequency and processed using Visual 3D (C-Motion, Inc., Germantown, MD). All kinematic and kinetic data were smoothed using a Butterworth filter with a 10 Hz cut-off and ground reaction force was filtered using a 50 Hz cut-off frequency.[27] Kinematic data using the array-based marker-set were collected with a 18-camera Vicon motion capture system and Vicon Nexus 2.5 software (Vicon, Inc., Centennial, CO) at 240 Hz and time synchronized with kinetic data collected at 960 Hz collected from on a force plate (Advanced Mechanical Technology Incorporated, Boston, MA) embedded flush with the second step of a three-step staircase.

Joint position sense was tested in three positions: seated OKC knee extension, and standing CKC double-leg squat, and single-leg squat (SLS). Ten repetitions[21] to a demonstrated reference angle of 30°, as described by Suner-Keklik et al,[28] from the resting position (90°) were completed during each test. The reference angle was measured using a goniometer by a single, board-certified Athletic Trainer experienced in goniometric measurement. The stationary arm was placed in line with the greater trochanter of the femur, fulcrum at the lateral epicondyle of the knee, and movement arm was placed in line with the lateral malleolus. In the OKC test, subjects felt the reference angle by the examiner passively extending the knee from the resting position to the reference angle. In both CKC tests, the subject performed a squat starting from 0° of knee flexion (standing upright in full knee extension) and was instructed to squat and flex, then maintain the reference angle indicated by the examiner. In all three conditions, the subject was not explicitly given the reference angle, but instructed to “familiarize themselves” with the produced angle for 10 seconds. Subsequent trials were completed without visual aid. The order in which subjects completed each test, as well as the order in which each limb was tested, was randomized. Randomization was chosen to eliminate testing bias for dominant and non-dominant limbs, as well as to eliminate the bias of a potential learning effect that might occur. Only 10 double-leg squat repetitions were completed for the CKC test to the demonstrated reference angle from the standing position due to redundancy of simultaneous knee flexion actions. For each trial, subjects were instructed to reproduce the demonstrated angle and maintain the position for three seconds.

Angular error of knee angles during JPS testing was calculated using: 1) Absolute Angular Error (AAE) and 2) Relative Angular Error (RAE), defined by Arvin et al.[29] The AAE measures JPS without considering the direction of error (flexion/extension are both positive values), whereas RAE is directional (flexed past 30° is negative and extended is positive). The average of 10 trials was determined to assign an error to each limb. Greater error would indicate less proprioceptive ability, i.e., inability to reproduce a specific joint position. Mathematical calculations are presented below:

1) *Absolute Angular Error*

   AAE= [| (target position−trial 1) |+| (target position−trial 2) |+| (target position−trial 3) …|]/10

2) *Relative Angular Error*

   RAE= [(target position−trial 1) +(target position−trial 2) +(target position−trial 3) …]/10

Three methods of leg stiffness (K_{leg}) were utilized : 1) Uniplanar, described by McMahon and Cheng[5] 2) vertical stiffness (K_{vert}), also described by McMahon and Cheng,[5] and 3) 3D leg stiffness (3D K_{leg}), described by Liew et al[10] and scaled to a dimensionless unit of %BW/LL. The dot product of the resultant ground reaction force (GRF) vector and the unit vector of the leg is taken in order to calculate the proportion of the resultant GRF that is in line with the leg. In doing so, a projected GRF vector is created as a scalar quantity at each frame. The projected GRF can then be compared to the leg vector frame by frame. The peak of this projection (the vector of greatest magnitude that is most in line with the leg vector) can then be used to estimate stiffness. The leg vector is taken as a line from the COP to the estimated hip joint center. Equations for each measure of leg stiffness are listed below:

$$1)\ \ Kleg = \frac{Fmax}{\Delta \mathrm{L}}$$

Where $\Delta L = \Delta y + L_0(1 - \cos\theta)\ and\ \theta = \sin^{-1}(ut_c\,/2L_0)$; L_0 = standing leg length θ = half angle of the arc swept by the leg, u = horizontal velocity, and t_c = ground contact time

$$2)\ \ Kvert = Fmax/\Delta y$$

Where F_{max} is the maximum vertical ground reaction force and Δy is the maximum vertical displacement of the center of mass

$$3)\quad 3D\ Kleg = \frac{Peak\ Projected\ GRF}{\Delta L}$$

Where peak projected GRF is the peak scalar quantity of the dot product of the resultant GRF vector and the 3D leg vector and ΔL is the resultant change in the leg vector.

1.1.3 Statistical Analysis

Paired samples t-tests were conducted between dominant and non-dominant limbs for all JPS and leg stiffness variables; descriptive statistics are reported for each method of leg stiffness and JPS during each test. Pearson correlations were calculated to determine relationships between angular error measures and each measure of leg stiffness on both the dominant and non-dominant limbs. Correlations were interpreted as small (0.1 to 0.3), medium (0.3 to 0.5), and large (0.5 to 1.0).[30] Cohen's d was calculated for between-limb effect sizes and interpreted as small (0.2), medium (0.5), and large (0.8).[31] Normality and equality of error variances were assessed with Shapiro-Wilk and Levene's Test's, respectively. Alpha level was set a priori at $p<0.05$ for all analyses. Power analysis was conducted a priori using G*Power 3.1.9.2; for paired t-test analyses, a sample size of 15 was required to obtain an effect size of 0.80 with power of 0.80 at alpha level of $p<0.05$; for bivariate correlation analyses, a sample size of 29 was required to obtain a correlation of 0.50 with power of 0.80 at alpha level of $p<0.05$. A sample size of 29 was reached; however, due to the amendment and addition of the study protocol to include JPS testing in a SLS test, an additional 29 subjects were recruited for subsequent analyses.

1.2 Results

Descriptive statistics and paired samples t-test results for JPS and leg stiffness between dominant and non-dominant limbs are reported in Table 1.1. Pearson correlations between measures of JPS and leg stiffness calculations are reported in Table 1.2. Joint position sense and leg stiffness were not statistically significant between limbs. A small effect was detected for AAE between limbs in the SLS test ($p=0.088$, $d=0.335$). No statistically significant correlations were found between measures of knee JPS and any method of leg stiffness calculation. The group was normally distributed for height, but not for age ($W=0.825$, $p<0.001$), body mass

(W=0.897, p<0.001), or BMI (W=0.903, p<0.001). Several variables violated the assumption of normality, which are reported in Table 1.3. Equal error variances were assumed for all variables.

1.3 Discussion

This study presents novel findings regarding the relationships between knee joint position sense and various measures of leg stiffness in a healthy, uninjured population. The current study also provides novel findings of leg stiffness for a variety of methods during stair descent. Surprisingly, no statistically significant correlations were found between leg stiffness and JPS in this analysis. Knee JPS averages were similar to previously reported data for the age range included in this study.[17] To our knowledge, leg stiffness has not been previously reported in this manner during the task of stair descent. The importance of the knee during similar tasks that require eccentric control has been demonstrated previously, especially in patients undergoing anterior cruciate ligament reconstruction (ACLR)[18, 32-34] and knee osteoarthritis (OA).[35-37] Furthermore, leg stiffness has been posited as a biomechanical expression of neuromuscular control,[4] which can be modulated to accommodate for the stiffness of the surface being traversed.[38, 39] The results of the current study suggest that knee JPS in a healthy population may not be adequate to thoroughly explain whole limb compression behavior during this task.

Bilateral sensorimotor coordination differences have recently been shown to be associated with limb dominance,[40] albeit observed with postural control rather than knee JPS. Weight shifting onto the non-dominant limb demonstrated detrimental decreases in postural control; therefore, lower JPS ability (greater angular error) at the knee was expected in the non-dominant limb in this analysis. In the current study, a similar task to weight shifting was the SLS test. Although no significant differences were observed, a small effect between limbs was detected, with the non-dominant limb demonstrating approximately 2° greater absolute angular error in this position. Given that CKC tests incorporate multiple joints and ground-foot interface, these other avenues of proprioceptive input inevitably influence whole limb neuromuscular control. This may be a result of the inclusion of a healthy population and examination of only the proprioceptive ability to reproduce a knee joint position. Additional evidence examining injury risk association with leg dominance is inconclusive, with both dominant and non-dominant limbs having demonstrated biomechanical deficiencies in various studies.[41] The results of the current study suggest that perceived limb dominance does not influence leg stiffness during the task of stair descent, nor does it influence JPS ability in any of the tested positions.

There are several possible explanations for the lack of statistically significant leg stiffness and JPS correlations: 1) the subjects are healthy adults with no recent reported history of injury. Studies examining JPS ability have focused exclusively on injured populations, particularly ACLR patients,[18, 34, 42] and knee OA patients.[16, 22, 37, 43-45] These studies highlight proprioceptive deficits at the knee as either a result of the injury or as a possible contributing factor to injury development, though the latter is speculative given that JPS ability was not determined prior to injury. The current sample was uninjured, with no known neurologic or musculoskeletal conditions, and it is reasonable to assume that healthy adults will not display any observable signs of proprioceptive differences between limbs that would contribute to decreases in leg compression ability. 2) knee JPS was measured in the sagittal plane with a reference angle of 30° of knee flexion and is a single joint within the lower extremity. The reference angle of 30° of knee flexion was selected because it is a commonly experienced degree of flexion during walking and stair descent activities,[29] although many knee OA patients are able to reach knee flexion angles beyond 90° during stair descent.[46] Still, those populations reaching that peak knee flexion angle must demonstrate the ability to move through that degree of knee excursion in order to reach it. Despite this commonly experienced degree of flexion, knee JPS around this angle may not be sufficient in explaining limb compression behavior as previously hypothesized. It is difficult to examine JPS ability of multiple joints simultaneously; however, the roles of the foot, ankle, and hip cannot be discredited regarding total limb compression. The degree to which each joint's proprioceptive ability correlates with whole limb compression should be investigated further. 3) Knee JPS was measured in the sagittal plane. Due to the larger total range of motion available at the knee in this plane, it is surprising that sagittal leg stiffness was not correlated with JPS ability. This again suggests that sagittal knee JPS may not be sufficient to explain total limb compression.

Several limitations should be acknowledged in the current analysis. Healthy subjects were recruited from a convenience sample and may not show deficits in proprioceptive ability. Half of the subjects completed single leg squat JPS testing, whereas all subjects completed the seated and double leg squat JPS testing. This lower sample included in the correlation analysis for those variables may explain the lack of statistical significance observed. A final limitation is the self-reporting of limb dominance. Physiological determinants of limb dominance may be necessary for a more accurate assessment of between-limb differences.

1.4 Conclusions

This study reports novel findings of both correlations between leg stiffness and knee JPS, as well as stair descent leg stiffness normative data in a healthy, uninjured population. No statistically significant differences were found in leg stiffness or knee JPS ability between limbs, nor were any statistically significant correlations found between leg stiffness and knee JPS ability in three JPS testing methods.

1.6 List of Tables

Table 1.1 Between Limb Differences for Angular Error and Leg Stiffness

	Dominant Limb			Non-Dominant Limb				
	Mean	SD	SEM	Mean	SD	SEM	p	d
3D K_{leg}	15.38	± 2.72	0.36	15.08	± 3.63	0.49	0.382	0.118
Uniplanar K_{leg}	30.58	± 11.86	1.57	32.50	± 10.79	1.43	0.154	0.192
K_{vert}	56.31	± 8.11	1.07	56.16	± 8.84	1.17	0.796	0.034
Seated AAE (°)	7.48	± 4.76	0.63	7.00	± 3.86	0.51	0.442	0.103
Seated RAE (°)	4.49	± 7.62	1.01	4.25	± 6.74	0.89	0.792	0.035
Squat AAE (°)	12.03	± 7.30	0.97	12.19	± 7.37	0.98	0.644	0.062
Squat RAE (°)	-10.99	± 8.78	1.16	-11.00	± 9.02	1.20	0.986	0.002
SLS AAE (°)	6.68	± 4.45	0.84	8.65	± 5.50	1.04	0.088	0.335
SLS RAE (°)	-3.36	± 7.31	1.38	-4.23	± 9.41	1.78	0.560	0.112

AAE=Absolue Angular Error; RAE=Relative Angular Error; SLS=Single Leg Squat;
d=Cohen's d effect size; SD= standard deviation; SEM=standard error of mean; p=p-value (α=0.05)

Table 1.2. Correlations Between Leg Stiffness and Angular Error

	3D K_{leg}				K_{vert}				Uniplanar K_{leg}			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p	r	p	r	p
Seated AAE (°)	0.062	0.648	-0.166	0.217	0.102	0.449	-0.063	0.640	0.061	0.651	0.200	0.136
Seated RAE (°)	-0.139	0.308	-0.008	0.954	-0.067	0.621	0.043	0.751	0.126	0.351	0.199	0.137
Standing AAE (°)	0.010	0.943	0.008	0.952	0.008	0.954	-0.081	0.548	-0.121	0.369	-0.036	0.790
Standing RAE (°)	-0.035	0.799	0.073	0.591	-0.034	0.799	0.135	0.318	0.019	0.886	-0.085	0.532
SLS AAE (°)	0.120	0.543	0.148	0.452	0.257	0.187	0.260	0.182	-0.143	0.468	0.094	0.634
SLS RAE (°)	-0.142	0.471	0.073	0.713	-0.144	0.465	-0.050	0.800	-0.172	0.382	-0.323	0.093

AAE=Absolue Angular Error; RAE=Relative Angular Error; SLS=Single Leg Squat; r=Pearson correlation coefficient; p=p-value (α=0.05)

Table 1.3 Bilateral Normality Statistics for Angular Error and Leg Stiffness

	Dominant Limb		Non-Dominant Limb	
	W	p	W	p
3D K_{leg}	0.976	0.338	0.981	0.523
Uniplanar K_{leg}	0.978	0.411	***0.956***	***0.041****
K_{vert}	***0.855***	***<0.001****	***0.816***	***<0.001****
Seated AAE (°)	***0.915***	***0.026****	0.937	0.095
Seated RAE (°)	0.986	0.963	0.974	0.704
Squat AAE (°)	0.951	0.204	0.960	0.357
Squat RAE (°)	0.966	0.471	0.962	0.395
SLS AAE (°)	***0.906***	***0.016****	0.935	0.082
SLS RAE (°)	0.985	0.955	0.955	0.263

AAE=Absolue Angular Error; RAE=Relative Angular Error;
SLS=Single Leg Squat; W=Shapiro-Wilk statistic; p=p-value (α=0.05)
* = Significant Shapiro-Wilk Test(p<0.05)

CHAPTER 2:

CROSS-SECTIONAL ANALYSIS OF KNEE BIOMECHANICS AND JOINT POSITION SENSE RELATIONSHIPS BETWEEN LIMBS OF HEALTHY ADULTS DURING STAIR NEGOTIATION

Abstract

Background: It is unknown if joint position sense (JPS) as a measure of knee joint proprioception is associated with biomechanical risk factors of knee osteoarthritis (OA). Biomechanical risk factors have been determined to predict and identify onset and progression of knee OA; however, the degree to which these risk factors are associated with knee joint proprioception in either a healthy or injured population has not been examined. The goal was to determine between-limb differences of JPS and stair negotiation biomechanics, and to determine correlations between JPS ability and stair negotiation biomechanics of the dominant and non-dominant limbs. **Methods:** Fifty-seven subjects with no history of injury or surgery within the last 6 months were included. Five trials of stair ascent and descent capturing data on each limb were measured via 3D motion capture. Movement trial data were processed using Visual3Dv4. This cross-sectional study determined correlations between biomechanical risk factors of knee OA and joint position sense (JPS) during stair negotiation in healthy individuals on both dominant and non-dominant limbs. Differences of biomechanical variables and JPS between-limbs were also assessed. Biomechanical variables included: Peak Knee Flexion Moment (KFM), Knee Adduction Moment (KAM), KFM and KAM Impulses, 1st Peak and Peak KFM Rate and KAM Rate, Loading Rate (LR), Peak Knee Varus and Flexion Angles, and Peak Varus Velocity. Descriptive statistics are reported as mean ± standard deviation for all variables. Paired-sample T-tests were conducted on all variables between the dominant and non-dominant limbs. Pearson's correlations were calculated to determine relationships between all three JPS methods and biomechanical variables on each limb. **Findings**: During stair ascent, the non-dominant limb demonstrated greater 1st peak KFM (p=0.008, d=0.364), greater KFM impulse (p=0.048, d=0.268), and greater peak knee extensor power (p<0.001, d=0.601). During stair descent, the non-dominant limb demonstrated greater peak KFM (p=0.003, d=0.405), greater 1st peak KFM (p=0.035, d=0.285), greater peak knee extensor power (p=0.004, d=0.393), and lower peak knee varus velocity (p<0.001, d=0.488). Knee JPS showed small-moderate correlations with measures of medial knee joint loading. **Interpretation:** In this sample of healthy, uninjured subjects, the non-dominant limb demonstrated greater measures of quadriceps performance than the dominant limb during stair negotiation. This challenges the notion that the dominant limb is superior in neuromuscular control; however, associations between knee loading biomechanics and JPS ability were small-moderate.

2. Introduction

Proprioception and joint kinesthesia relay vital information regarding motion and joint position sense (JPS) during both open-kinetic chain (OKC) and closed-kinetic chain (CKC) activities. Diminished JPS ability may indicate poor neuromuscular control, which influences injury development during OKC and CKC activities.[1] Sensory afferent pathways relay information to the central nervous system regarding joint position, weight-bearing tolerance, pain, and muscle length and tension via stimulation of mechanoreceptors within muscles, tendons, and joint tissues in order to elicit a motor response. Various methods for evaluating proprioception have been validated and are reliable, though the population being tested may influence the testing procedure.[2, 47-49] Younger populations are able to complete more functional, active tests and older populations may require more passive tests.[2] Assessment of JPS, when conducted in a single joint test such as knee extension, accounts for mechanoreceptors acting on and within the joint itself. Joint position sense tests in CKC methods incorporate multijoint mechanoreceptor activity, i.e. the foot, ankle, and hip. Due to increased afferent signal, this may reduce the JPS ability of the joint being tested and may demonstrate less proprioceptive acuity because of greater input from other joints.[50] While the assessment of a single joint may be benefical in understanding the proprioceptive ability of that joint, a more practical and functional application may be the use of CKC testing to mimic activities of daily living (ADLs).

Previous evidence suggests that JPS ability is diminished in knee osteoarthritis (OA) patients, and patients have demonstrated age-related proprioceptive declines.[45] While balance and fall risk may be indicators for proprioceptive decline,[1, 51, 52] they do not give insight into knee JPS influenced by articular mechanoreceptors and muscles surrounding the joint. The degree to which these proprioceptive deficits are either the cause or effect of aberrant movement patterns is unclear. In order to better understand the movement patterns of knee OA patients, several biomechanical factors have been identified as predictors or indicators of medial knee loading and quadriceps performance during functional activities such as stair negotiation.[46, 53, 54] Despite the conclusions made by previous researchers suggesting that neuromuscular control deficits contribute to these biomechanical deficiencies,[1] relationships between JPS and biomechanical risk factors of knee OA have not been established in either a healthy or pathologic population. Pain may convolute proprioceptive input and further diminish JPS ability with pathologic populations;[1, 37] therefore, it may be beneficial to first establish these relationships in a healthy, uninjured population.

In addition to pain convoluting proprioceptive input in pathologic populations, there is little evidence examining the effect of self-reported limb dominance on proprioceptive ability in either healthy or injured populations. Decreased postural control has been has previously associated with increased load bearing onto the non-dominant limb, despite similarities of knee strength between limbs;[40] however, postural control does not account for proprioceptive acuity of the knee itself. Furthermore, knee OA patients have demonstrated increased prevalence of OA development on the non-dominant limb,[55] though biomechanical factors associated with knee OA were not examined and a small sample size limits the interpretation of these findings. It is unknown if biomechanical risk factors of knee OA are associated with JPS ability or if limb dominance influence these relationships. Therefore, the purpose of this study was to examine relationships between knee JPS in OKC and CKC tests and biomechanical risk factors of knee OA in a healthy population during stair negotiation. The second purpose of this study was to determine differences in biomechanical variables and JPS ability between the dominant and non-dominant limbs. In this exploratory analysis, we hypothesize that knee JPS ability will be significantly correlated with biomechanical risk factors of knee OA, and that the non-dominant limb will demonstrate less proprioceptive acuity and less neuromuscular control when assessed with biomechanical variables.

2.1 Methods

2.1.1 Subjects

Fifty-seven healthy volunteers (28 male, 29 female; age: 22.26 ± 3.23 years; mass: 71.21 ± 16.63 kg; height: 1.69 ± 0.09 m; BMI: 24.64 ± 4.21) were recruited from the community. Fifty-eight subjects were recruited; however, one participant was removed from the analysis due to inadequate left limb thigh and shank segment resolution. Subjects were screened using a standard, Office of Human Studies approved, health-history questionnaire. Subjects were recruited via convenience sampling in University classroom settings and were excluded if pregnant, had a known neurologic condition, had a previous history of a hip, knee, or ankle ligament reconstruction, or had a history of recent lower extremity musculoskeletal injury (within 6 months) that hindered the ability to extend the knee, squat, or negotiate stairs. Subjects were asked to self-report limb dominance. Prior to enrollment in the study, all patients signed informed consent and the study was approved by the Institution's Committee on Human Studies.

2.1.2 Procedures

All biomechanical analyses were conducted in the University of Hawaii Human Performance and Biomechanics Laboratory. Motion capture data was collected using 29 retroreflective markers placed on the thorax, pelvis, and lower extremities and four marker-based arrays placed on the thigh and shank segments (Appendix A). For stair ascent and descent motion capture, methods were similar to Vallabhajosula et al.[26] Five successful trails were collected, indicated by successful foot strike without the use of the handrail for assistance. All kinetic data were smoothed using a Butterworth filter with a 10 Hz cutoff frequency and processed using Visual 3D (C-Motion, Inc., Germantown, MD). All kinematic and kinetic data were smoothed using a Butterworth filter with a 10 Hz cut-off and ground reaction force was filtered using a 50 Hz cut-off frequency[27]. External joint moments were calculated using inverse dynamics based on marker trajectories and kinetic data which was also filtered using a 10 Hz cut-off frequency.[56] Kinematic data using the array-based marker-set were collected with a Vicon motion capture system and Vicon Nexus software (Vicon, Inc., Centennial, CO) at 240 Hz and time synchronized with kinetic data collected at 960 Hz collected from on a force plate (Advanced Mechanical Technology Incorporated, Boston, MA) embedded flush with the second step of a three-step staircase.

Joint position sense was tested in three positions: open-kinetic chain (OKC) knee extension (seated), closed-kinetic chain (CKC) double-leg squat (standing), and closed-kinetic chain single-leg squat (SLS). Ten repetitions[21] to a demonstrated reference angle (30°)[28] from the resting position (90°) were completed during each test. The reference angle was measured using a goniometer by a single, board-certified Athletic Trainer experienced in goniometric measurement. In the OKC test, subjects were shown the reference angle by the examiner passively extending the knee from the resting position to the reference angle. In both CKC tests, the subject performed a squat and was instructed to maintain the reference angle indicated by the examiner. In all three conditions, the subject was not explicitly given the reference angle, but instructed to "familiarize themselves" with the produced angle for 10 seconds. Subsequent trials were completed without visual aid. The order in which subjects completed each test, as well as the order in which each limb was tested, was randomized. Randomization was chosen to eliminate testing bias for dominant and non-dominant limbs, as well as to eliminate the bias of a potential learning effect that may occur. Ten repetitions were completed for each limb during each test.[21] Only 10 double-leg squat repetitions were completed for the CKC test to the demonstrated reference angle from the standing position due to redundancy of simultaneous knee flexion actions. For each trial, subjects were instructed to reproduce the demonstrated angle and maintain the position for three seconds.

Angular error of knee angles during JPS testing was calculated using: Absolute Angular Error (AAE) and Relative Angular Error (RAE).[28] The AAE measures JPS without considering the direction of error (flexion/extension both positive), whereas RAE is directional (flexed past 30° is negative and extended is positive). The average of 10 trials was determined to assign an error to each limb. Greater error would, in theory, indicate less proprioceptive ability, i.e., a greater inability to reproduce a specific joint position. Mathematical calculations are presented below:

1) *Absolute Angular Error*

$$\text{AAE}= [|(\text{target position} - \text{trial } 1)| + |(\text{target position} - \text{trial } 2)| + |(\text{target position} - \text{trial } 3) \ldots|]/10$$

2) *Relative Angular Error*

$$\text{RAE}= [(\text{target position} - \text{trial } 1) + (\text{target position} - \text{trial } 2) + (\text{target position} - \text{trial } 3) \ldots]/10$$

2.1.3 Statistical Analysis

Pearson correlations were calculated to determine relationships between angular error measures (AAE and RAE) and biomechanical variables on both the dominant and non-dominant limbs. Paired-samples t-tests were conducted to determine the between-limb differences of knee JPS and biomechanical variables between the dominant and non-dominant limbs. Pearson correlation coefficients were interpreted as small (0.1 to 0.3), medium (0.3 to 0.5), and large (0.5 to 1.0).[30] Cohen's d was calculated for between-limb effect sizes and interpreted as small (0.2), medium (0.5), and large (0.8).[31] Normality and equality of error variances were assessed with Shapiro-Wilk and Levene's Tests, respectively. Alpha level was set a priori at $p<0.05$ for all analyses. Power analysis was conducted a priori using G*Power 3.1.9.2; for paired t-test analyses, a sample size of 15 was required to obtain an effect size of 0.80 with power of 0.80 at alpha level of $p<0.05$; for bivariate correlation analyses, a sample size of 29 was required to obtain a correlation of 0.50 with power of 0.80 at alpha level of $p<0.05$. A sample size of 29 was reached; however, due to the amendment and addition of the study protocol to include JPS testing in a SLS test, an additional 29 subjects were recruited for subsequent analyses.

2.2 Results

The group was normally distributed for height, but not for age (W=0.825, $p<0.001$), body mass (W=0.897, $p<0.001$), or BMI (W=0.903, $p<0.001$). Several variables violated the assumption of normality and are reported in Table 2.6 (Ascent) and Table 2.7 (Descent). Equality of error variances were assumed for all variables. Between-limb differences of knee JPS during each test are reported in Table 2.1. No significant differences of knee JPS were detected between limbs. A small effect was detected for AAE in the SLS test (p=0.088, d=0.335). Descriptive statistics and between-limb differences of biomechanical variables during stair ascent and descent are reported in Table 2.2 and Table 2.3, respectively. During stair ascent, the non-dominant limb demonstrated greater 1st peak KFM (p=0.008, d=0.364), greater KFM impulse (p=0.048, d=0.268), and greater peak knee extensor power ($p<0.001$, d=0.601). During stair descent, the non-dominant limb demonstrated greater peak KFM (p=0.003, d=0.405), greater 1st peak KFM (p=0.035, d=0.285), greater peak knee extensor power (p=0.004, d=0.393), and lower peak knee varus velocity ($p<0.001$, d=0.488).

Complete correlation data is reported in Table 2.4 (Ascent) and Table 2.5 (Descent). On the non-dominant limb during stair ascent, seated AAE was correlated with peak KAM Rate (r=-0.313, p=0.018), 1st peak KAM (r=-0.304, p=0.022), 1st peak KFM Rate (r=-0.285, p=0.032) and KFM Impulse (r=0.305, p=0.021); seated RAE was correlated with KAM Impulse (r=-0.268, p=0.044). On the dominant limb during stair ascent, standing AAE was correlated with 1st peak KAM Rate (r=-0.260, p=0.050), time to peak KFM (r=0.298, p=0.024), and time to 1st peak KFM (r=0.298, p=0.024); standing RAE was correlated with peak KAM Rate (r=0.285, p=0.032) and 1st peak KAM Rate (r=0.287, p=0.031); SLS AAE was correlated with peak varus angle (r=0.406, p=0.032).

On the dominant limb during stair descent, seated AAE was correlated with peak knee flexion angle (r=-0.361, p=0.006); seated RAE was correlated with peak KFM (r=-0.348, p=0.008) and KFM Impulse (r=-0.276, p=0.037). On the non-dominant limb, seated AAE was with time to peak KFM (r=-0.281, p=0.034) and time to 1st peak KFM (r=-0.324, p=0.014) On the dominant limb, standing RAE was correlated with KAM Impulse (r=-0.285, p=0.032). No other variables during stair descent demonstrated significant correlations with knee JPS.

2.3 Discussion

Several findings are important to note from this analysis. The first goal of this study was to evaluate differences between limbs for knee JPS ability and stair negotiation biomechanics. As expected, no differences were observed between limbs for knee JPS, given that this sample was healthy with no underlying musculoskeletal injuries or conditions, although a small effect was observed between limbs for the SLS test for absolute error. Biomechanical variables, however, demonstrated several between-limb differences during stair ascent and descent. During stair ascent the non-dominant limb demonstrated greater KFM rate at the first peak during loading, as well as greater KFM impulse throughout the period of stance. These differences were accompanied by greater peak concentric knee extensor power (Figure 2.6). This may suggest greater capacity for power generation, given the large effect observed. The peak KFM itself was not different between limbs; however, the rate of moment development was greater in the non-dominant limb, which may suggest early loading varied between limbs (Figure 2.4).

These findings were consistent during stair descent; however, peak KFM was greater in the non-dominant limb in addition to the 1st peak KFM (Figure 2.10). As with stair ascent, the non-dominant limb demonstrated greater peak knee extensor power (eccentric) during stair descent (Figure 2.12), as well as lower peak varus velocity (Figure 2.9). Subjects demonstrated greater quadriceps control on the non-dominant limb, which may serve as a mediator for lower varus velocity throughout stance. Bennell et al[57, 58] suggests that quadriceps function serves to mediate dynamic medial knee joint loads, albeit observed in knee OA patients. These findings during stair negotiation are not intuitive given the preconceived notion that the dominant limb demonstrates greater neuromuscular control and strength of the knee extensors. Kline et al[59] suggests that greater rate of torque development is desirable, given that lower rates significantly contribute to quadriceps avoidance gait patterns. When considering the faster rate of moment development in the non-dominant limb in the current study, this may suggest that greater quadriceps activity occurred in the non-dominant rather than the dominant limb; however, correlations with absolute error in the OKC knee extension test may suggest that the moment rate differences were attributed to the time component. Despite these small-moderate correlations, no significant between-limb differences were observed for the time components of these variables.

Within the many examinations of correlations in the current study, absolute and relative angular error demonstrated several small-moderate correlations with biomechanical variables during both stair ascent and descent. During stair ascent, greater AAE of the non-dominant limb was associated with greater sagittal plane moment exposure (KFM Impulse) but less medial loading (KAM rate and 1st peak KAM). The non-dominant limb also showed a small negative correlation between total frontal plane moment exposure and seated RAE. These findings may suggest that as subjects demonstrated greater error into knee extension, which equates to a positive relative angular error, total medial loading decreased during stair ascent. These correlations were accompanied by greater quadriceps control measured via sagittal plane biomechanics (Table 2.2). This supports the notion previously described by Chang et al[60] and Chehab et al[61] that greater quadriceps control may mediate medial knee loading that contributes to knee OA development; however, the current sample denied any history of injury. Fewer correlations between JPS ability and biomechanics were observed when examining the double-leg squat JPS test. The dominant limb standing RAE was positively correlated with peak KAM rate and 1st peak KAM rate; while AAE was negatively correlated with 1st peak KAM rate and positively correlated with the 1st peak and the peak KFM times. The observed correlations were small, though they may suggest that those subjects that remained more extended during the squatting JPS demonstrated faster medial loading on the dominant limb during stair ascent. Small associations with absolute error and the time components may indicate that faster time to a peak KFM may accompany faster medial loading. Although the magnitude of these associations is small, these findings may suggest that diminished JPS ability may lead to faster moment development in the frontal plane, which has been previously associated with greater frequency of poor MRI scores and reduced medial tibiofemoral cartilage thickness.[62]

During stair descent, several correlations were also noted between biomechanical variables and JPS ability. In the dominant limb, seated RAE demonstrated negative correlations with total sagittal plane moment exposure and peak KFM. This may suggest that as subjects had greater error in the positive direction (further into extension), less total KFM and a lower peak KFM occurred during stance. This is supported by the dominant limb's negative correlation between overall error and peak knee flexion as well (reference AAE seated correlation with peak knee flexion dominant limb here). Translating this to a functional task such as stair descent, this would indicate that diminished proprioceptive ability was associated with less knee extensor muscle activation measured by external KFM and less sagittal plane excursion. These associations were not observed with the standing JPS test; however, the dominant limb demonstrated a weak negative correlation between standing RAE and KAM impulse. This may suggest that greater extension during a squat test is associated with less total exposure to external KAM.

Several factors may be considered as limitations in the current study. The lower magnitude and decreased frequency of associations between biomechanics and knee JPS ability measured by the CKC tests (squat and SLS) may be explained by the nature of CKC activity. Including the hip, foot, and ankle in the test adds more proprioceptive input, which may increase the error experienced at the knee. Additional proprioceptive input from other joints, muscle tension throughout the kinetic chain, input from joint mechanoreceptors, and input from the skin will have an effect of the JPS ability of one or more joints. While isolation of the knee may provide information regarding the proprioceptive ability of that joint, few functional activities involve isolated knee motion. Another consideration is the inclusion of only healthy volunteers. Although biomechanics were similar to previously reported normative data,[63, 64] it may be unlikely that obvious proprioceptive differences would be observed between the dominant and non-dominant limbs in this population. Despite this possibility, side-to-side correlational differences were still observed, although small-moderate in magnitude.

2.4 Conclusions

Healthy, uninjured subjects' non-dominant limb demonstrated greater quadriceps function via increased external KFM, increased knee power, and lower varus velocity during stair negotiation compared to the dominant limb. These differences occurred despite similarities in knee JPS in three different tests. Weak, negative associations between measures of JPS ability and stair negotiation biomechanics indicate weak relationships between knee joint proprioception and biomechanical loading characteristics, which may be masked by volunteers being healthy and uninjured. Similarities in knee JPS ability indicate that proprioceptive input from mechanoreceptors elsewhere in the kinetic chain may contribute to overall limb function during stair negotiation.

2.5 List of Tables

Table 2.1 Between Limb Differences for Angular Error

	Dominant Limb			Non-Dominant Limb				
	Mean	SD	SEM	Mean	SD	SEM	p	d
Seated AAE (°)	7.48	± 4.76	0.63	7.00	± 3.86	0.51	0.442	0.103
Seated RAE (°)	4.49	± 7.62	1.01	4.25	± 6.74	0.89	0.792	0.035
Squat AAE (°)	12.03	± 7.30	0.97	12.19	± 7.37	0.98	0.644	0.062
Squat RAE (°)	-10.99	± 8.78	1.16	-11.00	± 9.02	1.20	0.986	0.002
SLS AAE (°)	6.68	± 4.45	0.84	8.65	± 5.50	1.04	0.088	0.335
SLS RAE (°)	-3.36	± 7.31	1.38	-4.23	± 9.41	1.78	0.560	0.112

AAE=Absolue Angular Error; RAE=Relative Angular Error; SLS=Single Leg Squat;
d=Cohen's d effect size; SD= standard deviation; SEM=standard error of mean; p=p-value (α=0.05)

Table 2.2. Between-limb Differences of Stair Ascent Biomechanics

	Dominant Limb			Non-Dominant Limb				
	Mean	SD	SEM	Mean	SD	SEM	p	d
Peak KAM (N·m/kg)	0.31	± 0.13	0.02	0.35	± 0.19	0.02	0.147	0.195
Time to Peak KAM (s)	0.40	± 0.21	0.03	0.42	± 0.21	0.03	0.556	0.079
Peak KAM Rate (N·m/kg/s)	0.88	± 0.49	0.07	1.05	± 0.69	0.09	0.126	0.206
1st Peak KAM (N·m/kg)	0.28	± 0.11	0.02	0.31	± 0.13	0.02	0.170	0.184
Time to 1st Peak KAM (s)	0.29	± 0.09	0.01	0.29	± 0.07	0.01	0.836	0.028
1st Peak KAM Rate (N·m/kg/s)	1.04	± 0.53	0.07	1.20	± 0.60	0.08	0.117	0.211
KAM Impulse (∫N·m/kg/s)	0.12	± 0.09	0.01	0.14	± 0.08	0.01	0.212	0.167
Peak KFM (N·m/kg)	1.12	± 0.22	0.03	1.06	± 0.56	0.07	0.439	0.103
Time to peak KFM (s)	0.22	± 0.04	0.01	0.22	± 0.04	0.00	0.798	0.034
Peak KFM Rate (N·m/kg/s)	5.30	± 1.32	0.17	4.91	± 2.84	0.38	0.339	0.128
1st Peak KFM (N·m/kg)	***1.12***	***± 0.22***	***0.03***	***1.18***	***± 0.20***	***0.03***	***0.008****	***0.364***
Time to 1st Peak KFM (s)	0.22	± 0.04	0.01	0.21	± 0.04	0.00	0.442	0.102
1st Peak KFM Rate (N·m/kg/s)	5.37	± 1.32	0.18	5.68	± 1.04	0.14	0.052	0.263
KFM Impulse (∫N·m/kg/s)	***0.21***	***± 0.13***	***0.02***	***0.23***	***± 0.10***	***0.01***	***0.048****	***0.268***
Max vGRF (N)	821.10	± 195.96	25.96	816.91	± 179.09	23.72	0.620	0.066
Time to Max vGRF (s)	0.23	± 0.06	0.01	0.22	± 0.04	0.01	0.520	0.086
Loading Rate (N/s)	4742.04	± 2911.84	385.68	4701.42	± 2056.75	272.42	0.924	0.013
Peak Knee Extensor Power (W/kg)	***2.61***	***± 0.69***	***0.09***	***2.93***	***± 0.58***	***0.08***	***<0.001****	***0.601***
Peak Varus Angle (°)	7.65	± 5.24	0.69	7.81	± 4.58	0.61	0.819	0.031
Peak Varus Velocity (°/s)	48.53	± 20.28	2.69	53.05	± 37.39	4.95	0.365	0.121
Peak Knee Flex Angle (°)	70.91	± 6.56	0.87	72.16	± 4.75	0.63	0.139	0.199

N·m=Newton-meter; vGRF=vertical ground reaction force; KAM=knee adduction moment; KFM=knee flexion moment
kg=kilograms; s=seconds; ∫=integral; SD=standard deviation; p=p-value (α=0.05); d=Cohen's d effect size; *=significant at p<0.05

Table 2.3. Between-limb Differences of Stair Descent Biomechanics

	Dominant Limb			Non-Dominant Limb				
	Mean	SD	SEM	Mean	SD	SEM	p	d
Peak KAM (N·m/kg)	0.41	± 0.19	0.02	0.36	± 0.18	0.02	0.135	0.201
Time to Peak KAM (s)	0.35	± 0.17	0.02	0.31	± 0.17	0.02	0.093	0.227
Peak KAM Rate (N·m/kg/s)	1.62	± 1.24	0.16	1.52	± 1.16	0.15	0.639	0.062
1st Peak KAM (N·m/kg)	0.39	± 0.19	0.03	0.34	± 0.18	0.02	0.166	0.186
Time to 1st Peak KAM (s)	0.20	± 0.06	0.01	0.19	± 0.05	0.01	0.086	0.234
1st Peak KAM Rate (N·m/kg/s)	2.10	± 1.17	0.16	1.81	± 1.13	0.15	0.154	0.193
KAM Impulse (∫N·m/kg/s)	0.15	± 0.10	0.01	0.12	± 0.08	0.01	0.120	0.209
Peak KFM (N·m/kg)	***1.44***	***± 0.29***	***0.04***	***1.52***	***± 0.30***	***0.04***	***0.003****	***0.405***
Time to peak KFM (s)	0.58	± 0.19	0.03	0.56	± 0.20	0.03	0.202	0.171
Peak KFM Rate (N·m/kg/s)	2.92	± 1.74	0.23	3.27	± 2.17	0.29	0.105	0.218
1st Peak KFM (N·m/kg)	***1.03***	***± 0.33***	***0.04***	***1.10***	***± 0.35***	***0.05***	***0.035****	***0.285***
Time to 1st Peak KFM (s)	0.22	± 0.07	0.01	0.22	± 0.07	0.01	0.901	0.017
1st Peak KFM Rate (N·m/kg/s)	5.01	± 2.24	0.30	5.37	± 2.32	0.31	0.074	0.242
KFM Impulse (∫N·m/kg/s)	0.59	± 0.17	0.02	0.61	± 0.15	0.02	0.182	0.179
Max vGRF (N)	933.08	± 223.23	29.57	926.42	± 224.77	29.77	0.470	0.096
Time to Max vGRF (s)	0.14	± 0.02	0.00	0.15	± 0.03	0.00	0.226	0.162
Loading Rate (N/s)	6657.48	± 1941.69	257.18	6397.39	± 1709.68	226.45	0.184	0.178
Peak Knee Extensor Power (W/kg)	***-4.54***	***± 0.85***	***0.11***	***-4.79***	***± 0.82***	***0.11***	***0.004****	***0.393***
Peak Varus Angle (°)	3.86	± 4.02	0.53	2.83	± 3.78	0.50	0.122	0.208
Peak Varus Velocity (°/s)	***39.45***	***± 27.81***	***3.72***	***26.06***	***± 16.29***	***2.18***	***<0.001****	***0.488***
Peak Knee Flex Angle (°)	32.35	± 6.92	0.92	32.42	± 6.22	0.82	0.933	0.011

N·m=Newton-meter; vGRF=vertical ground reaction force; KAM=knee adduction moment; KFM=knee flexion moment kg=kilograms; s=seconds; ∫=integral; SD=standard deviation; p=p-value (α=0.05); d=Cohen's d effect size; *=significant at p<0.05

Table 2.4. Correlations Between Angular Error and Stair Ascent Biomechanics in Dominant and Non-dominant Limbs

	Seated AAE				Seated RAE			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p
Peak KAM (N·m/kg)	-0.100	0.458	-0.199	0.139	-0.140	0.298	-0.140	0.298
Time to Peak KAM (s)	0.035	0.793	0.108	0.424	0.102	0.448	-0.179	0.183
Peak KAM Rate (N·m/kg/s)	-0.034	0.801	**_-0.313*_**	**_0.018*_**	-0.079	0.561	-0.101	0.453
1st Peak KAM (N·m/kg)	-0.167	0.214	**_-0.304*_**	**_0.022*_**	-0.211	0.115	-0.200	0.137
Time to 1st Peak KAM (s)	0.160	0.234	-0.024	0.861	0.167	0.215	-0.076	0.573
1st Peak KAM Rate (N·m/kg/s)	-0.150	0.264	0.148	0.271	-0.126	0.349	0.143	0.289
KAM Impulse (∫N·m/kg/s)	-0.221	0.099	-0.214	0.110	-0.163	0.225	**_-0.268*_**	**_0.044*_**
Peak KFM (N·m/kg)	0.046	0.735	0.054	0.691	-0.137	0.308	-0.057	0.674
Time to Peak KFM (s)	-0.053	0.698	-0.137	0.310	-0.062	0.647	-0.160	0.233
Peak KFM Rate (N·m/kg/s)	0.088	0.514	0.105	0.438	-0.025	0.855	0.005	0.973
1st Peak KFM (N·m/kg)	0.046	0.735	0.040	0.768	-0.137	0.308	-0.042	0.756
Time to 1st Peak KFM (s)	-0.053	0.698	-0.146	0.279	-0.062	0.647	-0.194	0.149
1st Peak KFM Rate (N·m/kg/s)	0.086	0.523	**_-0.285*_**	**_0.032*_**	-0.032	0.811	-0.132	0.330
KFM Impulse (∫N·m/kg/s)	-0.125	0.354	**_0.305*_**	**_0.021*_**	-0.045	0.740	0.125	0.355
Max vGRF (N)	0.002	0.986	0.077	0.570	-0.067	0.619	0.119	0.377
Time to Max vGRF (s)	-0.113	0.404	-0.019	0.889	0.036	0.791	0.045	0.741
Loading Rate (N/s)	0.019	0.890	-0.079	0.558	-0.198	0.141	-0.194	0.147
Peak Knee Extensor Power (W/kg)	0.119	0.378	0.073	0.589	0.053	0.693	0.023	0.864
Peak Varus Velocity (°/s)	0.101	0.456	0.021	0.878	0.124	0.359	-0.065	0.632
Peak Varus Angle (°)	-0.216	0.106	-0.181	0.179	-0.178	0.186	-0.015	0.911
Peak Knee Flexion Angle (°)	-0.055	0.683	-0.069	0.612	0.039	0.773	-0.032	0.812

	Standing AAE				Standing RAE			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p
Peak KAM (N·m/kg)	-0.166	0.217	0.027	0.841	0.169	0.210	0.152	0.260
Time to Peak KAM (s)	0.085	0.530	0.061	0.655	-0.130	0.335	-0.044	0.743
Peak KAM Rate (N·m/kg/s)	-0.233	0.082	-0.008	0.950	**_0.285*_**	**_0.032*_**	0.100	0.457
1st Peak KAM (N·m/kg)	-0.165	0.221	0.106	0.432	0.193	0.150	-0.122	0.367
Time to 1st Peak KAM (s)	0.211	0.116	0.032	0.812	-0.232	0.082	-0.078	0.564
1st Peak KAM Rate (N·m/kg/s)	**_-0.260*_**	**_0.050*_**	0.040	0.767	**_0.287*_**	**_0.031*_**	-0.045	0.740
KAM Impulse (∫N·m/kg/s)	-0.053	0.698	0.192	0.153	0.048	0.723	-0.213	0.111
Peak KFM (N·m/kg)	0.022	0.870	0.133	0.323	-0.074	0.585	-0.178	0.185
Time to Peak KFM (s)	**_0.298*_**	**_0.024*_**	0.075	0.580	-0.234	0.080	0.000	0.999
Peak KFM Rate (N·m/kg/s)	-0.192	0.153	0.095	0.480	0.108	0.424	-0.151	0.263
1st Peak KFM (N·m/kg)	0.022	0.870	0.157	0.244	-0.074	0.585	-0.157	0.244
Time to 1st Peak KFM (s)	**_0.298*_**	**_0.024*_**	0.098	0.470	-0.234	0.080	-0.144	0.285
1st Peak KFM Rate (N·m/kg/s)	-0.187	0.164	0.061	0.653	0.100	0.460	-0.021	0.877
KFM Impulse (∫N·m/kg/s)	0.193	0.151	0.229	0.086	-0.206	0.125	-0.197	0.142
Max vGRF (N)	-0.109	0.422	-0.022	0.873	0.058	0.668	-0.008	0.952
Time to Max vGRF (s)	0.113	0.404	-0.142	0.293	-0.103	0.444	0.086	0.526
Loading Rate (N/s)	-0.018	0.893	0.184	0.170	0.028	0.838	-0.186	0.166
Peak Knee Extensor Power (W/kg)	0.008	0.956	0.134	0.319	-0.064	0.638	-0.110	0.417
Peak Varus Velocity (°/s)	0.119	0.379	-0.088	0.514	-0.105	0.439	0.078	0.566
Peak Varus Angle (°)	0.020	0.882	-0.093	0.492	0.039	0.776	0.034	0.801
Peak Knee Flexion Angle (°)	0.041	0.760	0.085	0.530	-0.056	0.679	-0.087	0.522

	SLS AAE				SLS RAE			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p
Peak KAM (N·m/kg)	0.202	0.302	0.014	0.945	0.080	0.684	0.191	0.331
Time to Peak KAM (s)	0.027	0.891	0.028	0.889	-0.123	0.534	0.058	0.771
Peak KAM Rate (N·m/kg/s)	0.150	0.445	-0.009	0.964	0.146	0.460	0.035	0.859
1st Peak KAM (N·m/kg)	0.248	0.203	-0.036	0.855	0.108	0.585	-0.153	0.437
Time to 1st Peak KAM (s)	-0.207	0.292	0.103	0.601	0.077	0.696	-0.074	0.709
1st Peak KAM Rate (N·m/kg/s)	0.312	0.106	-0.087	0.658	0.004	0.985	-0.119	0.545
KAM Impulse (∫N·m/kg/s)	0.182	0.353	-0.004	0.983	0.169	0.389	-0.206	0.294
Peak KFM (N·m/kg)	-0.145	0.462	0.222	0.257	-0.065	0.743	-0.165	0.401
Time to Peak KFM (s)	-0.113	0.566	0.155	0.432	0.185	0.345	0.165	0.403
Peak KFM Rate (N·m/kg/s)	-0.056	0.777	0.187	0.340	-0.152	0.439	-0.174	0.375
1st Peak KFM (N·m/kg)	-0.145	0.462	0.220	0.260	-0.065	0.743	-0.096	0.626
Time to 1st Peak KFM (s)	-0.113	0.566	0.103	0.601	0.185	0.345	-0.113	0.569
1st Peak KFM Rate (N·m/kg/s)	-0.037	0.850	0.134	0.496	-0.154	0.434	-0.004	0.983
KFM Impulse (∫N·m/kg/s)	-0.164	0.405	0.147	0.455	-0.099	0.616	0.080	0.686
Max vGRF (N)	0.221	0.259	0.189	0.334	-0.326	0.091	-0.146	0.458
Time to Max vGRF (s)	0.002	0.992	-0.008	0.968	0.152	0.439	0.053	0.789
Loading Rate (N/s)	0.123	0.534	0.064	0.745	-0.224	0.252	-0.166	0.400
Peak Knee Extensor Power (W/kg)	-0.132	0.503	0.102	0.604	-0.065	0.743	0.067	0.733
Peak Varus Velocity (°/s)	-0.166	0.398	0.027	0.894	-0.033	0.868	-0.078	0.693
Peak Varus Angle (°)	**_0.406*_**	**_0.032*_**	0.069	0.728	-0.006	0.975	-0.226	0.247
Peak Knee Flexion Angle (°)	-0.083	0.675	-0.140	0.478	0.168	0.394	0.039	0.845

N·m=Newton-meter; vGRF=vertical ground reaction force; KAM=knee adduction moment; KFM=knee flexion moment
kg=kilograms; s=seconds; ∫=integral; p=p-value (α=0.05); *=significant at p<0.05

Table 2.5. Correlations Between Angular Error and Stair Descent Biomechanics in Dominant and Non-dominant Limbs

	Seated AAE				Seated RAE			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p
Peak KAM (N·m/kg)	-0.082	0.543	-0.119	0.376	-0.171	0.204	0.077	0.570
Time to Peak KAM (s)	-0.060	0.660	-0.073	0.592	0.027	0.842	0.043	0.753
Peak KAM Rate (N·m/kg/s)	-0.056	0.678	-0.048	0.723	-0.089	0.510	0.042	0.759
1st Peak KAM (N·m/kg)	-0.043	0.753	-0.106	0.432	-0.148	0.272	0.070	0.604
Time to 1st Peak KAM (s)	0.028	0.836	-0.203	0.129	-0.109	0.425	-0.091	0.500
1st Peak KAM Rate (N·m/kg/s)	-0.015	0.913	-0.011	0.933	-0.044	0.747	0.113	0.404
KAM Impulse (∫N·m/kg/s)	-0.061	0.655	-0.163	0.226	-0.090	0.506	0.023	0.864
Peak KFM (N·m/kg)	-0.114	0.398	-0.135	0.316	***-0.348****	***0.008****	-0.213	0.112
Time to Peak KFM (s)	0.028	0.835	***-0.281****	***0.034****	-0.052	0.700	-0.178	0.185
Peak KFM Rate (N·m/kg/s)	-0.072	0.597	0.124	0.356	-0.170	0.206	0.032	0.814
1st Peak KFM (N·m/kg)	-0.166	0.218	0.149	0.270	-0.215	0.109	-0.017	0.898
Time to 1st Peak KFM (s)	-0.188	0.162	***-0.324****	***0.014****	-0.164	0.222	-0.117	0.385
1st Peak KFM Rate (N·m/kg/s)	-0.024	0.859	0.248	0.063	-0.115	0.393	0.050	0.713
KFM Impulse (∫N·m/kg/s)	-0.242	0.070	-0.130	0.337	***-0.276****	***0.037****	-0.247	0.064
Max vGRF (N)	0.081	0.548	0.092	0.496	-0.038	0.777	0.126	0.349
Time to Max vGRF (s)	-0.104	0.443	-0.016	0.908	-0.047	0.728	0.103	0.446
Loading Rate (N/s)	0.084	0.535	0.083	0.539	-0.028	0.837	0.054	0.691
Peak Knee Extensor Power (W/kg)	-0.127	0.348	0.119	0.377	0.260	0.051	0.250	0.060
Peak Varus Velocity (°/s)	-0.034	0.806	-0.023	0.867	-0.143	0.295	0.044	0.743
Peak Varus Angle (°)	-0.212	0.113	-0.025	0.854	-0.106	0.433	-0.024	0.862
Peak Knee Flexion Angle (°)	***-0.361****	***0.006****	0.069	0.612	-0.100	0.457	0.036	0.791

	Standing AAE				Standing RAE			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p
Peak KAM (N·m/kg)	0.131	0.330	-0.128	0.342	-0.150	0.266	0.119	0.380
Time to Peak KAM (s)	0.081	0.548	0.026	0.850	-0.041	0.761	-0.058	0.668
Peak KAM Rate (N·m/kg/s)	0.035	0.796	-0.090	0.507	-0.069	0.611	0.086	0.523
1st Peak KAM (N·m/kg)	0.129	0.340	-0.098	0.470	-0.142	0.292	0.085	0.530
Time to 1st Peak KAM (s)	0.184	0.174	0.180	0.181	-0.148	0.276	-0.200	0.135
1st Peak KAM Rate (N·m/kg/s)	0.054	0.690	-0.135	0.317	-0.085	0.531	0.132	0.327
KAM Impulse (∫N·m/kg/s)	0.255	0.056	-0.113	0.402	***-0.285****	***0.032****	0.109	0.419
Peak KFM (N·m/kg)	-0.061	0.654	-0.093	0.491	-0.016	0.907	0.010	0.939
Time to Peak KFM (s)	0.128	0.344	0.121	0.371	-0.068	0.613	-0.116	0.391
Peak KFM Rate (N·m/kg/s)	-0.090	0.508	-0.147	0.277	0.015	0.913	0.113	0.403
1st Peak KFM (N·m/kg)	0.068	0.616	0.067	0.619	-0.155	0.251	-0.071	0.600
Time to 1st Peak KFM (s)	-0.133	0.324	-0.108	0.424	0.182	0.176	0.041	0.762
1st Peak KFM Rate (N·m/kg/s)	0.095	0.483	0.068	0.614	-0.182	0.174	-0.042	0.757
KFM Impulse (∫N·m/kg/s)	0.201	0.134	0.123	0.362	-0.258	0.053	-0.189	0.160
Max vGRF (N)	-0.018	0.897	0.061	0.651	-0.009	0.946	-0.075	0.581
Time to Max vGRF (s)	0.101	0.455	0.152	0.259	-0.044	0.743	-0.173	0.198
Loading Rate (N/s)	-0.054	0.687	-0.043	0.749	0.005	0.972	0.046	0.735
Peak Knee Extensor Power (W/kg)	0.134	0.321	0.079	0.559	-0.109	0.422	0.023	0.867
Peak Varus Velocity (°/s)	-0.128	0.347	-0.022	0.869	0.053	0.698	-0.027	0.842
Peak Varus Angle (°)	-0.012	0.931	-0.042	0.754	0.015	0.911	-0.032	0.813
Peak Knee Flexion Angle (°)	-0.047	0.728	0.011	0.934	-0.051	0.704	-0.020	0.882

	SLS AAE				SLS RAE			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p
Peak KAM (N·m/kg)	0.203	0.301	0.080	0.685	-0.176	0.370	-0.021	0.917
Time to Peak KAM (s)	-0.084	0.671	-0.142	0.472	0.023	0.908	-0.073	0.713
Peak KAM Rate (N·m/kg/s)	0.156	0.429	0.223	0.254	-0.136	0.490	-0.066	0.740
1st Peak KAM (N·m/kg)	0.219	0.264	0.121	0.538	-0.191	0.331	-0.042	0.833
Time to 1st Peak KAM (s)	0.044	0.823	-0.069	0.728	-0.150	0.447	-0.118	0.551
1st Peak KAM Rate (N·m/kg/s)	0.161	0.413	0.218	0.265	-0.108	0.583	-0.073	0.711
KAM Impulse (∫N·m/kg/s)	0.295	0.127	-0.037	0.850	-0.199	0.310	0.102	0.606
Peak KFM (N·m/kg)	-0.051	0.798	-0.152	0.439	0.034	0.865	0.023	0.908
Time to Peak KFM (s)	-0.110	0.578	0.025	0.900	0.106	0.593	-0.131	0.507
Peak KFM Rate (N·m/kg/s)	0.139	0.482	-0.105	0.595	-0.101	0.611	0.134	0.497
1st Peak KFM (N·m/kg)	0.114	0.563	-0.099	0.615	-0.070	0.725	0.092	0.643
Time to 1st Peak KFM (s)	-0.202	0.302	-0.155	0.432	0.264	0.175	0.015	0.940
1st Peak KFM Rate (N·m/kg/s)	0.146	0.458	-0.055	0.779	-0.100	0.613	0.104	0.597
KFM Impulse (∫N·m/kg/s)	0.033	0.869	-0.019	0.923	-0.044	0.824	-0.147	0.456
Max vGRF (N)	0.240	0.220	0.281	0.148	-0.327	0.089	-0.222	0.255
Time to Max vGRF (s)	-0.142	0.470	-0.038	0.849	0.023	0.908	-0.049	0.805
Loading Rate (N/s)	0.283	0.144	0.322	0.095	-0.295	0.128	-0.203	0.300
Peak Knee Extensor Power (W/kg)	0.168	0.393	0.105	0.596	-0.118	0.550	0.034	0.864
Peak Varus Velocity (°/s)	-0.095	0.638	-0.228	0.243	-0.018	0.930	0.001	0.995
Peak Varus Angle (°)	0.193	0.326	-0.168	0.391	0.099	0.614	-0.153	0.438
Peak Knee Flexion Angle (°)	0.051	0.797	-0.286	0.139	0.057	0.772	0.188	0.338

N·m=Newton-meter; vGRF=vertical ground reaction force; KAM=knee adduction moment; KFM=knee flexion moment
kg=kilograms; s=seconds; ∫=integral; p=p-value (α=0.05); *=significant at p<0.05

Table 2.6 Bilateral Normality Statistics for Stair Ascent Biomechanics

	Dominant Limb		Non-Dominant Limb	
	W	p	W	p
Peak KAM (N·m/kg)	0.951	0.209	***0.752***	***<0.001****
Time to Peak KAM (s)	***0.827***	***<0.001****	***0.853***	***0.001****
Peak KAM Rate (N·m/kg/s)	0.983	0.623	***0.912***	***0.001****
1st Peak KAM (N·m/kg)	0.967	0.501	0.948	0.180
Time to 1st Peak KAM (s)	0.931	0.066	**0.922**	**0.039***
1st Peak KAM Rate (N·m/kg/s)	0.954	0.249	0.972	0.640
KAM Impulse (∫N·m/kg/s)	0.958	0.312	0.948	0.178
Peak KFM (N·m/kg)	***0.879***	***0.004****	***0.624***	***<0.001****
Time to peak KFM (s)	0.961	0.364	0.940	0.114
Peak KFM Rate (N·m/kg/s)	0.980	0.467	***0.574***	***<0.001****
1st Peak KFM (N·m/kg)	***0.879***	***0.004****	***0.908***	***0.018****
Time to 1st Peak KFM (s)	0.961	0.364	0.975	0.712
1st Peak KFM Rate (N·m/kg/s)	0.945	0.146	0.964	0.437
KFM Impulse (∫N·m/kg/s)	0.972	0.632	0.985	0.948
Max vGRF (N)	***0.897***	***0.010****	***0.879***	***0.004****
Time to Max vGRF (s)	0.976	0.742	0.977	0.785
Loading Rate (N/s)	***0.898***	***0.010****	***0.868***	***0.002****
Peak Knee Extensor Power (W/kg)	0.946	0.161	***0.921***	***0.038****
Peak Varus Angle (°)	0.965	0.451	0.959	0.336
Peak Varus Velocity (°/s)	***0.897***	***0.010****	***0.704***	***<0.001****
Peak Knee Flex Angle (°)	0.980	0.839	***0.924***	***0.044****

N·m=Newton-meter; vGRF=vertical ground reaction force; kg=kilograms; KAM=knee adduction moment; KFM=knee flexion moment; s=seconds; ∫=integral; W=Shapiro-Wilk statistic; p=p-value; *=significant at $p<0.05$

Table 2.7 Bilateral Normality Statistics for Stair Descent Biomechanics

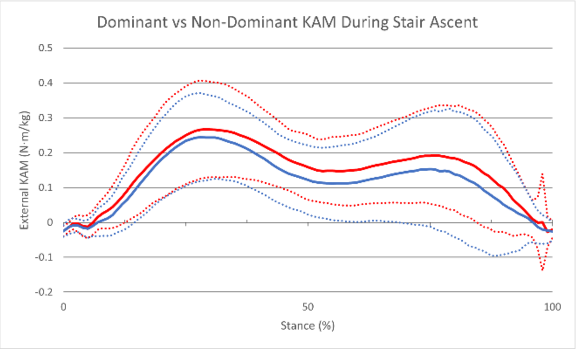
	Dominant Limb		Non-Dominant Limb	
	W	p	W	p
Peak KAM (N·m/kg)	0.986	0.763	***0.901***	***<0.001****
Time to Peak KAM (s)	***0.912***	***<0.001****	***0.839***	***<0.001****
Peak KAM Rate (N·m/kg/s)	***0.887***	***<0.001****	***0.821***	***<0.001****
1st Peak KAM (N·m/kg)	0.983	0.622	***0.910***	***0.001****
Time to 1st Peak KAM (s)	***0.877***	***<0.001****	***0.955***	***0.039****
1st Peak KAM Rate (N·m/kg/s)	***0.949***	***0.021****	***0.842***	***<0.001****
KAM Impulse (∫N·m/kg/s)	0.972	0.232	***0.950***	***0.023****
Peak KFM (N·m/kg)	***0.908***	***<0.001****	***0.890***	***<0.001****
Time to peak KFM (s)	***0.946***	***0.015****	***0.940***	***0.009****
Peak KFM Rate (N·m/kg/s)	***0.685***	***<0.001****	***0.581***	***<0.001****
1st Peak KFM (N·m/kg)	***0.849***	***<0.001****	***0.893***	***<0.001****
Time to 1st Peak KFM (s)	***0.717***	***<0.001****	***0.741***	***<0.001****
1st Peak KFM Rate (N·m/kg/s)	***0.866***	***<0.001****	***0.895***	***<0.001****
KFM Impulse (∫N·m/kg/s)	***0.929***	***0.003****	***0.938***	***0.007****
Max vGRF (N)	***0.914***	***<0.001****	***0.901***	***<0.001****
Time to Max vGRF (s)	0.961	0.071	0.967	0.139
Loading Rate (N/s)	***0.911***	***0.006****	***0.943***	***0.012****
Peak Knee Extensor Power (W/kg)	***0.879***	***<0.001****	***0.913***	***0.001****
Peak Varus Angle (°)	0.962	0.077	0.972	0.221
Peak Varus Velocity (°/s)	***0.939***	***0.008****	0.983	0.627
Peak Knee Flex Angle (°)	0.964	0.096	0.989	0.888

N·m=Newton-meter; vGRF=vertical ground reaction force; kg=kilograms; KAM=knee adduction moment; KFM=knee flexion moment; s=seconds; ∫=integral; W=Shapiro-Wilk statistic; p=p-value; *=significant at $p<0.05$

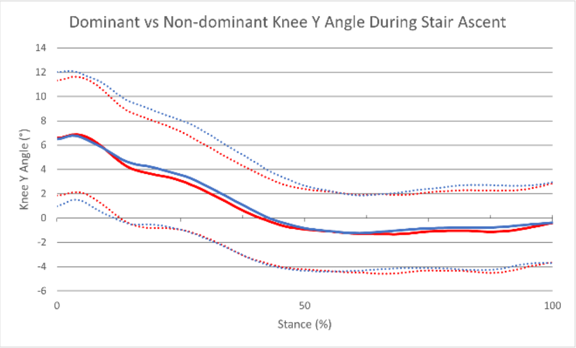
2.6 Figures: Stair Ascent Biomechanics

Figures 2.1-2.3. Dominant (blue) vs Non-dominant (red) limb mean (solid line) and standard deviation (dotted lines) for stance phase frontal plane biomechanics during stair ascent: 2.1) external KAM, 2.2) knee Y angle, 2.3) knee Y velocity

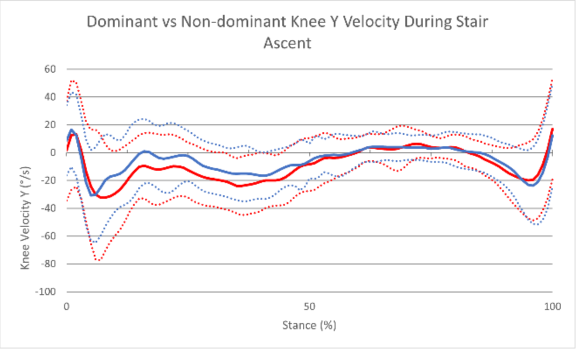
2.1)



2.2)

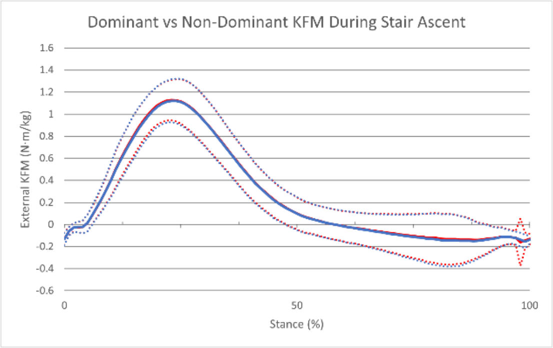


2.3)



Figures 2.4-2.6. Dominant (blue) vs Non-dominant (red) limb mean (solid line) and standard deviation (dotted lines) for stance phase sagittal plane biomechanics during stair ascent: 2.4) external KFM, 2.5) knee X angle, 2.6) sagittal knee power

2.4)



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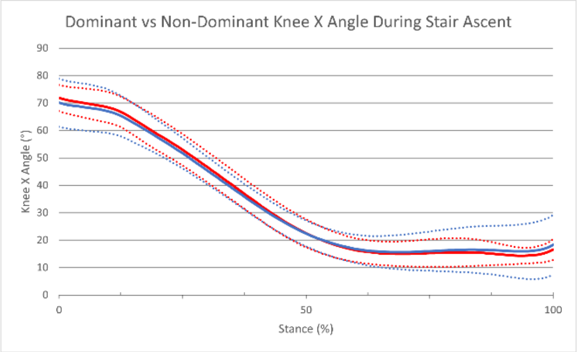

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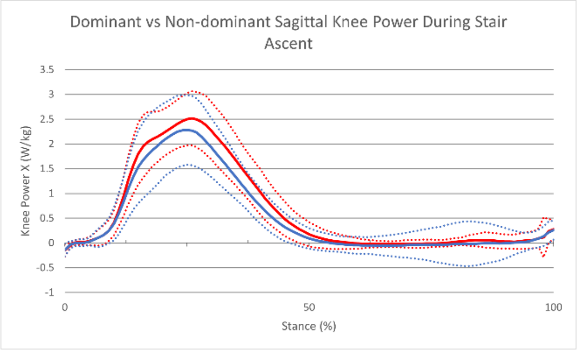
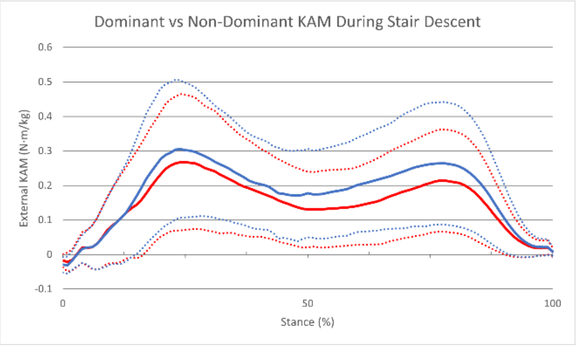


2.7 Figures: Stair Descent Biomechanics

Figures 2.7-2.9. Dominant (blue) vs Non-dominant (red) limb mean (solid line) and standard deviation (dotted lines) for stance phase frontal plane biomechanics during stair ascent: 2.7) external KAM, 2.8) knee Y angle, 2.9) knee Y velocity

2.7)



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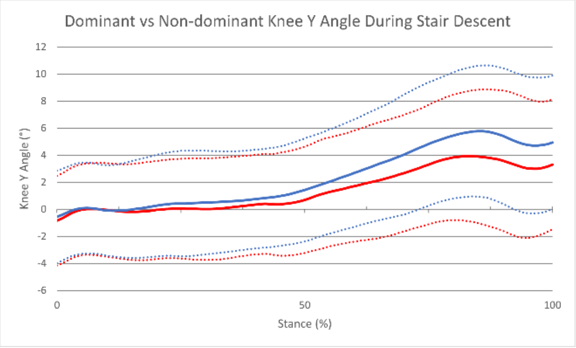

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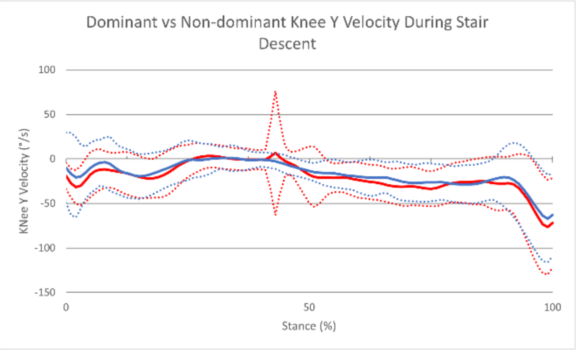
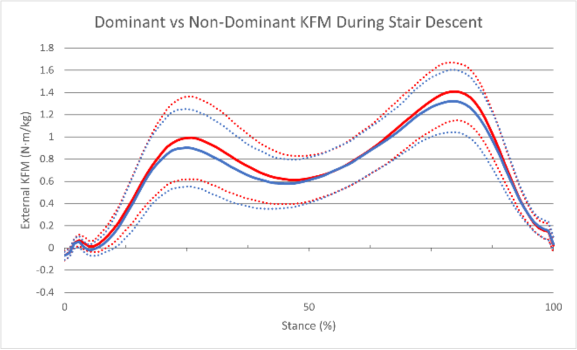


Figures 2.10-2.12. Dominant (blue) vs Non-dominant (red) limb mean (solid line) and standard deviation (dotted lines) for stance phase sagittal plane biomechanics during stair ascent: 2.10) external KFM, 2.11) knee X angle, 2.12) sagittal knee power

2.10)



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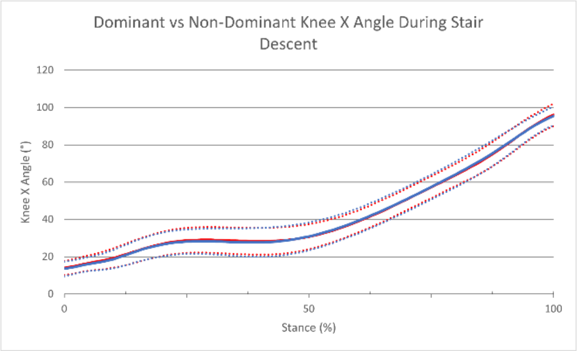

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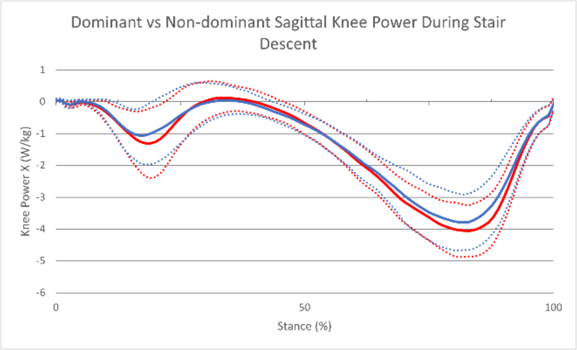
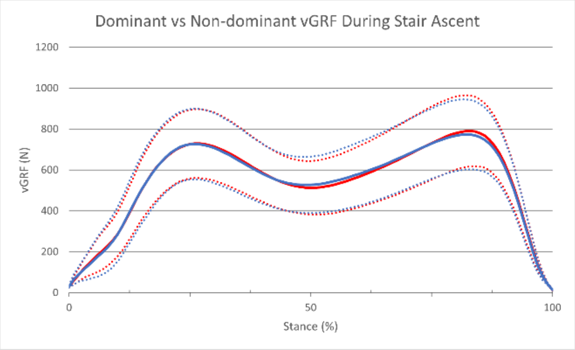


2.8 Figures: Stair Negotiation vGRF

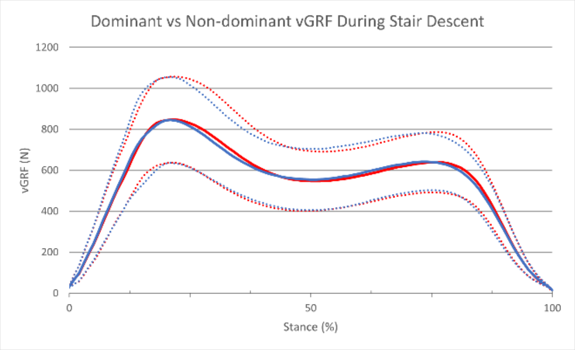
Stair Ascent vGRF

2.13)



Stair Descent vGRF

2.14)



CHAPTER 3:

CROSS-SECTIONAL ANALYSIS OF LEG STIFFNESS AND KNEE BIOMECHANICS BETWEEN LIMBS OF HEALTHY ADULTS DURING STAIR DESCENT

Abstract

Background: Abnormal loading patterns and proprioception during dynamic tasks are linked to osteoarthritis (OA) development and progression. Stair descent is often difficult for OA patients and allows modeling limbs as spring-mass systems to assess load-bearing characteristics through various leg stiffness calculations. Currently, no evidence exists examining relationships between leg stiffness and biomechanical risk factors of knee OA in injured or healthy populations. The objective of this study was to examine relationships between three leg stiffness calculations and biomechanical risk factors of knee OA. **Methods:** Fifty-seven subjects with no history of injury or surgery within the last 6 months were included. Five trials of stair descent capturing data on each limb were measured via 3D motion capture. Movement trial data were processed using Visual3Dv4. This cross-sectional study determined correlations between three methods of leg stiffness calculation and biomechanical risk factors of knee OA during stair descent in healthy individuals on both dominant and non-dominant limbs. Leg stiffness calculations included 3D leg stiffness (3D K_{leg}), uniplanar leg stiffness (K_{leg}), and vertical stiffness (K_{vert}). Biomechanical variables included: Peak Knee Flexion Moment (KFM), Knee Adduction Moment (KAM), KFM and KAM Impulses, 1st Peak and Peak KFM Rate and KAM Rate, Loading Rate (LR), Peak Knee Varus and Flexion Angles, and Peak Varus Velocity. Descriptive statistics are reported as mean ± standard deviation for all variables. Paired-sample T-tests were conducted on all leg stiffness and biomechanical variables between the dominant and non-dominant limbs. Pearson's correlations were calculated to determine relationships between all three leg stiffness methods and biomechanical variables on each limb. **Findings**: No differences in leg stiffness were observed between-limbs. Peak external knee flexion moment (KFM) was significantly greater in the non-dominant limb (1.52 ± 0.30 N·m/kg) compared to the dominant limb (1.44 ± 0.29 N·m/kg, p=0.003, d=0.405). The 1st peak KFM was greater in the non-dominant limb (1.10 ± 0.35 N·m/kg) compared to the dominant limb (1.03 ± 0.33 N·m/kg, p=0.035, d=0.285). Peak eccentric knee extensor power was greater in the non-dominant limb (-4.79 ± 0.82 W/kg) than the dominant limb (-4.54 ± 0.85 W/kg, p=0.004, d=0.393) Peak varus velocity was significantly greater in the dominant limb (39.45 ± 27.81°) compared to the non-dominant limb (26.06 ± 16.29°, p<0.001, d=0.488). Leg stiffness showed several small and moderate correlations with measures of medial knee loading and quadriceps performance. **Interpretation:** Overall leg stiffness was not different between limbs; however, the non-dominant limb demonstrated greater

measures of quadriceps performance as measured via sagittal plane biomechanics. Given the moderate association between leg stiffness and medial knee loading in this healthy, uninjured sample, further biomechanical investigation of neuromuscular control is warranted in injured populations to examine between-limb loading patterns.

3. Introduction

Loading mechanics of the knee have been well-researched in the context of identifying patterns contributing to knee osteoarthritis (OA) development and progression. Kinetic and kinematic indicators of medial knee loading include measures of frontal plane torque development, such as peak external knee adduction moment (KAM),[60, 61] as well as the peak varus angle and velocity[65] experienced during the stance phase of gait. Indeed, greater external KAM and dynamic knee loading have been associated with medial compartment cartilage degeneration and are also associated with worsening grades of knee OA.[57, 66] Biomechanical assessment of knee biomechanics has given insight into loading of the knee; however, overall limb behavior beyond kinematic and kinetic analysis[67-69] of OA patients has not been examined.

Quantitative assessment of leg stiffness is grounded in the spring-mass model (Hooke's Law) but can be calculated using a variety of different methods. Leg stiffness models have been collectively described as neuromuscular variables due to their application in modeling limb compression for sport performance and injury;[4] however, current evidence focuses on applications in running, walking, and stationary hopping activities.[9, 11, 12, 70] Leg stiffness models have not been applied to other activities of daily living that require limb compliance, such as stair descent, which is a common functional goal of knee OA patients during rehabilitation.[71] Despite the potential application for observing limb compliance and neuromuscular control in this manner, leg stiffness models have not been examined in knee OA patients and it is unclear how overall leg stiffness contributes to biomechanical loading patterns associated with knee OA. In the context of knee loading patterns, relationships between leg stiffness measures and biomechanical variables during stair descent have not been examined. Furthermore, it is unknown if self-reported limb dominance influences the relationships between biomechanical variables and leg stiffness. Previous evidence suggests that athletes experience more frequent injuries and have a greater prevalence of knee OA on the non-dominant limb,[55] though small sample sizes make the observed differences marginal. Small differences of cartilage volume between limbs have been observed in healthy subjects.[72] Although limb dominance was not correlated with cartilage volume and thickness, side-to-side differences in thigh muscle cross-sectional area were significantly correlated with side-to-side differences in cartilage morphology, suggesting varying neuromuscular adaptations between limbs. Biomechanical data, however, has not been examined.

Normative biomechanical data during stair negotiation from a healthy population is lacking and is necessary for future comparison in injured populations. Given the paucity of evidence examining the associations of leg stiffness and predictive knee OA biomechanical variables, relationships should be established in healthy individuals to determine how overall limb behavior might influence knee biomechanics. Therefore, the purpose of this study was to examine between-limb differences and relationships of three leg stiffness measures and biomechanical risk factors of knee OA in a healthy population during stair descent. We hypothesized that non-dominant limbs would show increased leg stiffness and that leg stiffness would be positively correlated with frontal plane knee biomechanics, while negatively correlated with sagittal plane knee biomechanics.

3.1 Methods

3.1.1 Subjects

Fifty-eight healthy volunteers (29 male, 29 female; age: 22.24 ± 3.2 years; mass: 71.47 ± 16.60 kg; height: 1.69 ± 0.08 m) were recruited from the community. Subjects were screened using a standard, Office of Human Studies approved, health-history questionnaire. Subjects were recruited via convenience sampling in University of Hawaii classroom settings and were excluded if pregnant, had a known neurologic condition, had a previous history of a hip, knee, or ankle ligament reconstruction, or had a history of recent lower extremity musculoskeletal injury (within 6 months) that hindered the ability to extend the knee, squat, or negotiate stairs. Subjects were asked to self-report limb dominance. Prior to enrollment in the study, all patients signed informed consent and the study was approved by the Institution's Committee on Human Studies.

3.1.2 Procedures

All biomechanical analyses were conducted in the University of Hawaii Human Performance and Biomechanics Laboratory. Motion capture data was collected using 29 retroreflective markers placed on the thorax, pelvis, and lower extremities and four marker-based arrays placed on the thigh and shank segments (Appendix A). For stair descent motion capture, methods were similar to Vallabhajosula et al.[26] Five successful trails were collected, indicated by successful foot strike without the use of the handrail for assistance. All kinetic data were smoothed using a Butterworth filter with a 10 Hz cutoff frequency and processed using Visual 3D (C-Motion, Inc., Germantown, MD). All kinematic and kinetic data were smoothed using a Butterworth filter with a 10 Hz cut-off and ground reaction force was filtered using a 50 Hz cut-off frequency.[27] Kinematic data using the array-based marker-set were collected with a Vicon motion capture system and Vicon Nexus software (Vicon, Inc., Centennial, CO) at 240 Hz and time synchronized with kinetic data collected at 960 Hz collected from on a force plate (Advanced Mechanical Technology Incorporated, Boston, MA) embedded flush with the second step of a three-step staircase.

Three methods of leg stiffness were utilized : 1) Uniplanar (K_{leg}), described by McMahon and Cheng[5] 2) vertical stiffness (K_{vert}), also described by McMahon and Cheng,[5] and 3) 3D leg stiffness (3D K_{leg}), described by Liew et al[10] and scaled to a dimensionless unit of %BW/LL. The dot product of the resultant ground reaction force (GRF) vector and the unit vector of the leg is taken in order to calculate the proportion of the resultant GRF that is in line with leg. In doing so, a projected GRF vector is created as a scalar quantity at each frame. The projected GRF can then be compared to the leg vector frame by frame. The peak of this projection (the vector of greatest magnitude that is most in line with the vector) can then be used to estimate stiffness. The leg vector is taken as a line from the COP to the estimated hip joint center. Equations for each measure of leg stiffness are listed below:

1) $Kleg = \frac{Fmax}{\Delta L}$

Where $\Delta L = \Delta y + L_0(1 - \cos\theta)\ and\ \theta = \sin^{-1}(ut_c/2L_0)$; L_0 = standing leg length θ = half angle of the arc swept by the leg, u = horizontal velocity, and t_c = ground contact time

$$2)\ Kvert = Fmax/\Delta y$$

Where F_{max} is the maximum vertical ground reaction force and Δy is the maximum vertical displacement of the center of mass

$$3)\ 3\text{D}\ Kleg = \frac{Peak\ Projected\ GRF}{\Delta L}$$

Where peak projected GRF is the peak scalar quantity of the dot product of the resultant GRF vector and the 3D leg vector and ΔL is the resultant change in the leg vector.

3.1.3 Statistical Analysis

Pearson correlations were calculated to determine relationships between stair descent biomechanics three measures of leg stiffness on both the dominant and non-dominant limbs. Paired-samples t-tests were conducted to determine the between-limb differences of biomechanical variables and leg stiffness between the dominant and non-dominant limbs. Pearson correlation coefficients were interpreted as small (0.1 to 0.3), medium (0.3 to 0.5), and large (0.5 to 1.0).[30] Cohen's d was calculated for between-limb effect sizes and interpreted as small (0.2), medium (0.5), and large (0.8).[31] Normality and equality of error variances were assessed with Shapiro-Wilk and Levene's Test, respectively. Alpha level was set a priori at $p<0.05$ for all analyses. Power analysis was conducted a priori using G*Power 3.1.9.2; for paired t-test analyses, a sample size of 15 was required to obtain an effect size of 0.80 with power of 0.80 at alpha level of $p<0.05$; for bivariate correlation analyses, a sample size of 29 was required to obtain a correlation of 0.50 with power of 0.80 at alpha level of $p<0.05$. A sample size of 29 was reached; however, due to the amendment and addition of the study protocol to include JPS testing in a SLS test, an additional 29 subjects were recruited for subsequent analyses.

3.2 Results

3.2.1 Between-Limb Comparisons

Descriptive statistics and paired sample T-test results for stair descent leg stiffness and biomechanics are reported in Table 3.1. Several variables violated the assumption of normality, and are reported in Table 3.3. Equality of error variances were assumed for all variables. Peak external knee flexion moment (KFM) was significantly greater in the non-dominant limb (1.52 ± 0.30 N·m/kg) compared to the dominant limb (1.44 ± 0.29 N·m/kg, p=0.003, d=0.405). The 1st peak KFM was greater in the non-dominant limb (1.10 ± 0.35 N·m/kg) compared to the dominant limb (1.03 ± 0.33 N·m/kg, p=0.035, d=0.285). Peak eccentric knee extensor power was greater in the non-dominant limb (-4.79 ± 0.82 W/kg) than the dominant limb (-4.54 ± 0.85 W/kg, p=0.004, d=0.393). Peak varus velocity was significantly greater in the dominant limb (39.45 ± 27.81°) compared to the non-dominant limb (26.06 ± 16.29°, p<0.001, d=0.488). No other statistically significant differences between limbs were observed.

3.2.2 Leg Stiffness and Biomechanics Correlations

Pearson correlations between leg stiffness and biomechanical variables are presented in Table 3.2. Significant correlations were observed between 3D K_{leg} and biomechanical variables in the dominant limb: peak KAM (r= 0.335, p=0.011), time to peak KAM (r=-0.310, p=0.020, peak KAM Rate (r=0.343, p=0.010), 1st peak KAM (r=0.329, p=0.013), 1st peak KAM Rate (r=0.335, p=0.012), 1st peak KFM (r=0.277, p=0.039), time to 1st peak KFM (r=-0.279, p=0.037), 1st peak KFM Rate (r=0.416, p<0.001), time to max vGRF (r=-0.273, p=0.042), Loading Rate (r=0.302, p=0.024), and peak eccentric knee extensor power (r=-0.405, p=0.002); Significant correlations with 3D K_{leg} were also observed on the non-dominant limb: peak KAM (r=0.336, p=0.011), peak KAM Rate (r=0.368, p=0.005), 1st peak KAM (r=0.303, p=0.022), time to 1st peak KAM (r=-0.432, p=0.008), 1st peak KAM Rate (r-0.428, p<0.001), time to peak KFM (r=-0.302, p=0.022), peak KFM Rate (r=0.312, p=0.018), time to 1st peak KFM (r=-0.304, p=0.022), 1st peak KFM Rate (r=0.308, p=0.020), KFM impulse (r=-0.330, p= 0.012), time to max vGRF (r=-0.520, p<0.001), and Loading Rate (r=0.438, p<0.001).

Significant correlations were also observed between K_{vert} and biomechanical variables in the dominant limb: peak KAM (r=0.365, p=0.005), time to peak KAM (r=-0.340, p=0.009), peak KAM Rate (r=0.438, p<0.001), 1st peak KAM (r=0.364, p=0.005), time to 1st peak KAM (r=-0.337, p=0.011), 1st peak KAM Rate (r=0.476, p<0.001), time to peak KFM (r=-0.454, p<0.001), peak KFM Rate (r=0.547, p<0.001), 1st peak KFM (r=0.456, p<0.001), time to 1st peak KFM (r=-0.273, p=0.040), 1st Peak KFM Rate (r=0.584, p<0.001), time to max vGRF (r=-0.298, p=0.024), Loading Rate (r=0.339, p<0.001), peak eccentric knee extensor power (r=-0.384, p=0.003), and peak varus velocity (r=-0.332, p=0.012); Significant correlations were also observed K_{vert} on the non-dominant limb: peak KAM (r=0.307, p=0.020), peak KAM Rate (r=0.379, p=0.004), 1st peak KAM (r=0.276, p=0.038), time to 1st peak KAM (r=-0.378, p=0.004), 1st peak KAM Rate (r=0.413, p<0.001), time to peak KFM (r=-0.448, p<0.001), peak KFM Rate (r=0.569, p<0.001), 1st peak KFM (r=-0.473, p<0.001), time to 1st peak KFM (r=-0.273, p=0.040), 1st peak KFM Rate (r=0.559, p<0.001), time to max vGRF (r=-0.508, p<0.001), Loading Rate (r=0.563, p<0.001), and peak eccentric knee extensor power (r=-0.391, p=0.003).

Uniplanar K_{leg} was significantly correlated with variables in the dominant limb: time to peak KAM (r=-0.275, p=0.038), time to 1st peak KAM (r=-0.344, p=0.009), time to peak KFM (r=-0.303, p=0.020), time to 1st peak KFM (r=-0.438, p<0.001), 1st peak KFM Rate (r=0.280, p-0.035), KFM Impulse (r=-0.285, p=0.032), and peak varus velocity (r=-0.318, p=0.017). The non-dominant limb uniplanar K_{leg} was also correlated with biomechanical variables: time to 1st peak KFM (r=-0.349, p=0.008) and peak knee flexion angle (r=0.267, p=0.045).

3.3 Discussion

To our knowledge, this is the first study to examine correlations between various methods of leg stiffness and biomechanical variables associated with knee OA. It is also the first study to incorporate a recently updated model of triplanar leg stiffness proposed by Liew et al.[10] Analysis of between-limb differences in leg stiffness revealed no side-to-side differences in this healthy cohort. Biomechanical variable differences were observed in the sagittal plane, with the non-dominant limb demonstrating increased quadriceps performance and lower peak varus velocity during stance. Recent evidence suggests that shifting weight onto the non-dominant limb results in decreases of postural control, with no differences in knee strength in trained skiers.[40] The increased external peak KFM of the non-dominant limb in the current sample perhaps suggests that subjects adopted greater knee extensor muscle activity than the dominant limb. This was accompanied by increase eccentric knee extensor power on the non-dominant limb. It is therefore not surprising that subjects demonstrated lower peak varus velocity, perhaps indicating stability facilitated by greater quadriceps activity. Evidence comparing between-limb biomechanical deficiencies is lacking for activities such as stair descent; however, there is wide variability in biomechanical deficiencies noted previously in change of direction activities.[41] Varus thrust (varus velocity) has been found to be predictive of knee OA progression and development,[65, 73, 74] so it is important to note that subjects exhibited greater peak varus velocity on the dominant limb.

The most compelling findings of the current study are the significant correlations observed between various leg stiffness methods and biomechanics associated with knee OA (Table 3.2). A common pattern observed between each measure of leg stiffness was that stiffness was moderately correlated with peak moment development and rate of moment development, while being negatively correlated with the time component of peak moment development. These relationships were present in both 3D K_{leg} and K_{vert} but less so in uniplanar K_{leg}. Increased leg stiffness was associated with increased first peak and the overall peak KAM, suggesting that as subjects navigated stair descent with a stiffer loading pattern, greater medial loads were experienced. Those relationships were relatively consistent between limbs; however, the non-dominant limb demonstrated strong correlations between loading rate and the time to max vGRF. It is reasonable to assume some level of correlation between loading rate and leg stiffness, given the inclusion of the GRF signal in the determination of leg stiffness. Therefore, it is also reasonable to then assume some level of correlation between kinetic variables, due to the inclusion of the GRF signal in the calculation of moments through the inverse dynamics process. Interestingly, however, no significant correlations were observed with max vGRF and leg stiffness. This indicates that the time it takes to reach a peak vGRF is more indicative of dynamic loading than the peak vGRF itself, particularly since increased magnitude and rate of loading have different effects on macroscopic and microscopic cartilage damage.[75] Although Henoa-Murillo et al[75] examined mechanical loading properties of articular cartilage, the concept of dynamic loading of the knee in the manner of the current study may provide insight into movement patterns that may contribute to knee OA development. An important consideration is that the current sample is healthy and denied any history of injury; therefore, conclusions cannot be drawn regarding risk of knee OA development.

The traditional application of uniplanar K_{leg} showed few correlations with biomechanical variables on either limb. Leg stiffness through this method is modelled in the sagittal plane, with the included factor of the arc of the leg sweep during the period of stance. This method showed small-moderate correlations with sagittal plane variables as well as peak varus velocity in the dominant limb. Using this model of stiffness, the correlation with the KFM impulse suggests that stiffer legs undergo less exposure to an external knee flexion moment in the dominant limb. This supports previous conclusions from these analyses that the dominant limb demonstrates less quadriceps control compared to the non-dominant limb. The negative correlation to varus velocity suggests, similarly to K_{vert}, that a stiffer loading pattern was associated with a slower movement in the varus direction. On the non-dominant limb, a small but significant correlation was observed for peak knee flexion angle, which may suggest that whole limb compression ability was not associated with lower knee flexion angles.

Although there were no between-limb differences of leg stiffness, biomechanical indicators of medial joint loading, and quadriceps performance were moderately correlated with

3D K_{leg} and K_{vert}. Important differences between limbs for measures of quadriceps performance were also correlated with these measures of leg stiffness. Previous evidence of leg stiffness asymmetry in rested, healthy individuals has also been reported during running;[76] although, symmetry was not examined in the current study, no differences were detected between-limbs. This may be a consequence of task selection (stair descent). Further investigation may be warranted to examine symmetry between limbs in this task. Given that leg stiffness has been previously described as a neuromuscular variable, in that individuals have the ability to modulate stiffness based on the traversed surface,[4] the results of the current study do not support the notion the non-dominant limb demonstrated greater neuromuscular control than the dominant limb. However, given the observed differences in sagittal plane biomechanical measures, the non-dominant limb demonstrated greater quadriceps control. These contradictory findings should be further investigated to further elucidate differences in neuromuscular control between dominant and non-dominant limbs.

Several limitations should be acknowledged. The first limitation is that the included sample is healthy with no reported history of lower limb surgery, nor any injury within the previous six months; however, activity level and physical condition was not a factor included in this analysis. These results are not generalizable to athletes with regimented training programs or injured populations. Although knee OA predictive biomechanical variables were analyzed, these results may not be generalized to knee OA patients, since knee pain and severity of knee OA may be confounding factors in gait analysis. The second limitation is that limb dominance was self-reported. The validity of this limb dominance reporting strategy is unknown. A final limitation that should be acknowledged is the dependency of biomechanical variables to leg stiffness calculations. Some degree of correlation might be expected between leg stiffness and kinetic/kinematic variables due to the shared signals with which each is calculated through an inverse dynamics approach; however, given the novelty of this analysis, the degree to which these variables should be correlated is unknown.

3.4 Conclusions

Various measures of leg stiffness are correlated with biomechanical movement patterns associated with knee OA. Subjects demonstrated increased quadriceps performance on the non-dominant limb, measured via sagittal plane biomechanical indicators of loading and movement, while demonstrating similar leg stiffness strategies between limbs. Leg stiffness was moderately and positively correlated with medial loading kinetics and sagittal plane measures of external KFM.

3.5 List of Tables

Table 3.1. Between-limb Differences of Leg Stiffness and Stair Descent Biomechanics

	Dominant Limb	Non-Dominant Limb	p	d
3D K_{leg}	15.40 ± 2.68	15.12 ± 3.70	0.412	0.141
Uniplanar K_{leg}	30.56 ± 11.76	32.33 ± 10.78	0.184	0.176
K_{vert}	56.28 ± 8.05	57.29 ± 12.28	0.443	0.101
Peak KAM (N·m/kg)	0.41 ± 0.19	0.36 ± 0.18	0.154	0.190
Peak KAM Rate (N·m/kg/s)	1.60 ± 1.24	1.51 ± 1.15	0.682	0.054
Peak KFM (N·m/kg)	1.43 ± 0.30	1.51 ± 0.30	**_0.002*_**	0.426
Peak KFM Rate (N·m/kg/s)	2.91 ± 1.73	3.24 ± 2.16	0.113	0.211
1st Peak KAM (N·m/kg)	0.39 ± 0.19	0.34 ± 0.18	0.203	0.169
1st Peak KFM (N·m/kg)	1.02 ± 0.33	1.09 ± 0.35	**_0.025*_**	0.303
1st Peak KAM Rate (N·m/kg/s)	2.08 ± 1.18	1.84 ± 1.15	0.166	0.186
1st Peak KFM Rate (N·m/kg/s)	4.98 ± 2.23	5.35 ± 2.30	0.060	0.251
KAM Impulse (∫N·m/kg/s)	0.15 ± 0.11	0.12 ± 0.08	0.167	0.184
KFM Impulse (∫N·m/kg/s)	0.58 ± 0.17	0.61 ± 0.15	0.098	0.221
Peak Varus Angle (°)	4.20 ± 4.77	2.63 ± 4.02	0.067	0.245
Peak Varus Velocity (°/s)	39.45 ± 27.81	26.06 ± 16.29	**_0.001*_**	0.488
Peak Knee Flex Angle (°)	32.39 ± 6.87	32.27 ± 6.26	0.925	0.019
Loading Rate (N/s)	6661.12 ± 1924.78	6387.51 ± 1696.29	0.156	0.189
Max vGRF (N)	932.42 ± 221.32	929.02 ± 223.67	0.723	0.047

N·m=Newton-meter; vGRF=vertical ground reaction force; KAM=knee adduction moment; KFM=knee flexion moment
kg=kilograms; s=seconds; ∫=integral; p=p-value (α=0.05); d=Cohen's d effect size; *=significant at p<0.05

Table 3.2. Correlations Between Leg Stiffness and Stair Descent Biomechanics in Dominant and Non-dominant Limbs

	3D K_{leg}				K_{vert}				Uniplanar K_{leg}			
	Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb		Dominant Limb		Non-Dominant Limb	
	r	p	r	p	r	p	r	p	r	p	r	p
KFM Impulse (∫N·m/kg/s)	-0.077	0.566	-0.123	0.358	-0.122	0.361	-0.091	0.498	**-0.270**	***0.040****	-0.091	0.498
KAM Impulse (∫N·m/kg/s)	0.111	0.406	-0.006	0.963	0.102	0.444	0.075	0.578	-0.107	0.425	0.075	0.578
Peak KAM (N·m/kg)	***0.296***	***0.024****	***0.340***	***0.009****	***0.365***	***0.005****	0.181	0.174	-0.049	0.716	-0.034	0.800
Peak KFM (N·m/kg)	0.162	0.225	-0.069	0.608	0.205	0.123	-0.017	0.898	-0.110	0.409	-0.240	0.070
Peak KAM Rate (N·m/kg/s)	***0.322***	***0.014****	***0.372***	***0.004****	***0.439***	***<0.001†***	0.232	0.079	0.106	0.430	0.060	0.655
Peak KFM Rate (N·m/kg/s)	0.243	0.066	***0.322***	***0.014****	***0.548***	***<0.001†***	***0.335***	***0.010****	0.132	0.323	-0.057	0.672
1st Peak KAM Rate (N·m/kg/s)	***0.335***	***0.011****	0.233	0.079	***0.475***	***<0.001†***	0.232	0.080	0.068	0.618	0.232	0.080
1st Peak KFM Rate (N·m/kg/s)	***0.419***	***0.001****	***0.331***	***0.011****	***0.584***	***<0.001†***	***0.346***	***0.008****	***0.280***	***0.033****	***0.346***	***0.008****
Peak Varus Velocity (°/s)	-0.117	0.397	0.094	0.481	***-0.318***	***<0.001†***	0.037	0.781	***-0.332***	***0.012****	-0.050	0.709
Peak Varus Angle (°)	-0.122	0.361	0.157	0.239	-0.027	0.841	-0.237	0.073	-0.046	0.731	-0.074	0.582
Peak Knee Flexion Angle (°)	-0.195	0.142	-0.105	0.435	0.068	0.612	-0.106	0.430	0.236	0.075	***0.283***	***0.031****
Loading Rate (N/s)	***0.301***	***0.022****	***0.401***	***0.002****	***0.338***	***0.010****	***0.371***	***0.004****	-0.095	0.477	***0.371***	***0.004****
Max vGRF (N)	0.190	0.156	0.046	0.733	0.239	0.071	0.203	0.127	-0.109	0.417	-0.148	0.268

N·m=Newton-meter; vGRF=vertical ground reaction force; KAM=knee adduction moment; KFM=knee flexion moment; kg=kilograms; s=seconds; ∫=integral; p=p-value (α=0.05)
r=Pearson correlation coefficient; *=significant at p<0.05; †=significant at p<0.001

Table 3.3 Bilateral Normality Statistics for Stair Descent Biomechanics & Leg Stiffness

	Dominant Limb		Non-Dominant Limb	
	W	p	W	p
3D K_{leg}	0.976	0.338	0.981	0.523
Uniplanar K_{leg}	0.978	0.411	***0.956***	***0.041****
K_{vert}	***0.855***	***<0.001****	***0.816***	***<0.001****
Peak KAM (N·m/kg)	0.986	0.763	***0.901***	***<0.001****
Time to Peak KAM (s)	***0.912***	***<0.001****	***0.839***	***<0.001****
Peak KAM Rate (N·m/kg/s)	***0.887***	***<0.001****	***0.821***	***<0.001****
1st Peak KAM (N·m/kg)	0.983	0.622	***0.910***	***0.001****
Time to 1st Peak KAM (s)	***0.877***	***<0.001****	***0.955***	***0.039****
1st Peak KAM Rate (N·m/kg/s)	***0.949***	***0.021****	***0.842***	***<0.001****
KAM Impulse (∫N·m/kg/s)	0.972	0.232	***0.950***	***0.023****
Peak KFM (N·m/kg)	***0.908***	***<0.001****	***0.890***	***<0.001****
Time to peak KFM (s)	***0.946***	***0.015****	***0.940***	***0.009****
Peak KFM Rate (N·m/kg/s)	***0.685***	***<0.001****	***0.581***	***<0.001****
1st Peak KFM (N·m/kg)	***0.849***	***<0.001****	***0.893***	***<0.001****
Time to 1st Peak KFM (s)	***0.717***	***<0.001****	***0.741***	***<0.001****
1st Peak KFM Rate (N·m/kg/s)	***0.866***	***<0.001****	***0.895***	***<0.001****
KFM Impulse (∫N·m/kg/s)	***0.929***	***0.003****	***0.938***	***0.007****
Max vGRF (N)	***0.914***	***<0.001****	***0.901***	***<0.001****
Time to Max vGRF (s)	0.961	0.071	0.967	0.139
Loading Rate (N/s)	***0.911***	***0.006****	***0.943***	***0.012****
Peak Knee Extensor Power (W/kg)	***0.879***	***<0.001****	***0.913***	***0.001****
Peak Varus Angle (°)	0.962	0.077	0.972	0.221
Peak Varus Velocity (°/s)	***0.939***	***0.008****	0.983	0.627
Peak Knee Flex Angle (°)	0.964	0.096	0.989	0.888

N·m=Newton-meter; vGRF=vertical ground reaction force; kg=kilograms; KAM=knee adduction moment; KFM=knee flexion moment; s=seconds; ∫=integral; W=Shapiro-Wilk statistic; p=p-value; *=significant at $p<0.05$

CHAPTER 4:

LONGITUDINAL EVALUATION OF KNEE EXTENSOR STRENGTH AND BIOMECHANICS OF KNEE ARTHROPLASTY PATIENTS DURING WALKING AND STAIR NEGOTIATION

Abstract

Background: The purpose of this study was to determine differences of frontal and sagittal plane knee biomechanics and isometric knee extension strength between total knee arthroplasty (TKA) patients and unicompartmental knee arthroplasty (UKA) patients receiving a medial knee compartment implant. ***Methods:*** Twenty-three patients (30 knees) undergoing TKA were randomly assigned either a single-radius (GetAroundKnee™, Stryker Orthopedics, Mahwah, NJ) or multi-radius (Balanced Knee® System, Ortho Development Corporation, Draper, UT) femoral component, and 10 patients (15 knees) receiving medial UKA (Oxford® Partial Knee Implant, Zimmer Biomet Orthopedics, Warsaw, IN) were included in this study. Walking and stair negotiation biomechanics were assessed via 3D motion capture (Vicon). Knee extensor isometric strength was evaluated using a handheld dynamometer (HHD) strapped to a table. Clinical scores were evaluated with the UCLA Activity Questionnaire. Patients reported for data collection within one month prior to surgery as well as 6-months and 12-months after surgery. ***Findings:*** Strength and UCLA scores increased linearly following surgery, but there were no differences between implant groups. The UKA group demonstrated greater neuromuscular control of the quadriceps following surgery evaluated via sagittal plane biomechanics but also demonstrated greater medial loading evaluated via frontal plane biomechanics. ***Interpretation:*** Both TKA and UKA subjects demonstrate comparable strength and overall function following surgery, but key gait parameters measured in the sagittal plane indicate that UKA patients demonstrate greater quadriceps control post-operatively during walking and while navigating stairs. Despite the desired overall decrease in peak KAM following surgery for all patients, UKA patients demonstrated greater levels of medial knee loading following surgery, particularly during walking and stair descent tasks.

4. Introduction

End-stage knee osteoarthritis (OA) patients eligible for total knee arthroplasty (TKA) may receive a popular alternative, a unicompartmental knee arthroplasty (UKA). This alternative was introduced for patients' knee OA isolated to either the medial or lateral compartment. Clinical outcomes for TKA patients are generally good, with patients reporting significant increases in quality of life and functional scores.[77-79] Similar trends are observed with UKA patients.[80] Given the similarity in patient reported outcomes between the two procedures, UKA is advantageous due to faster recovery time attributed to the lesser degree of invasiveness.[81] Another advantage of UKA is the retention of functional stability afforded by the cruciate ligaments, which provide critical mechanoreceptor activity for neuromuscular control of the knee.[1, 81] End-stage knee OA patients receiving a posterior-stabilized (PS) TKA do not have retention of the cruciate ligaments, and therefore may be at a disadvantage for regaining post-operative neuromuscular control.

The advantages of UKA over TKA are clear, and clinical scores for both procedures are excellent, likely due to significant decreases in pain.[71, 77, 82] In addition to pain reduction, restoration of the extensor mechanism is a primary goal following knee arthroplasty. Given that UKA retains the cruciate ligaments, greater neuromuscular control of the knee has been theorized as the reason for greater functional outcomes.[80] Knee extensor strength of UKA patients has been shown to be less than[83] and comparable to[84] the non-operative limb up to 1-year post operatively, but long-term follow-up of 5-years indicates comparable between-limb strength.[85] Similarly, TKA patients have demonstrated lower strength in the operated limb up to 1-year post-operatively but regained full strength after a 3-year follow-up.[86] Despite similar longitudinal trends compared to healthy controls, evidence directly comparing knee extensor strength between TKA and UKA patients is limited. Further understanding long-term quadriceps muscle function may provide insight into how knee OA patients' neuromuscular control recovers following either TKA or UKA.

Knee extensor strength may dictate performance following knee arthroplasty; however, the manifestation of knee OA is a product of aberrant knee loading in one or both compartments and should therefore be investigated further. Frontal and sagittal plane knee biomechanics that indicate knee loading in either plane have been well established. Peak external knee adduction moment (KAM) is widely viewed as a proxy for medial knee loading, while peak external knee flexion moment (KFM) can describe the performance of the knee extensors during gait.[60, 61] During walking gait, similarities of kinetic and kinematic parameters have been observed between TKA and UKA patients, with UKA patients showing greater post-operative function.[87] A more challenging task such as stair negotiation may be required to elicit biomechanical differences between TKA and UKA patients, given that no differences were reported in level and sloped walking[88]. Indeed, UKA patients have demonstrated greater transverse plane rotation compared to TKA, but no differences in sagittal and frontal plane peak moments during stair negotiation.[89]

Although peak KAM and KFM begin to describe medial knee loading and quadriceps activity, respectively, they insufficiently describe neuromuscular control of the knee because they do not encapsulate dynamic motion. More sensitive measures of impact attenuation may be necessary to describe neuromuscular control and loading of the knee. Both KAM rate and total moment exposure, or impulse, have been previously associated with medial knee OA progression.[90] When accounting for KAM rate, the relationship between OA progression and KAM is diminished, which suggests a multifactorial approach should be used to evaluate medial knee loading.[62] In the sagittal plane, rate of torque develoment has been previously associated with quadriceps avoidance gait.[59] In conjunction with KFM impulse and rate, comprehensive evaluation of KFM can best describe the eccentric loading of the quadriceps and impact attenuation during stance. Therefore, the purpose of this study was to determine longitudinal changes in knee extensor strength, clinical scores, and frontal and sagittal plane knee biomechanics between TKA and UKA patients during walking and stair negotiation pre-operatively and 6- and 12-months post-operatively. We hypothesized that UKA patients would show greater knee extensor strength, clinical scores, and KFM variables, as well as decreased KAM variables compared to TKA patients at all time points.

4.1 Methods

4.1.1 Subjects & Surgical Procedure

Twenty-three patients (30 knees) undergoing TKA were randomly assigned either a single-radius (GetAroundKnee™, Stryker Orthopedics, Mahwah, NJ) or multi-radius (Balanced Knee® System, Ortho Development Corporation, Draper, UT) femoral component, and 10 patients (15 knees) receiving medial UKA (Oxford® Partial Knee Implant, Zimmer Biomet Orthopedics, Warsaw, IN) were included in this study. Both TKA implants were posterior stabilized design and all UKA were cruciate retaining. All patients received patellar resurfacing. A single, board certified surgeon performed all surgeries under general anesthesia using a medial parapatellar approach in all patients. The preoperative diagnosis of all patients was osteoarthritis. Therefore, if OA was present in the contralateral limb in these patients, it had not yet progressed to cause pain or functional deficiencies. Patients were included if: 1) under 75 years of age at time of surgery, 2) no previous history of lower extremity fracture, osteotomy, or joint replacement, 3) undergoing a unilateral or simultaneous bilateral TKA for the treatment of OA, and 4) able to walk without an aid. Prior to enrollment in the study, all patients signed informed consent and the study was approved by the Institution's Committee on Human Studies.

4.1.2 Biomechanical Analysis & Strength Testing

All strength and biomechanical analyses were conducted at the University Gait Laboratory. Walking gait biomechanics were collected at a self-selected pace using a 29-retroreflective array-based marker set (Appendix A). Kinematic data were collected with a Vicon motion capture system and Vicon Nexus software (Nexus 2.5, Vicon, Inc., Centennial, CO) at 240 Hz and time synchronized with kinetic data. Kinetic data from the operated limb were collected at 960 Hz from a force plate (Advanced Mechanical Technology Incorporated, Boston, MA) embedded flush with the floor. All kinematic data were smoothed using a low-pass Butterworth filter using a 10 Hz cut-off frequency and ground reaction forces (GRF) were filtered using a 50 Hz cut-off frequency. Joint moments were calculated using inverse dynamics based on marker trajectories and filtered with a 10 Hz cut-off frequency.[27, 56] Three successful trials for walking were collected, indicated by placement of the entire foot on the force plate without a visible change in gait in an attempt to target the force plate. Stair negotiation trials were completed following the walking trials and were conducted in similar fashion to Vallabhajosula et al [26] Five acceptable trials were collected for stair negotiation. Subjects performed the minimum number of trials necessary to obtain acceptable trials for the involved limb undergoing TKA. All data was processed using Visual 3D (C-Motion, Inc., Germantown, MD) and moments are expressed as external.

Following movement trials, strength tests were conducted using a MicroFET2 hand-held dynamometer (Hoggan Health Industries, West Jordan, UT) and performed in a gravity dependent position. Knee extensor strength was assessed while the patient was seated with the knee placed at 60°of knee flexion and their trunk in a recumbent position. The hand-held dynamometer was placed on the anterior shank, at a point 80% of the distance from the lateral knee joint line to the lateral malleolus and secured with a strap. The dynamometer was fixed to the lower tibia to increase the validity and reliability of the measure[91, 92] and is a recommended alternative technique to the isokinetic dynamometer for evaluating knee strength.[91, 92] Subjects were instructed to build force over three seconds, holding the maximal contraction for two seconds. Two trials of maximal effort were completed. A third trial was completed if the second trial did not measure within 10% force output of the first trial. All successful trials were averaged for each participant on the involved limb and calculated as Newton-meters per kilogram (N·m/kg). Verbal encouragement was provided to help elicit maximal force production by the participant during strength testing. Clinical and functional outcomes were evaluated using the UCLA Activity Questionnaire, which has been shown to be an adequate predictor of routine post-TKA activity levels.[93] Data were analyzed preoperatively within one month pre-operatively, and 6-months and 12-months post-operatively.

4.1.3 Statistical Analysis

Descriptive statistics are reported for patient demographics. Independent samples t-tests were conducted between TKA and UKA groups pre-operatively to determine differences in group characteristics. All variables were tested for normality using the Shapiro-Wilk test. Repeated-measures Analysis of Variance (RM-ANOVA) was conducted between TKA and UKA groups over time (pre-operative, 6-months, and 12-months post-operative) during walking and stair negotiation. Strength, UCLA scores, and biomechanical variables at each time point were tested for sphericity and homogeneity of variance using Mauchly's Test of Sphericity and Levene's Test, respectively. Univariate main effects were interpreted without corrections and if the sphericity and/or homogeneity of variance assumptions were violated, Greenhouse-Geisser corrections were used. Bonferroni corrections were used to adjust for multiple comparisons in post-hoc pairwise analyses. Effect sizes were calculated using partial eta squared (ηp^2) and reported as small (0.02), medium (0.13), and large (0.26).[31, 94] All statistical analyses were performed using SPSS version 26.0 (IBM, Armonk, NY, USA). A priori power analysis (G*Power 3.1.9.2) for RM-ANOVA between two groups across three measurement intervals indicated a total sample size requirement of 26 to achieve power of 0.80 with a large effect (ηp^2) of 0.26. Alpha level was set a priori to $p<0.05$ for all analyses.

4.2 Results

4.2.1 Patient Demographics

Pre-operative patient demographics are presented Table 4.1. Body mass index was not normally distributed (W=0.842; p=0.046). Subjects in the TKA and UKA groups did not differ for age, height, body mass or body mass index (BMI). A large effect was detected for age but was not statistically significant, with UKA subjects being younger than TKA subjects (TKA: 67.87 ± 3.68; UKA: 64.80 ± 5.49 years; p=0.068; d=0.716), no statistically significant differences were observed.

4.2.2 Clinical Scores & Knee Extensor Strength

Longitudinal clinical scores for the UCLA Activity Questionnaire and knee extensor strength are presented in Table 4.2. The UCLA Activity scores violated the assumption of homogeneity of variance, therefore the Greenhouse-Geisser correction was interpreted for main effects. Scores were not normally distributed in the UKA group pre-operatively (W=0.721, p=0.002). A significant within-subject main effect was detected (F (2, 64) =22.453; p<0.001;

np^2=0.412). A significant between-subjects effect was detected at 6-months post-operatively, with UKA patients indicating higher UCLA scores (7.30 ± 1.64) than TKA patients (5.79 ± 1.25; mean difference= -1.508, p=0.006, np^2=0.211, 95% CI: -2.559, -0.458). All subjects reported lower scores pre-operatively (4.82 ± 1.93) than 6-months (6.24 ± 1.51, p<0.001) and 12-months post-operatively (6.76 ± 1.71, p<0.001).

Knee extensor strength violated the assumption of sphericity, therefore the Greenhouse-Geisser correction was interpreted for main effects. Additionally, the TKA showed a non-normal distribution at 6-months (W=0.927; p=0.040) and the UKA group showed a non-normal distribution at 12-months (W=0.843; p=0.014). A significant within-subject effect was detected (F (1.735, 74.615) =8.538, p<0.001, np^2=0.166); however, no between group interactions were detected at any time point. All subjects demonstrated greater strength 12-months post-operatively (1.20 ± 0.36 N·m/kg) compared to 6-months post-operatively (1.10 ± 0.32 N·m/kg, p=0.016) and pre-operatively (0.99 ± 0.38 N·m/kg, p<0.001).

4.2.3 Walking Biomechanics

Thirty knees were included in the TKA group and 15 knees were included in the UKA group. Descriptive statistics for walking biomechanics over time for each group are presented in Table 4.3. Several variables violated the assumption of normality and are reported in Table 4.6. Sphericity was assumed for all variables, with exception of peak KAM (W=0.316, p<0.001), peak KAM rate (W=0.754, p=0.003), peak knee flexion angle (W=0.669, p<0.001), peak knee varus angle (W=0.532, p<0.001), KAM impulse (W=0.199, p<0.001), KFM impulse (W=0.682, p<0.001), and walking velocity (W=0.675, p<0.001). Equality of variance between groups was assumed for all variables at each time interval, with exception of pre-operative peak KAM (W=8.848, p=0.005), time to peak KAM (W=7.620, p=0.008), peak KAM rate (W=6.225, p=0.017), peak knee varus angle (W=8.012, p=0.007); 6-month peak KFM (W=4.895, p=0.032), peak KFM rate (W=5.611, p=0.022), loading rate (W=6.447, p=0.015), peak eccentric knee extensor power (W=7.609, p=0.008), walking velocity (W=10.778, p=0.002); and 12-month walking velocity (W=6.431, p=0.015).

Significant within-subject univariate effects for time (in all subjects) were detected for peak KAM (F (1.188, 51.078)=28.740, p<0.001, np^2=0.401), time to peak KAM (F (2, 86)=5.066, p=0.008, np^2=0.105), peak KAM rate (F (1.605, 69)=4.055, p=0.030, np^2=0.086), KAM impulse (F (1.110, 47.745)=38.257, p<0.001, np^2=0.471), peak KFM (F (2, 86)=6.489, p=0.002, np^2=0.131), max vGRF (F (2, 80.290)=11.482, p<0.001, np^2=0.211), max vGRF/kg (F (2, 83.574)=9.331, p<0.001, np^2=0.178), peak eccentric sagittal knee power (F (2, 86)=13.608, p<0.001, np^2=0.240), peak knee flexion angle (F (1.503, 64.630)=7.933, p=0.002, np^2=0.156), and peak knee varus angle (F (1.362, 76.436)=7.933, p=0.002, np^2=0.525), and walking velocity (F (1.509, 64.890)=25.474, p<0.001, np^2=0.372). One significant univariate implant x time interaction effect was detected for peak KAM rate ((F (1.605, 69) =3.427, p=0.048, np^2=0.074).

Significant between-subject pairwise comparisons were observed at each time point during walking and are presented in Table 4.3. For peak KAM, were comparable pre-operatively and at 6-months post-operatively, and lower at 12-months post-operatively (mean difference: -0.077 N·m/kg, SE: 0.032, p=0.023, ηp^2=0.115, 95% CI: -0.142, -0.011). For peak KAM rate, TKA were lower than UKA subjects pre-operatively (mean difference: -1.678 N·m/kg/s, SE: 0.507, p=0.002, ηp^2=0.203, 95% CI: -2.699, -0.656), lower than UKA subjects 6-months post-operatively (mean difference: -1.511 N·m/kg/s, SE: 0.581, p=0.013, ηp^2=0.136, 95% CI: -2.683, -0.339) and lower than UKA subjects 12-months post-operatively (mean difference: -1.490 N·m/kg/s, SE: 0.265, p<0.001, 95% CI: -2.025 , -0.956). For KAM impulse, TKA subjects were lower than UKA subjects pre-operatively (mean difference: -0.098 ∫N·m/kg/s, SE: 0.036, p=0.009, $\eta p^2=0.147$, 95% CI: -0.171 , -0.025), 6-months post-operatively (mean difference: -0.045 ∫N·m/kg/s, SE: 0.018, p=0.016, $\eta p^2=0.127$, 95% CI: -0.082 , -0.009), and 12-months post-operatively (mean difference: -0.053 ∫N·m/kg/s, SE: 0.017, p=0.003, $\eta p^2=0.186$, 95% CI: -0.087 , -0.019). For peak KFM rate, TKA subjects were significantly lower pre-operatively (mean difference: -2.030 N·m/kg/s, SE: 0.711, p=0.007, $\eta p^2=0.127$, 95% CI: -3.465, -0.595), lower than UKA subjects 6-months post-operatively (mean difference: -1.511 N·m/kg/s, SE: 0.581, p=0.013, $\eta p^2=0.136$, 95% CI: -2.683, -0.339), and lower than UKA subjects 12-months post-operatively (mean difference: -1.719 N·m/kg/s, SE: 0.609, $\eta p^2=0.156$, 95% CI: -2.947, -0.491). For KFM impulse, TKA subjects were significantly lower 6-months post-operatively (mean difference: -0.053 ∫N·m/kg/s, SE: 0.023, p=0.027, $\eta p^2=0.108$, 95% CI: -0.100, -0.006) but equal pre-operatively and 12-months post-operatively. For peak knee flexion, TKA subjects were significantly lower 12-months post-operatively (mean difference: -4.344°, SE: 1.867, p=0.025, $\eta p^2=0.112$, 95% CI: -8.110, -0.578) but comparable to UKA pre-operatively and 6-months post-operatively. For peak knee varus, TKA and UKA subjects were equal pre-operatively and at 6-months post-operatively but TKA subjects were significantly lower at 12-months post-operatively (mean difference: -3.275°, SE: 1.150, p=0.007, 95% CI: -5.595, -0.956). For peak knee varus velocity, TKA and UKA subjects were equal pre-operatively and 12-months post-operatively but TKA subjects were significantly higher at 6-months post-operatively (mean difference: 19.606°/s, SE: 8.922, p=0.033, $\eta p^2=0.101$, 95% CI: 1.613, 37.598).

4.2.4 Stair Ascent Biomechanics

Twenty-one knees were included in the TKA group, and 14 knees were included in the UKA group. Several subjects were unable to negotiate stairs at one or more data collection periods and were removed from the RM-ANOVA: Six TKA subjects were unable to ascend stairs pre-operatively, one unable at 6-months post-operatively (9 knees total); one UKA subject was unable to ascend stairs pre-operatively (one knee total). Stair ascent biomechanics across time points for each group are presented in Table 4.4. Several variables violated the assumption of normality and are reported in Table 4.7. Sphericity was assumed for all variables except for peak KAM (W=0.809, p=0.033), KAM impulse (W=0.601, p<0.001), time to peak KFM (W=0.136, p<0.001), peak KFM Rate (W=0.128, p<0.001), KFM impulse (W=0.470, p<0.001), max vGRF (W=0.596, p<0.001), time to max vGRF (W=0.501, p<0.001), and stance time (W=0.158, p<0.001). Greenhouse-Geisser corrections were used for interpreting univariate effects in all variables that violated the sphericity assumption.

Significant within-subject univariate effects for time (for all subjects) were detected for peak KAM (F (1.679, 55.401)=15.772, p<0.001, np^2=0.323), time to peak KAM (F (2, 66)=14.717, p<0.001, np^2=0.308), KAM impulse (F (1.430, 47.176)=19.048, p<0.001, np^2=0.366), time to peak KFM (F (1.073, 61.104)=11.130, p=0.002, np^2=0.252), max vGRF (F (1.425, 47.017)=3.799, p=0.043, np^2=0.103), and peak knee flexion angle (F (2, 66)=5.005, p=0.009, np^2=0.009). Significant univariate time x implant interaction effects were detected for peak KAM rate (F (2, 66)=4.703, p=0.012, np^2=0.125), KAM impulse (F (1.430, 47.176)=3.791, p=0.043, np^2=0.103), peak KFM (F (2, 66)=5.922, p=0.004, np^2=0.148), KFM impulse (F (1.376, 35.256)=3.644, p=0.049, np^2=0.097), and peak knee flexion (F (2, 66)=3.916, p=0.025, np^2=0.103).

Between-subject pairwise comparisons at each time point are presented in Table 4.4. For peak KFM, TKA subjects were significantly lower than UKA subjects pre-operatively (mean difference: -0.204 N·m/kg, SE: 0.072, p=0.008, ηp^2= 0.196, 95% CI: -0.350, -0.058) and were equal at 6-months and 12-months post-operatively. For time to peak KFM, groups were comparable pre-operatively and 12-months post-operatively, but TKA subjects were significantly lower than UKA subjects 6-months post-operatively (mean difference: -0.050 s, SE: 0.020, p=0.018, ηp^2=0.157, 95% CI: -0.091, -0.009). For KFM impulse, TKA subjects and UKA subjects were equal pre-operatively and 12-months post-operatively, but TKA subjects were significantly lower at 6-months post-operatively (mean difference: -0.088 ∫N·m/kg/s, SE: 0.036, p=0.022, ηp^2=0.149, 95% CI: -0.162, -0.014). For time to peak KAM, TKA subjects were comparable pre-operatively and 12-months post-operatively but were lower than UKA subjects at 6-months post-operatively (mean difference: -0.125 s, SE: 0.054, p=0.026, ηp^2=0.141, 95% CI: -0.234, -0.016). For peak KAM rate, TKA and UKA subjects were comparable pre-operatively and 12-months post-operatively, but TKA subjects were significantly greater 6-months post-operatively (mean difference: 0.318 N·m/kg/s, SE: 0.149, p=0.041, ηp^2=0.121, 95% CI: 0.014, 0.621).

4.2.5 Stair Descent Biomechanics

Twenty knees were included in the TKA group and 11 knees were included in the UKA group. Several subjects were unable to negotiate stairs at one or more data collection periods and were removed from the RM-ANOVA: Six TKA subjects were unable to descend stairs pre-operatively, one unable at 6-months post-operatively and one unable at 12-months post-operatively (10 knees total); three UKA subjects were unable to descend stairs pre-operatively, and one 6-months post-operatively (4 knees total). Stair descent biomechanics for each group across time are presented in Table 4.5. Several variables violated the assumption of normality and are reported in Table 4.8. Equal variances were assumed between-groups at each time interval for all variables, with exception of peak knee varus angle pre-operatively (W=8.719, p=0.006) and peak knee flexion angle post-operatively at 6-months (W=6.055, p=0.020) and 12-months (W=7.784, p=0.009). All variables violated the sphericity assumption, with exception of peak KAM, peak KFM, and peak knee flexion angle. Greenhouse-Geisser corrections were used for: time to peak KAM (W=0.357, p<0.001), peak KAM rate (W=0.761, p=0.022), KAM impulse (W=0.459, p<0.001), time to peak KFM (W=0.322, p<0.001), peak KFM rate (W=0.771, p=0.026), KFM impulse (W=0.757, p=0.020), max vGRF (W=0.516, p<0.001), time to max vGRF (W=0.516, p<0.001), loading rate (W=0.480, p<0.001), peak eccentric knee extensor power (W=0.727, p=0.012), peak knee varus angle (W=0.646, p=0.002), and peak knee varus velocity (W=0.396, p<0.001).

Significant within-subject univariate effects for time (for all subjects) were detected for peak KAM (F (2, 58)=15.365, p<0.001, np^2=0.361), time to peak KAM (F (1.217, 35.299)=6.782, p=0.010, np^2=0.190), KAM impulse (F (1.298, 37.361)=26.881, p<0.001, np^2=0.481), peak KFM (F (2, 58)=4.064, p=0.022, np^2=0.123), time to peak KFM (F (1.192, 34.558)=4.822, p=0.029, np^2=0.143), peak KFM rate (F (1.628, 47.201)=7.840, p=0.002, np^2=0.213), KFM impulse (F (1.609, 46.654)=5.241, p=0.013, np^2=0.153), max vGRF (F (1.348, 39.095)=9.530, p=0.002, np^2=0.247), max vGRF/kg (F (1.376, 39.914)=7.501, p=0.005, np^2=0.206), Loading Rate (F (1.316, 38.166)=5.579, p=0.016, np^2=0.161), peak eccentric knee extensor power (F (1.571, 45.567)=4.894, p=0.018, np^2=0.144), peak knee flexion angle (F (2, 58)=6.694, p=0.002, np^2=0.188), peak knee varus angle (F (1.477, 42.824)=41.961, p<0.001, np^2=0.591), and stance time (F (1.182, 35.457)=20.584, p<0.001, np^2=0.407). A single significant univariate implant x time interaction effect was detected for peak knee flexion angle (F (2, 58) =3.655, p=0.032, np^2=0.112).

Between-subject pairwise comparisons at each time point presented in Table 4.5. For peak KAM, TKA and UKA subjects were comparable pre-operatively and 6-months post-operatively, but TKA subjects were significantly lower at 12-months post-operatively (mean difference: -0.216 N·m/kg, SE: 0.075, p=0.008, ηp^2=0.221, 95% CI: -0.371, -0.062). For peak KFM, TKA and UKA subjects were comparable pre-operatively and 12-months post-operatively, but TKA subjects were significantly lower 6-months post-operatively (mean difference: -0.205 N·m/kg, SE: 0.087, p=0.026, ηp^2=0.159, 95% CI: -0.384, -0.026). For peak eccentric knee extensor power, TKA subjects were lower than UKA subjects pre-operatively (mean difference: -0.205 N·m/kg, SE: 0.348, p=0.039, ηp^2=0.139, 95% CI: 0.042, 1.465) and lower 6-months post-operatively (mean difference: 0.573 N·m/kg, SE: 0.251, p=0.030, ηp^2=0.152, 95% CI: 0.059, 1.087), but were comparable to UKA subjects 12-months post-operatively. For Loading Rate, TKA subjects were significantly lower than UKA subjects pre-operatively (mean difference: -3244.097 N/s, SE: 1502.473, p=0.039, ηp^2=0.138, 95% CI: -6316.998, -171.195) but were comparable 6 and 12-months post-operatively.

4.3 Discussion

The goal of this study was to evaluate longitudinal changes in knee extensor strength, clinical scores, and knee biomechanics within the frontal and sagittal plane for TKA and UKA patients. The results described and discussed below indicate that early intervention of medial knee OA with the utilization of UKA restores early function and allows for more expedient recovery of the quadriceps muscle group, which is critical for ADLs and quality of life following knee arthroplasty.

4.3.1 Strength and Clinical Scores

This analysis reports novel findings comparing TKA and UKA strength gains, in conjunction with biomechanical outcomes during walking and stair negotiation. Strength and clinical scores generally improved for all patients after knee arthroplasty; however, the implant did not appear to have a detectable influence on knee extensor strength and overall function as measured on the UCLA Activity Questionnaire. These results agree with previous evidence; however, we expected UKA patients to demonstrate greater knee extensor strength due to the procedure being less invasive and the retention of the cruciate ligaments providing for a more naturally occurring knee anatomy. Contrary to early evidence suggesting that knee extensor strength does not exceed pre-operative levels,[95, 96] both TKA and UKA subjects showed greater strength 12-months after surgery, which agrees with more recent evidence.[97-99] Li et al[85] observed resumption of knee extensor strength and power up to five years post-UKA; however, comparisons were not made between TKA and UKA patients.

4.3.2 Walking Biomechanics

During walking, several important findings should be noted. In the frontal plane, peak KAM, as well as other indicators of medial loading, were greater in the UKA subjects post-operatively. Peak KAM generally decreased following surgery for all subjects, but TKA subjects maintained a decreasing peak KAM, while UKA subjects showed a slight resurgence in peak KAM at 12-months post-operatively (Figures 4.7-4.9). Given the etiology that only the medial compartment is affected in UKA patients such that a UKA is indicated over a TKA, this may not be surprising. Consequently, UKA subjects demonstrated greater KAM impulse than TKA at all time points. Overall larger peak KAM experienced during stance may explain this, particularly since both groups demonstrated comparable stance times and walking velocity throughout the study duration. Interestingly, the UKA group also demonstrated greater peak knee varus at 12-months (Figure 4.12). Despite the restoration of frontal plane alignment being one of the goals of knee arthroplasty,[100] these results may indicate that UKA subjects maintain a greater varus alignment than TKA subjects during gait. Further examination of mechanical alignment between groups' implant designs may be warranted for future analyses.

In the sagittal plane, there was some evidence observed during walking that would indicate superior quadriceps performance of the UKA group. At 6-months post-operatively, UKA subjects demonstrated greater total exposure to external KFM than TKA subjects but this difference was not observed pre-operatively or 12-months post-operatively. The UKA group did, however, demonstrate greater rate of peak KFM development across all three time points. Kline et al[59] suggests that greater quadriceps torque development during gait is desirable due to lower rates significantly contributing to quadriceps avoidance gait patterns. Previous evidence also suggests that rate of torque development of the quadriceps is associated with walking speed in knee OA patients.[101] Given the similarities in walking velocity between the groups at each time interval, it does not appear that differences in KFM rate of development are not attributed to changes in walking velocity post-surgery. Regarding quadriceps activity, all subjects did, however, improve over time, evidenced by linear increases in peak KFM and greater peak KFM rate. The increase in external KFM variables across time points coincide with increases in knee extensor strength, which supports the notion that quadriceps performance can be evaluated by external KFM. The peak KFM also indicates an increased willingness to load the limb;[60] however, both groups demonstrated comparable vertical GRF's and loading rates throughout stance. The UKA subjects appear to demonstrate greater quadriceps function both pre- and post-operatively; however, walking may not be a challenging enough task to elicit differences in loading. The TKA group also demonstrated lower peak knee flexion angles 12-months post-operatively (Figure 4.6), which may be long-term consequence of TKA procedures.[102]

4.3.3 Stair Ascent Biomechanics

During stair ascent, several important findings were observed between TKA and UKA subjects regarding sagittal plane biomechanics. The TKA group was generally lower for all metrics at one time point or another. It should be noted that TKA subjects showed lower baseline quadriceps activity measured by peak KFM and peak concentric knee extensor power but recovered to comparable levels as UKA subjects at both times post-operatively. The TKA group showed lower overall KFM impulse at 6-months post-operatively; however, both TKA and UKA subjects showed comparable exposure to an external KFM pre-operatively and 12-months post-operatively. This may suggest that TKA patients continue to show less willingness to load the limb[60, 61] while climbing stairs up to 6-months after surgery compared to UKA patients. Equal peak KFM measures were observed but a lower KFM impulse would suggest that the exposure to an external KFM occurred over a shorter period of stance. Similar patterns have been observed in the frontal plane, indicating an antalgic gait pattern.[103] On the contrary, subjects demonstrated comparable stance times throughout the study duration, suggesting that differences in total moment exposure was a consequence of the magnitude of moment development rather than the stance duration. Interestingly, TKA and UKA subjects demonstrated comparable strength increases post-operatively, with no interaction effects observed between groups over time. Despite the comparable increases in post-operative strength and KFM, total exposure to a KFM seems to suggest that TKA patients have not yet regained full neuromuscular control of the quadriceps as early as 6-months following surgery. Furthermore, TKA subjects demonstrated lower peak knee flexion 12-months post-operatively but comparable flexion angles to UKA patients pre-operatively and 6-months post-operatively. This may not be a consequence of inadequate neuromuscular control of the quadriceps, given the motion required during stair ascent. Greater degrees of knee flexion are required to climb stairs successfully[104] and diminished post-operative knee flexion may be a negative consequence of the more invasive nature of TKA compared to UKA. The degree to which this mediates quadriceps control post-operatively is unknown; however, UKA has demonstrated clear early clinical advantages.[85, 93]

The significant interaction main effect observed for peak KAM rate is of some importance. The TKA group showed greater rate of moment development at 6-months post-operatively; however, both the pre-operative and 12-month post-operative times showed comparable rates between groups. This may be explained by the faster time taken to reach the peak moment by the TKA subjects compared to UKA subjects, particularly since each group showed similar peak moments. Morgenroth et al[62] suggests that greater frequency of subchondral bone marrow lesions is associated with greater rates of medial knee loading, even after adjusting for peak moment; therefore, a faster time to develop a peak frontal plane moment should be considered an undesirable occurrence. Despite these relatively short-term adaptations, no differences between groups were observed at the 12-month post-operative data collection.

4.3.4 Stair Descent Biomechanics

During stair descent, few differences were observed between TKA and UKA patients regarding sagittal plane biomechanics and quadriceps control. Lower peak KFM was observed in TKA patients 6-months following surgery compared to UKA, suggesting that overall quadriceps activity was lower at this time point; however, groups showed comparable peak KFM rates. Despite comparable peak KFM at 12-months post-operatively, a small-medium effect was detected at the same time point. While not statistically significant, this effect may indicate a lower peak KFM rate in TKA subjects up to 12-months post-surgery. Given that lower rate of torque development of the quadriceps has been shown to be a significant contributor to quadriceps avoidance gait,[59] greater peak KFM rate would be desired to demonstrate greater neuromuscular control of the quadriceps. This finding is important to note, as it demonstrates that peak KFM alone may not adequately describe quadriceps activity. Previous research suggests that improvements in KFM can be attributed to an increased willingness to load the limb,[60, 61] so it is feasible that that contributes to greater stair descent ability. The UKA group demonstrated moderately greater max vGRF loading rate prior to surgery, with a small-medium but non-significant effect for max vGRF. This in conjunction with greater eccentric knee extensor power pre-operatively may indicate that UKA subjects were more willing to load their limbs prior to surgery than TKA subjects. The UKA group maintained greater eccentric knee extensor power up to 6-months post-operatively, but groups were comparable at 12-months post-surgery. This provides further evidence that UKA patients regain function earlier than TKA, which is in agreement with early clinical advantages and indications.[105]

In the frontal plane, peak KAM was greater in the UKA group at 12-months post-operatively (Figure 4.51) but comparable pre-operatively and 6-months post-surgery. This may not be surprising, given the small-medium effect detected pre-operatively. It seems feasible that since isolated medial compartment replacement is generally indicated for UKA patients, they may have greater levels of baseline medial loading. While all subjects generally showed the desired decrease in KAM levels following surgery, the UKA group showed overall greater levels of peak KAM across all time points. Further examination of the severity of knee OA in these groups is needed, however. It is possible that the TKA group demonstrated equal severity in the medial compartment, but with severe enough OA present in the lateral compartment to warrant a

TKA procedure. Considering the etiology of medial compartment knee OA, both groups showed comparable levels of medial compartment loading 6-months post-operatively, but UKA patients reverted back to greater peak KAM 12-months post-operatively, albeit lower than pre-operative levels.

4.3.6 Limitations

Several limitations should be acknowledged. In the TKA group, patients receiving both multi-radius and single-radius femoral components were included. The degree to which the implant influences the knee extensor mechanism during these tasks is unknown. Additionally, follow-up from patients and some patients' inability to navigate stairs limited the sample size of each group, particularly during stair negotiation. Only patients that completed both the 6-month and 12-month data collections were included in this repeated-measures analysis; however, this may also increase statistical power via removal of the within-subjects error term in the determination of the F statistic. Another limitation is that only medial UKA patients were included, therefore the results may not be generalizable to lateral compartment UKA patients. Lastly, TKA and UKA may not be comparable due to different etiologies of knee OA manifestation. Isolated medial compartment knee OA is a primary indicator for medial UKA; however, TKA is often considered if the lateral compartment shows enough cartilage degradation.

4.4 Conclusions

Both TKA and UKA subjects demonstrate comparable strength and overall function following surgery, but key gait parameters measured in the sagittal plane indicate that UKA patients demonstrate greater quadriceps control post-operatively. Despite the desired overall decrease in peak KAM following surgery for all patients, UKA patients demonstrated greater levels of medial knee loading following surgery, particularly during walking and stair descent tasks.

4.5 Tables and Figures

Table 4.1. Baseline TKA and UKA Patient Demographics

	TKA (n=23)	UKA (n=10)	p	d
Age (y)	67.87 ± 3.68	64.80 ± 5.49	0.068	0.716
Height (m)	1.63 ± 0.09	1.68 ± 0.09	0.145	0.567
Body Mass (kg)	79.53 ± 17.11	83.30 ± 16.03	0.557	0.225
Body Mass Index	29.90 ± 5.30	29.28 ± 3.89	0.742	0.126

	TKA (n=23)	UKA (n=10)	χ2(1,33)
Males	12	6	0.172, p=0.678
Females	11	4	
Bilateral Cases	7	5	1.153, p=0.283
Unilateral Cases	16	5	

TKA=total knee arthroplasty; m=meters; kg=kilograms; y=years; UKA=unicompartmental knee arthroplasty; p=p-value (α=0.05); χ2=Pearson's Chi Square ($p<0.05$)

Table 4.2. Knee Extensor Strength and UCLA Activity Questionnaire Scores Pairwise Comparisons Across Time

	TKA (n=23 patients) (n=30 knees)	UKA (n=10 patients) (n=15 knees)	Mean Difference	SE	p	np^2	95% CI	
Pre-operative							Lower	Upper
Strength (N·m/kg)	0.92 ± 0.37	1.12 ± 0.37	-0.196	0.117	0.100	0.062	-0.432	0.039
UCLA Score	4.71 ± 1.37	5.10 ± 2.96	-0.392	0.734	0.598	0.009	-1.888	1.104
6-months Post-operative								
Strength (N·m/kg)	1.07 ± 0.33	1.16 ± 0.31	-0.098	0.102	0.340	0.021	-0.303	0.107
UCLA Score	5.79 ± 1.25	7.30 ± 1.64	***-1.508****	0.516	***0.006****	***0.211***	-2.559	-0.458
12-months Post-operative								
Strength (N·m/kg)	1.16 ± 0.34	1.27 ± 0.39	-0.112	0.113	0.328	0.022	-0.340	0.116
UCLA Score	6.46 ± 1.53	7.50 ± 1.96	-1.042	0.626	0.106	0.080	-2.316	0.233

N·m=Newton-meter; kg=kilograms; SE=standard error; p=p-value (α=0.05); CI=confidence interval of the difference

np^2=partial eta squared effect size; *=significant interaction effect ($p<0.05$)

Table 4.3. TKA vs UKA Walking Biomechanics Pairwise Comparisons Across Time

	Implant Group		Mean				95% CI	
Pre-operative	TKA (n=30)	UKA (n=15)	Difference	SE	p	ηp^2	Lower	Upper
Peak KAM (N·m/kg)	0.50 ± 0.22	0.61 ± 0.13	-0.114	0.061	0.069	0.075	-0.237	0.009
Time to Peak KAM (s)	0.27 ± 0.12	0.30 ± 0.12	-0.024	0.039	0.539	0.009	-0.102	0.054
Peak KAM Rate (N·m/kg/s)	***2.30 ± 1.48***	***3.98 ± 1.83***	***-1.678*†***	***0.507***	***0.002*†***	***0.203***	***-2.699***	***-0.656***
KAM Impulse (∫N·m/kg/s)	***0.21 ± 0.13***	***0.31 ± 0.08***	***-0.098****	***0.036***	***0.009****	***0.147***	***-0.171***	***-0.025***
Peak KFM (N·m/kg)	0.61 ± 0.24	0.59 ± 0.22	0.020	0.075	0.794	0.002	-0.131	0.171
Time to Peak KFM (s)	0.19 ± 0.10	0.17 ± 0.09	0.015	0.031	0.630	0.005	-0.048	0.079
Peak KFM Rate (N·m/kg/s)	***2.96 ± 2.18***	***4.99 ± 2.39***	***-2.030****	***0.711***	***0.007****	***0.159***	***-3.464***	***-0.595***
KFM Impulse (∫N·m/kg/s)	0.20 ± 0.10	0.21 ± 0.12	-0.007	0.033	0.834	0.001	-0.073	0.059
Max vGRF (N)	770.41 ± 146.82	827.33 ± 159.22	-56.922	47.740	0.240	0.032	-153.199	39.354
Max vGRF (N/kg)	10.11 ± 1.19	9.83 ± 0.38	0.282	0.317	0.378	0.018	-0.356	0.921
Time to Max vGRF (s)	0.35 ± 0.15	0.41 ± 0.16	-0.056	0.049	0.252	0.030	-0.154	0.042
Loading Rate (N/s)	3124.48 ± 1990.94	2613.34 ± 1479.11	511.135	581.858	0.385	0.018	-662.294	1684.564
Peak Negative Ext. Power (W/kg)	-1.25 ± 0.52	-1.31 ± 0.55	0.068	0.167	0.686	0.004	-0.268	0.404
Peak Knee Flexion (°)	45.11 ± 9.41	45.96 ± 4.40	-0.847	2.571	0.743	0.003	-6.031	4.337
Peak Knee Varus (°)	6.81 ± 7.45	10.18 ± 3.53	-3.373	2.036	0.105	0.060	-7.480	0.733
Peak Knee Varus Velocity (°/s)	88.35 ± 32.57	84.17 ± 39.22	4.181	11.028	0.706	0.003	-18.058	26.421
Stance Time (s)	0.71 ± 0.11	0.75 ± 0.14	-0.038	0.038	0.330	0.022	-0.115	0.040
Walking Velocity (m/s)	0.97 ± 0.20	0.94 ± 0.18	0.030	0.060	0.615	0.006	-0.091	0.152

			Mean				95% CI	
6-months Post-operative	TKA (n=30)	UKA (n=15)	Difference	SE	p	ηp^2	Lower	Upper
Peak KAM (N·m/kg)	0.39 ± 0.12	0.41 ± 0.11	-0.018	0.037	0.624	0.006	-0.093	0.056
Time to Peak KAM (s)	0.22 ± 0.12	0.25 ± 0.09	-0.027	0.034	0.433	0.014	-0.097	0.042
Peak KAM Rate (N·m/kg/s)	2.32 ± 1.37	2.87 ± 0.70	-0.548	0.377	0.153	0.047	-1.308	0.212
KAM Impulse (∫N·m/kg/s)	***0.15 ± 0.05***	***0.19 ± 0.06***	***-0.045****	***0.018***	***0.016****	***0.127***	***-0.082***	***-0.009***
Peak KFM (N·m/kg)	0.67 ± 0.22	0.71 ± 0.10	-0.047	0.059	0.430	0.015	-0.166	0.072
Time to Peak KFM (s)	0.16 ± 0.10	0.14 ± 0.03	0.019	0.027	0.474	0.012	-0.035	0.073
Peak KFM Rate (N·m/kg/s)	***3.89 ± 2.09***	***5.40 ± 1.16***	***-1.511****	***0.581***	***0.013****	***0.136***	***-2.683***	***-0.339***
KFM Impulse (∫N·m/kg/s)	***0.19 ± 0.08***	***0.24 ± 0.07***	***-0.053****	***0.023***	***0.027****	***0.108***	***-0.100***	***-0.006***
Max vGRF (N)	784.39 ± 163.62	855.21 ± 181.98	-70.822	53.700	0.194	0.039	-179.119	37.474
Max vGRF (N/kg)	10.49 ± 1.04	10.01 ± 0.19	0.479	0.272	0.086	0.067	-0.070	1.027
Time to Max vGRF (s)	0.36 ± 0.15	0.41 ± 0.15	-0.054	0.047	0.257	0.030	-0.148	0.041
Loading Rate (N/s)	3456.12 ± 2535.87	2720.92 ± 1076.56	735.198	686.606	0.290	0.026	-649.475	2119.871
Peak Negative Ext. Power (W/kg)	-1.65 ± 0.53	-1.64 ± 0.23	-0.015	0.145	0.918	0.000	-0.308	0.278
Peak Knee Flexion (°)	47.09 ± 8.03	50.10 ± 6.18	-3.004	2.365	0.211	0.036	-7.774	1.766
Peak Knee Varus (°)	0.68 ± 3.83	2.42 ± 3.16	-1.740	1.147	0.137	0.051	-4.054	0.574
Peak Knee Varus Velocity (°/s)	***84.40 ± 28.29***	***64.80 ± 28.06***	***19.606****	***8.922***	***0.033****	***0.101***	***1.613***	***37.598***
Stance Time (s)	0.66 ± 0.05	0.69 ± 0.09	-0.037	0.021	0.081	0.069	-0.078	0.005
Walking Velocity (m/s)	1.08 ± 0.15	1.05 ± 0.07	0.030	0.040	0.452	0.013	-0.050	0.111

			Mean				95% CI	
12-months Post-operative	TKA (n=30)	UKA (n=15)	Difference	SE	p	ηp^2	Lower	Upper
Peak KAM (N·m/kg)	***0.37 ± 0.09***	***0.44 ± 0.13***	***-0.077****	***0.032***	***0.023****	***0.115***	***-0.142***	***-0.011***
Time to Peak KAM (s)	0.26 ± 0.14	0.22 ± 0.09	0.041	0.039	0.300	0.025	-0.038	0.121
Peak KAM Rate (N·m/kg/s)	***1.81 ± 0.84***	***3.30 ± 0.83***	***-1.490*†***	***0.265***	***<0.001*†***	***0.424***	***-2.025***	***-0.956***
KAM Impulse (∫N·m/kg/s)	***0.14 ± 0.05***	***0.19 ± 0.06***	***-0.053****	***0.017***	***0.003****	***0.186***	***-0.087***	***-0.019***
Peak KFM (N·m/kg)	0.70 ± 0.23	0.65 ± 0.19	0.054	0.070	0.446	0.014	-0.087	0.194
Time to Peak KFM (s)	0.15 ± 0.08	0.14 ± 0.03	0.018	0.022	0.417	0.015	-0.027	0.064
Peak KFM Rate (N·m/kg/s)	***3.47 ± 1.97***	***5.19 ± 1.83***	***-1.719****	***0.609***	***0.007****	***0.156***	***-2.947***	***-0.491***
KFM Impulse (∫N·m/kg/s)	0.20 ± 0.07	0.19 ± 0.06	0.009	0.020	0.660	0.005	-0.032	0.050
Max vGRF (N)	790.11 ± 163.80	864.83 ± 191.69	-74.723	54.826	0.180	0.041	-185.290	35.843
Max vGRF (N/kg)	10.38 ± 1.13	10.12 ± 0.35	0.261	0.300	0.390	0.017	-0.345	0.867
Time to Max vGRF (s)	0.37 ± 0.15	0.38 ± 0.16	-0.013	0.048	0.793	0.002	-0.109	0.084
Loading Rate (N/s)	3180.73 ± 2015.04	3313.86 ± 1791.77	-133.129	615.115	0.830	0.001	-1373.627	1107.369
Peak Negative Ext. Power (W/kg)	-1.65 ± 0.53	-1.57 ± 0.29	-0.084	0.148	0.573	0.007	-0.382	0.214
Peak Knee Flexion (°)	***48.03 ± 5.78***	***52.37 ± 6.17***	***-4.344****	***1.867***	***0.025****	***0.112***	***-8.110***	***-0.578***
Peak Knee Varus (°)	***1.12 ± 3.51***	***4.40 ± 3.88***	***-3.275****	***1.150***	***0.007****	***0.159***	***-5.595***	***-0.956***
Peak Knee Varus Velocity (°/s)	86.07 ± 33.22	79.02 ± 30.08	7.049	10.193	0.493	0.011	-13.507	27.604
Stance Time (s)	0.65 ± 0.06	0.68 ± 0.07	-0.025	0.020	0.212	0.036	-0.065	0.015
Walking Velocity (m/s)	1.08 ± 0.15	1.08 ± 0.09	0.004	0.043	0.925	0.000	-0.082	0.090

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; SE=standard error; CI=confidence interval of the difference

ηp^2=partial eta squared effect size

* = Significant between-subjects effect (p<0.05)

† = Significant interaction effect (p<0.05)

Table 4.4 TKA vs UKA Stair Ascent Biomechanics Pairwise Comparisons Across Time

	Implant Group		Mean Difference	SE	p	ηp^2	95% CI	
Pre-operative	TKA (n=21)	UKA (n=14)					Lower	Upper
Peak KAM (N·m/kg)	0.46 ± 0.23	0.57 ± 0.17	-0.105	0.073	0.156	0.060	-0.253	0.042
Time to peak KAM (s)	0.71 ± 0.29	0.75 ± 0.23	-0.037	0.093	0.691	0.005	-0.226	0.151
Peak KAM Rate (N·m/kg/s)	0.75 ± 0.40	0.87 ± 0.30	-0.121	0.127	0.346	0.027	-0.378	0.137
KAM Impulse (∫N·m/kg/s)	0.24 ± 0.22	0.36 ± 0.14	-0.117	0.067	0.089	0.085	-0.252	0.019
Peak KFM (N·m/kg)	***0.59 ± 0.20***	***0.80 ± 0.23***	***-0.204*†***	***0.072***	***0.008*†***	***0.196***	***-0.350***	***-0.058***
Time to peak KFM (s)	0.35 ± 0.27	0.35 ± 0.17	-0.002	0.081	0.980	0.000	-0.166	0.162
Peak KFM Rate (N·m/kg/s)	3.61 ± 5.30	2.86 ± 1.30	0.752	1.450	0.608	0.008	-2.199	3.702
KFM Impulse (∫N·m/kg/s)	0.21 ± 0.18	0.34 ± 0.23	-0.122	0.069	0.084	0.088	-0.262	0.017
Max vGRF (N)	826.76 ± 176.34	876.92 ± 148.12	-50.167	57.206	0.387	0.023	-166.553	66.220
Max vGRF (N/kg)	10.61 ± 0.68	10.39 ± 0.71	0.218	0.239	0.368	0.025	-0.269	0.705
Time to max vGRF (s)	0.34 ± 0.12	0.31 ± 0.18	0.029	0.050	0.575	0.010	-0.074	0.131
Loading Rate (N/s)	4723.10 ± 3802.75	7013.63 ± 4682.52	-2290.527	1439.318	0.121	0.071	-5218.841	637.786
Peak Concentric Knee Ext. Power (W/kg)	***1.45 ± 0.53***	***1.87 ± 0.57***	***-0.425****	***0.188***	***0.031****	***0.134***	***-0.808***	***-0.042***
Peak Knee Flexion (°)	71.01 ± 5.11	68.72 ± 4.83	2.286	1.726	0.194	0.050	-1.226	5.797
Peak Knee Varus (°)	9.86 ± 9.01	11.98 ± 4.21	-2.121	2.586	0.418	0.020	-7.383	3.140
Peak Knee Varus Velocity (°/s)	69.41 ± 26.22	69.55 ± 24.05	-0.140	8.688	0.987	0.000	-17.796	17.515
Stance Time (s)	1.20 ± 0.41	1.24 ± 0.30	-0.041	0.131	0.758	0.003	-0.308	0.226

			Mean Difference	SE	p	ηp^2	95% CI	
6-months Post-operative	TKA (n=21)	UKA (n=14)					Lower	Upper
Peak KAM (N·m/kg)	0.38 ± 0.13	0.34 ± 0.17	0.039	0.051	0.451	0.017	-0.065	0.142
Time to peak KAM (s)	***0.49 ± 0.16***	***0.61 ± 0.14***	***-0.125****	***0.054***	***0.026***	***0.141****	***-0.234***	***-0.016***
Peak KAM Rate (N·m/kg/s)	***0.94 ± 0.49***	***0.62 ± 0.33***	***0.318*†***	***0.149***	***0.041***	***0.121*†***	***0.014***	***0.621***
KAM Impulse (∫N·m/kg/s)	0.17 ± 0.09	0.16 ± 0.12	0.010	0.035	0.772	0.003	-0.060	0.081
Peak KFM (N·m/kg)	0.58 ± 0.18	0.69 ± 0.19	-0.110	0.064	0.093	0.083	-0.240	0.019
Time to peak KFM (s)	***0.21 ± 0.06***	***0.26 ± 0.06***	***-0.050****	***0.020***	***0.018***	***0.157****	***-0.091***	***-0.009***
Peak KFM Rate (N·m/kg/s)	3.23 ± 1.57	2.94 ± 1.02	0.285	0.477	0.555	0.011	-0.685	1.254
KFM Impulse (∫N·m/kg/s)	***0.16 ± 0.11***	***0.24 ± 0.11***	***-0.088****	***0.036***	***0.022***	***0.149****	***-0.162***	***-0.014***
Max vGRF (N)	846.07 ± 194.32	949.45 ± 207.05	-103.384	68.810	0.142	0.064	-243.380	36.612
Max vGRF (N/kg)	11.02 ± 0.52	11.04 ± 0.69	-0.016	0.205	0.937	0.000	-0.433	0.400
Time to max vGRF (s)	0.30 ± 0.10	0.27 ± 0.08	0.031	0.032	0.339	0.028	-0.035	0.097
Loading Rate (N/s)	4279.47 ± 3169.97	6622.37 ± 4745.07	-2342.899	1334.520	0.088	0.085	-5058.000	372.203
Peak Concentric Knee Ext. Power (W/kg)	1.47 ± 0.52	1.70 ± 0.49	-0.227	0.174	0.202	0.049	-0.581	0.127
Peak Knee Flexion (°)	65.95 ± 5.97	70.43 ± 8.09	-4.483	2.376	0.068	0.097	-9.316	0.351
Peak Knee Varus (°)	5.28 ± 4.73	6.03 ± 4.93	-0.753	1.660	0.653	0.006	-4.129	2.624
Peak Knee Varus Velocity (°/s)	66.67 ± 19.10	86.08 ± 46.55	-19.414	11.100	0.089	0.083	-41.971	3.143
Stance Time (s)	0.99 ± 0.17	1.00 ± 0.12	-0.014	0.054	0.802	0.002	-0.125	0.097

			Mean Difference	SE	p	ηp^2	95% CI	
12-months Post-operative	TKA (n=21)	UKA (n=14)					Lower	Upper
Peak KAM (N·m/kg)	0.38 ± 0.14	0.39 ± 0.14	-0.017	0.048	0.729	0.004	-0.114	0.080
Time to peak KAM (s)	0.56 ± 0.21	0.58 ± 0.18	-0.023	0.069	0.744	0.003	-0.162	0.117
Peak KAM Rate (N·m/kg/s)	0.81 ± 0.41	0.75 ± 0.28	0.068	0.126	0.592	0.009	-0.188	0.324
KAM Impulse (∫N·m/kg/s)	0.17 ± 0.10	0.17 ± 0.09	-0.002	0.034	0.961	0.000	-0.071	0.068
Peak KFM (N·m/kg)	0.67 ± 0.21	0.66 ± 0.17	0.005	0.067	0.937	0.000	-0.130	0.141
Time to peak KFM (s)	0.23 ± 0.07	0.22 ± 0.05	0.009	0.022	0.664	0.006	-0.035	0.054
Peak KFM Rate (N·m/kg/s)	3.36 ± 1.64	3.17 ± 1.10	0.190	0.500	0.706	0.004	-0.827	1.208
KFM Impulse (∫N·m/kg/s)	0.20 ± 0.11	0.18 ± 0.09	0.021	0.034	0.547	0.011	-0.049	0.091
Max vGRF (N)	859.92 ± 210.62	958.59 ± 212.06	-98.670	72.866	0.185	0.053	-246.918	49.578
Max vGRF (N/kg)	10.84 ± 0.64	11.14 ± 0.79	-0.297	0.242	0.230	0.043	-0.790	0.197
Time to max vGRF (s)	0.28 ± 0.08	0.23 ± 0.07	0.053	0.027	0.054	0.108	-0.001	0.107
Loading Rate (N/s)	5111.67 ± 4175.50	8091.67 ± 5475.27	-2980.004	1632.129	0.077	0.092	-6300.596	340.588
Peak Concentric Knee Ext. Power (W/kg)	1.70 ± 0.65	1.70 ± 0.48	-0.001	0.204	0.998	0.000	-0.415	0.414
Peak Knee Flexion (°)	67.92 ± 4.93	71.94 ± 6.87	-4.021	1.991	0.052	0.110	-8.071	0.030
Peak Knee Varus (°)	5.75 ± 5.62	8.69 ± 5.99	-2.931	1.991	0.150	0.062	-6.983	1.120
Peak Knee Varus Velocity (°/s)	72.19 ± 35.30	62.57 ± 24.44	9.616	10.800	0.380	0.023	-12.332	31.564
Stance Time (s)	0.97 ± 0.15	0.94 ± 0.10	0.033	0.047	0.486	0.015	-0.062	0.129

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; SE=standard error; CI=confidence interval of the difference

ηp^2=partial eta squared effect size

* = Significant between-subjects effect (p<0.05)

† = Significant interaction effect (p<0.05)

Table 4.5. TKA & UKA Stair Descent Biomechanics Pairwise Comparisons Across Time

	Implant Group		Mean				95% CI	
Pre-operative	TKA (n=20)	UKA (n=11)	Difference	SE	p	ηp^2	Lower	Upper
Peak KAM (N·m/kg)	0.55 ± 0.26	0.74 ± 0.26	-0.193	0.097	0.056	0.120	-0.392	0.005
Time to Peak KAM (s)	0.31 ± 0.17	0.33 ± 0.14	-0.020	0.060	0.742	0.004	-0.143	0.103
Peak KAM Rate (N·m/kg/s)	2.71 ± 1.93	3.39 ± 2.59	-0.686	0.818	0.408	0.024	-2.359	0.987
KAM Impulse (∫N·m/kg/s)	0.36 ± 0.32	0.45 ± 0.12	-0.096	0.100	0.344	0.031	-0.300	0.108
Peak KFM (N·m/kg)	0.86 ± 0.29	1.00 ± 0.35	-0.149	0.119	0.221	0.051	-0.391	0.094
Time to Peak KFM (s)	0.31 ± 0.13	0.32 ± 0.19	-0.014	0.058	0.818	0.002	-0.133	0.106
Peak KFM Rate (N·m/kg/s)	4.01 ± 2.52	4.69 ± 2.79	-0.684	0.983	0.492	0.016	-2.694	1.327
KFM Impulse (∫N·m/kg/s)	0.66 ± 0.28	0.86 ± 0.37	-0.198	0.118	0.104	0.089	-0.440	0.043
Max vGRF (N)	1004.32 ± 290.37	1256.24 ± 438.85	-251.918	130.925	0.064	0.113	-519.690	15.853
Max vGRF (N/kg)	13.03 ± 2.82	14.32 ± 3.65	-1.292	1.174	0.280	0.040	-3.694	1.110
Time to max vGRF (s)	0.14 ± 0.02	0.13 ± 0.03	0.010	0.009	0.296	0.038	-0.009	0.028
Loading Rate (N/s)	***7705.56 ± 2989.07***	***10949.65 ± 5429.88***	***-3244.097****	***1502.473***	***0.039****	***0.138***	***-6316.998***	***-171.195***
Peak Eccentric Knee Ext. Power (W/kg)	***-3.00 ± 1.05***	***-3.75 ± 0.62***	***0.753****	***0.348***	***0.039****	***0.139***	***0.042***	***1.465***
Peak Knee Flexion (°)	30.96 ± 7.60	28.36 ± 10.03	2.602	3.196	0.422	0.022	-3.934	9.138
Peak Knee Varus (°)	10.00 ± 7.78	11.32 ± 3.37	-1.326	2.478	0.597	0.010	-6.394	3.742
Peak Knee Varus Velocity (°/s)	53.43 ± 68.80	37.46 ± 33.56	15.966	22.174	0.477	0.018	-29.386	61.318
Stance Time (s)	1.35 ± 0.73	1.29 ± 0.31	0.061	0.223	0.786	0.003	-0.394	0.517

			Mean				95% CI	
6-months Post-operative	TKA (n=20)	UKA (n=11)	Difference	SE	p	ηp^2	Lower	Upper
Peak KAM (N·m/kg)	0.40 ± 0.17	0.45 ± 0.18	-0.057	0.064	0.381	0.027	-0.189	0.074
Time to Peak KAM (s)	0.24 ± 0.11	0.22 ± 0.06	0.021	0.036	0.562	0.012	-0.053	0.096
Peak KAM Rate (N·m/kg/s)	2.25 ± 1.41	2.33 ± 1.22	-0.082	0.505	0.873	0.001	-1.114	0.951
KAM Impulse (∫N·m/kg/s)	0.18 ± 0.12	0.19 ± 0.13	-0.004	0.046	0.924	0.000	-0.098	0.089
Peak KFM (N·m/kg)	***0.94 ± 0.23***	***1.15 ± 0.23***	***-0.205****	***0.087***	***0.026****	***0.159***	***-0.384***	***-0.026***
Time to Peak KFM (s)	0.27 ± 0.22	0.24 ± 0.08	0.027	0.069	0.703	0.005	-0.115	0.169
Peak KFM Rate (N·m/kg/s)	5.14 ± 2.46	6.00 ± 2.09	-0.855	0.878	0.338	0.032	-2.651	0.941
KFM Impulse (∫N·m/kg/s)	0.63 ± 0.26	0.69 ± 0.09	-0.058	0.081	0.474	0.018	-0.223	0.106
Max vGRF (N)	1155.70 ± 393.70	1377.37 ± 418.48	-221.670	151.059	0.153	0.069	-530.620	87.280
Max vGRF (N/kg)	14.99 ± 2.25	15.38 ± 2.94	-0.394	0.943	0.679	0.006	-2.323	1.534
Time to max vGRF (s)	0.12 ± 0.02	0.13 ± 0.02	-0.008	0.007	0.316	0.035	-0.023	0.008
Loading Rate (N/s)	10319.80 ± 4826.02	11420.66 ± 4480.92	-1100.861	1767.987	0.538	0.013	-4716.801	2515.078
Peak Eccentric Knee Ext. Power (W/kg)	***-3.29 ± 0.57***	***-3.86 ± 0.82***	***0.573****	***0.251***	***0.030****	***0.152***	***0.059***	***1.087***
Peak Knee Flexion (°)	30.80 ± 5.62	34.06 ± 9.22	-3.255	2.656	0.230	0.049	-8.687	2.176
Peak Knee Varus (°)	2.28 ± 3.73	2.79 ± 2.49	-0.509	1.258	0.689	0.006	-3.083	2.064
Peak Knee Varus Velocity (°/s)	25.10 ± 18.39	35.82 ± 27.41	-10.718	8.228	0.203	0.055	-27.547	6.110
Stance Time (s)	1.05 ± 0.60	0.94 ± 0.17	0.115	0.179	0.525	0.014	-0.251	0.481

			Mean				95% CI	
12-months Post-operative	TKA (n=20)	UKA (n=11)	Difference	SE	p	ηp^2	Lower	Upper
Peak KAM (N·m/kg)	***0.37 ± 0.15***	***0.58 ± 0.27***	***-0.216****	***0.075***	***0.008****	***0.221***	***-0.371***	***-0.062***
Time to Peak KAM (s)	0.25 ± 0.15	0.19 ± 0.03	0.052	0.045	0.257	0.044	-0.040	0.144
Peak KAM Rate (N·m/kg/s)	2.09 ± 1.29	3.22 ± 1.94	-1.131	0.580	0.061	0.116	-2.317	0.055
KAM Impulse (∫N·m/kg/s)	0.16 ± 0.12	0.19 ± 0.10	-0.030	0.042	0.484	0.017	-0.116	0.056
Peak KFM (N·m/kg)	1.03 ± 0.24	1.18 ± 0.51	-0.151	0.134	0.269	0.042	-0.424	0.123
Time to Peak KFM (s)	0.24 ± 0.14	0.18 ± 0.05	0.065	0.043	0.138	0.074	-0.022	0.153
Peak KFM Rate (N·m/kg/s)	5.45 ± 2.07	7.51 ± 3.72	-2.057	1.034	0.056	0.120	-4.172	0.059
KFM Impulse (∫N·m/kg/s)	0.62 ± 0.18	0.62 ± 0.22	0.008	0.073	0.911	0.000	-0.140	0.157
Max vGRF (N)	1152.49 ± 429.39	1435.94 ± 387.88	-283.446	155.987	0.080	0.102	-602.475	35.583
Max vGRF (N/kg)	14.39 ± 2.33	15.99 ± 1.85	-1.600	0.818	0.060	0.116	-3.273	0.074
Time to max vGRF (s)	0.12 ± 0.02	0.12 ± 0.02	-0.001	0.007	0.921	0.000	-0.015	0.014
Loading Rate (N/s)	10125.93 ± 5258.94	12309.06 ± 4137.77	-2183.132	1839.876	0.245	0.046	-5946.101	1579.837
Peak Eccentric Knee Ext. Power (W/kg)	-3.84 ± 1.13	-4.35 ± 1.65	0.508	0.501	0.319	0.034	-0.516	1.531
Peak Knee Flexion (°)	32.65 ± 5.11	35.18 ± 8.99	-2.538	2.517	0.322	0.034	-7.685	2.609
Peak Knee Varus (°)	2.11 ± 3.20	3.24 ± 4.32	-1.136	1.360	0.411	0.023	-3.918	1.646
Peak Knee Varus Velocity (°/s)	29.61 ± 26.34	38.35 ± 21.37	-8.745	9.286	0.354	0.030	-27.738	10.248
Stance Time (s)	0.97 ± 0.46	0.83 ± 0.12	0.140	0.137	0.315	0.034	-0.140	0.420

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; SE=standard error; CI=confidence interval of the difference

ηp^2=partial eta squared effect size

* = Significant between-subjects effect (p<0.05)

† = Significant interaction effect (p<0.05)

Table 4.6 Normality Statistics For TKA and UKA Biomechanics During Walking

	TKA						UKA					
	Pre-operative		6-month		12-month		Pre-operative		6-month		12-month	
	W	p	W	p	W	p	W	p	W	p	W	p
Peak KAM (N·m/kg)	0.950	0.212	0.930	0.068	0.967	0.513	0.909	0.128	0.933	0.301	0.927	0.243
Time to Peak KAM (s)	***0.919***	***0.038****	***0.822***	***<0.001****	***0.881***	***0.005****	0.943	0.425	***0.816***	***0.006****	***0.798***	***0.003****
Peak KAM Rate (N·m/kg/s)	***0.909***	***0.021****	***0.891***	***0.008****	0.951	0.229	0.925	0.230	0.924	0.220	0.973	0.906
KAM Impulse (∫N·m/kg/s)	0.951	0.221	0.979	0.832	0.978	0.812	0.942	0.412	***0.860***	***0.024****	0.912	0.147
Peak KFM (N·m/kg)	0.979	0.838	0.966	0.505	0.972	0.645	0.948	0.490	0.914	0.155	0.924	0.218
Time to Peak KFM (s)	***0.762***	***<0.001****	***0.537***	***<0.001****	0.981	0.880	***0.840***	***0.012****	***0.853***	***0.019****	***0.855***	***0.020****
Peak KFM Rate (N·m/kg/s)	***0.849***	***0.001****	0.940	0.122	***0.918***	***0.035****	0.946	0.461	0.979	0.959	0.904	0.108
KFM Impulse (∫N·m/kg/s)	0.951	0.226	0.964	0.456	0.950	0.217	0.982	0.980	0.953	0.581	0.968	0.824
Max vGRF (N)	0.980	0.853	0.979	0.847	0.970	0.603	0.944	0.429	0.954	0.583	0.971	0.868
Max vGRF (N/kg)	***0.684***	***<0.001****	***0.707***	***<0.001****	***0.631***	***0.000***	0.966	0.795	0.953	0.570	0.960	0.693
Time to Max vGRF (s)	0.955	0.285	0.926	0.055	***0.886***	***0.007****	***0.859***	***0.023****	0.933	0.300	0.916	0.167
Loading Rate (N/s)	***0.796***	***<0.001****	***0.786***	***<0.001****	***0.809***	***0.002****	***0.733***	***<0.001****	0.895	0.080	***0.831***	***0.001****
Peak Negative Ext. Power (W/kg)	0.985	0.956	0.972	0.658	0.966	0.503	***0.878***	***0.044****	0.978	0.955	0.899	0.093
Peak Knee Flexion (°)	***0.850***	***0.001****	0.964	0.464	0.962	0.419	0.965	0.779	0.959	0.677	***0.877***	***0.042****
Peak Knee Varus (°)	0.957	0.315	0.964	0.462	0.968	0.542	0.969	0.837	0.943	0.426	***0.863***	***0.027****
Peak Knee Varus Velocity (°/s)	0.936	0.097	0.943	0.148	0.958	0.328	0.931	0.282	0.888	0.062	0.927	0.245
Stance Time (s)	0.937	0.101	***0.916***	***0.032****	0.928	0.063	0.944	0.435	***0.835***	***0.011****	***0.864***	***0.027****
Walking Velocity (m/s)	0.952	0.240	0.959	0.344	0.957	0.633	0.914	0.158	0.985	0.949	0.937	0.348

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; KAM=knee adduction moment;KFM=knee flexion moment; vGRF=vertical ground reaction force
W=Shapiro-Wilk statistic; p=p-value
* = Significant Shapiro-Wilk Test(p<0.05)

Table 4.7 Normality Statistics For TKA and UKA Biomechanics During Stair Ascent

	TKA						UKA					
	Pre-operative		6-month		12-month		Pre-operative		6-month		12-month	
	W	p	W	p	W	p	W	p	W	p	W	p
Peak KAM (N·m/kg)	0.969	0.711	0.964	0.598	0.943	0.246	0.924	0.280	0.890	0.099	***0.854***	***0.032****
Time to Peak KAM (s)	***0.862***	***0.007****	0.948	0.312	0.945	0.277	0.958	0.729	0.899	0.130	0.874	0.059
Peak KAM Rate (N·m/kg/s)	0.964	0.593	0.930	0.138	0.942	0.235	0.924	0.288	0.926	0.298	0.924	0.282
KAM Impulse (∫N·m/kg/s)	0.971	0.761	0.952	0.375	0.920	0.086	0.937	0.413	0.870	0.052	0.936	0.411
Peak KFM (N·m/kg)	0.977	0.870	0.987	0.987	0.952	0.374	0.914	0.208	0.948	0.573	0.943	0.495
Time to Peak KFM (s)	***0.797***	***<0.001****	0.961	0.531	0.955	0.421	0.871	0.055	0.906	0.161	***0.858***	***0.036****
Peak KFM Rate (N·m/kg/s)	***0.464***	***<0.001****	***0.897***	***0.031****	0.932	0.154	0.957	0.709	0.951	0.611	0.965	0.833
KFM Impulse (∫N·m/kg/s)	0.936	0.181	0.980	0.921	0.974	0.817	0.948	0.572	0.928	0.318	0.968	0.869
Max vGRF (N)	0.954	0.406	0.970	0.730	0.964	0.608	0.973	0.931	0.924	0.280	0.955	0.675
Max vGRF (N/kg)	0.940	0.218	0.968	0.688	0.943	0.250	0.929	0.332	0.926	0.298	0.958	0.722
Time to Max vGRF (s)	***0.891***	***0.024****	0.946	0.290	***0.890***	***0.022****	***0.803***	***0.007****	0.968	0.865	0.974	0.940
Loading Rate (N/s)	***0.759***	***<0.001****	***0.781***	***<0.001****	***0.751***	***<0.001****	0.935	0.401	***0.829***	***0.015****	0.896	0.117
Peak Concentric Ext. Power (W/kg)	0.928	0.124	0.965	0.613	0.913	0.063	0.949	0.578	0.962	0.788	0.958	0.718
Peak Knee Flexion (°)	0.968	0.685	0.926	0.115	0.974	0.826	0.891	0.099	0.921	0.262	0.906	0.161
Peak Knee Varus (°)	0.925	0.110	0.950	0.335	***0.901***	***0.037****	0.970	0.898	0.928	0.319	0.939	0.444
Peak Knee Varus Velocity (°/s)	***0.877***	***0.013****	0.939	0.208	***0.780***	***<0.001****	0.934	0.389	0.871	0.055	***0.849***	***0.028****
Stance Time (s)	***0.773***	***<0.001****	***0.824***	***0.002****	0.918	0.080	***0.717***	***<0.001****	***0.846***	***0.025****	0.940	0.452

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; KAM=knee adduction moment;KFM=knee flexion moment; vGRF=vertical ground reaction force
W=Shapiro-Wilk statistic; p=p-value
* = Significant Shapiro-Wilk Test(p<0.05)

Table 4.8 Normality Statistics For TKA and UKA Biomechanics During Stair Descent

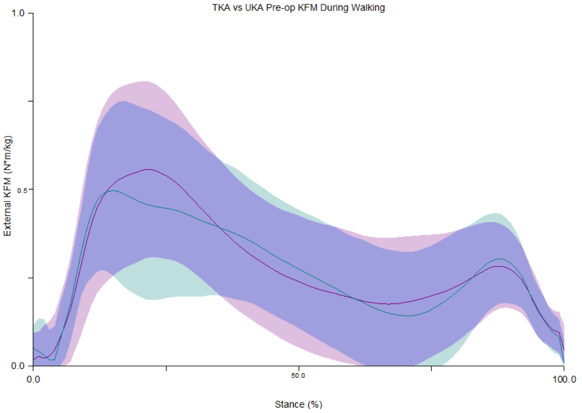
	TKA						UKA					
	Pre-operative		6-month		12-month		Pre-operative		6-month		12-month	
	W	p	W	p	W	p	W	p	W	p	W	p
Peak KAM (N·m/kg)	0.984	0.977	0.974	0.831	0.965	0.648	0.872	0.083	0.945	0.583	***0.785***	***0.006****
Time to Peak KAM (s)	***0.880***	***0.018****	***0.797***	***<0.001****	***0.714***	***<0.001****	0.911	0.248	***0.836***	***0.028****	0.932	0.427
Peak KAM Rate (N·m/kg/s)	0.905	0.051	***0.876***	***0.015****	0.931	0.164	***0.814***	***0.014****	0.851	0.044	***0.723***	***0.001****
KAM Impulse (∫N·m/kg/s)	***0.844***	***0.004****	0.968	0.721	***0.867***	***0.010****	0.969	0.874	0.950	0.647	0.907	0.225
Peak KFM (N·m/kg)	0.974	0.831	0.984	0.978	0.965	0.654	***0.812***	***0.014****	0.939	0.509	***0.746***	***0.002****
Time to Peak KFM (s)	0.950	0.365	***0.554***	***<0.001****	***0.726***	***<0.001****	0.875	0.091	0.919	0.311	***0.740***	***0.002****
Peak KFM Rate (N·m/kg/s)	0.914	0.077	0.941	0.248	0.941	0.250	0.928	0.389	0.921	0.326	0.856	0.051
KFM Impulse (∫N·m/kg/s)	0.954	0.430	***0.876***	***0.015***	0.970	0.760	0.954	0.700	0.983	0.980	***0.817***	***0.016****
Max vGRF (N)	0.949	0.352	***0.888***	***0.025****	***0.844***	***0.004****	0.865	0.067	0.965	0.832	0.948	0.616
Max vGRF (N/kg)	0.948	0.343	0.973	0.820	0.956	0.458	0.871	0.081	0.898	0.175	0.926	0.376
Time to Max vGRF (s)	0.964	0.624	0.971	0.774	0.956	0.459	0.947	0.605	0.946	0.599	0.973	0.918
Loading Rate (N/s)	0.965	0.646	***0.885***	***0.022****	***0.830***	***0.002****	0.864	0.065	0.901	0.191	0.956	0.726
Peak Eccentric Ext. Power (W/kg)	0.874	0.014	0.943	0.276	0.915	0.079	0.869	0.076	0.967	0.859	***0.726***	***0.001****
Peak Knee Flexion (°)	0.964	0.631	0.971	0.784	0.958	0.508	0.947	0.609	0.968	0.867	0.947	0.611
Peak Knee Varus (°)	0.926	0.128	0.935	0.190	0.955	0.452	0.916	0.289	0.937	0.482	***0.845***	***0.037****
Peak Knee Varus Velocity (°/s)	***0.550***	***<0.001****	***0.815***	***<0.001****	***0.893***	***0.030****	0.922	0.332	0.922	0.332	0.857	0.052
Stance Time (s)	***0.688***	***<0.001****	***0.572***	***<0.001****	***0.641***	***<0.001****	0.931	0.425	0.943	0.558	0.919	0.311

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; KAM=knee adduction moment;KFM=knee flexion moment; vGRF=vertical ground reaction force
W=Shapiro-Wilk statistic; p=p-value
* = Significant Shapiro-Wilk Test(p<0.05)

4.6 Figures: Walking Biomechanics

Figures 4.1-4.3. TKA (Purple) vs UKA (cyan) mean (line) and standard deviation (shade) for external KFM during walking at 4.1) pre-operative, 4.2) 6-months post-operative, 4.3) 12-months post-operative.

4.1)



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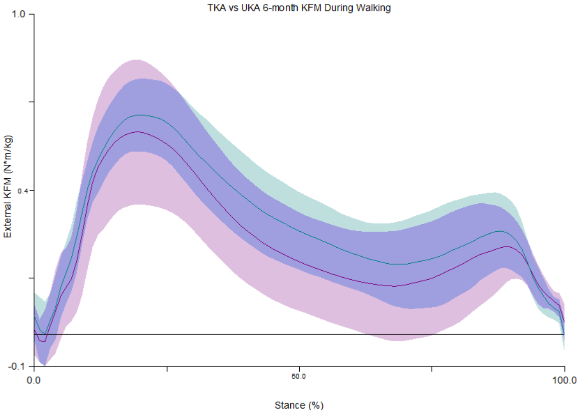

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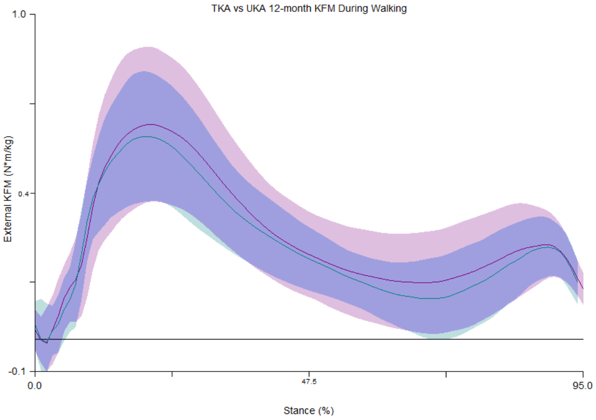
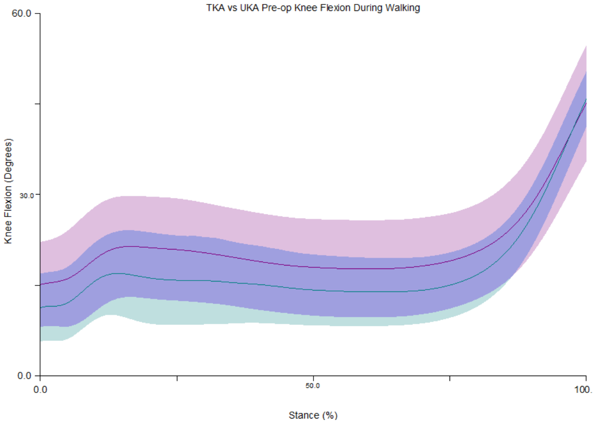


Figures 4.4-4.6. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for knee flexion during walking at 4.4) pre-operative, 4.5) 6-months post-operative, 4.6) 12-months post-operative.

4.4)



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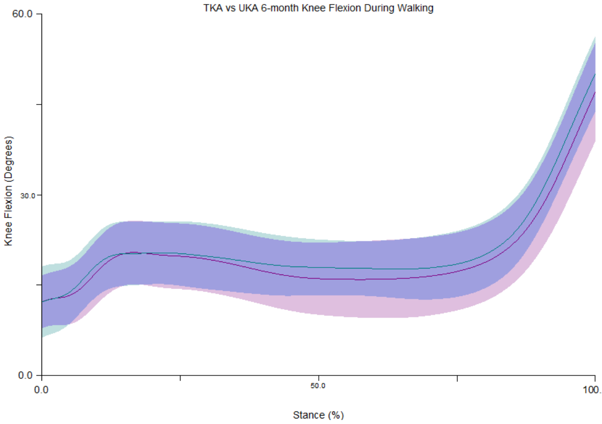
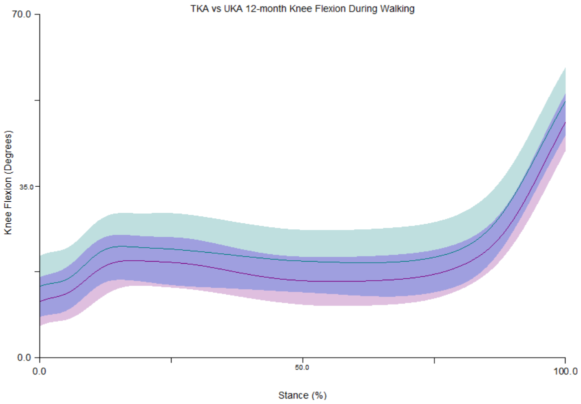
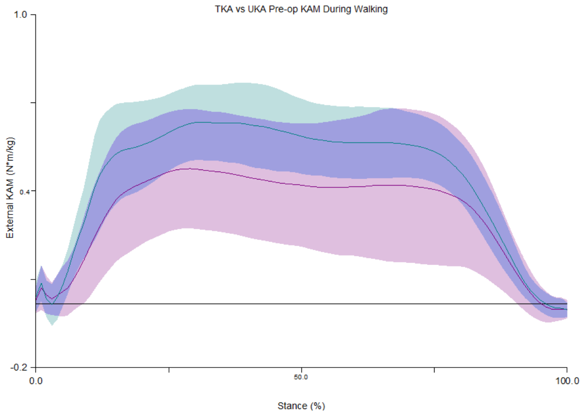


4.6)



Figures 4.7-4.9. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for external KAM during walking at 4.7) pre-operative, 4.8) 6-months post-operative, 4.9) 12-months post-operative.

4.7)



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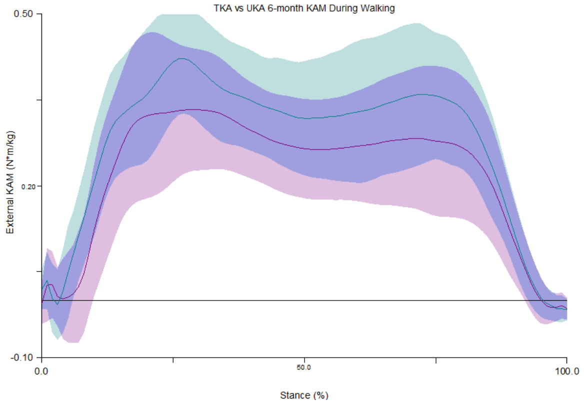
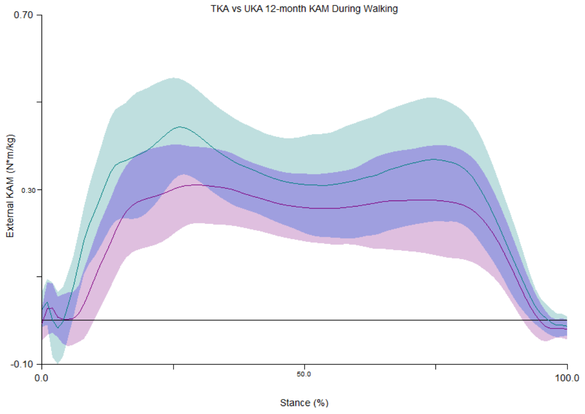
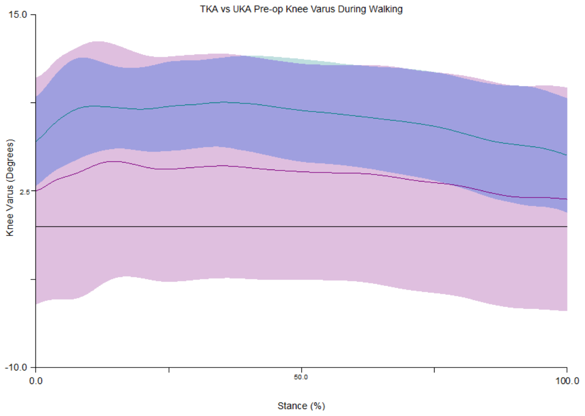


4.9)



Figures 4.10-4.12. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for knee varus angle during walking at 4.10) pre-operative, 4.11) 6-months post-operative, 4.12) 12-months post-operative.

4.10)



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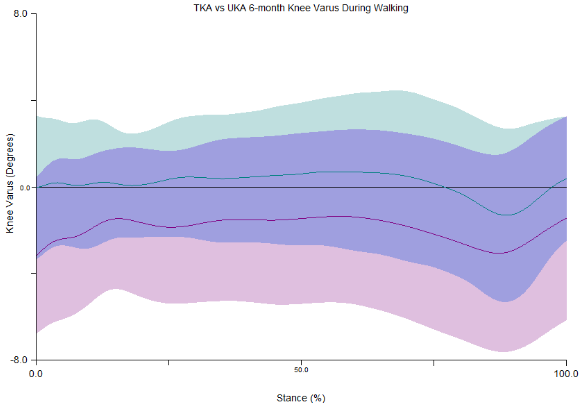

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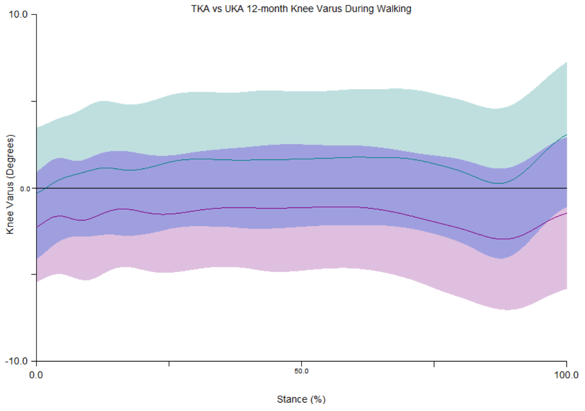
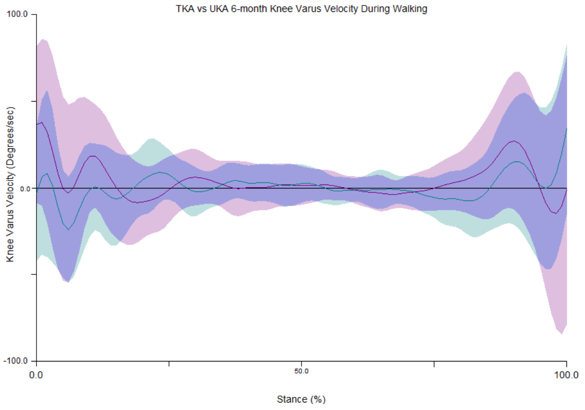


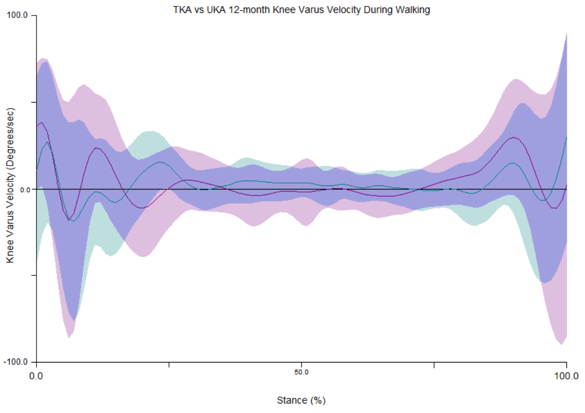
Figures 4.13-4.15. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for knee varus velocity during walking at 4.13) pre-operative, 4.14) 6-months post-operative, 4.15) 12-months post-operative.

4.13)

4.14)

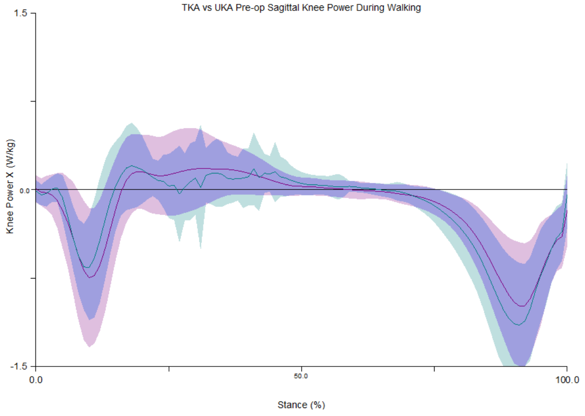


4.15)



Figures 4.16-4.18. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for sagittal knee power during walking at 4.16) pre-operative, 4.17) 6-months post-operative, 4.18) 12-months post-operative.

4.16)



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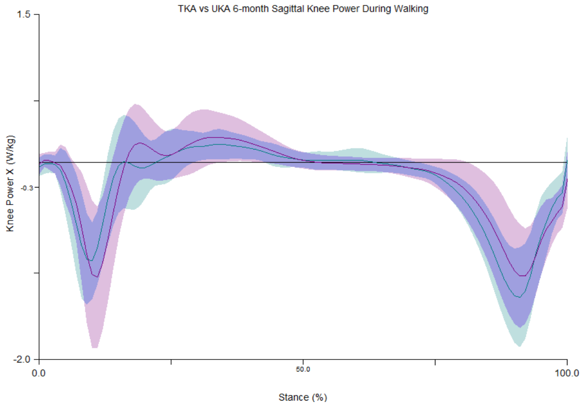
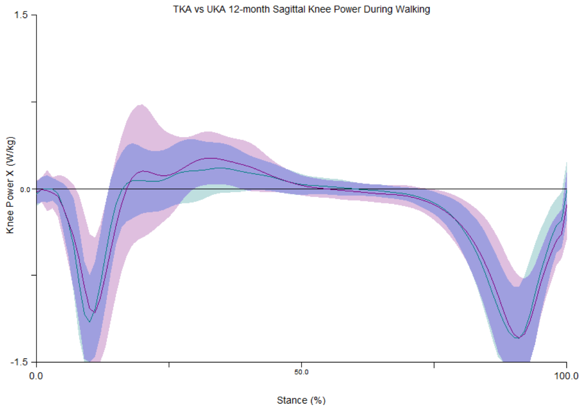
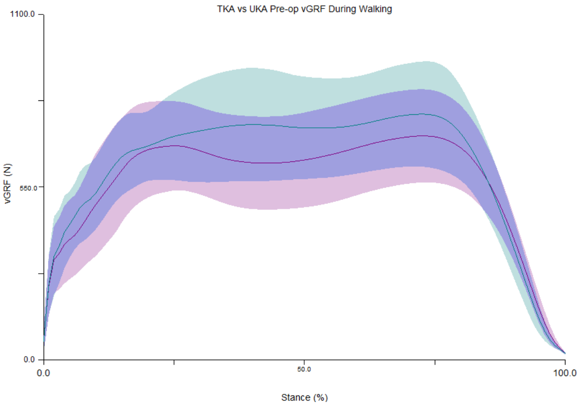


4.18)



Figures 4.19-4.21. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for raw vGRF during walking at 4.19) pre-operative, 4.20) 6-months post-operative, 4.21) 12-months post-operative.

4.19)



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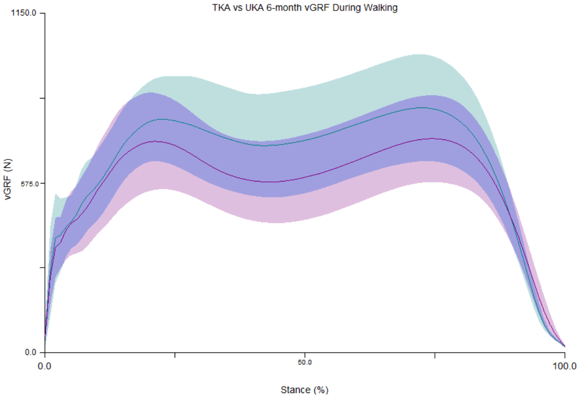

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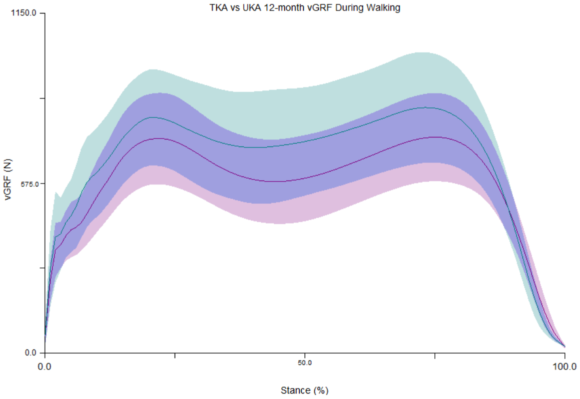
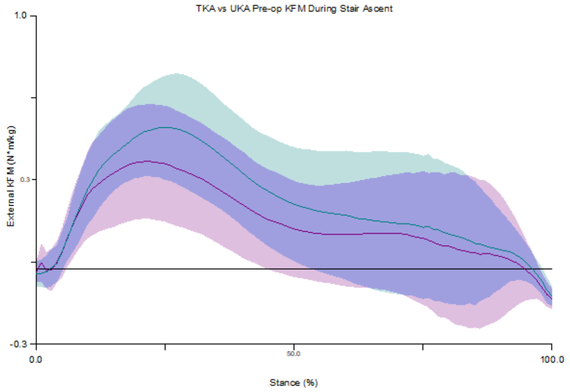


4.7 Figures: Ascent Biomechanics

Figures 4.22-4.24. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for external KFM during stair ascent at 4.22) pre-operative, 4.23) 6-months post-operative, 4.24) 12-months post-operative.

4.22)



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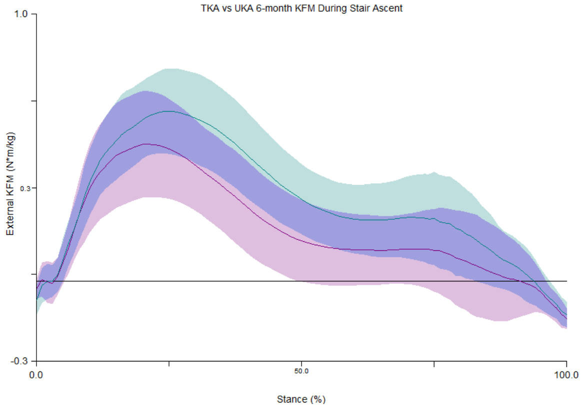

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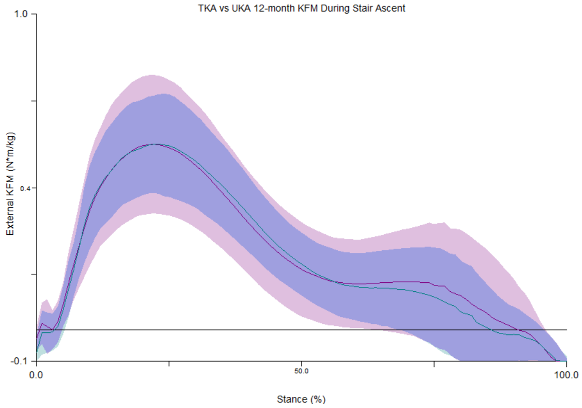
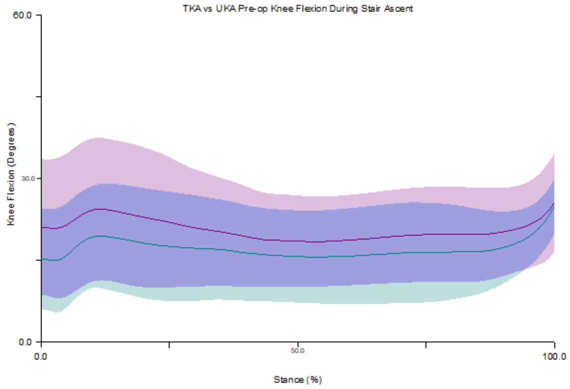


Figures 4.24-4.27. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for knee flexion angle during stair ascent at 4.25) pre-operative, 4.26) 6-months post-operative, 4.27) 12-months post-operative.

4.25)



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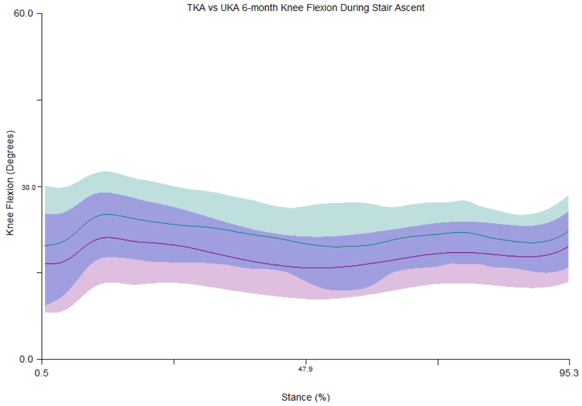

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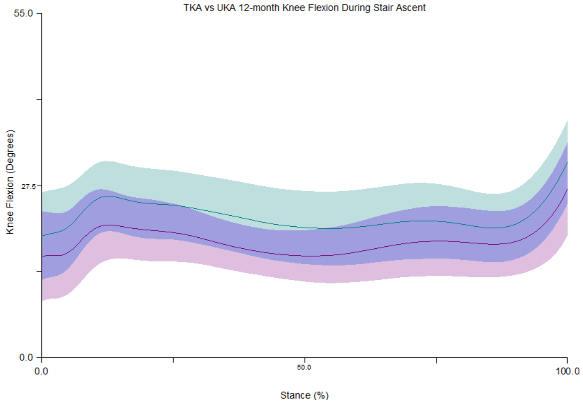
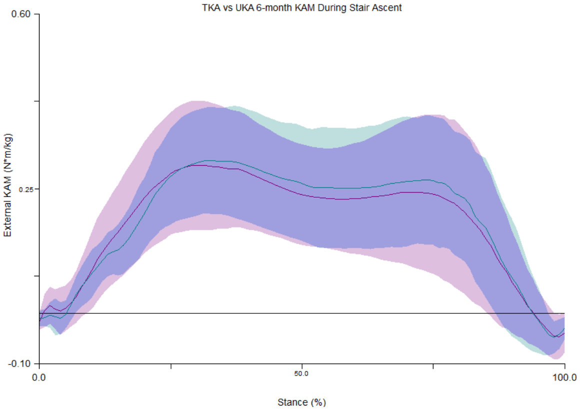


Figures 4.28-4.30. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for external KAM during stair ascent at 4.28) pre-operative, 4.29) 6-months post-operative, 4.30) 12-months post-operative.

4.28)

4.29)



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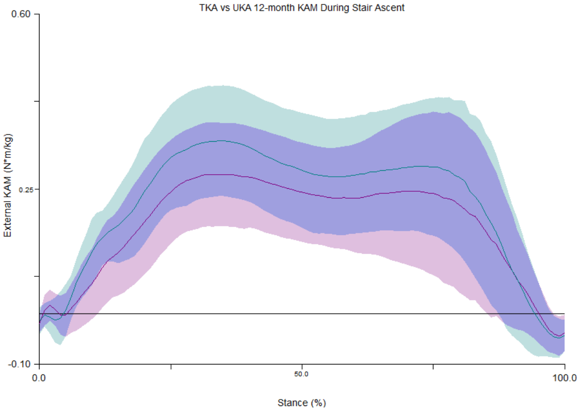
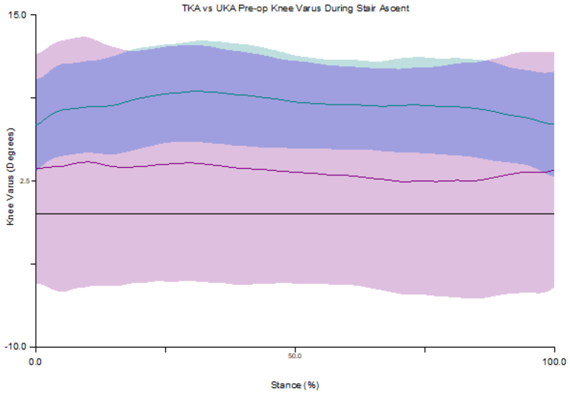


Figures 4.31-4.33. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for knee varus angle during stair ascent at 4.31) pre-operative, 4.32) 6-months post-operative, 4.33) 12-months post-operative.

4.31)



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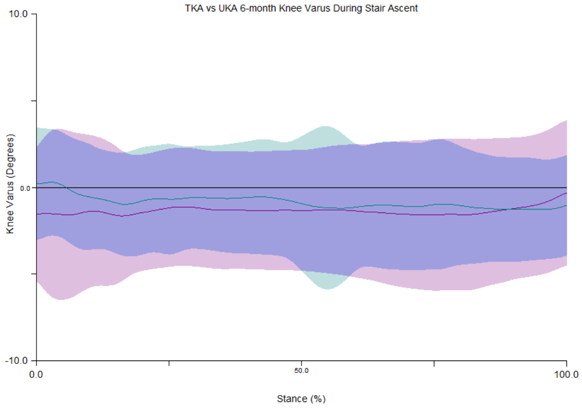

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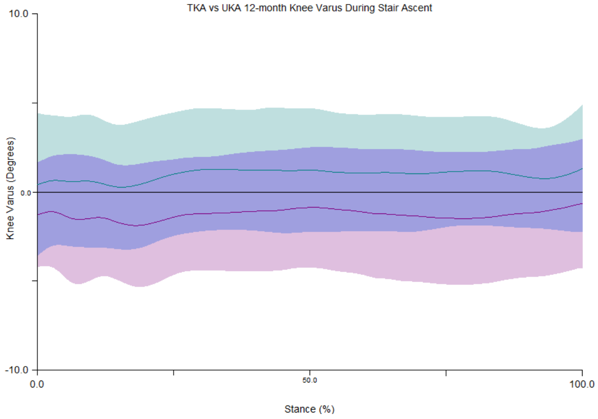
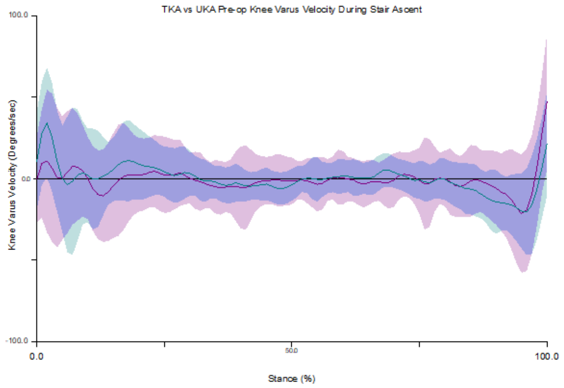


Figures 4.34-4.36. TKA (purple) vs UKA (cyan) mean (line) and standard deviation (shade) for knee varus velocity during stair ascent at 4.34) pre-operative, 4.35) 6-months post-operative, 4.36) 12-months post-operative.

4.34)



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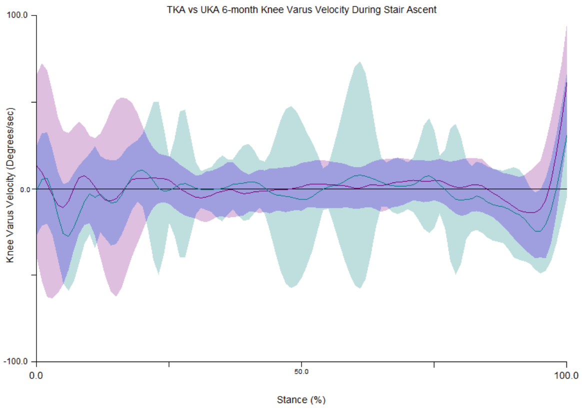

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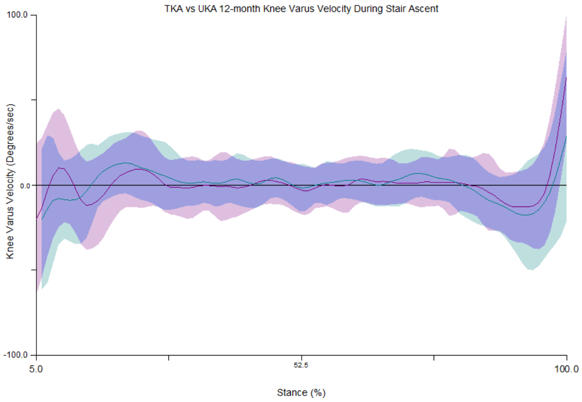

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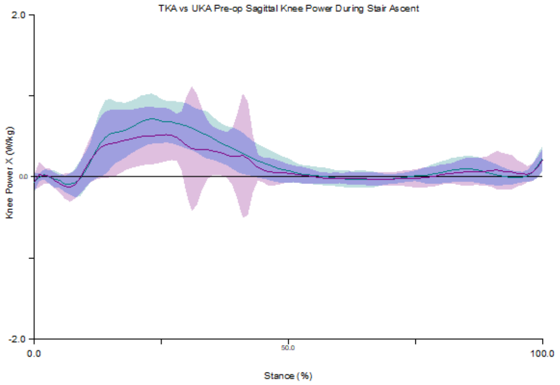

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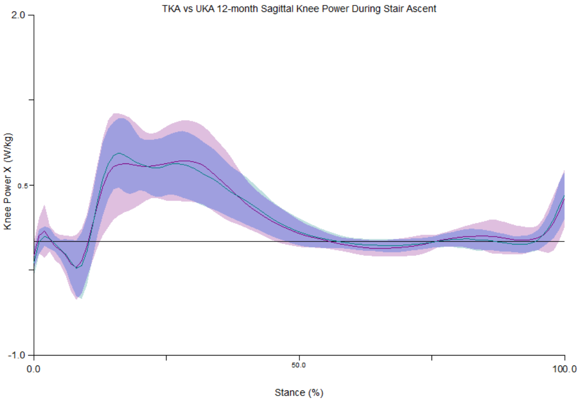

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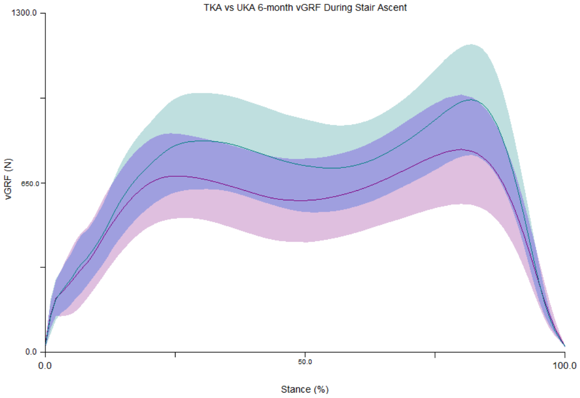

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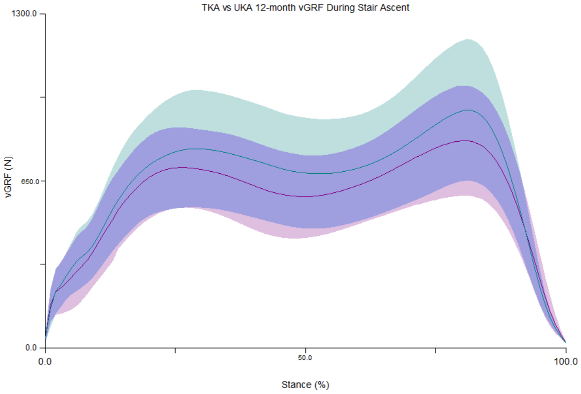

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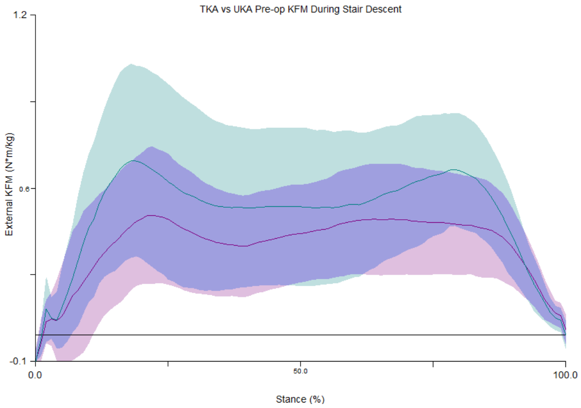

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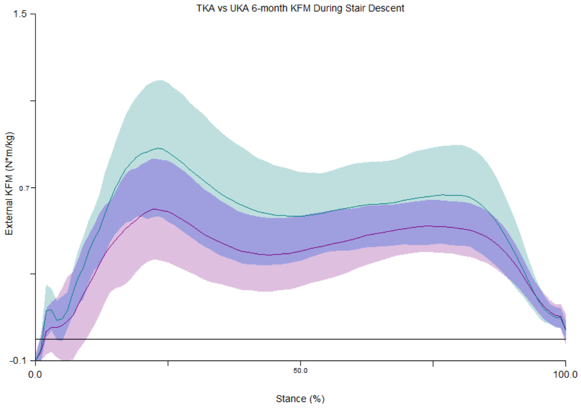

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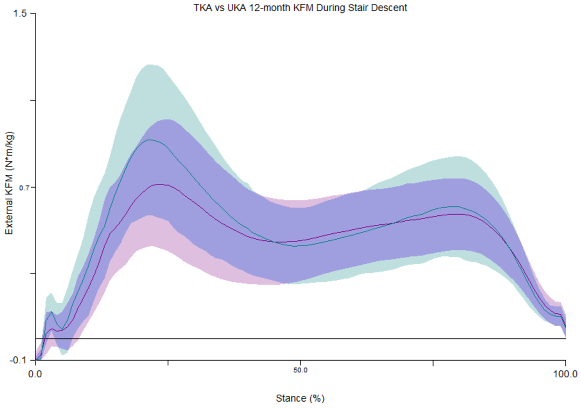

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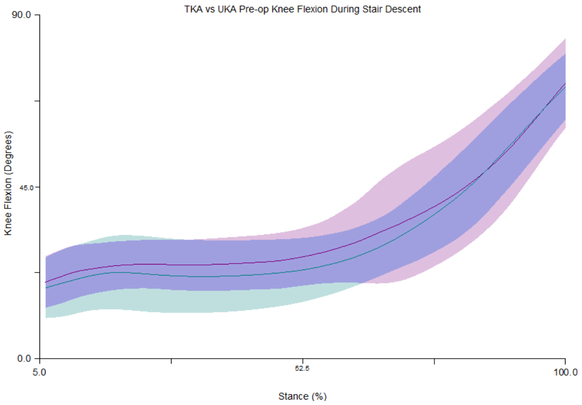

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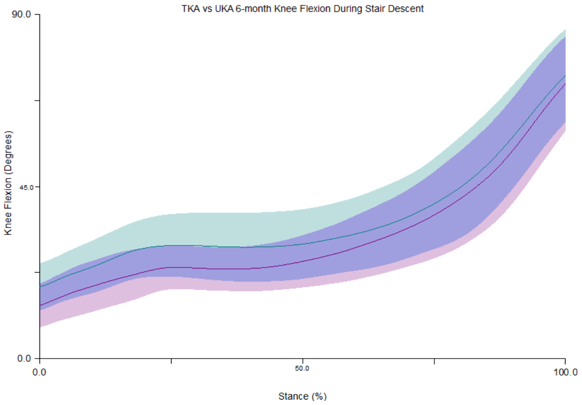

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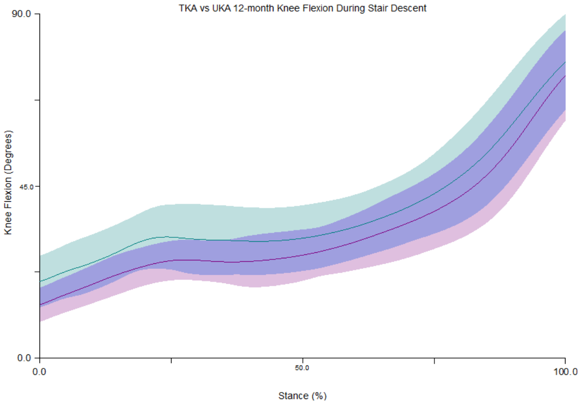

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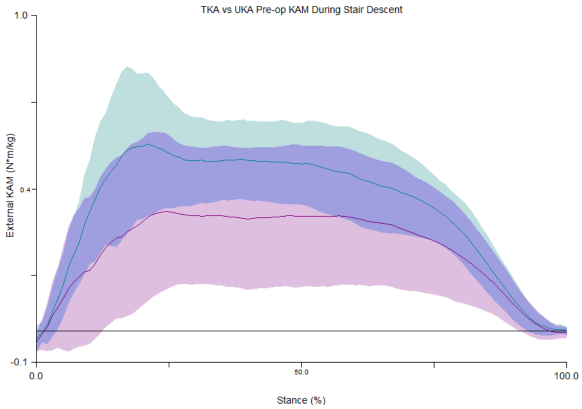

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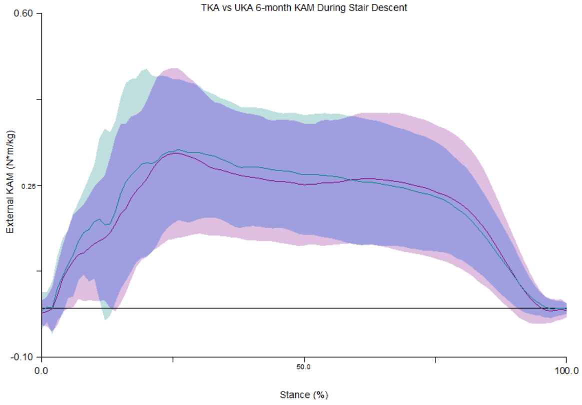

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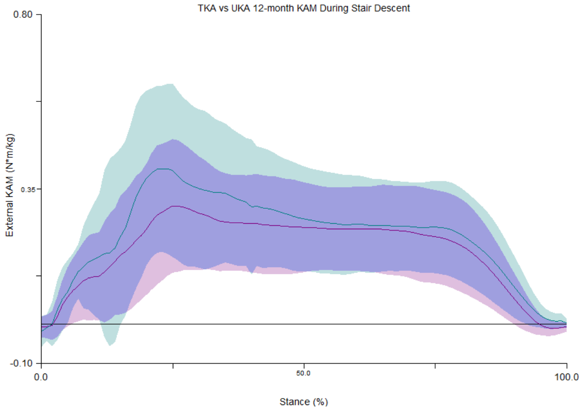

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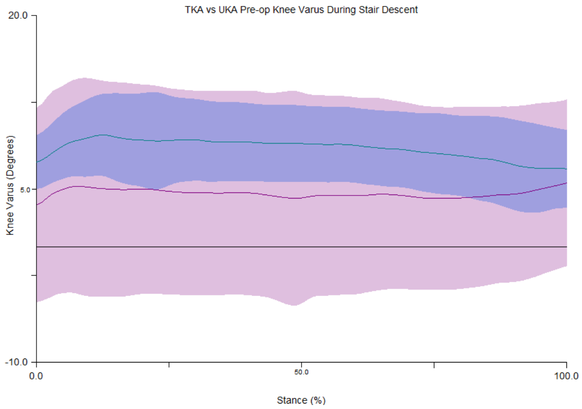

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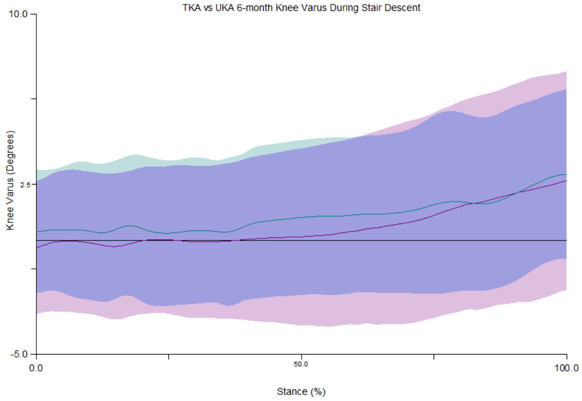

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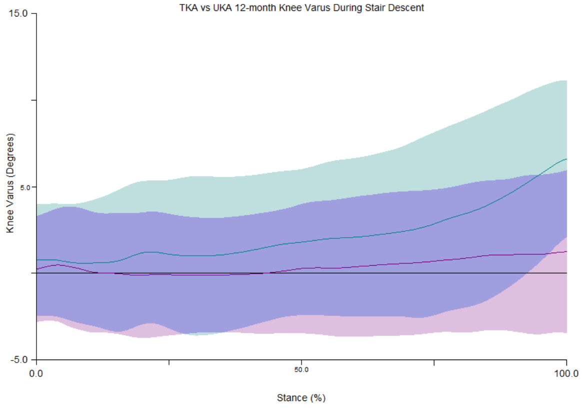

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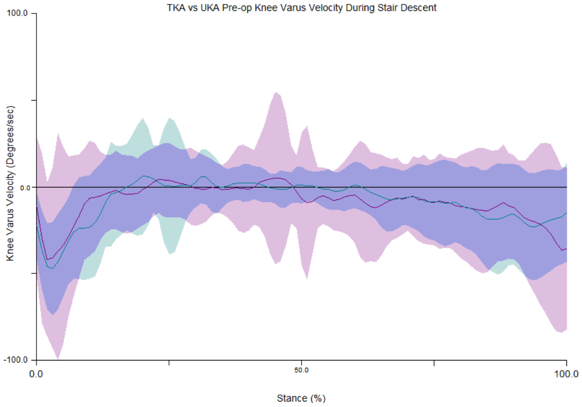

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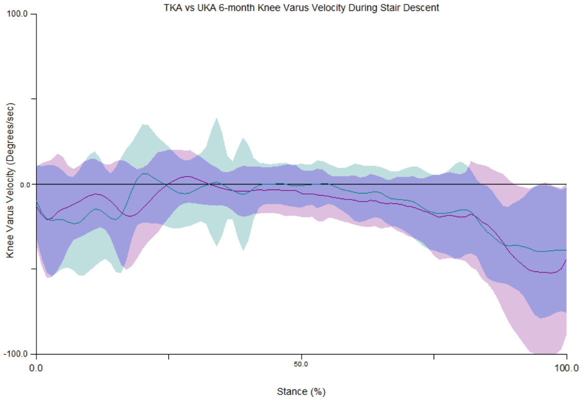

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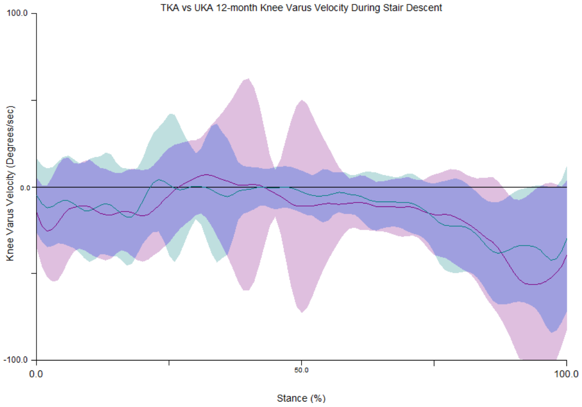

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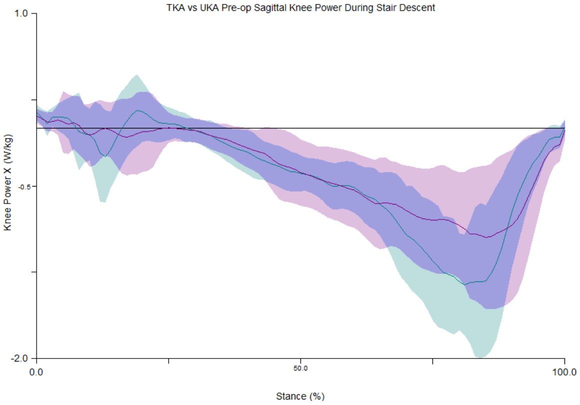

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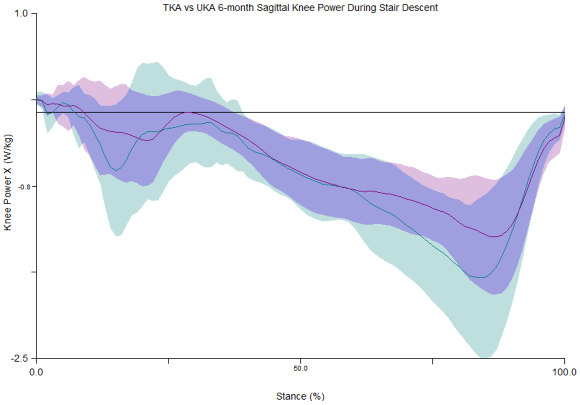

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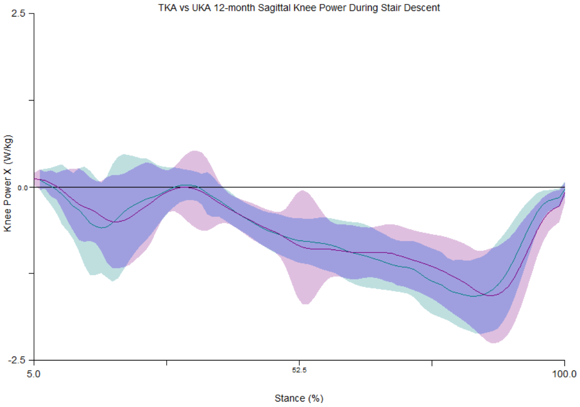

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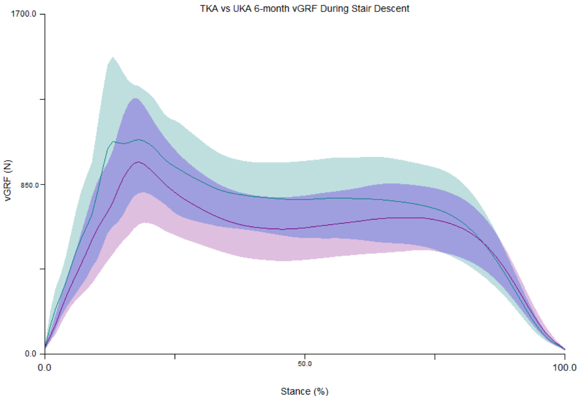

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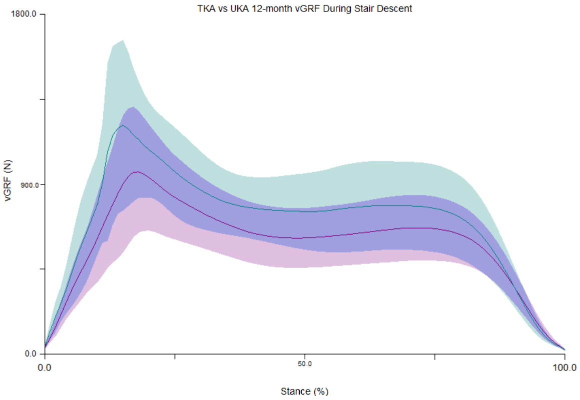


CHAPTER 5:

KNEE EXTENSOR STRENGTH AND SAGITTAL PLANE BIOMECHANICS OF MULTI-RADIUS AND SINGLE-RADIUS TOTAL KNEE ARTHROPLASTY PATIENTS

Abstract

Objective: The purpose of this study was to determine differences of isometric quadriceps strength, knee sagittal plane biomechanics, and clinical cores between total knee arthroplasty (TKA) patients receiving either single-radius (SR) or multi-radius (MR) femoral component implants during level walking and stair negotiation. ***Methods:*** Ten patients (14 knees) undergoing SR-TKA (GetAroundKnee™, Stryker Orthopedics, Mahwah, NJ) and 13 patients (16 knees) undergoing MR-TKA (Balanced Knee® System, Ortho Development Corporation, Draper, UT) were included in this study. Knee extensor strength was evaluated using a handheld dynamometer (HHD). Biomechanics during level walking and stair negotiation were examined using an 18-camera 3D motion capture system (Vicon). Data were analyzed pre-operatively as well as 6-months and 12-months post-operatively. Repeated-measures Analysis of Variance (RM-ANOVA) was conducted to determine between-subjects and within-subject effects over time between MR and SR groups during walking and stair negotiation. ***Results:*** Both groups improved post-operatively for sagittal plane biomechanical measures. No interaction main effects were detected between implant groups over time. ***Conclusions:*** The results of this study do not support the theoretical advantage that has been suggested for SR implants. Neither implant demonstrated superiority in restoring strength or neuromuscular control assessed via 3D motion capture and sagittal plane biomechanics.

5. Introduction

Many patients undergoing total knee arthroplasty (TKA) for the treatment of severe knee osteoarthritis (OA) receive excellent clinical results[77-79]; however, studies have reported post-operative instability relating to revision rates[106] and weakness of the quadriceps femoris remaining after TKA[95, 107]. Quadriceps strength is related to extensor mechanism function, and muscle recovery of the mechanism after TKA plays an important role in obtaining good clinical outcomes and restoring activities of daily living (ADL)[108, 109]. Therefore, maintaining stability and the function of the extensor mechanism is important for facilitating adequate neuromuscular control following TKA. These outcomes may be influenced by the implant design, given the wide variety of available designs and their various theoretical advantages.

Multi-radius (MR) and single-radius (SR) femoral components are commonly used during TKA. Multi-radius designs are intended to reproduce the migrating knee joint axis of rotation, which shifts from a flexion axis to an extension axis at approximately 30° of knee flexion[110]. Compared to MR implants, SR implants have a more posterior and stationary axis of rotation, which contributes to increase the moment arm of the patellar tendon. Studies have reported that SR patients require less quadriceps muscle force to obtain an equivalent knee extension moment in MR patients.[111, 112] Regarding instability after TKA, previous research has referred to the feeling of instability, specifically between 30° and 45° of knee flexion, which causes dissatisfaction in patients with an MR knee implant.[113] Biomechanical confirmation of sagittal plane instability between MR and SR implants is limited and a multivariate approach is necessary to thoroughly evaluate quadriceps performance and neuromuscular control during both simple and complex ADL's such as walking and stair negotiation.

Relatively few studies have compared strength and biomechanical analysis of MR and SR femoral designs,[114, 115] with studies focusing on clinical outcome scores comparing each implant design.[82, 116] External knee flexion moment (KFM) can be used to biomechanically assess quadriceps muscle activity during gait.[117] Lower KFM during gait may suggest decreased power absorption and torque production, while lower rates of KFM development has been identified as a primary contributor to quadriceps avoidance gait patterns.[59] The total external KFM moment exposure, or impulse, during gait may also provide biomechanical evaluation of load attenuation throughout stance, given the theoretical advantage of SR implant designs, which increases joint surface contact area and consistent ligament tension throughout a range of motion.[118] Despite this theoretical advantage, and excellent clinical outcomes between each implant, it is still debatable whether SR implants improve the effectiveness of the extensor mechanism, thereby improving extensor strength and sagittal plane neuromuscular control compared to MR implants. Therefore, the purpose of this study was to examine clinical scores, quadriceps strength, and sagittal plane knee biomechanics during level walking between patients receiving either MR or SR TKA designs pre-operatively and 6-months and 12-months post-operatively.

5.1 Methods

5.1.1 Subjects & Surgical Procedure

Ten patients (14 knees) undergoing SR-TKA (GetAroundKnee™, Stryker Orthopedics, Mahwah, NJ) and 13 patients (16 knees) undergoing MR-TKA (Balanced Knee® System, Ortho Development Corporation, Draper, UT) were included in this study. Both implants are posterior stabilized design. All patients received patellar resurfacing. A single, board certified surgeon performed all surgeries under general anesthesia using a medial parapatellar approach in all patients. The preoperative diagnosis of all patients was osteoarthritis. Therefore, if OA was present in the contralateral limb in these patients, it had not yet progressed to cause pain or functional deficiencies. Patients were included if: 1) under 75 years of age at time of surgery, 2) no previous history of lower extremity fracture, osteotomy, or joint replacement, 3) undergoing a unilateral or simultaneous bilateral TKA for the treatment of OA, and 4) able to walk without an aid. Prior to enrollment in the study, all patients signed informed consent and the study was approved by the Institution's Committee on Human Studies.

5.1.2 Strength Testing, Biomechanical Analysis, and Clinical Scores

All strength and biomechanical analyses were conducted at the University Gait Laboratory. Walking gait biomechanics were collected at a self-selected pace using a 29-retroreflective array-based marker set (Appendix A). Landmarks were located on the thorax (including the xiphoid process, jugular notch, C7 spinous process, T10 spinous process, apex of the inferior angle of the scapula and bilateral acromioclavicular joints), the pelvis (including bilateral anterior superior iliac spine and posterior superior iliac spine), and lower extremities (including bilateral head of the 1st metatarsal, head of the 2nd metatarsal, head of the 5th metatarsal, base of the 5th metatarsal, posterior calcaneus, medial malleoli, lateral malleoli, medial femoral epicondyles, lateral femoral epicondyles). Additionally, four 4-marker arrays were secured bilaterally on the mid-thigh and mid-shank segments. The medial epicondyle, medial malleolus, and head of the 1st metatarsal were used for static calibration only and removed for movement trials. Kinematic data were collected with a Vicon motion capture system and Vicon Nexus software (Nexus 2.5, Vicon, Inc., Centennial, CO) at 240 Hz and time synchronized with kinetic data. Kinetic data from the operated limb were collected at 960 Hz from a force plate (Advanced Mechanical Technology Incorporated, Boston, MA) embedded flush with the floor. All kinematic data were smoothed using a low-pass Butterworth filter using a 10 Hz cut-off frequency and ground reaction forces (GRF) were filtered using a 50 Hz cut-off frequency. Joint moments were calculated using inverse dynamics based on marker trajectories and filtered with a 10 Hz cut-off frequency.[27] Three successful trials for walking were collected, indicated by placement of the entire foot on the force plate without a visible change in gait in an attempt to target the force plate. Subjects performed the minimum number of trials necessary to obtain acceptable trials for the involved limb undergoing TKA. All data was processed using Visual 3D (C-Motion, Inc., Germantown, MD) and moments are expressed as external.

Following movement trials, strength tests were conducted using a MicroFET2 hand-held dynamometer (Hoggan Health Industries, West Jordan, UT) and performed in a gravity dependent position. Knee extensor strength was assessed while the patient was seated with the knee placed at 60°of knee flexion and their trunk in a recumbent position. The hand-held dynamometer was placed on the anterior shank, at a point 80% of the distance from the lateral knee joint line to the lateral malleolus and secured with a strap. The dynamometer was fixed to the lower tibia to increase the validity and reliability of the measure[91, 92] and is a recommended alternative technique to the isokinetic dynamometer for evaluating knee strength.[92] Subjects were instructed to build force over three seconds, holding the maximal contraction for two seconds. Two trials of maximal effort were completed. A third trial was completed if the second trial did not measure within 10% force output of the first trial. All successful trials were averaged for each participant on the involved limb and calculated as Newton-meters per kilogram (N·m/kg). Verbal encouragement was provided to help elicit maximal force production by the participant during strength testing. Clinical scores were evaluated using the UCLA Activity Questionnaire, which is a predictor of routine post-TKA activity.[93] Data were analyzed preoperatively within two weeks pre-operatively, and 6-months and 12-months post-operatively.

5.1.3 Statistical Analysis

Descriptive statistics are reported for patient demographics. Independent samples t-tests were conducted between MR and SR groups pre-operatively to determine differences in group characteristics. Levene's Test was used to test for homogeneity of variance between groups for baseline demographics. Repeated-measures Analysis of Variance (RM-ANOVA) was conducted between MR and SR groups over time (pre-operative, 6-months, and 12-months post-operative) during walking and stair negotiation. Strength, UCLA scores, and biomechanical variables at each time point were tested for sphericity and homogeneity of variance using Mauchly's Test of Sphericity and Levene's Test, respectively.

Univariate main effects were interpreted with Greenhouse-Geisser corrections when sphericity or homogeneity of variance assumptions were violated. Bonferroni corrections were used to adjust for multiple comparisons in post-hoc pairwise analyses. Additionally, Pearson's Chi-Square was conducted to determine the proportions of males and females, as well as the number of bilateral cases in each group at baseline. Effect sizes were calculated using partial eta squared (ηp^2) and reported as small (0.02), medium (0.13), and large (0.26).[31, 94] All statistical analyses were performed using SPSS version 25.0 (IBM, Armonk, NY, USA). A priori power analysis (G*Power 3.1.9.2) for RM-ANOVA between two groups across three measurement intervals indicated a total sample size requirement of 26 to achieve power of 0.80 with a large effect (ηp^2) of 0.26. Alpha level was set a priori to $p<0.05$ for all analyses.

5.2 Results

5.2.1 Demographics, Clinical Scores, and Isometric Quadriceps Strength

Baseline demographics including age, gender, body mass, height, and body mass index (BMI) are reported in Table 5.4. No significant differences of baseline demographics were observed between MR and SR groups. The proportion of males to females in each group was not statistically significantly different ($\chi^2(1, 23) = 0.434$, $p=0.510$), as was the number of bilateral cases in each group ($\chi^2(1, 23) = 0.765$, $p=0.382$). The UCLA Activity Scores for subjects at each time point are presented in Table 5.2. Sphericity and homogeneity of variance were assumed for UCLA Activity Scores. There were no significant differences of UCLA Activity Scores between implant groups at any time period; however, a significant within-subject main effect was observed for time ($F(2, 20) = 13.387$, $p<0.001$, $\eta p^2=0.572$). No significant interaction between implant groups and time was observed. Subjects demonstrated significantly higher UCLA Activity scores 6-months (5.78±1.28, p=0.001) and 12-months (6.48±1.56, p=0.001) post-operatively compared to pre-operatively (4.74±1.39).

Knee extensor strength violated the sphericity assumption; therefore, Huynh-Feldt corrections were interpreted for within-subject univariate effects. A significant univariate effect for time was observed for knee extensor strength ($F(1.51, 42.34) = 9.21$, $p=0.001$, $\eta p^2=0.248$). Descriptive statistics of knee extensor strength over time are presented in Table 5.2. No between-subject differences were observed between implant groups at any time point. Over time, subjects demonstrated significantly greater strength 12-months postoperatively (1.16 ± 0.34 N·m/kg) compared to pre-operatively (0.92 ± 0.37 N·m/kg, p=0.002) and compared to 6-months post-operatively (1.07 ± 0.33 N·m/kg, p=0.018).

5.2.2 Sagittal Plane Walking Biomechanics

The RM-ANOVA for walking included 16 knees in 13 patients in the MR group and 14 knees in 10 patients in the SR group. Descriptive statistics at each time point biomechanical variables are reported in Table 5.3. Several variables violated the assumption of normality and are reported in Table 5.6. Sphericity was assumed for all variables with exception of peak KAM rate (W=0.691, p=0.007), KFM impulse (W=0.642, p=0.003), stance time (W=0.311, p<0.001), and walking velocity (W=0.543, p<0.001). Equal variances were assumed between groups at each time interval for all variables except for walking velocity pre-operatively (W=4.297, p=0.047) and 12-months post-operatively (W=4.448, p=0.044).

Significant univariate effects for time (in all subjects) were detected for peak KFM (F (2, 56)=4.795, p=0.012, ηp^2=0.146), and peak KFM Rate (F (1.528, 42.791)=3.912, p=0.038, ηp^2=0.123), max vGRF (F (2, 56)=3.833, p=0.028, ηp^2=0.120), max vGRF/kg (F (2, 56)=8.208, p=0.001, ηp^2=0.227), peak eccentric knee extensor power (F (2, 56)=12.956, p<0.001, ηp^2=0.316), walking velocity (F (1.373, 38.445)=18.279, p<0.001, ηp^2=0.395), and stance time (F (1.184, 33.156)=7.640, p=0.007, ηp^2=0.214). No significant between-subjects interaction effects were detected. Pairwise comparisons between subjects at each time interval are presented in Table 5.3. For time to max vGRF, MR subjects were significantly greater 6-months post-operatively (mean difference: 0.117 s, SE:0.050, p=0.020, ηp^2=0.165, 95% CI: 0.015, 0.219) but comparable to SR subjects pre-operatively and 12-months post-operatively. For stance time, MR subjects were significantly greater 12-months post-operatively (mean difference: 0.046 s, SE: 0.021, p=0.035, ηp^2=0.149, 95% CI: 0.003, 0.088).

5.2.3 Sagittal Plane Stair Ascent Biomechanics

The RM-ANOVA for stair ascent included 11 knees in the MR group and 10 knees in the SR group. Several subjects were unable to negotiate stairs at one or more data collection periods and were removed from the RM-ANOVA: Six TKA subjects were unable to ascend stairs pre-operatively, one unable at 6-months post-operatively (9 knees total). Several variables violated the normality assumption and are reported in Table 5.7. Sphericity was assumed for all variables with exception of time to peak KFM (W=0.082, $p<0.001$), KFM impulse (W=0.483, p=0.001), peak KFM rate (W=0.075, $p<0.001$), max vGRF (W=0.613, p=0.012), and stance time (W=0.166, $p<0.001$). Equality of error variances between groups was assumed for all variables at each time interval with exception of stance time at 6-months post-operatively (W=28.964, $p<0.001$).

Significant univariate within-subjects effects (in all subjects) were detected for time to peak KFM (F (2, 38)=6.795, p=0.016, $\eta p^2=0.263$), max vGRF (F (1.442, 27.390)=4.258, p=0.036, $\eta p^2=0.183$), max vGRF/kg (F (2, 38)=4.618, p=0.016, $\eta p^2=0.196$), time max vGRF (F (2, 38)=5.253, p=0.010, $\eta p^2=0.217$), peak knee flexion (F (2, 38)=6.309, p=0.004, $\eta p^2=0.249$), peak concentric knee extensor power (F (2, 38)=3.529, p=0.039, $\eta p^2=0.150$), and stance time (F (1.091, 20.723)=9.902, p=0.004, $\eta p^2=0.343$). No significant univariate interactions were detected; pairwise comparisons between groups at each time interval are presented in Table 5.4. For time to peak KFM, MR subjects were significantly lower than SR subjects 6-months post-operatively (mean difference: 0.067 s, SE: 0.022, p=0.008, $\eta p^2=0.318$, 95% CI: 0.020, 0.114) but comparable pre-operatively and 12-months post-operatively. No other significant differences were observed.

5.2.4 Sagittal Plane Stair Descent Biomechanics

The RM-ANOVA for stair descent included 10 knees in both the MR and SR groups. Several subjects were unable to negotiate stairs at one or more data collection periods and were removed from the RM-ANOVA: six TKA subjects were unable to descend stairs pre-operatively, one unable at 6-months post-operatively, and one unable at 12-months post-operatively (10 knees total). Several variables violated the normality assumption and are reported in Table 5.8. Sphericity was assumed for all variables with exception of time to peak KFM (W=0.309, $p<0.001$), max vGRF (W=0.291, $p<0.001$), max vGRF/kg (W=0.429, $p<0.001$), time to max vGRF (W=0.330, $p<0.001$), loading rate (W=0.470, p=0.002), and stance time (W=0.330, $p<0.001$). Equality of error variances was assumed for all variables at each time interval, with exception of 6-month KFM impulse (W=17.517, $p<0.001$), and stance time at 6-months (W=9.632, p=0.006) and 12-months (W=7.213, p=0.015).

A significant univariate effect for time (in all subjects) was detected for peak KFM (F (2, 36)=3.450; p=0.043, ηp^2=0.161), max vGRF (1.170,)=5.775, p=0.021, ηp^2=0.243), max vGRF/kg (F (1.273,)=5.661; p=0.020, ηp^2=0.239), time to max vGRF (F (1.197,)=5.460; p=0.024, ηp^2=0.233), loading rate (F (1.307,)=6.952; p=0.010, ηp^2=0.279), peak eccentric knee extensor power (F (2, 36)=5.571; p=0.008, ηp^2=0.236), and stance time (F (1.198,)=9.332, p=0.004, ηp^2=0.341). No significant univariate interaction effects were detected. Pairwise comparisons between groups at each time interval are presented in Table 5.5. For loading rate, SR subjects were significantly greater pre-operatively (mean difference: -2697.763 N/s, SE: 1217.313, p=0.040, ηp^2=0.214, 95% CI: -5255.242, -140.283) and 6-months post-operatively (mean difference: -4259.917 N/s, SE: 1977.048, p=0.045, ηp^2=0.205, 95% CI: -8413.540, -106.293) but comparable 12-months post-operatively. No other significant differences were observed.

5.3 Discussion

The most important finding of this study was that SR implants did not show the proposed theoretical advantage compared to MR implants. In this study, there were no significant differences in muscle strength, sagittal plane biomechanics, and clinical scores between SR and MR posterior stabilized implants. As expected, within-subjects changes over time were detected following surgery; however, the implant design did not have any detected effects on neuromuscular control during walking, stair ascent, or stair descent.

5.3.1 Clinical Scores and Knee Extensor Strength

Satisfaction of TKA is well documented, with good to excellent patient-reported results.[79, 119, 120] A meta-analysis of outcomes compared 948 SR-TKA with 1361 MR-TKA and reported there were no differences in clinical scores, clinical outcomes, complications, quadriceps strength, and survival rate[121] Survival rates have also been reported to be high as long as 5+ years post-TKA.[120] Based on previous findings, no differences between groups were expected for clinical scores, which was demonstrated in the results. Patients in the current study exceeded pre-operative UCLA activity scores, which is not surprising since pain is a primary contributor to lower activity levels prior to surgery.

Conflicting evidence regarding knee extensor strength and recovery post-TKA has been reported; however, based on previous findings, no differences in knee extensor strength were expected. One study reported SR-TKA patients had better quadriceps recovery and clinical outcomes in short term follow-up using a dynamometer;[122] however, the study had some significant limitations, such as a difference between follow-up periods of the SR and MR groups and the lack of pre-operative values. A study utilizing electromyography reported SR-TKA patients had more efficient quadriceps activation during both sit-to-stand and stand-to-sit movements.[123, 124] In contrast, a study assessing the ability to rise from a chair without assistance reported no difference between SR and MR-TKA patients.[125] Studies utilizing a dynamometer also reported there were no differences between MR and SR groups for quadriceps recovery and clinical results throughout the follow-up period; however, not all patients received patellar resurfacing.[115] Some studies reported quadriceps strength after TKA remained weaker than or hardly recovered to pre-operative levels,[95, 96, 107] while more recent evidence suggests that strength exceeds pre-operative levels.[97-99] The results of the current study agree with more recently published literature that suggests TKA patients exceed pre-operative strength at 6-months and 12-months post-operatively.

5.3.2 Sagittal Plane Walking Biomechanics

In the current sample, few differences were observed between MR and SR implants at any time point. External KFM is an important indicator of the forces acting on the knee joint, with a larger KFM demonstrating an increase in joint loading and a willingness to load the knee.[126] Subjects demonstrated linear increases in knee extensor strength and external KFM; however, no interaction was observed between implant design and time. This suggests that while subjects increased their willingness to load and demonstrated muscle strength improvements following TKA, the implant design did not appear to influence these gains. Given that no differences were detected between implants for peak KFM, peak KFM rate, and KFM impulse, these results suggest that neither implant is superior in influencing the extensor mechanism during walking. While peak KFM and KFM impulse may indicate quadriceps activity, willingness to load the knee, and load attenuation, greater KFM rate of development may indicate greater quadriceps activation and reduce quadriceps avoidance gait.[59] Greater rate of quadriceps torque development has also been previously associated with faster walking speed.[101]

As subjects showed similar walking velocities post-operatively, and similar rates of KFM development, it does not appear that the implant design had an influence on quadriceps performance during walking.

The SR subjects did however demonstrate significantly faster time to reach their max vGRF at the 6-month time point, which may explain the small-medium (but non-significant) effect observed for loading rate. Although these differences were not apparent at 12-months post-surgery, this result may show evidence of earlier willingness to load in SR patients during walking. Despite that difference, peak loading did not significantly differ between groups. Additionally, SR subjects showed significantly shorter stance time at 12-months post-operatively; however, a difference of 0.04s may not be clinically relevant, given that the total exposure to external KFM did not differ at the same time interval.

5.3.3 Stair Negotiation Sagittal Plane Biomechanics

The results of the stair ascent analysis are less clear than during walking. The only observed significant difference between groups was at 6-months post-surgery, with time to peak KFM. The MR group was slower, which may have contributed to the small effect in peak KFM rate. Previous evidence indicates that greater co-contraction of the quadriceps and hamstring musculature is required during stair ascent,[127] which may attenuate the rate of external KFM development. Subjects demonstrated linear increases in peak KFM across time (Figures 5.1-5.3), indicating that quadriceps activity and willingness to load consistently increased after surgery, which agrees with the linear increases in strength that patients exhibited. In this analysis, the implant design did not have a detectable influence on sagittal plane biomechanics that may explain quadriceps control. The SR group demonstrated a small-medium but non-significant effect for peak knee flexion 6-months post-operatively, potentially indicating greater available range of motion during early stance (Figure 5.5). It is feasible that subjects had not fully regained functional range of motion to climb stairs, which is common following TKA procedures. [102] This small effect suggested an approximate 3° greater peak knee flexion in the SR group; therefore, the effect may not be clinically relevant.

During stair descent, few differences were observed between groups. Lower sample sizes during stair descent may speak to the difficulty of the task and the eccentric quadriceps control that is required to descend stairs smoothly and efficiently.[46] Given the inability of several patients to descend stairs without assistance, this may have been a confounding factor leading to an underpowered analysis. Still, external KFM showed a similar trend of increasing linearly following surgery (Figures 5.25-5.27). This indicates that all subjects, regardless of implant design, demonstrated greater willingness to load the limb and greater eccentric quadriceps activity during stair descent. This coincided with a medium effect of eccentric knee extensor power at the 6-month time period, with the SR patients demonstrating greater power absorption (Figure 5.20). In conjunction with similarities in peak knee flexion angles across all time points, this may indicate greater quadriceps function in the SR group up to 6-months post-surgery. This effect, however, was not observed at 12-months post-TKA. Differences in the rate of peak KFM development trended towards significance across time points, although pairwise comparison did not reveal any differences when adjusting for multiple comparisons. It should be noted that there was as a medium effect noted for max vGRF, coupled with significant differences in max vGRF loading rate both pre-operatively and 6-months post-operatively. The SR group was greater for both of these variables, which may indicate a greater baseline willingness to load; however, this effect was not present when vGRF was scaled to body mass. Considering these results, it is difficult to ascertain any meaningful differences between MR and SR implant designs during stair descent. With greater standard errors and divergences from normality during stair descent, the variability in how patients navigate stairs may be a confounding factor.

5.3.4 Limitations

Several limitations of this study should be acknowledged. First, because some patients were not available for follow-up, or were unable to ascend or descend stairs without the use of the handrail, the smaller sample sizes included for stair negotiation may have impacted the results. Second, although all subjects underwent similar rehabilitation protocols following surgery, each protocol was individualized and unstandardized, based on patient-oriented goals. It is unknown how post-operative rehabilitation played a role in muscle strength development over the course of recovery. Third, a single board-certified surgeon performed all surgeries, therefore expertise level in conducting TKA was not a factor and the results may not be generalizable to different surgical approaches. Lastly, patients undergoing unilateral and bilateral surgery were included. Although the proportion of bilateral cases was not different between the two groups, it may be prudent to evaluate bilateral cases separately from unilateral cases due to potential compensatory gait patterns observed in each condition.

5.4 Conclusion

In this study, there were no significant differences between muscle strength recovery and clinical scores between SR and MR posterior-stabilized implants. Both implant groups demonstrated comparable increases in muscle strength and exceeded pre-operative strength by 6-months and 12-months post-operatively. Sagittal plane biomechanics did not indicate superior stability or neuromuscular control by subjects receiving either MR or SR implant designs during the tasks of walking, stair ascent and stair descent.

5.5 List of Tables

Table 5.1. MR vs SR Patient Demographics at Baseline

	MR (n=13)	SR (n=10)	p
Age (y)	68.85 ± 3.13	66.60 ± 4.12	0.151
Height (m)	1.61 ± 0.07	1.65 ± 0.12	0.386
Mass (kg)	76.93 ± 17.13	82.90 ± 17.39	0.419
Body Mass Index (BMI)	29.57 ± 6.27	30.34 ± 3.98	0.738
Chi-Square Test	MR (n=13)	SR (n=10)	χ2(1,23)
Males	6	6	0.434, p=0.510
Females	7	4	
Bilateral Cases	3	4	0.765, p=0.382
Unilateral Cases	10	6	

y=years; m=meters; kg=kilograms; MR=Multi-radius; SR=Single-radius
p=p-value (α=0.05); χ2=Pearson's Chi-Square (p<0.05)

Table 5.2. MR vs SR Knee Extensor Strength and UCLA Activity Questionnaire Score Pairwise Comparisons Across Time

	MR (n=13 patients)	SR (n=10 patients)	Mean Difference	SE	p	np^2	95% CI	
Pre-operative	(n=16 knees)	(n=14 knees)					Lower	Upper
Strength (N·m/kg)	0.93 ± 0.37	0.91 ± 0.38	0.021	0.138	0.879	0.001	-0.261	0.303
UCLA	4.55 ± 1.37	4.00 ± 0.67	0.545	0.478	0.268	0.064	-0.455	1.546
6-months Post-operative								
Strength (N·m/kg)	1.10 ± 0.39	1.03 ± 0.24	0.070	0.121	0.566	0.012	-0.177	0.318
UCLA (different n)	5.91 ± 1.30	6.00 ± 1.15	-0.091	0.539	0.868	0.001	-1.219	1.037
12-months Post-operative								
Strength (N·m/kg)	1.15 ± 0.43	1.17 ± 0.21	-0.019	0.127	0.885	0.001	-0.280	0.243
UCLA (different n)	6.91 ± 1.51	6.20 ± 1.55	0.709	0.669	0.302	0.056	-0.691	2.109

N·m=Newton-meter; kg=kilograms; SE=standard error; p=p-value (α=0.05); CI=confidence interval of the difference
np^2=partial eta squared effect size

Table 5.3. MR vs SR Walking Biomechanics Pairwise Comparisons Across Time

	Implant Group						95% CI	
Pre-operative	MR (n=16 knees)	SR (n=14 knees)	Mean Difference	SE	p	ηp^2	Lower	Upper
Peak KFM (N·m/kg)	0.62 ± 0.30	0.59 ± 0.17	0.038	0.090	0.679	0.006	-0.147	0.222
Time to peak KFM	0.19 ± 0.09	0.18 ± 0.11	0.011	0.038	0.779	0.003	-0.067	0.088
PKFM Rate (N·m/kg/s)	2.90 ± 2.59	3.03 ± 1.69	-0.129	0.810	0.875	0.001	-1.788	1.531
KFM Impulse (∫N·m/kg/s)	0.21 ± 0.10	0.19 ± 0.10	0.014	0.036	0.690	0.006	-0.059	0.087
Max vGRF (N)	747.88 ± 132.04	796.16 ± 163.19	-48.284	53.914	0.378	0.028	-158.722	62.155
Max vGRF (N/kg)	10.29 ± 1.59	9.90 ± 0.40	0.385	0.437	0.386	0.027	-0.510	1.281
Time to Max vGRF (s)	0.37 ± 0.16	0.34 ± 0.14	0.024	0.056	0.669	0.007	-0.090	0.138
Loading Rate (N/s)	3073.34 ± 2226.15	3182.92 ± 1765.60	-109.586	741.217	0.884	0.001	-1627.901	1408.728
Peak Eccentric Knee Ext. Power (W/kg)	-1.24 ± 0.62	-1.25 ± 0.39	0.012	0.193	0.953	0.000	-0.384	0.407
Peak Knee Flexion (°)	21.71 ± 9.47	22.32 ± 5.66	-0.607	2.904	0.836	0.002	-6.556	5.341
Walking Velocity (m/s)	0.94 ± 0.22	1.00 ± 0.16	-0.055	0.072	0.449	0.021	-0.203	0.092
Stance Time (s)	0.73 ± 0.11	0.69 ± 0.12	0.041	0.042	0.335	0.033	-0.045	0.128
							95% CI	
6-months Post-operative	MR (n=16 knees)	SR (n=14 knees)	Mean Difference	SE	p	ηp^2	Lower	Upper
Peak KFM (N·m/kg)	0.68 ± 0.22	0.65 ± 0.21	0.028	0.080	0.728	0.004	-0.136	0.192
Time to peak KFM	0.16 ± 0.11	0.15 ± 0.09	0.010	0.037	0.797	0.002	-0.067	0.086
PKFM Rate (N·m/kg/s)	3.72 ± 2.48	4.08 ± 1.60	-0.358	0.775	0.647	0.008	-1.945	1.229
KFM Impulse (∫N·m/kg/s)	0.20 ± 0.08	0.18 ± 0.07	0.017	0.028	0.537	0.014	-0.040	0.074
Max vGRF (N)	760.15 ± 145.53	812.09 ± 183.66	-51.944	60.141	0.395	0.026	-175.137	71.250
Max vGRF (N/kg)	10.67 ± 1.34	10.27 ± 0.49	0.401	0.379	0.300	0.038	-0.377	1.178
Time to Max vGRF (s)	***0.41 ± 0.14***	***0.30 ± 0.13***	***0.117****	***0.050***	***0.026****	***0.165***	***0.015***	***0.219***
Loading Rate (N/s)	2663.24 ± 2197.05	4362.27 ± 2667.88	-1699.025	888.205	0.066	0.116	-3518.431	120.380
Peak Eccentric Knee Ext. Power (W/kg)	-1.59 ± 0.58	-1.73 ± 0.49	0.139	0.197	0.487	0.017	-0.265	0.543
Peak Knee Flexion (°)	20.02 ± 5.68	22.08 ± 5.36	-2.057	2.024	0.318	0.036	-6.202	2.089
Walking Velocity (m/s)	1.05 ± 0.17	1.12 ± 0.10	-0.071	0.053	0.187	0.061	-0.179	0.037
Stance Time (s)	0.67 ± 0.06	0.64 ± 0.04	0.030	0.018	0.110	0.089	-0.007	0.068
							95% CI	
12-months Post-operative	MR (n=16 knees)	SR (n=14 knees)	Mean Difference	SE	p	ηp^2	Lower	Upper
Peak KFM (N·m/kg)	0.70 ± 0.25	0.70 ± 0.21	0.007	0.087	0.932	0.000	-0.170	0.185
Time to peak KFM	0.13 ± 0.04	0.18 ± 0.11	-0.047	0.030	0.125	0.082	-0.108	0.014
PKFM Rate (N·m/kg/s)	3.28 ± 2.11	3.69 ± 1.84	-0.413	0.729	0.575	0.011	-1.906	1.080
KFM Impulse (∫N·m/kg/s)	0.20 ± 0.06	0.20 ± 0.07	0.004	0.024	0.854	0.001	-0.045	0.054
Max vGRF (N)	765.03 ± 147.24	818.77 ± 182.15	-53.744	60.155	0.379	0.028	-176.966	69.477
Max vGRF (N/kg)	10.64 ± 1.45	10.07 ± 0.49	0.570	0.407	0.173	0.065	-0.265	1.404
Time to Max vGRF (s)	0.39 ± 0.15	0.34 ± 0.14	0.048	0.054	0.386	0.027	-0.063	0.159
Loading Rate (N/s)	2906.50 ± 2012.22	3494.13 ± 2046.35	-587.630	742.222	0.435	0.022	-2108.002	932.742
Peak Eccentric Knee Ext. Power (W/kg)	-1.60 ± 0.61	-1.71 ± 0.44	0.111	0.197	0.578	0.011	-0.292	0.514
Peak Knee Flexion (°)	21.26 ± 6.03	19.79 ± 4.31	1.477	1.940	0.453	0.020	-2.496	5.451
Walking Velocity (m/s)	1.08 ± 0.19	1.09 ± 0.11	-0.008	0.057	0.892	0.001	-0.124	0.108
Stance Time (s)	***0.67 ± 0.06***	***0.63 ± 0.05***	***0.046****	***0.021***	***0.035****	***0.149***	***0.003***	***0.088***

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; SE=standard error; CI=confidence interval of the difference

ηp^2=partial eta squared effect size

* = Significant between-subjects effect (p<0.05)

† = Significant interaction effect (p<0.05)

Table 5.4. MR vs SR Stair Ascent Biomechanics Pairwise Comparisons Across Time

	Implant Group							
	MR	SR	Mean				95% CI	
Pre-operative	(n=11 knees)	(n=10 knees)	Difference	SE	p	np^2	Lower	Upper
Peak KFM (N·m/kg)	0.65 ± 0.17	0.53 ± 0.21	0.128	0.083	0.137	0.112	-0.045	0.301
Time to Peak KFM (s)	0.42 ± 0.31	0.27 ± 0.21	0.148	0.115	0.215	0.080	-0.093	0.389
PKFM Rate (N·m/kg/s)	2.47 ± 1.55	4.85 ± 7.49	-0.199	0.175	0.269	0.055	-0.564	0.167
KFM Impulse (∫N·m/kg/s)	0.26 ± 0.20	0.16 ± 0.14	0.099	0.076	0.211	0.081	-0.061	0.258
Max vGRF (N)	775.31 ± 182.41	883.35 ± 159.18	-108.043	75.065	0.166	0.098	-265.156	49.069
Max vGRF (N/kg)	10.66 ± 0.76	10.55 ± 0.63	0.103	0.305	0.738	0.006	-0.535	0.742
Time to Max vGRF (s)	0.35 ± 0.14	0.31 ± 0.09	0.040	0.053	0.459	0.029	-0.070	0.150
Loading Rate (N/s)	3801.80 ± 3320.82	5736.54 ± 4207.57	-1934.740	1645.908	0.254	0.068	-5379.666	1510.186
Peak Concentric Knee Ext. Power (W/kg)	1.42 ± 0.58	1.36 ± 0.60	0.063	0.252	0.806	0.003	-0.463	0.588
Peak Knee Flexion (°)	68.08 ± 5.03	67.74 ± 5.08	-2.996	2.184	0.186	0.090	-7.568	1.575
Stance Time (s)	1.28 ± 0.52	1.10 ± 0.27	0.184	0.184	0.331	0.050	-0.202	0.570

	MR	SR	Mean				95% CI	
6-months Post-operative	(n=11 knees)	(n=10 knees)	Difference	SE	p	np^2	Lower	Upper
Peak KFM (N·m/kg)	0.58 ± 0.12	0.57 ± 0.23	0.016	0.080	0.847	0.002	-0.153	0.184
Time to Peak KFM (s)	***0.24 ± 0.06***	***0.17 ± 0.04***	***0.067****	***0.022***	***0.008****	***0.318***	***0.020***	***0.114***
PKFM Rate (N·m/kg/s)	2.82 ± 0.96	3.71 ± 1.97	0.084	0.217	0.702	0.087	-0.370	0.539
KFM Impulse (∫N·m/kg/s)	0.17 ± 0.12	0.14 ± 0.09	0.037	0.047	0.441	0.032	-0.061	0.134
Max vGRF (N)	782.13 ± 201.55	916.39 ± 168.18	-134.256	81.484	0.116	0.125	-304.804	36.292
Max vGRF (N/kg)	10.93 ± 0.58	11.13 ± 0.44	-0.199	0.228	0.396	0.038	-0.677	0.280
Time to Max vGRF (s)	0.33 ± 0.12	0.28 ± 0.07	0.050	0.043	0.267	0.064	-0.041	0.141
Loading Rate (N/s)	3871.29 ± 3248.90	4728.48 ± 3189.76	-857.197	1407.366	0.550	0.019	-3802.849	2088.455
Peak Concentric Knee Ext. Power (W/kg)	1.47 ± 0.48	1.46 ± 0.56	0.002	0.221	0.991	0.000	-0.459	0.464
Peak Knee Flexion (°)	69.58 ± 4.85	72.58 ± 5.15	-5.023	2.417	0.051	0.185	-10.081	0.036
Stance Time (s)	1.05 ± 0.22	0.91 ± 0.05	0.141	0.072	0.064	0.170	-0.009	0.291

	MR	SR	Mean				95% CI	
12-months Post-operative	(n=11 knees)	(n=10 knees)	Difference	SE	p	np^2	Lower	Upper
Peak KFM (N·m/kg)	0.66 ± 0.23	0.67 ± 0.19	-0.008	0.093	0.930	0.000	-0.202	0.186
Time to Peak KFM (s)	0.25 ± 0.07	0.21 ± 0.07	0.039	0.031	0.214	0.080	-0.025	0.104
PKFM Rate (N·m/kg/s)	3.02 ± 1.37	3.76 ± 1.82	-0.051	0.184	0.786	0.056	-0.435	0.334
KFM Impulse (∫N·m/kg/s)	0.19 ± 0.13	0.20 ± 0.08	-0.017	0.048	0.731	0.006	-0.116	0.083
Max vGRF (N)	786.16 ± 213.31	941.07 ± 184.54	-154.912	87.472	0.093	0.142	-337.994	28.170
Max vGRF (N/kg)	10.81 ± 0.58	10.89 ± 0.73	-0.077	0.286	0.790	0.004	-0.675	0.521
Time to Max vGRF (s)	0.29 ± 0.09	0.28 ± 0.07	0.005	0.036	0.885	0.001	-0.071	0.082
Loading Rate (N/s)	5606.71 ± 4325.50	4567.12 ± 4162.47	1039.591	1856.546	0.582	0.016	-2846.204	4925.386
Peak Concentric Knee Ext. Power (W/kg)	1.62 ± 0.68	1.73 ± 0.64	-0.108	0.285	0.709	0.007	-0.704	0.487
Peak Knee Flexion (°)	63.55 ± 4.99	68.58 ± 6.07	0.347	2.208	0.877	0.001	-4.274	4.968
Stance Time (s)	1.02 ± 0.18	0.93 ± 0.10	0.096	0.066	0.160	0.101	-0.041	0.233

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; SE=standard error; CI=confidence interval of the difference

np^2=partial eta squared effect size

* = Significant between-subjects effect (p<0.05)

† = Significant interaction effect (p<0.05)

Table 5.5. MR vs SR Stair Descent Biomechanics Pairwise Comparisons Across Time

	Implant Group							
	MR	SR	Mean				95% CI	
Pre-operative	(n=10 knees)	(n=10 knees)	Difference	SE	p	np^2	Lower	Upper
Peak KFM (N·m/kg)	0.81 ± 0.36	0.90 ± 0.23	-0.085	0.134	0.533	0.022	-0.366	0.196
Time to Peak KFM (s)	0.34 ± 0.14	0.29 ± 0.12	0.051	0.059	0.400	0.040	-0.074	0.176
Peak KFM Rate (N·m/kg/s)	3.70 ± 2.72	4.32 ± 2.41	-0.625	1.149	0.593	0.016	-3.039	1.790
KFM Impulse (∫N·m/kg/s)	0.73 ± 0.35	0.59 ± 0.19	0.140	0.126	0.281	0.064	-0.125	0.404
Max vGRF (N)	880.38 ± 232.34	1128.27 ± 299.79	-247.887	119.940	0.053	0.192	-499.871	4.097
Max vGRF (N/kg)	12.49 ± 2.62	13.56 ± 3.04	-1.072	1.269	0.410	0.038	-3.738	1.595
Time to Max vGRF (s)	0.14 ± 0.02	0.13 ± 0.02	0.010	0.009	0.313	0.057	-0.010	0.030
Loading Rate (N/s)	***6356.67 ± 2594.11***	***9054.44 ± 2844.14***	***-2697.763****	***1217.313***	***0.040****	***0.214***	***-5255.242***	***-140.283***
Peak Eccentric Knee Ext. Power (W/kg)	-2.80 ± 0.73	-3.20 ± 1.31	0.392	0.475	0.420	0.037	-0.605	1.390
Peak Knee Flexion (°)	31.40 ± 8.50	30.52 ± 7.01	0.881	3.484	0.803	0.004	-6.439	8.202
Stance Time (s)	1.51 ± 0.94	1.19 ± 0.43	0.320	0.327	0.340	0.051	-0.366	1.007

	MR	SR	Mean				95% CI	
6-months Post-operative	(n=10 knees)	(n=10 knees)	Difference	SE	p	np^2	Lower	Upper
Peak KFM (N·m/kg)	0.91 ± 0.28	0.98 ± 0.19	-0.073	0.105	0.500	0.026	-0.294	0.149
Time to Peak KFM (s)	0.35 ± 0.29	0.20 ± 0.07	0.149	0.095	0.135	0.120	-0.051	0.350
Peak KFM Rate (N·m/kg/s)	4.47 ± 2.62	5.82 ± 2.21	-1.354	1.085	0.228	0.080	-3.632	0.925
KFM Impulse (∫N·m/kg/s)	0.70 ± 0.33	0.55 ± 0.14	0.152	0.113	0.194	0.092	-0.085	0.388
Max vGRF (N)	997.62 ± 285.11	1313.78 ± 436.35	-316.168	164.828	0.071	0.170	-662.460	30.123
Max vGRF (N/kg)	14.28 ± 1.87	15.69 ± 2.48	-1.405	0.981	0.169	0.102	-3.466	0.657
Time to Max vGRF (s)	0.13 ± 0.02	0.11 ± 0.02	0.014	0.008	0.123	0.127	-0.004	0.031
Loading Rate (N/s)	***8189.84 ± 3005.45***	***12449.76 ± 5482.19***	***-4259.917****	***1977.048***	***0.045****	***0.205***	***-8413.540***	***-106.293***
Peak Eccentric Knee Ext. Power (W/kg)	-3.07 ± 0.48	-3.51 ± 0.59	0.446	0.242	0.082	0.159	-0.062	0.953
Peak Knee Flexion (°)	29.54 ± 4.38	32.07 ± 6.63	-2.526	2.514	0.328	0.053	-7.808	2.757
Stance Time (s)	1.25 ± 0.81	0.85 ± 0.13	0.398	0.260	0.143	0.115	-0.149	0.944

	MR	SR	Mean				95% CI	
12-months Post-operative	(n=10 knees)	(n=10 knees)	Difference	SE	p	np^2	Lower	Upper
Peak KFM (N·m/kg)	1.04 ± 0.27	1.02 ± 0.22	0.021	0.108	0.852	0.002	-0.207	0.248
Time to Peak KFM (s)	0.29 ± 0.17	0.19 ± 0.07	0.096	0.059	0.121	0.128	-0.028	0.219
Peak KFM Rate (N·m/kg/s)	4.97 ± 1.88	5.94 ± 2.24	-0.963	0.925	0.311	0.057	-2.907	0.980
KFM Impulse (∫N·m/kg/s)	0.69 ± 0.19	0.56 ± 0.15	0.135	0.075	0.090	0.151	-0.023	0.293
Max vGRF (N)	1003.88 ± 362.03	1301.11 ± 457.27	-297.223	184.434	0.124	0.126	-684.704	90.257
Max vGRF (N/kg)	13.98 ± 2.35	14.80 ± 2.37	-0.822	1.055	0.446	0.033	-3.037	1.394
Time to Max vGRF (s)	0.13 ± 0.02	0.12 ± 0.02	0.010	0.008	0.257	0.071	-0.008	0.027
Loading Rate (N/s)	8385.50 ± 4266.63	11866.36 ± 5783.63	-3480.864	2272.764	0.143	0.115	-8255.763	1294.035
Peak Eccentric Knee Ext. Power (W/kg)	-3.96 ± 1.42	-3.73 ± 0.81	-0.230	0.518	0.663	0.011	-1.319	0.859
Peak Knee Flexion (°)	33.44 ± 4.93	31.85 ± 5.42	1.592	2.317	0.501	0.026	-3.276	6.461
Stance Time (s)	1.12 ± 0.62	0.82 ± 0.11	0.297	0.201	0.157	0.108	-0.125	0.718

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; SE=standard error; CI=confidence interval of the difference

np^2=partial eta squared effect size

* = Significant between-subjects effect ($p<0.05$)

† = Significant interaction effect ($p<0.05$)

Table 5.6 Normality Statistics for MR and SR Biomechanics During Walking

	MR						SR					
	Pre-operative		6-month		12-month		Pre-operative		6-month		12-month	
	W	p	W	p	W	p	W	p	W	p	W	p
Peak KFM (N·m/kg)	0.976	0.931	0.984	0.990	0.951	0.544	0.951	0.647	0.886	0.104	0.980	0.984
Time to Peak KFM (s)	**_0.864_**	**_0.028*_**	**_0.510_**	**_<0.001*_**	0.968	0.828	**_0.621_**	**_<0.001*_**	**_0.563_**	**_<0.001*_**	0.944	0.552
Peak KFM Rate (N·m/kg/s)	**_0.812_**	**_0.005*_**	0.909	0.129	**_0.870_**	**_0.033*_**	0.880	0.088	0.949	0.621	0.918	0.269
KFM Impulse (∫N·m/kg/s)	0.926	0.240	0.967	0.818	0.933	0.302	0.946	0.585	0.894	0.134	0.961	0.795
Max vGRF (N)	0.938	0.356	0.962	0.729	0.968	0.820	0.878	0.083	**_0.861_**	**_0.050*_**	0.923	0.308
Max vGRF (N/kg)	**_0.742_**	**_<0.001*_**	**_0.768_**	**_0.001*_**	**_0.708_**	**_<0.001*_**	0.970	0.906	0.875	0.076	0.925	0.328
Time to Max vGRF (s)	0.947	0.483	**_0.800_**	**_0.004*_**	**_0.830_**	**_0.009*_**	0.926	0.342	0.956	0.731	0.927	0.350
Loading Rate (N/s)	**_0.780_**	**_0.002*_**	**_0.689_**	**_<0.001*_**	**_0.802_**	**_0.004*_**	0.764	0.004	**_0.852_**	**_0.039*_**	**_0.818_**	**_0.015*_**
Peak Eccentric Knee Ext. Power (W/kg)	0.978	0.958	0.930	0.273	0.948	0.495	0.955	0.717	0.936	0.452	0.950	0.632
Peak Knee Flexion (°)	0.944	0.557	0.944	0.557	0.949	0.618	0.970	0.894	0.878	0.125	0.963	0.820
Stance Time (s)	0.949	0.511	0.941	0.395	0.897	0.086	**_0.793_**	**_<0.001*_**	0.891	0.121	0.931	0.388
Walking Velocity (m/s)	0.905	0.115	0.934	0.308	0.970	0.860	0.946	0.581	0.932	0.407	0.922	0.299

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; KFM=knee flexion moment; vGRF=vertical ground reaction force; N=newton; MR=multiradius
SR=single radius; W=Shapiro-Wilk statistic; p=p-value
* = Significant Shapiro-Wilk Test(p<0.05)

Table 5.7 Normality Statistics for MR and SR Biomechanics During Stair Ascent

	MR						SR					
	Pre-operative		6-month		12-month		Pre-operative		6-month		12-month	
	W	p	W	p	W	p	W	p	W	p	W	p
Peak KFM (N·m/kg)	0.882	0.110	0.956	0.726	0.930	0.410	0.889	0.165	0.984	0.982	0.939	0.546
Time to Peak KFM (s)	**_0.834_**	**_0.026*_**	0.954	0.701	0.882	0.112	**_0.746_**	**_0.003*_**	0.872	0.106	0.968	0.876
Peak KFM Rate (N·m/kg/s)	0.869	0.076	0.962	0.791	0.898	0.172	**_0.521_**	**_<0.001*_**	0.922	0.375	0.853	0.064
KFM Impulse (∫N·m/kg/s)	0.937	0.480	0.915	0.282	0.955	0.708	0.932	0.473	0.868	0.094	0.951	0.676
Max vGRF (N)	0.953	0.685	0.944	0.570	0.914	0.270	0.903	0.238	0.929	0.434	0.950	0.663
Max vGRF (N/kg)	0.956	0.721	0.973	0.917	0.977	0.947	0.892	0.177	0.903	0.239	0.872	0.106
Time to Max vGRF (s)	0.886	0.123	0.962	0.798	**_0.803_**	**_0.010*_**	0.970	0.891	0.918	0.341	0.925	0.399
Loading Rate (N/s)	**_0.644_**	**_<0.001*_**	**_0.739_**	**_0.001*_**	**_0.840_**	**_0.032*_**	**_0.839_**	**_0.043*_**	**_0.750_**	**_0.004*_**	**_0.532_**	**_<0.001*_**
Peak Concentric Knee Ext. Power (W/kg)	**_0.807_**	**_0.012*_**	**_0.824_**	**_0.020*_**	0.897	0.170	0.936	0.513	0.940	0.557	0.923	0.385
Peak Knee Flexion (°)	0.930	0.411	0.926	0.375	0.945	0.587	0.970	0.894	0.878	0.125	0.963	0.820
Stance Time (s)	**_0.801_**	**_0.010*_**	0.876	0.091	0.949	0.635	0.823	0.027	0.955	0.729	0.914	0.311

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; KFM=knee flexion moment; vGRF=vertical ground reaction force; N=newton; MR=multiradius
SR=single radius; W=Shapiro-Wilk statistic; p=p-value
* = Significant Shapiro-Wilk Test(p<0.05)

Table 5.8 Normality Statistics for MR and SR Biomechanics During Stair Descent

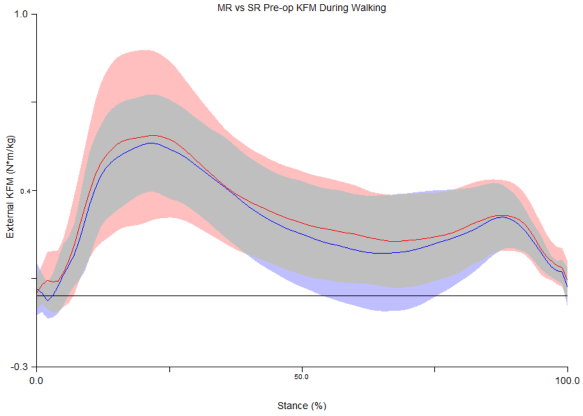
	MR						SR					
	Pre-operative		6-month		12-month		Pre-operative		6-month		12-month	
	W	p	W	p	W	p	W	p	W	p	W	p
Peak KFM (N·m/kg)	0.960	0.784	0.986	0.990	0.966	0.849	0.948	0.645	0.928	0.425	0.941	0.563
Time to Peak KFM (s)	0.953	0.709	**_0.613_**	**_<0.001*_**	**_0.758_**	**_0.004*_**	0.940	0.558	0.909	0.274	**_0.832_**	**_0.035*_**
Peak KFM Rate (N·m/kg/s)	0.903	0.234	0.942	0.570	0.888	0.160	**_0.840_**	**_0.044*_**	**_0.804_**	**_0.016*_**	**_0.829_**	**_0.032*_**
KFM Impulse (∫N·m/kg/s)	0.960	0.787	0.852	0.061	0.920	0.358	0.922	0.377	0.978	0.956	0.975	0.935
Max vGRF (N)	0.969	0.881	**_0.844_**	**_0.049*_**	0.865	0.088	0.881	0.135	**_0.829_**	**_0.032*_**	**_0.731_**	**_0.002*_**
Max vGRF (N/kg)	0.869	0.098	0.969	0.883	0.960	0.784	0.979	0.959	0.920	0.356	**_0.809_**	**_0.019*_**
Time to Max vGRF (s)	0.974	0.923	0.979	0.959	0.923	0.379	0.891	0.174	0.966	0.850	0.900	0.220
Loading Rate (N/s)	0.958	0.767	0.899	0.213	**_0.806_**	**_0.017*_**	0.889	0.165	0.933	0.483	**_0.780_**	**_0.008*_**
Peak Eccentric Knee Ext. Power (W/kg)	0.909	0.271	0.934	0.489	0.915	0.320	**_0.807_**	**_0.018*_**	0.912	0.294	0.948	0.650
Peak Knee Flexion (°)	0.975	0.937	0.931	0.462	0.950	0.674	0.911	0.291	0.947	0.636	0.964	0.835
Stance Time (s)	**_0.710_**	**_0.001*_**	**_0.697_**	**_<0.001*_**	**_0.762_**	**_0.005*_**	**_0.729_**	**_0.002*_**	**_0.813_**	**_0.021*_**	0.952	0.697

N·m=Newton-meter; kg=kilograms; s=seconds; °=degrees; ∫=integral; KFM=knee flexion moment; vGRF=vertical ground reaction force; N=newton; MR=multiradius
SR=single radius; W=Shapiro-Wilk statistic; p=p-value
* = Significant Shapiro-Wilk Test(p<0.05)

5.6 Figures: Walking Biomechanics

Figures 5.1-5.3. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for external KFM during walking at 5.1) pre-operative, 5.2) 6-months post-operative, 5.3)12-months post-operative.

5.1)



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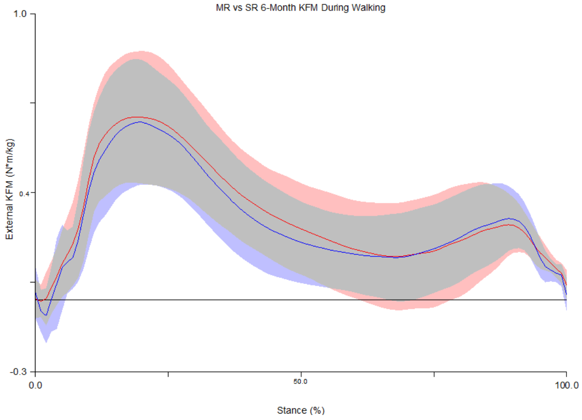

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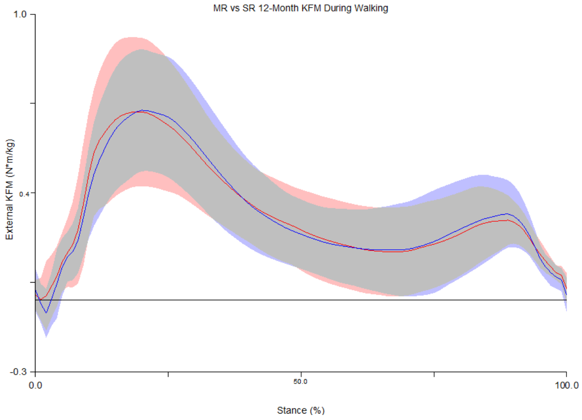
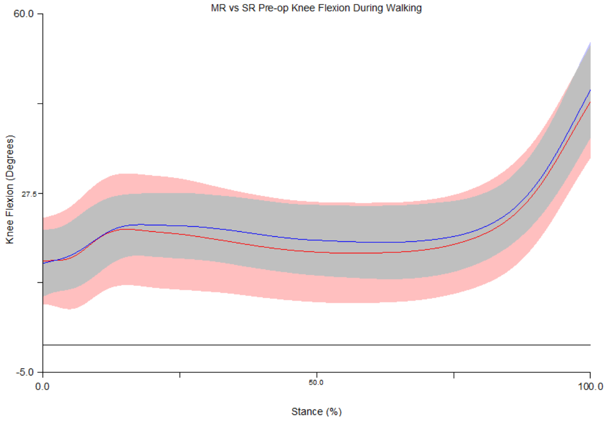


Figures 5.4-5.6. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for knee flexion angle during walking at 5.4) pre-operative, 5.5) 6-months post-operative, 5.6) 12-months post-operative.

5.4)



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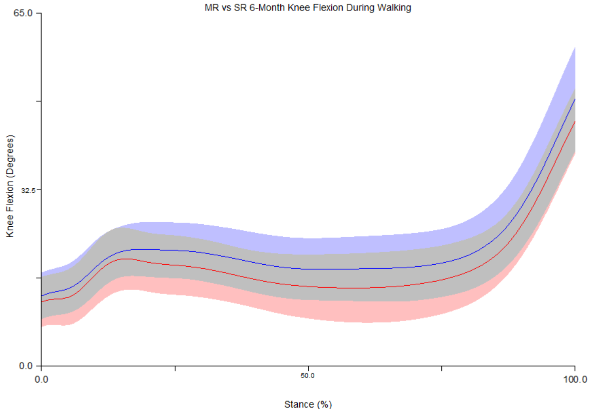

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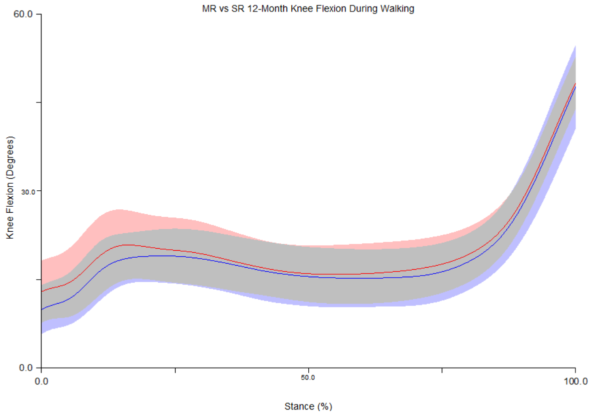
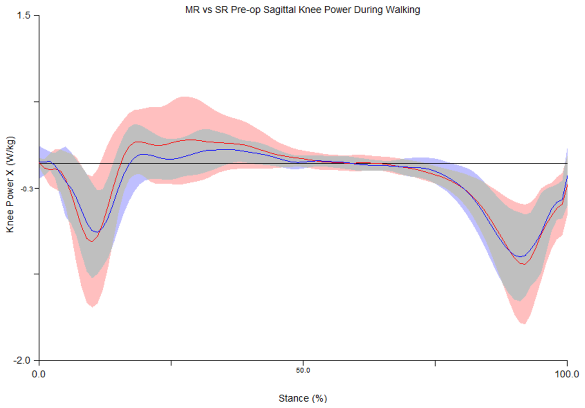


Figures 5.7-5.9. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for sagittal knee power during walking at 5.7) pre-operative, 5.8) 6-months post-operative, 5.9) 12-months post-operative.

5.7)



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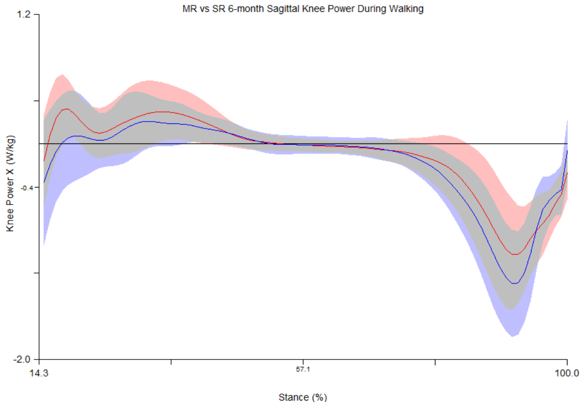

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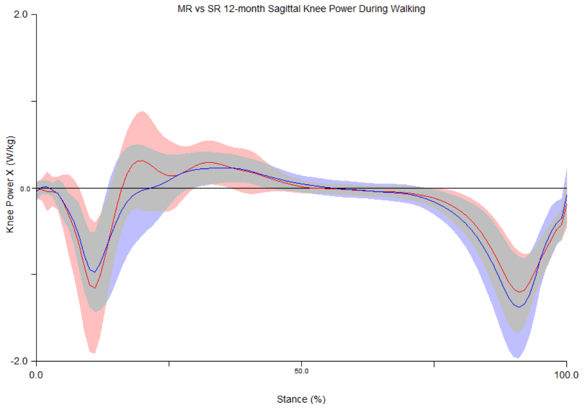
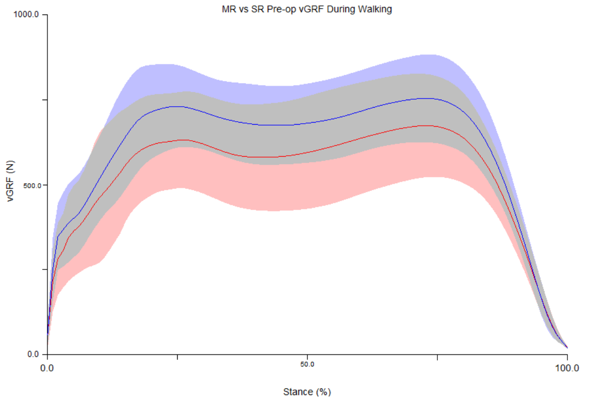


Figures 5.10-5.12. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for raw vGRF during walking at 5.10) pre-operative, 5.11) 6-months post-operative, 5.12) 12-months post-operative.

5.10)



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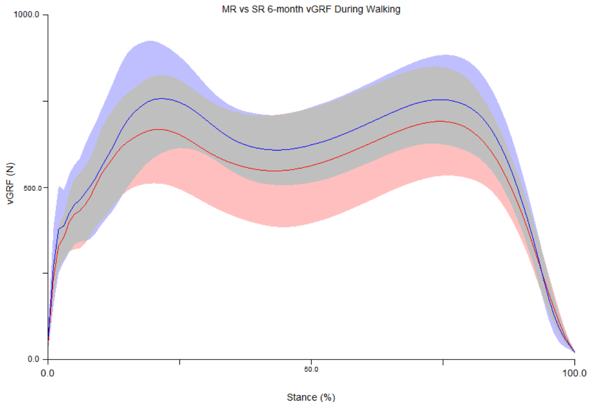

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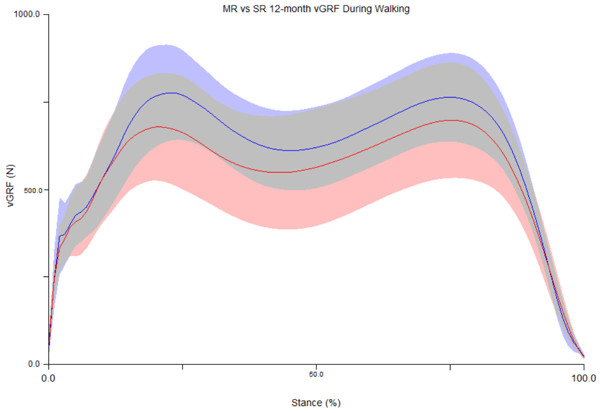
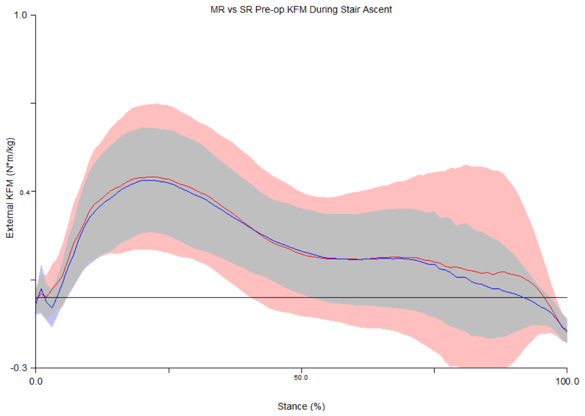


5.7 List of Figures: Ascent Biomechanics

Figures 5.13-5.15. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for external KFM during stair ascent at 5.13) pre-operative, 5.14) 6-months post-operative, 5.15) 12-months post-operative.

5.13)



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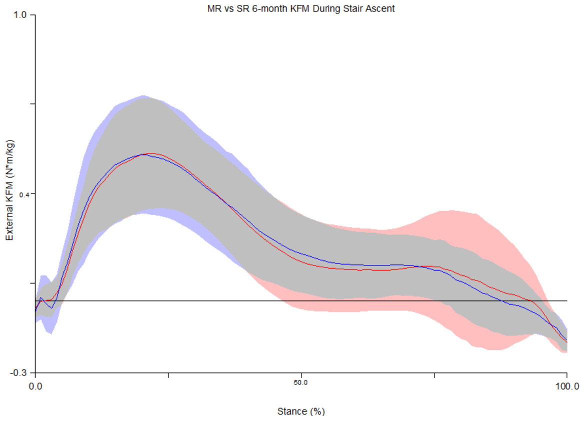

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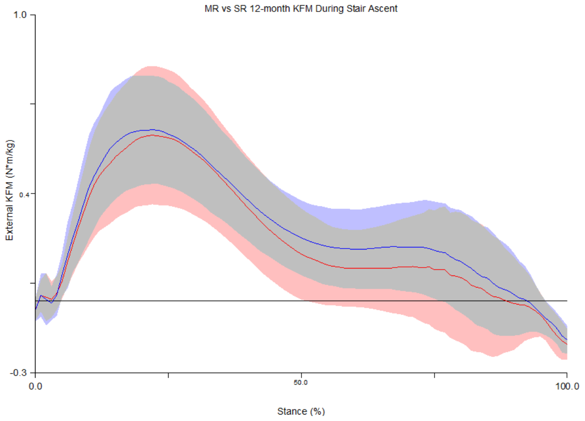
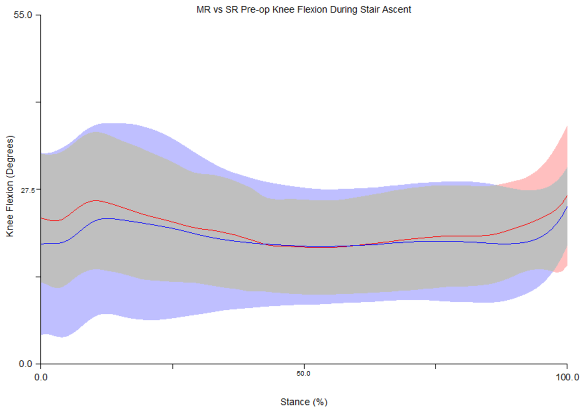


Figures 5.16-5.18. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for knee flexion angle during stair ascent at 5.16) pre-operative, 5.17) 6-months post-operative, 5.18) 12-months post-operative.

5.16)



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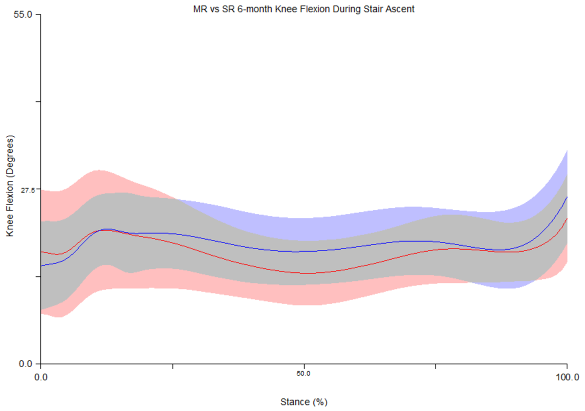

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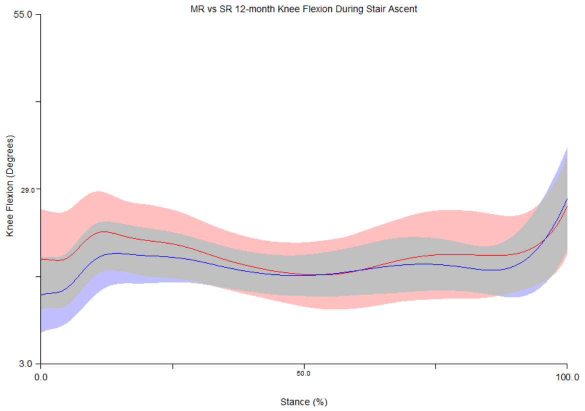
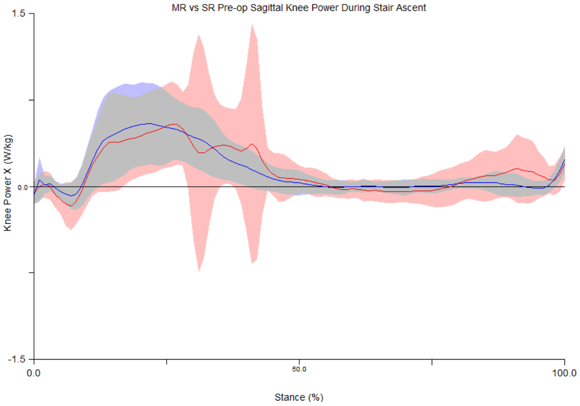


Figures 5.19-5.21. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for sagittal knee power during stair ascent at 5.19) pre-operative, 5.20) 6-months post-operative, 5.21) 12-months post-operative.

5.19)



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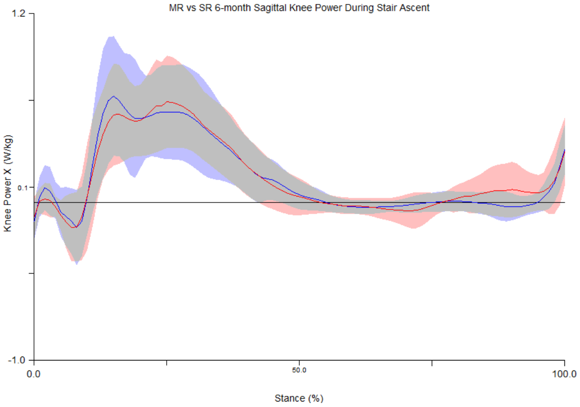

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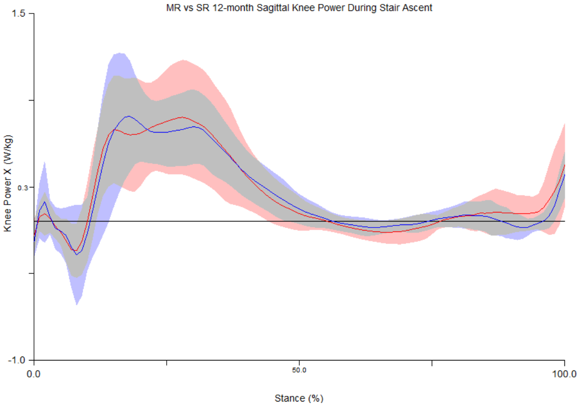
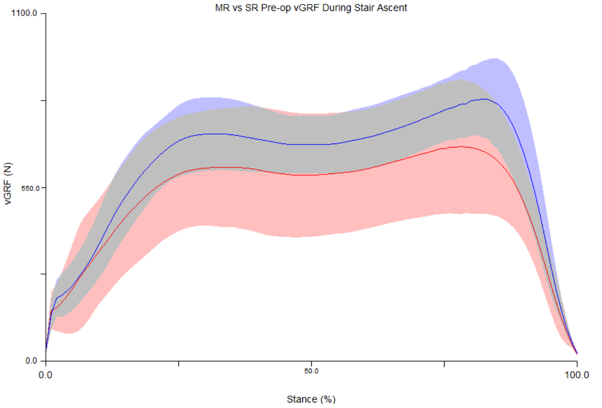


Figures 5.22-5.24. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for raw vGRF during stair ascent at 5.22) pre-operative, 5.23) 6-months post-operative, 5.24) 12-months post-operative.

5.22)



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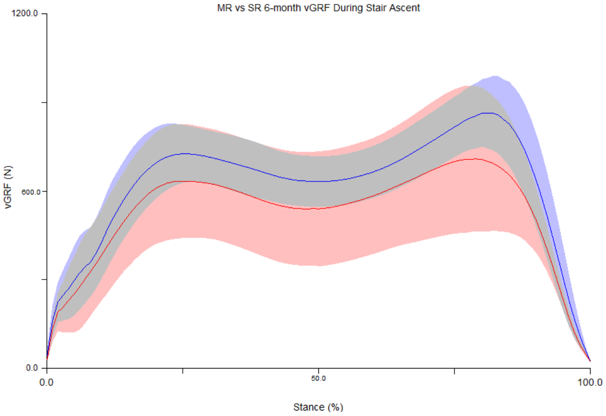

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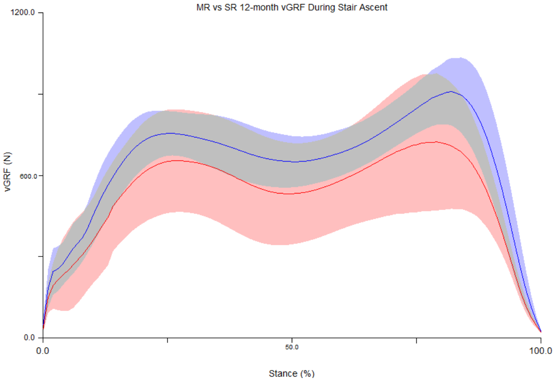
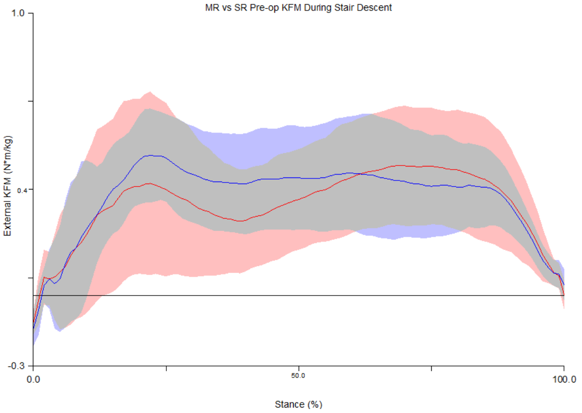


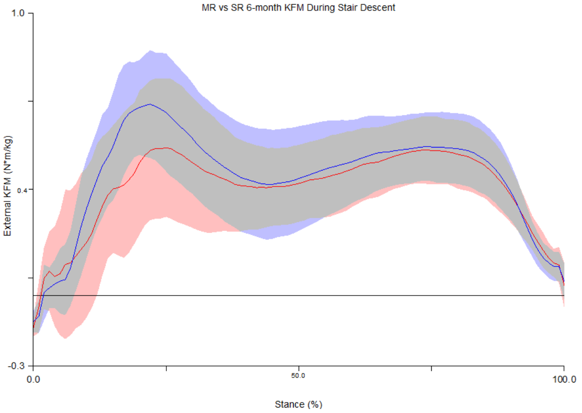
5.8 List of Figures: Descent Biomechanics

Figures 5.25-5.27. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for external KFM during stair descent at 5.25) pre-operative, 5.26) 6-months post-operative, 5.27) 12-months post-operative.

5.25)



5.26)



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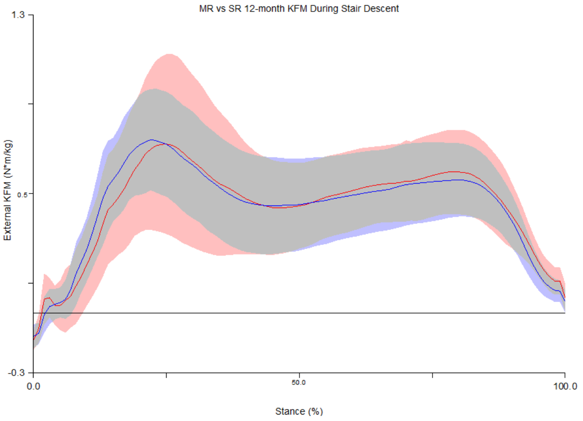
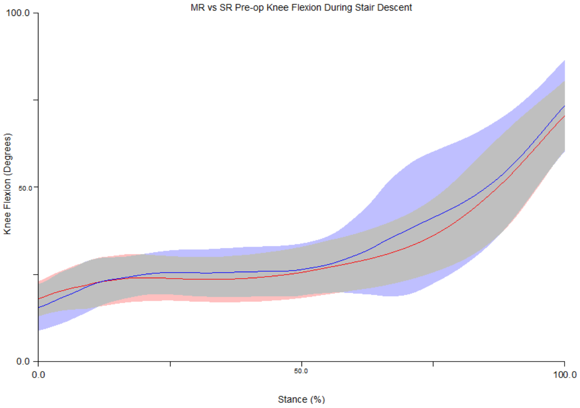


Figures 5.28-5.30. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for knee flexion angle during stair descent at 5.16) pre-operative, 5.17) 6-months post-operative, 5.18) 12-months post-operative.

5.28)



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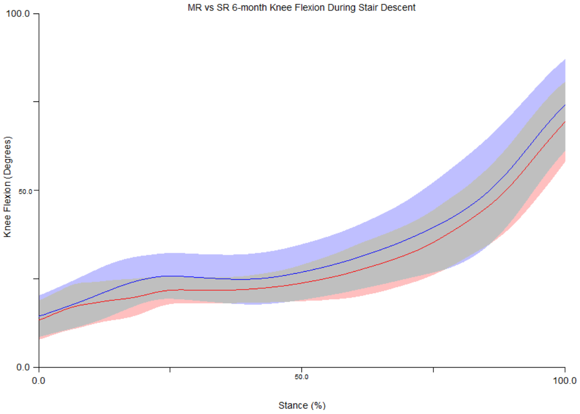

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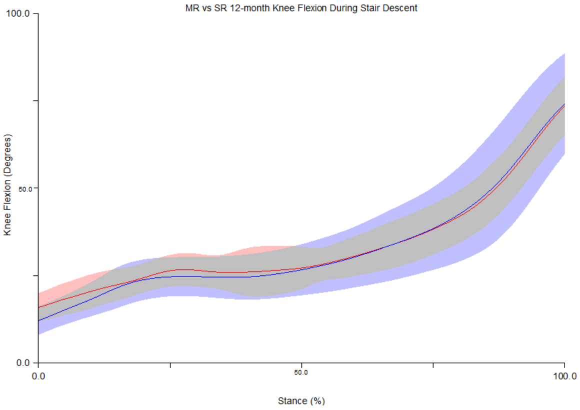
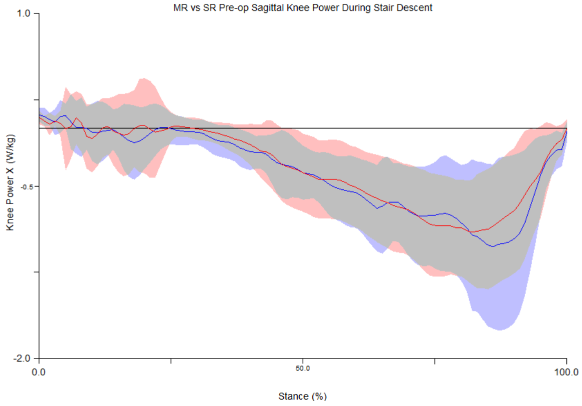


Figures 5.31-5.33. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for sagittal knee power angle during stair descent at 5.31) pre-operative, 5.32) 6-months post-operative, 5.33) 12-months post-operative.

5.31)



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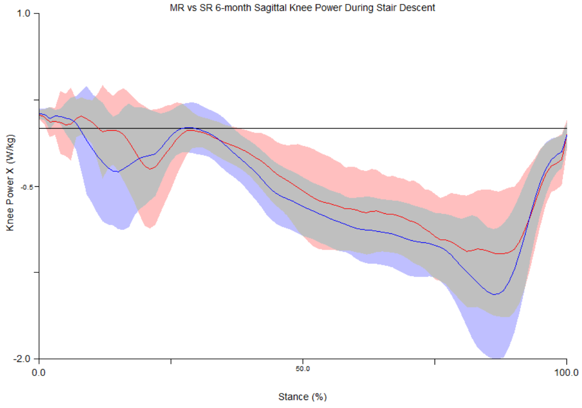

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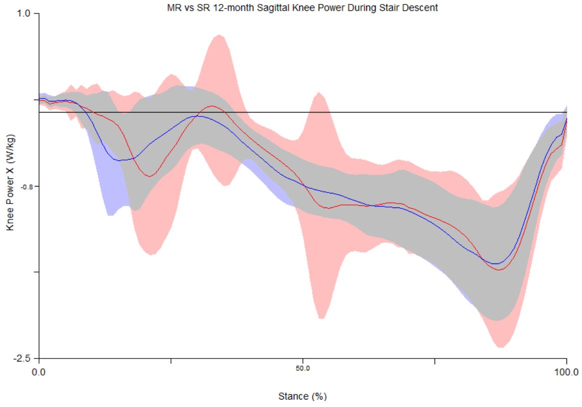
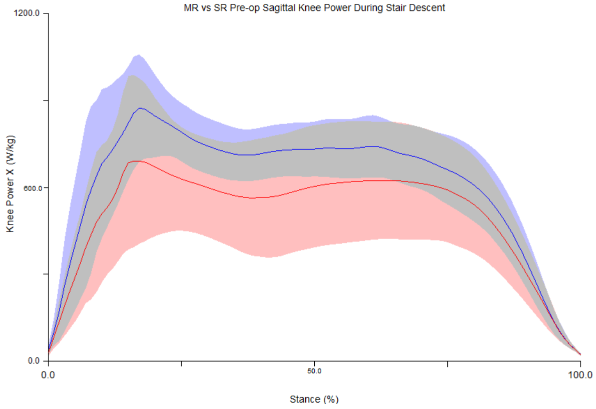


Figures 5.34-5.36. MR (red) vs SR (blue) mean (line) and standard deviation (shade) for raw vGRF angle during stair descent at 5.34) pre-operative, 5.35) 6-months post-operative, 5.36) 12-months post-operative.

5.34)



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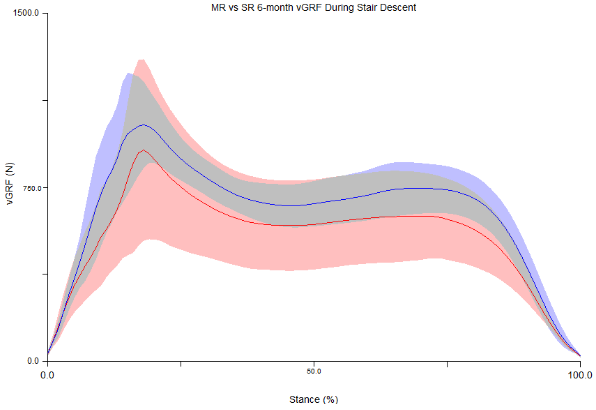

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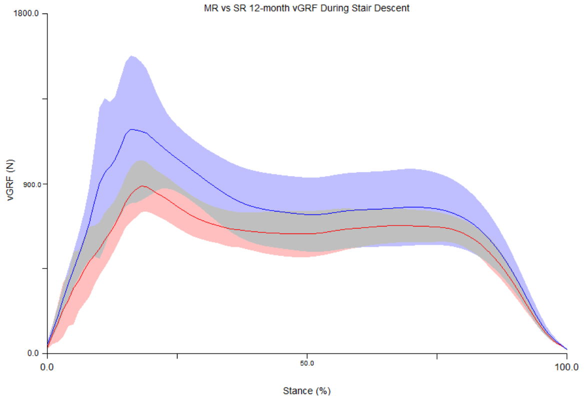


CHAPTER 6:

LONGITUDINAL MIXED-EFFECTS MODEL OF LEG STIFFNESS DURING STAIR DESCENT BETWEEN TOTAL AND UNICOMPARTMENTAL KNEE ARTHROPLASTY PATIENTS

Abstract

Background: Deficits in neuromuscular control are thought to contribute to functional deficiencies in knee osteoarthritis (OA) and knee arthroplasty patients. Following either total (TKA) or unicompartmental (UKA) knee arthroplasty, descending stairs is a primary goal for patients and requires dynamic function of the limb for efficient and safe stair navigation. To understand neuromuscular control of the limb, various leg stiffness measurements can be applied; however, limited evidence examining various techniques during stair negotiation is limited. The goal of this study was to examine three methods of leg stiffness during this task longitudinally, before and after knee arthroplasty in TKA and UKA patients. ***Methods:*** 3D gait analysis of stair descent was conducted for 27 patients receiving TKA and 16 patients receiving UKA. Leg stiffness was modeled as 3D K_{leg}, uniplanar K_{leg}, vertical stiffness (K_{vert}). A Generalized Linear Mixed Model was chosen to model longitudinal growth of individuals and each group across time pre-operatively, and post-operatively at 6-weeks, 3-months, 6-months, and 12-months. ***Findings:*** Significant fixed effects were observed for time in the 3D K_{leg} analysis, but not for uniplanar K_{leg} or K_{vert}. Each method demonstrated significant random variation of subjects across time. Neither group demonstrated superiority in leg stiffness measured with these three methods. ***Interpretation:*** Subjects generally increased in leg stiffness following surgery. The increase was significant for 3D K_{leg} but there was no between-group interaction. This may be a consequence of increased willingness to load following surgery. Given the significant between subject variation in leg stiffness for each method, further research is needed to determine the appropriate estimation during stair descent.

6.1 Introduction

A primary concern for knee osteoarthritis (OA) patients is the deterioration of neuromuscular control associated with cartilage degeneration and pain [128]. Neuromuscular deficits in the involved limb in knee OA patients have been observed as balance abnormalities[51, 129], decreased knee joint proprioception[48, 129], and diminished quadriceps strength[97, 98]. These deficits contribute to diminished activities of daily living (ADLs) and lower quality of life.[79] Current evidence evaluating these aspects of neuromuscular control in knee OA patients is available; however, longitudinal gait changes are less well understood following surgery. Furthermore, evidence examining how these neuromuscular components translate to more complex tasks such as stair descent is absent. To restore function and eliminate pain, total knee arthroplasty (TKA) is the current gold standard for knee OA patients; however, patients may receive the popular unicompartmental knee arthroplasty (UKA) option when cartilage degeneration is isolated to a single compartment. Given that UKA retains the cruciate ligaments, advantages in neuromuscular control have been theorized for these and similar implants. [130] Patients receiving a posterior stabilized TKA may therefore be at a disadvantage for regaining post-operative neuromuscular control due to the lack of cruciate ligament retention.

Post-operative neuromuscular control has been previously evaluated by examining muscle strength[127, 131] and biomechanical gait parameters. [59] While these aspects of post-operative recovery are important for return to ADLs and understanding knee loading, they inadequately describe neuromuscular control of the limb. Leg stiffness has been described as a neuromuscular variable, as it models limb compression as a spring under using various models. [11]Despite its application to sport performance and injury recovery,[4, 12] leg stiffness has not been used to investigate neuromuscular recovery before and after knee arthroplasty for osteoarthritis.

Still, increased leg stiffness has been associated with decreased total joint work[15] and may be a compensation for muscle fatigue to increase muscle efficiency.[76] The degree to which leg stiffness increases or decreases following knee arthroplasty has not been documented, and analyzing changes post-operatively may give insight into the efficacy of TKA and UKA implant designs.

Analysis of leg stiffness and limb compression behavior has been well established during activities such as walking and running;[4, 8-12] however, limb compression during more challenging tasks such as stair descent has been less documented. Furthermore, methods of determining leg stiffness have largely been uniplanar or biplanar[5, 7] or have determined leg stiffness as an aggregate of whole-body compression, such as vertical stiffness.[9, 13, 14] While these approaches have successfully modeled the lower extremity as a spring, they do not consider the lower extremity as a 3-dimensional (3D) unit. More recent methods employed by Liew et al[10, 15] that model both a 3D ground reaction force (GRF) vector and 3D leg vector have attempted to do so. Given the paucity of evidence examining any of these methods during stair negotiation, it is prudent to examine which may give insight into leg compression during this activity. Given that stair negotiation is relevant to reported goals of knee OA patients, examining neuromuscular recovery in TKA and UKA patients may give insight into which arthroplasty technique is recommended. Therefore, the purpose of this study was to assess longitudinal changes of three different leg stiffness determinants during stair descent between TKA and UKA patients pre-operatively and post-operatively at 6-weeks, 3-months, 6-months, and 12-months. We hypothesized that UKA patients would have lower leg stiffness at baseline and would have lower leg stiffness over time compared to TKA patients.

6.2 Methods

6.2.1 Subjects & Surgical Procedure

Forty-three patients (58 knees) were included in this analysis. Twenty-seven patients (36 knees) undergoing TKA were randomly assigned either a single-radius (GetAroundKnee™, Stryker Orthopedics, Mahwah, NJ) or multi-radius (Balanced Knee® System, Ortho Development Corporation, Draper, UT) femoral component, and 16 patients (22 knees) receiving medial UKA (Oxford® Partial Knee Implant, Zimmer Biomet Orthopedics, Warsaw, IN) were included in this study. Both TKA implants were posterior stabilized design and all UKA were cruciate retaining. Only medial UKA patients were included. All patients received patellar resurfacing. A single, board certified surgeon performed all surgeries under general anesthesia using a medial parapatellar approach in all patients. The preoperative diagnosis of all patients was osteoarthritis. Therefore, if OA was present in the contralateral limb in these patients, it had not yet progressed to cause pain or functional deficiencies. Patients were included if: 1) under 75 years of age at time of surgery, 2) no previous history of lower extremity fracture, osteotomy, or joint replacement, 3) undergoing a unilateral or simultaneous bilateral TKA/UKA for the treatment of OA, and 4) able to walk without an aid. Prior to enrollment in the study, all patients signed informed consent and the study was approved by the Institution's Committee on Human Studies.

6.2.2 Biomechanical Analysis

All biomechanical analyses were conducted at the University Gait Laboratory. Stair descent biomechanics were collected at a self-selected pace using a 29-retroreflective array-based marker set. Landmarks were located on the thorax (including the xiphoid process, jugular notch, C7 spinous process, T10 spinous process, apex of the inferior angle of the scapula and bilateral acromioclavicular joints), the pelvis (including bilateral anterior superior iliac spine and posterior superior iliac spine), and lower extremities (including bilateral head of the 1st metatarsal, head of the 2nd metatarsal, head of the 5th metatarsal, base of the 5th metatarsal, posterior calcaneus, medial malleoli, lateral malleoli, medial femoral epicondyles, lateral femoral epicondyles). Additionally, four 4-marker arrays were secured bilaterally on the mid-thigh and mid-shank segments. The medial epicondyle, medial malleolus, and head of the 1st metatarsal were used for static calibration only and removed for movement trials. Kinematic data were collected with a Vicon motion capture system and Vicon Nexus software (Nexus 2.5, Vicon, Inc., Centennial, CO) at 240 Hz and time synchronized with kinetic data. Kinetic data from the operated limb were collected at 960 Hz from a force plate (Advanced Mechanical Technology Incorporated, Boston, MA) embedded flush with the floor. All kinematic data were smoothed using a low-pass Butterworth filter using a 10 Hz cut-off frequency and ground reaction forces (GRF) were filtered using a 50 Hz cut-off frequency.[27, 56] Stair descent trials were completed following the walking trials and were conducted in similar fashion to Vallabhajosula et al [26] Up to five acceptable trials were collected for stair negotiation. Subjects performed the minimum number of trials necessary to obtain acceptable trials for the involved limb undergoing surgery. All data was processed using Visual 3D (C-Motion, Inc., Germantown, MD).

6.2.3 Leg Stiffness Calculations

Leg Stiffness Model 1[10]

$$Kleg = \frac{Peak\ Projected\ GRF}{\Delta L}$$

Where Peak Projected GRF = the peak scalar value of the dot product of the resultant GRF vector and the unit vector of the leg, and ΔL is the change in leg vector length between corresponding frames, measured as a line from the center of pressure (COP) to the estimated hip joint center. K_{leg} is then expressed as a Dimensionless Kleg (DK_{leg}):

$$DKleg = Kleg/(BW * LL^{-1})$$

Where the scaling factor ($BW*LL^{-1}$) is a ratio of body weight (BW) to standing leg length (LL)

Leg Stiffness Model 2[5]

$$Kleg = \frac{Fmax}{\Delta L}$$

Where $\Delta L = \Delta y + L_0(1 - \cos\theta)\ and\ \theta = \sin^{-1}(ut_c\ /2L_0)$; L_0 = standing leg length θ = half angle of the arc swept by the leg, u = horizontal velocity, and t_c = ground contact time.

Leg Stiffness Model 3[5]

$$Kvert = Fmax/\Delta y$$

Where Fmax = maximum vertical ground reaction force and Δy = maximum vertical displacement of the center of mass (COM)

6.2.4 Statistical Analysis

Descriptive statistics are reported for patient demographics. Independent samples t-tests were conducted between TKA and UKA groups pre-operatively to determine differences in group demographics. Shapiro-Wilk and Levene's Tests were used to assess normality and equality of variances, respectively, between groups. Leg stiffness variables were examined with a generalized linear mixed model (GLMM) analysis to determine fixed effects for time and the time*surgery interaction for each measure of leg stiffness. A random intercept (subjects) and random slope for time were included. The pre-operative time was coded "0", six weeks was coded "0.125", 3-months was coded "0.25", 6-months was coded "0.50" and 12-months was coded "1" for the fixed effect for time. This was done to examine time as a linear model between time codes 0 and 1 while also observing variances at each other time point. The TKA group was coded "0" and the UKA group was coded "1" for the fixed interaction effect. All statistical analyses were performed using SPSS version 26.0 (IBM, Armonk, NY, USA). Alpha level was set a priori to $p<0.05$ for all analyses. The p-values for variance components were interpreted as one-tailed tests.

6.3 Results

6.3.1 Patient Demographics

Demographics for TKA and UKA patients are presented in Table 6.1. Age, height, mass, and body mass index (BMI) were normally distributed and equal variances were assumed between each group. Groups were comparable for age, height, body mass, and body mass index (BMI), with no significant differences observed in baseline characteristics.

6.3.2 Longitudinal Mixed Effect Model Analysis of Leg Stiffness

Descriptive statistics for each measure of leg stiffness are presented in Table 6.2 and the GLMM results are presented in Table 3. For Leg Stiffness Model 1, a significant fixed effect was observed for time (F (1, 205) =10.419, p=0.001) but not for the time*surgery interaction (F (1, 205) =0.963, p=0.328). The TKA group at baseline showed an average stiffness of 15.976 and significantly increased stiffness by an average of 1.840 by 12-months (t=1.975, p=0.050, 95% CI: 0.004 – 3.676). The UKA group increased stiffness by an additional 1.493 by 12-months, but this interaction was not significant (t=0.981, p=0.328, 95% CI: -1.507 – 4.492). A significant random effect was observed for the average variance at baseline in all subjects (estimate=11.878, z=3.709, p<0.001, 95% CI: 7.002 – 20.149) and at 12-months (estimate=17.654, z=2.529, p=0.006, 95% CI: 8.133 – 38.319).

For Leg Stiffness Model 2, no significant fixed effects for time or the time*surgery interaction were observed for this measure of leg stiffness. A significant random effect of the average variance for all subjects was observed at baseline (estimate=135.995, z=4.147, p<0.001, 95% CI: 84.777 – 218.155) but not at 12-months.

For Leg Stiffness Model 3, a significant fixed effect for time was observed for vertical stiffness (F (1, 205) =15.842, p<0.001) but not for the time*surgery interaction. The TKA group showed significant variation at baseline with average stiffness of 58.855 (t=35.646, p<0.001, 95% CI: 55.600 – 62.110) and increased stiffness by an average of 4.169 at 12-months, but this effect was not significant (t=1.855, p=0.065, 95% CI: -0.262 – 8.601). The UKA group also increased stiffness by an additional 6.812 by 12-months, but this interaction effect was not significant (t=1.857, p=0.065, 95% CI: -0.419 – 14.043). Significant random effects were observed for the average variance of all subjects at baseline (estimate=124.423, z=4.516, p<0.001, 95% CI: 80.612 – 192.045) and at 12-months (estimate=72.828, z=1.975, p=0.024, 95% CI: 26.992 – 196.499).

6.4 Discussion

The key finding in this analysis is that UKA patients did not demonstrate significantly lower leg stiffness for any of the three measures during stair descent. The GLMM estimated that over time, although TKA and UKA patients both increased for 3D K_{leg}, the UKA group did not increase significantly more than the TKA group. Contrary to our experimental hypothesis, TKA generally showed lower leg stiffness over time compared to UKA patients. Both groups also showed relatively greater stiffness post-operatively, but only for 3D K_{leg}. Although descriptive statistics (Table 6.2) may suggest that subjects increased in stiffness for uniplanar K_{leg} and K_{vert}, the lack of significant fixed effects for time may be due to random variation between individuals for each measure (Table 6.3). Although means tended to increase between baseline and 12-month time periods for all three measures, the random variation between individuals should be interpreted with caution. Group means with standard deviations, as well as individual trajectories are illustrated for each method of leg stiffness across all time points. Fixed and random effects were only determined between baseline and 12-month time periods due to the assumption that the earlier post-operative time points are not linear in nature. Each measure of leg stiffness attempts to describe the compression of the limb, but each is calculated using various different components; therefore, they are discussed independently below.

6.4.1 Leg Stiffness Model 1 (3D K_{leg})

Leg Stiffness Model 1 is the most recently proposed model described by Liew et al.[10] This measure of leg stiffness is relatively new, although it is considered an expansion of the method developed by Coleman et al[7] to model the limb as a multi-dimensional unit. The hip joint center and center of pressure (COP) are used, rather than the greater trochanter and COP, for determining leg length. Reliability of gait data has been improved when using the functional landmark of the hip to estimate the hip joint center.[132] The resultant GRF is derived from vertical, anterior-posterior, and medial-lateral components of the GRF vector. The dot product of the 3D leg vector and the resultant 3D GRF vector is calculated to determine a projected GRF vector. This projection is created as a scalar quantity at each frame. The projected GRF can then be compared to the leg vector frame by frame. The peak of this projection (the vector of greatest magnitude that is most in line with the leg vector) can then be used to estimate stiffness. Therefore, the peak projected GRF and leg length change are used when determining leg stiffness. The K_{leg} coefficient is then scaled to the dimensionless expression of (%BW/LL) where BW is body weight in Newtons (N) and LL is standing leg length. This creates a relative dimensionless K_{leg} tailored to individual differences in BW and LL.

In this analysis, significant changes over time were observed for both TKA and UKA groups. Significant random variation in subjects at both the baseline and 12-month post-operative time points demonstrate the wide range of function exhibited by knee OA patients during stair descent. In examining leg stiffness with this method, subjects demonstrated significant increases in leg stiffness following surgery (Figure 6.1). Although statistically significant, these increases may not necessarily be clinically relevant in terms of neuromuscular control, given the increase in K_{leg} less than 2 %BW/LL for the TKA group and less than 3.5 %BW/LL for the UKA group between baseline and 12-month time points. Consideration for the variability between individuals should be given. Figure 6.2 illustrates the variability in K_{leg} of the TKA and UKA groups. Although there is high variability among subjects, a general trend can be observed that shows leg stiffness increasing over time for most subjects. Relative to Leg Stiffness Models 2 and 3, this method appeared to have the least amount of variability between individuals, despite the significant random effects. Furthermore, the groups do not increase at different rates. This occurrence is counterintuitive, particularly since leg stiffness describes the ability of the limb to compress under a load. We hypothesized that stiffness would decrease following surgery, given the reduction in pain and relative restoration of knee joint surface contact area provided by the implants.

6.4.2 Leg Stiffness Model 2 (Uniplanar K_{leg})

Leg Stiffness Model 2 is described by McMahon and Cheng.[5] Leg stiffness calculated in this manner considers several additional factors that appear to be more appropriate to human gait. Moderate to good reliability has been reported during running tasks,[14, 133] and a field based approach has been developed and validated.[134] Standing leg length, as well as the sweeping of the leg during the time of ground contact, are also integrated into the spring constant calculation. Incorporating these descriptors of the movement provides a richer, more in-depth characterization of the behavior of the limb during human gait. This method, however, has not been examined for its usefulness in describing limb motion during stair descent.

Analysis of K_{leg} with this method yielded interesting results. Although Figure 6.3 appears to illustrate significant changes over time, there is considerable variability between individuals (Figure 6.4). Some trends can be observed in the individual trajectories in Figure 6.4. Leg stiffness appears to decrease at the 6-week time point but returns to baseline by the 12-month time period. The UKA group shows the trend of being greater in leg stiffness than the TKA group; however, the variability between individuals makes this interpretation difficult. The raw calculation of K_{leg} using this method may speak to the variability observed in subjects. Far more components are necessary to capture the arc of leg motion that this method attempts to model; therefore, there may be more variation in K_{leg} due to variation in any combination of those individual components. Further examination of this method during stair descent is warranted due to the greater random variation between individuals.

6.4.3 Leg Stiffness Model 3 (K_{vert})

Leg Stiffness Model 3 is also described by McMahon and Cheng[5] as vertical stiffness (K_{vert}). Vertical stiffness describes the vertical displacement of the center of mass (COM) in response to a vertical ground reaction force during a task.[9, 11, 12, 14, 135, 136] It has previously been used to also describe K_{leg};[4] however, it is important to note that K_{vert} has been traditionally applied to vertically oriented tasks, such as hopping, jumping, and landing. It has also been applied to running. [9, 13, 39] The model estimates the COM displacement of the whole body, thereby including the trunk and upper limbs. Only the vertical component of the GRF is accounted for when determining deformation of the body. The use of these two components (Fmax and Δy) also inherently assumes a linear relationship between COM displacement and ground reaction force, described by Butler et al.[4] Certainly, this would necessitate the peak displacement occurring at the instant of peak force application, which may not always be the case. The use of this calculation may be appropriate for stair negotiation, seeing as it is a more vertically oriented task than level walking; however, it has not been observed in the literature during stair descent or step-down tasks.

When examining K_{vert} in the current sample, no significant changes over time were observed between the groups. Although a significant fixed effect was observed for time, this is an overall effect accounting for the total subject pool. Individual groups did not significantly change over time, though trends are observed between groups that might suggest the UKA group increased in stiffness relative to the TKA group (Figure 6.5). As with Leg Stiffness Model 2, the individual subject variability in K_{vert} was significant at baseline and the 12-month time period (Figure 6.6). Leg stiffness tended to increase, but the variability between subjects makes this interpretation difficult. These trends suggest that although UKA subjects may appear to be higher in leg stiffness, they also show greater variation in the amount of leg stiffness between individuals. Although direct comparison cannot be done between these measures of leg stiffness, we speculate that K_{vert} overestimated leg stiffness given the moderate differences in spring constant values between the measures.

6.4.4 Limitations

Several limitations should be acknowledged. In the TKA group, patients receiving both multi-radius and single-radius femoral components were included. The degree to which the implant influences leg compression ability during loading is unclear. Given previously theorized influences on the extensor mechanism, this may introduce an additional confounding factor. Another limitation is that only medial UKA patients were included, therefore the results may not be generalizable to lateral compartment UKA patients. Additionally, measures of leg stiffness were not standardized, caution should be used when interpreting different leg stiffness methods. Given that each calculation is measured with various metrics, the results may not be generalizable to studies incorporating different measures of leg stiffness. Leg stiffness measures that are modeled with direct kinetic-kinematic methods may be subject to changes in GRF associated with willingness to load; therefore, increases in stiffness observed following surgery may have been a consequence of patients increased willingness to load, rather than neuromuscular control of the limb. Lastly, random variation between individuals may be seen as a limitation. This may be explained by lack of standardization in rehabilitation protocols, logistical barriers dictating patient ADLs, or overall patient activity levels. Given the relatively limited sample size for the UKA group compared to the TKA group, this may have also contributed to the greater variation leg stiffness in the UKA group.

6.5 Conclusions

Both TKA and UKA subjects tended to increase in leg stiffness during stair descent between baseline and 12-month post-operative time points. Each method of leg stiffness demonstrated considerable variation between individuals, and this likely influenced between-group analysis across time. It is difficult to draw conclusions to recommend a method of leg stiffness to report during stair negotiation, due in part to the natural variability associated with stair negotiation trials

6.6 List of Tables

Table 6.1. TKA vs UKA Baseline Demographics

	TKA (n=27)	UKA (n=16)	p	g
Age (years)	64.26 ± 5.81	64.44 ± 4.90	0.919	0.032
Height (m)	1.65 ± 0.10	1.69 ± 0.08	0.148	0.457
Mass (kg)	80.90 ± 16.35	86.53 ± 16.30	0.281	0.338
BMI	29.72 ± 4.77	30.28 ± 5.08	0.720	0.112

Data are presented as mean ± standard deviation; m=meters; kg=kilograms; TKA=total knee arthroplasty; UKA=unicompartmental knee arthroplasty; p=p-value; g=Hedge's g effect size

Table 6.2. Descriptive Statistics of Leg Stiffness Models Across Time for TKA and UKA Patients During Stair Descent

	Model 1 (3D Kleg)				Model 2 (Uniplanar Kleg)		Model 3 (Kvert)	
	TKA		UKA		TKA	UKA	TKA	UKA
Pre-operative	n=25	15.07 ± 3.90	n=18	14.68 ± 4.76	26.97 ± 11.92	32.89 ± 14.87	57.24 ± 10.97	57.29 ± 13.36
Post-operative								
6-weeks	n=14	16.15 ± 3.55	n=16	18.44 ± 4.44	26.06 ± 12.89	31.05 ± 16.94	56.64 ± 8.12	65.55 ± 16.87
3-months	n=22	17.61 ± 3.99	n=16	16.68 ± 7.72	27.29 ± 16.15	36.37 ± 16.12	59.42 ± 10.35	66.72 ± 18.13
6-months	n=31	17.64 ± 4.22	n=16	18.01 ± 4.29	29.46 ± 15.10	40.66 ± 15.16	61.80 ± 10.75	66.90 ± 14.59
12-months	n=32	17.78 ± 4.19	n=16	20.19 ± 7.03	28.48 ± 12.20	41.32 ± 17.15	60.27 ± 9.30	72.77 ± 18.66

Data are expressed as mean ± standard deviation; n=sample size (knees); TKA= total knee arthroplasty; UKA=unicompartmental knee arthroplasty K=stiffness coefficient; 3D=three-dimensional

Table 6.3. Generalized Linear Mixed Model Fixed and Random Effects For Each Model of Leg Stiffness

	Model 1 (3D K_{leg})						Model 2 (Uniplanar K_{leg})						Model 3 (K_{vert})					
Fixed Effects																		
					95% CI						95% CI						95% CI	
	Coefficient	Std. Error	t	Sig.	Lower	Upper	Coefficient	Std. Error	t	Sig.	Lower	Upper	Coefficient	Std. Error	t	Sig.	Lower	Upper
Intercept	15.98	0.56	28.504		14.87	17.08	28.95	1.87	15.475		25.26	32.63	58.85	1.65	35.646		55.60	62.11
Time	1.84	0.93	1.975	**0.050**	0.00	3.68	1.04	2.44	0.427	0.670	-3.77	5.86	4.17	2.25	1.855	0.065	-0.26	8.60
Time*Group	1.49	1.52	0.981	0.328	-1.51	4.49	5.89	3.85	1.530	0.128	-1.70	13.48	6.81	3.67	1.857	0.065	-0.42	14.04
Random Effects																		
					95% CI						95% CI						95% CI	
Variance	Estimate	Std. Error	Z	Sig.	Lower	Upper	Estimate	Std. Error	Z	Sig.	Lower	Upper	Estimate	Std. Error	Z	Sig.	Lower	Upper
Baseline	11.88	3.20	3.709	**0.000**	7.00	20.15	135.99	32.79	4.147	**0.000**	84.78	218.15	124.42	27.55	4.516	**0.000**	80.61	192.04
12-months	17.65	6.98	2.529	**0.006**	8.13	38.32	23.45	49.59	0.473	0.318	0.37	1478.51	72.83	36.88	1.975	**0.024**	26.99	196.50

For Fixed Effects: Intercept is interpretted as the group coded "0" (TKA) at Time "0" (Baseline); Time is interpretted as TKA at Time "1" (12-months); Time*Group is interpretted as the group coded "1" (UKA) at 12-months.

For Random Effects: Baseline is interpretted as all subjects at Time "0" (Baseline); 12-months is interpretted as all subjects at Time "1" (12-months).

t= t-statistic; Z=z-stastic; CI=confidence interval; p=significance ($p \leq 0.05$).

6.7 List of Figures

Figure 1. Mean 3D K$_{leg}$ across time for TKA (blue) and UKA (green) operated knees. Error bars represent one standard deviation above and below the mean.

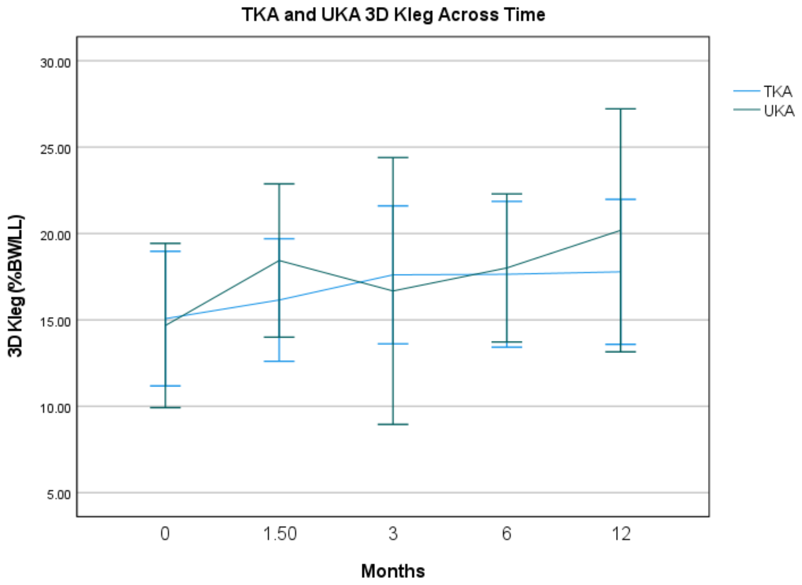


Figure 2. Individual 3D K$_{leg}$ across time. Each bar represents each individual subject (knee) during the stance phase of stair descent.

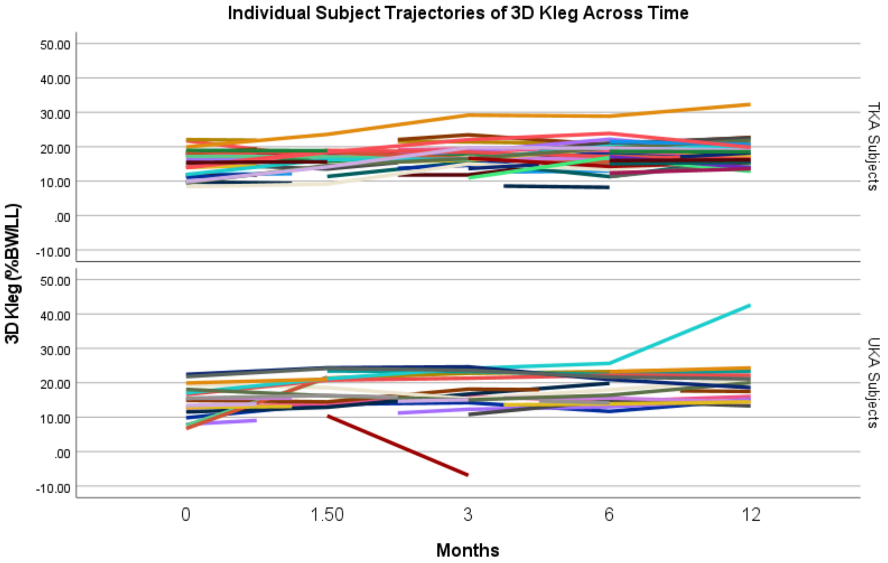


Figure 3. Mean uniplanar K_{leg} across time for TKA (blue) and UKA (green) operated knees. Error bars represent one standard deviation above and below the mean.

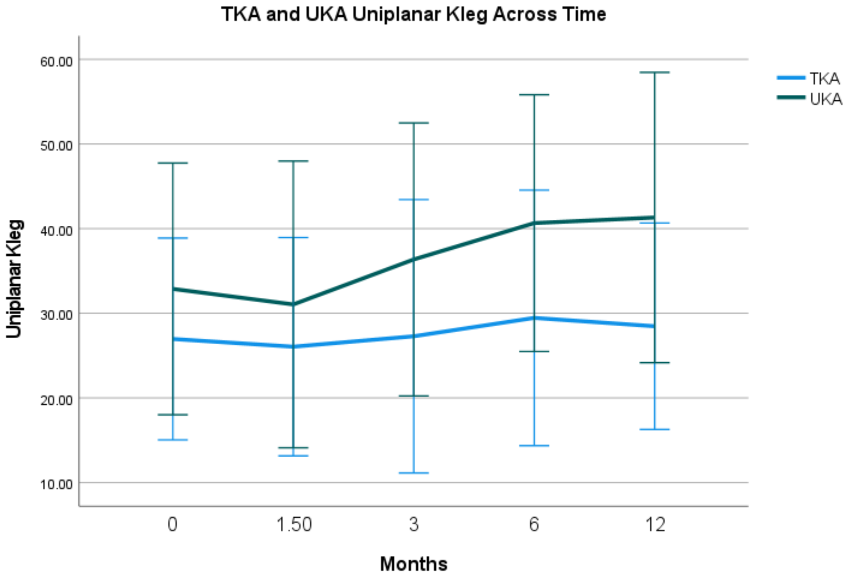
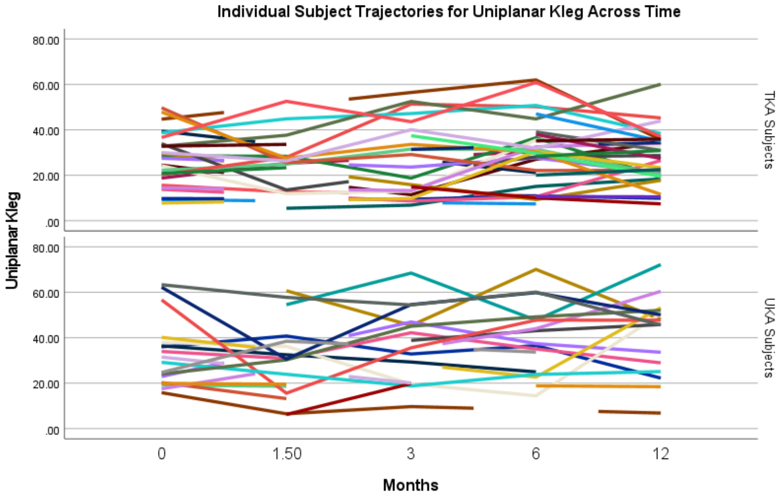


Figure 4. Individual uniplanar K_{leg} across time. Each bar represents each individual subject (knee) during the stance phase of stair descent.



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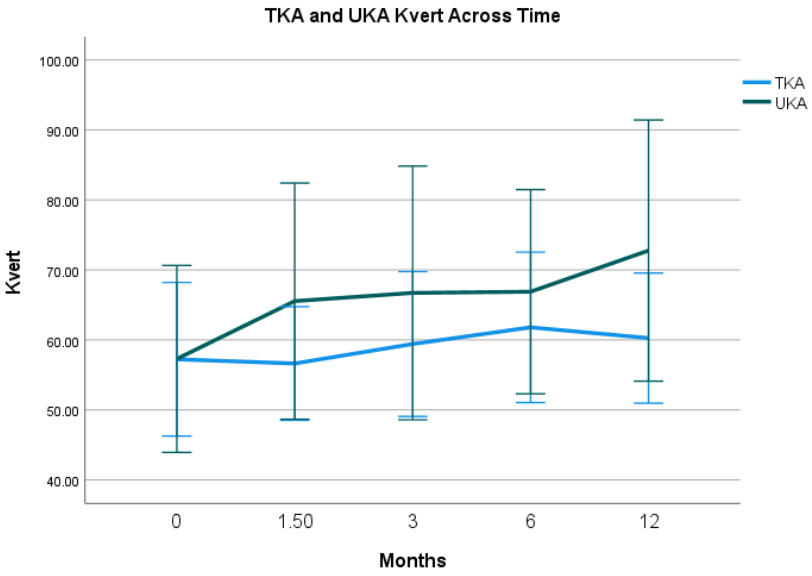
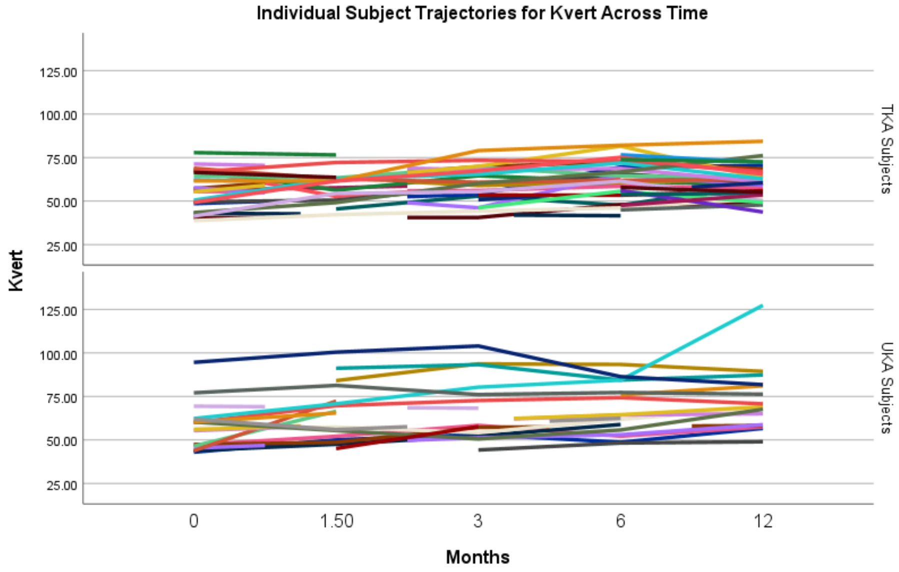


Figure 6. Individual K_{vert} across time. Each bar represents each individual subject (knee) during the stance phase of stair descent.



PART II

REVIEW OF LITERATURE

THE SENSORIMOTOR SYSTEM AND METHODS OF ASSESSING JOINT POSITION SENSE

The Sensorimotor System

The sensorimotor system that allows humans to control complex movements is vast and highly integrated. The work of Charles Sherrington provided significant contributions to understanding the integration of the peripheral nervous system with the central nervous system to facilitate balance, coordination, and movement.[137] Sherrington is largely credited for advancing the early mapping of the motor cortex[138] and for the later acceptance of the corticospinal tract's involvement with motor control.[139] The corticospinal tract consists of a white matter pathway from the primary motor cortex to various levels of the spinal cord. Axons descend from the precentral gyrus into the brainstem to form cerebral peduncles, where they continue into the medulla to form pyramids, or ridges, of the brainstem. The axonal fibers cross the brainstem at the base of these pyramids to form the lateral corticospinal tract. A small number of remaining fibers will not cross but continue ipsilaterally down the spinal cord to form the ventral corticospinal tract. Axons in the corticospinal tract will synapse with lower motor neurons to produce movement of muscles in the limbs, trunk, and neck. Given the array of originations in both the primary motor cortex and the somatosensory cortex, the corticospinal tract is involved with both efferent motor responses and afferent sensory stimulation conduction within the central nervous system.[139] The second pyramidal tract is termed, the corticobulbar tract, which also originates in the primary motor cortex of the frontal lobe. Whereas the corticospinal tract is responsible for motor response coordination to the limbs and trunk, the corticobulbar tract consists of motor pathways for controlling muscles of the face, head, and neck.[140]

Five traditional senses: taste, smell, vision, hearing, and touch, are widely accepted as the primary senses through which humans interact with the surrounding environment[1] describe the interaction of these systems in great detail. The interaction of the senses of position and movement of our limbs and trunk, the sense of effort, the sense of force, and the sense of heaviness are integrated via afferent input and effect motor responses. Balance via the vestibular system is also highly integrated with these other systems. Furthermore, a "sense of innervations" has previously been theorized and is now known as proprioception. Proprioception can be defined as the collective afferent input provided by receptors throughout the limb, including the joints, muscles, and surrounding connective tissues that provide for kinesthetic awareness of body or joint position.[1, 141] More specifically, joint kinesthesia pertains to limb position and the relative position of each joint within the specified segment. Kinesthesia is accepted as a surrogate of joint proprioception, as it is not necessary to actively contract a muscle and produce movement of a joint to be aware of its position.[1]

An intriguing concept of body perception has drawn some attention to how the brain and remaining sensorimotor system interact.[142, 143] Paillard et al[142] describe the concept of body image and body schema as it relates to dissociative awareness of the body. Body image versus body schema is a distinction that should be made; body image is the cognitive representation of the body based on stored knowledge and experience; whereas body schema is dependent on ongoing proprioceptive input and is concerned with body movements. Patients with specific afferent sensory deficits have been able to identify the area of skin that was stimulated by either thermal or tactile sensation, despite not having the capability to actually feel the applied stimulus. In a case report, Anema et al[143] indicate that individuals might rely on stored knowledge of the body predict movements. In the same case report, a different subject could not identify where on her arm she had been touched but could point to the exact location on a line drawing of the hand. This would indicate a disturbance in body schema.[143] The concept of body image vs schema seems to apply directly to proprioception and afferent stimulation and may be an underlying factor in determining how the vast sensorimotor system is integrated with the psyche. Awareness in this sense inherently requires either direct or indirect pathways to the motor cortex, along with the midbrain, hindbrain, spinal cord, and cerebellum for decision making and movements.[144-146] Cote et al[144] describes these complex interneural pathways as both a first-order and a last-order neuronal arrangement, depending on the context of the relative function, and that function is reflected in the pathway of nerve fibers that they connect. The peripheral and central nervous systems are meticulously interconnected by series of interneurons that serve as multifunctional mediators between afferent and efferent nerves.

Muscle Afferent Pathways

Proprioceptive input is extensive, particularly to load bearing joints. Knoop et al[48] discusses the importance knee joint mechanoreceptors and the consequences of knee OA. The knee is one of the primary load-bearing joints, if not *the* primary load-bearing joint.

Mechanoreceptors providing afferent sensory information within and around the tibiofemoral and patellofemoral joints are highly integrated with the spinal column and the brain itself. Mechanoreceptor communication to the spinal cord is accomplished via several different pathways, named as Group I-IV, as described by Lance and McLeod.[145] Group I afferents are characterized by the highest conduction velocity and largest diameter, being further subdivided into "A" and "B" subgroups. Group 1A muscle afferents originate in the primary endings of muscle spindles. The afferent signals travel within peripheral nerves and synapse within the gray matter of the spinal cord with alpha motor neurons. Group 1A afferents project to the cerebellum and the sensorimotor cortex, thereby playing a prominent role in joint kinesthesia.[144, 145] Group 1B afferent signals, on the other hand, originate from Golgi Tendon Organs (GTO). These mechanoreceptors are found primarily in muscle tendons, but also within muscle tissue in close proximity to the musculotendinous junction. A theory posited by Houk and Henneman[147] suggests that GTO's are an indicator of active contraction of each motor unit, which has led to the consensus that GTO's detect tension changes in muscle fibers, rather than muscle length changes. Group I afferents have been found to contribute to functional knee joint stability due to their high conduction velocity and sensitivity to muscle length changes associated with functional performance and the stretch reflex. This phenomenon is described by Proske et al[1] and Friemert et al.[24] Group II afferents originate from the secondary endings of muscle spindles but are only sensitive to changes in length of a muscle. This afferent pathway has been thought to be responsible for varying degrees of medium latency stretch reflexes, particularly during gait, observed in the lower extremity. Two examples of this phenomenon are described by Grey et al [148] with the gastrocnemius and by Roujeau et al[149] with the semitendinosus. The exact neuronal pathway has not been determined in these cases, however, emphasized by Cote et al's examination of interneural pathways.[144] The roles of Group III and IV afferent fibers have been questioned but seem to have a major role in autonomic responses during exercise.[150-152]

Smith et al[150] describes Group III and IV muscle afferents originating in skeletal muscle and being responsible for autonomic responses. They are stimulated by mechanical and metabolic receptors within muscle fibers themselves and have been shown to have inhibitory effects of central motor command. Stimulation will facilitate central fatigue. Nobrega et al[152] suggest that continuous afferent feedback is necessary in order to invoke ventilatory and circulatory responses that facilitate muscle function, highlighting the importance of peripheral function in regulating central control mechanisms during activity. Messlinger et al[153] and Porreca et al[154] suggest that Group III and IV afferent signals also include nociceptive receptor activity, or the sensation of pain, and may be responsible for sending signals of pain via peripheral nerves from structures within the knee joint, supported by Bendtsen et al.[155] Muscle pain is also thought to be generated by group III and IV afferent pathways via stimulation by the presence of various substances. Marcora et al[25] indicated lactic acid and Kaufman et al[156, 157] indicated chemical nociceptive mediators and mechanical stimulus of muscle lengthening would stimulate muscle pain via Group III and IV afferent pathways. These mechanoreceptors serve as the first order neuronal units that convey proprioceptive information to the spinal cord and are subsequently relayed through the central nervous system to the cerebellum via the spinocerebellar tract.[1] Their cell bodies lie within the dorsal root ganglion and travel via this pathway to the cerebellum, where unconscious proprioceptive information is processed.

The Roles of Mechanoreceptors

Muscle spindles embedded within intrafusal muscle fibers play a prominent role in the conscious and unconscious awareness of joint position, but GTO's within tendons and ligaments, skin receptors, and articular mechanoreceptors contribute in various ways.[2, 48, 158, 159] Muscle spindle activity is widely viewed as the primary proprioceptive mechanism, with varied importance of articular mechanoreceptor activity.[1] Velocity and acceleration changes associated with muscle length changes are also detected by muscle spindles. Primary and secondary endings of the spindle respond differently, with primary endings responding to velocity and magnitude of the length change. Secondary endings are sensitive only to length changes. Generally, muscle spindles have heightened sensitivity to low-speed manipulations, given the fact that both endings respond to length, while only primary endings respond to velocity.[1]

Golgi tendon organs are generally sensitive to tension rather than a mechanical stimulus of lengthening. The relationship between muscle spindle activity and GTO activity cannot be ignored, though Proske et al[1] suggest from their review that GTO activity is a primary contributor to proprioceptive input when undergoing an increased load. Interestingly, GTO presence is also observed in ligaments and synovial joint capsules, albeit in different saturations, indicated by Moore.[141] Consequently, GTO's can be stimulated by changing a joint angle, whether actively or passively. Due to their intimate relationship, GTO's and muscle spindles work synergistically to provide sensory feedback regarding whole muscle-tendon unit activities.

In addition to GTO activity, several other articular mechanoreceptors contribute to joint proprioception. Proske et al[1] and Moore[141] summarize articular mechanoreceptors. They can be broadly defined as either slow-adapting or quick-adapting in nature. Mostly engrained within the joint capsule, these mechanoreceptors respond to different stimuli. Pacinian corpuscles are observed in ligaments, joint capsule, and the menisci. These receptors are generally quick-adapting and respond to dynamic changes in tissue deformation, thereby mediating the sensation of joint movement during high velocity movement scenarios such as changes of direction. Ruffini endings are also observed within articular structures. Contrary to Pacinian corpuscles, Ruffini endings are slow-adapting and respond to intra-articular pressure changes. Straining of tissue associated with extreme joint angles will also serve as a stimulus. Bare nerve endings are found in various tissues in and around the knee joint and are stimulated by excessive tissue deformation, pain, and inflammation. When there is no strain placed on the joint capsule and ligaments, afferent neurons are relatively inactive; therefore, it is argued that joint afferent signals from articular mechanoreceptors serve as limit detectors. Craske[160] described this limit detection, given the overriding ability of the brain to falsely detect impossible joint positions when a tendon is vibrated. This may be a contributing factor to injuries received when tissue oscillation creates misinterpreted signals through afferent pathways.

Skin receptors are also considered part of the proprioceptive system. Proske et al[1] and Johansson et al[161] summarize these in their analyses of the proprioceptive system. Meissner corpuscles (quick-adapting), Pacinian corpuscles (quick-adapting), Merkel endings (slow-adapting), and Ruffini endings (slow-adapting) are the four specializations of mechanoreceptors found in skin and subcutaneous tissue. Skin receptors are able to sense changes in joint position via stretch responses that occur concomitantly with muscle action. Johansson et al[161] found that perceived joint motion can occur when skin is stretched without actual joint motion occurring. This is likely due to the vast network of previously described interneurons and the multiplicity of their functions.

Blalock et al[162] also describes the potential role that articular cartilage may play in joint stability. Articular cartilage is largely devoid of nerve innervation, vasculature, and overall cellular components. It is primarily composed of water. Loading of the cartilage and menisci stimulates chondrocytes and appears to disrupt the extracellular matrix, leading to degeneration. The exact role (and mechanism) that loading patterns on articular cartilage have in mechanoreceptor stimulation is not clear, though it is apparent that the end result is degeneration. The aging process may play a role in the diminishment of proprioceptive ability; however, conflicting results have been found between healthy and osteoarthritic knees in osteoarthritis (OA) patients, as described by Sharma et al.[22] Bennell et al[36, 37] found associations of sensorimotor dysfunction with poor loading characteristics and increased pain in OA patients. As articular cartilage is largely devoid of neurovascular structures and mechanoreceptors, the nature of observed proprioception deficits remains unclear; however, some authors suggest that subsequent joint laxity and pain may override articular mechanoreceptor afferent signals in the osteoarthritic knee, creating abhorrent loading and movement patterns that lead to cartilage degeneration.[1, 162]

Mechanoreceptors relay afferent signals that must be transmitted through corresponding nerve pathways to the level of the respective spinal root. Afferent signals are transmitted to the spinal column and efferent motor signals exit the spinal column through the levels of T1-L2.[163] Despite the afferent signal reaching the somatosensory cortex of the brain, much of the afferent signal is suppressed and many self-initiated movements do not reach conscious level. Blakemore et al[164] demonstrated this with the tickling response in situations of self-induced tickling versus external stimuli. The afferent (and efferent) responses vary drastically due to the feed forward mechanism that occurs in anticipation of efferent responses during self-initiated movements.

The aforementioned mechanoreceptors and their respective pathways contribute holistically to proprioception. It is clear that in this capacity, proprioception contributes to neuromuscular control of the joint during complex movement tasks. Three presumed functions of proprioception in this regard have been described:[1, 2, 47] 1) protection against excessive and possible injurious movements via reflex responses; 2) stabilization of the knee during static posture; and 3) coordination of complex movement systems and precise knee joint motions. The communication between peripheral afferent signals and central motor commands that execute these functions has been studied in both humans and other animals. Coordination of afferent stimuli and efferent motor responses is critical for optimal, healthy performance. In a sense, neuromuscular control of the lower extremity is the combined, coordinated effort of agonist and antagonist muscle function. The combined responses of muscle function and forces creating tissue deformation lead to reflexive reactions to both anticipatory movements and external stimuli, not in the least of which in the lower extremity.[19]

Methods of Assessing Proprioception

The assessment techniques designed to evaluate these various pathways have been reviewed[2, 17, 47, 159] in different healthy and pathologically impaired populations, including, but not limited to cerebral palsy,[23] osteoarthritis,[36, 37, 44, 48] stroke,[21, 165]and ligament and tendon injuries at the knee.[34, 166] Assessment of knee joint proprioception has been traditionally executed with one or more of several different techniques. Han et al[2] reviewed these methods and describes three commonly used methods as: threshold to motion detection, joint position reproduction, and active movement discrimination. Hillier et al[47] also describes these three methods as falling into two main proprioceptive functions: detecting static position and detection of motion. More recently, joint force sense has also been established as a method for assessing knee joint proprioceptive ability.[49, 167, 168]

Knee joint angle reproduction has been established as a valid and reliable technique to assess proprioceptive acuity.[3, 16, 28, 29, 169] Testing in this manner can be conducted in open (OKC) and closed kinetic chain (CKC) positions, though OKC testing has been more widely examined according to a review conducted by Hillier et al.[47] Arvin et al[29] assessed the reproducibility of these methods with 19 healthy older adults. Proprioception of the hip (flexion and abduction) and knee (flexion) were assessed in both legs using the "active-active" reproduction technique. Intraclass correlation coefficient (ICC), standard error of measurement (SEM), and limits of agreement (LOA) were estimated for relative angular error (RE), absolute angular error (AE), and variable angular error (VE). The ICC values ranged from 0.75 to 0.93, indicating excellent reliability of the JPS test. Similar findings were observed for AE with knee flexion and hip abduction of the left and right leg. The ICC results of VE showed poor reliability for both joints. The SEM and LOA values for hip abduction were generally lower than for hip and knee flexion, indicating lower measurement error or more precise scores for the proprioception test of hip abduction. Similar findings for JPS reliability have been found with different populations, and under various conditions, by Suner-Keklik et al,[28] Baert et al,[16] and in a series of studies conducted by Relph and Herrington.[3, 169] Additionally, this method has been validated and found reliable in a CKC position by Romero-Franco et al.[50] They examined 10 athletes (5 men and 5 women; 26.2 +/- 1.3 y, 71.7 +/- 12.4 kg; 1.75 +/- 0.09 m; 23.5 +/- 3.9 kg/m^2) and measured absolute angular error (AAE) of knee JPS in a CKC standing position. The ICC and SEM were calculated to determine the validity and reliability of the inclinometer. High agreement was detected (ICC = 1.0, SEM = 1.39, $p < 0.001$), and there was exceptionally good intra- and inter-tester reliability for reading the inclinometer (ICC = 1.0, SEM = 0.85, $p < 0.001$).

Joint position sense itself is comprised of several different methodological approaches for evaluating proprioception, and each approach has advantages and drawbacks. Muscle spindle activity is the primary contributor of afferent stimulation during reproduction of joint positions, regardless of the method. The most advantageous and functional method is active joint position reproduction, during which the subject actively attempts to reproduce an established reference position. Muscle spindles, skin stretch receptors, and articular mechanoreceptors are most excitable during active JPS. The obvious strength of this method is that it is representative of a more functional use of the proprioceptive receptors. An added benefit of this method is that additional pressure sensation from a device or clinician is given, which would be required during passive JPS and may convolute test results by influencing skin receptor activity.[170] Passive joint position detection involves the subject recognizing a previously established joint position when a device or clinician reproduces it passively. Muscle spindles continue to be the most excitable mechanoreceptor; however, Weerakkody et al theorized that secondary endings are the main contributor due to the lack of *active* muscle fiber length changes being induced.[158]

Passive motion detection threshold can also be assessed, which involves identifying the threshold (point at which motion is felt) that a subject feels a joint moving from the stationary position. The advantage of this method is that it does not require a working memory of the reference position; however, it is highly dependent on the velocity of the motion.[171] Thresholds for detection are much higher for slower movements. This method has been examined in OA patients,[44, 172] in order to better understand how articular mechanoreceptors might influence knee joint proprioception. In passive motion detection, active contractions of musculature are not exercised by the participant. It would seem that this essentially eliminates a large, though complexly quantifiable, amount of muscle spindle and GTO afferent feedback. Active joint angle reproduction also causes a greater amount of pain in individuals with OA, which can be avoided to a certain extent with passive motion detection. Cammarata et al[44] assessed proprioception in knee OA patients. The experimental group included 13 patients and 14 healthy age-matched controls. Proprioception was assessed in knee varus, valgus, flexion, and extension using threshold to detection of passive movement (TDPM) tests. Repeated-measures analysis of variance was used to assess differences in TDPM between the groups and across movement directions. Linear regression assessed the correlation of the TDPM between and within planes of movement. The TDPM was significantly higher ($P<0.05$) in the knee OA group than the control group for all directions. Differences in the TDPM between groups were consistent across all movement directions, with mean differences as follows: for valgus, 0.94 degrees (95% confidence interval [95% CI] 0.20-1.65 degrees); for varus, 0.92 degrees (95% CI 0.18-1.68 degrees); for extension, 0.93 degrees (95% CI 0.19-1.66 degrees); for flexion, 1.11 degrees (95% CI 0.38-1.85 degrees). Despite these results, only weak correlations were detected between the planes of movement.

The final method of assessing JPS is passive motion direction discrimination. This method involves a subject describing the direction of the motion, which may require awareness of movement about one or more axes of joint rotation, particularly when two joint muscles are involved. Brindle et al[173] examined this in ankle motion. Healthy volunteers sat with knees flexed at 20° and were passively rotated at three velocities (0.5, 2, or 10°/s). Detection of passive movement sense (DPMS) was the angular movement before activation of a thumb-switch. Significant differences ($P=0.003$) in the rate of change in DPMS across a variety of movement velocities was observed but shortening or elongation of the gastrocnemius did not affect DPMS. Gastrocnemius elongation/shortening did not appear to affect knee proprioception in this regard; however, these findings were observed in healthy subjects. The advantage of this technique is that it does not require active memory control of the joint being tested, which may be advantageous for understanding proprioceptive ability in populations who have varying degrees of memory deficits such as with stroke patients.[174] Memory impairments may have a deleterious effect on joint angle reproduction ability; therefore, a simpler method of detecting the direction of motion may be more advantageous in this population. This may also have benefits in identifying proprioceptive deficits in individuals with cerebral palsy.[23] The ability to discriminate the direction of a movement might give insight into the basal ganglia’s ability (or lack thereof) to relay messages to the muscles. This area has not been well researched, and further investigation into different methodological approaches is warranted.

Joint force sense (JFS) ability has emerged as a method for examining proprioception in an even greater functional capacity than joint angle reproduction that accounts for force accommodations made by muscles during activity.[168] Testing in this manner involves the subject either reproducing a force in the ipsilateral limb based on memory or reproduces the same target force in the contralateral limb. Joint force sense incorporates and emphasizes the afferent input of GTOs more readily than any other afferent mechanoreceptor at the knee. This is apparent through the GTO function of sensing tension changes in muscle, rather than muscle length changes. Niespodzinksi et al[168] employed this method to examine 17 elite adult gymnasts and 24 untrained, matched controls. Active reproduction (AR) and passive reproduction (PR) tasks and a force reproduction (FR) task at the elbow joint were examined. They evaluated between-group differences as well as correlations between JPS and FR tasks. Absolute error in the control group was higher during the PR task (7.15 ± 2.72°) than during the AR task (3.1 ± 1.93°). They also reported mean relative error in the control group was 61% higher in the elbow extensors than in the elbow flexors during 50% FR, while the gymnast group had similar results in both reciprocal muscles. No correlations between JPS and FR were observed; however, FR was negatively correlated with antagonist muscle strength.

Recent attempts have been made to standardize the application of JFS methods by using dynamometry but have demonstrated poor to fair interrater reliability with excellent intra-rater reliability.[16, 167] The primary limitation of JFS is the necessity for electromyography or isokinetic dynamometry equipment to measure maximum voluntary contractions of muscle groups more accurately. Furthermore, Han et al[175] suggests that the perception of force and motion are experienced through different pathways altogether, given no differences in finger pinch discrimination ability at varying elastic resistances. This could perhaps be because tactile and mechanoreceptor feedback are greater in the hands, demonstrated in the ability of an individual to perform fine-motor tasks with the hands and fingers. Another limitation of JFS is the variability in force resistance provided by individual assessors; without the use of an isokinetic device, muscle contractions are limited to isometric contractions.[16] Although this may be a limitation, the previously described function of GTOs might take precedence in a position of an isometric contraction, given their sensitivity to muscle tension increases. Further research should be conducted in this area to better understand the relative contribution that force detection has on proprioceptive ability.

Recently, CKC testing has emerged as a reliable method with regards to testing the knee joint.[28, 50] Clear advantages present themselves when conducting JPS testing in a CKC manner; though, complications arise in the interpretation of how knee joint mechanoreceptors, muscle spindles, and various other receptors interact. In an OKC position, the direction of motion may influence the proprioceptive input that the knee provides, mainly due to the muscle contraction necessary of the quadriceps to resist gravitational acceleration during active knee extension.[17] With emerging CKC testing, the direction of reproduction mimics a single leg squat (or double leg), which requires the knee to actively flex, essentially eliminating the resistance against gravitational acceleration. By placing the limb in a load-bearing position, additional peripheral afferents are signaled from articular mechanoreceptors in the knee joint itself, but also from the foot, ankle, and hip.[1, 50]

Each method of assessing knee joint proprioception has distinct advantages and disadvantages that may be applied to varying populations. The elderly population is of great interest due to the increased likelihood of falling.[51] The arthritic changes often experienced with aging may be associated with proprioception deficits. Gait asymmetries have also been observed, potentially contributing to fall risk.[176] Age related changes in proprioceptive ability have been attributed to arthritic changes about the knee joint[177], but the exact mechanism through which afferent signal disruption occurs is unclear. Postural sway relating to fall risk has been shown to be heightened in elderly patients, but the role that proprioception plays during balance activities is difficult to identify due to involvement of the vestibular system.[1, 178] The most well documented theory describes muscle spindle changes associated with aging in rats,[179, 180] and in humans.[181] Each of these authors describes the accompaniment of muscle spindle changes with loss of large, myelinated sensory fibers and distal mechanoreceptors. Shaffer and Harrison[181] observed widespread decreases in quantities and morphological changes of muscle spindles, GTO's, Ruffini endings, and Pacinian corpuscles have been observed in aging versus young adults. Mechanoreceptors lose their innate microcellular structures, thereby losing effective function. Examples include muscle spindles losing their conical, spring-like structure, Pacinian corpuscles flattening and losing surface area, and Ruffini endings becoming frayed. These changes likely contribute to proprioceptive deficits observed in elderly individuals.

EXAMINING LEG STIFFNESS AS A MEASURE OF NEUROMUSCULAR CONTROL

Leg Stiffness Principles

Lower extremity stiffness has been examined in various settings and the concept is grounded in the spring-mass model. The most basic definition of stiffness originates from Hooke's Law:

$$F = Kx$$

A force (F) required to deform a material or body is related to the spring constant (k) and the distance (x) that the material is deformed. The spring constant (K) can be termed "stiffness" and applied to the lower extremity during human gait.

Higher values of K would indicate a stiffer object, in this case the leg, whereas lower values of K would indicate a more compliant limb. Maloney and Fletcher[12] and Lorimer et al[11] conducted recent reviews of stiffness models. Vertical stiffness (K_{vert}) describes the vertical displacement of the center of mass (COM) in response to a vertical ground reaction force during a task[9, 12, 135] and was first described by McMahon and Cheng.[5] Vertical stiffness has previously been used to also describe leg stiffness (K_{leg}), though Butler et al[4] note that K_{vert} should be applied to vertical oriented tasks, such as hopping, jumping, and landing, though it has also been applied to running.[9, 13, 39]

Leg stiffness is a more precise measure and describes the compression of the leg itself, as if being modeled as a spring, in response to a force that attempts to deform it in any plane or direction.[4, 5, 8] Sagittal plane kinetics and kinematics have been evaluated primarily due to larger excursions through available ranges of motion observed during human walking and running gait; however, relatively recent models have attempted to account for multiplanar[7] and triplanar[10] joint contributions. Vertical and leg stiffness should be considered aggregate measures of lower extremity stiffness. Joint stiffness (K_{joint}) attempts to describe the angular displacement of a single joint in response to the moment at the joint.[182, 183] The determination of stiffness is largely dependent upon the computational method, evident in the wide array of calculations available;

therefore, it is important to consider both the task and computational method when calculating stiffness in order to avoid misrepresenting leg stiffness.[184]

Vertical Stiffness

In the earliest and simplest models of K_{vert}, stiffness is represented in several different ways.[5, 135, 185] All attempt to aggregate leg stiffness, though the most widely used definition is expressed as the ratio of maximum vertical ground reaction force (Fmax) and the maximum vertical displacement of the center of mass (Δy):[5]

$$Kvert = Fmax/\Delta y$$

Joseph et al[133] and Pappas et al[14] report good reliability for K_{vert} during running and hopping tasks. The most easily perceived advantage of this method is that it is relatively simple. While ease of calculation is apparent, it requires the use of a force platform and inverse dynamics to estimate the center of mass. A derivation from this method has been developed in an attempt to mitigate the necessity of a force platform by substituting a contact mat, as described by Dalleau et al.[186] Despite the ease of calculation and applicability to field research,[187] the most pressing drawback of this method is that it is not a true representation of leg compression. The model estimates the COM of the whole body, thereby including the trunk and upper limbs. Furthermore, only the vertical component of the ground reaction force is accounted for when determining deformation of the body. The use of these two components (Fmax and Δy) also inherently assumes a linear relationship between COM displacement and ground reaction force, described by Butler et al.[4] Certainly, this would necessitate the peak displacement occurring at the instant of peak force application, which is not always the case. Including only the maximum vertical force significantly limits the application to purely vertical tasks, as previously described.

Different calculations for Kvert have been proposed for various situations. McMahon et al[185] proposed one of these variations in K_{vert}, calculated as:

$$Kvert = m\omega_0^2$$

Where *m* is the mass of the body, and ω_0 is the natural frequency of oscillation of a rigid body. The natural frequency of oscillation is calculated using contact time and the time in the air between successive foot strikes, derived from two force plates or a treadmill; the latter of which can use foot switches rather than foot strike. The application of this method may be more appropriate during running, given that it incorporates contact time and in cases where perceived leg stiffness would be decreased, such as in Groucho running. Groucho running was demonstrated by McMahon et al[185] to increase the work required by the knee extensors to resist gravitational acceleration while also attenuating the spring constant compared to normal running gait. Using this method may be more applicable in scenarios that stiffness can be evaluated but the subject cannot run with a normal or typical running gait pattern that is frequently observed in healthy individuals.

Yet another form of K_{vert} was proposed by Cavagna et al.[136] In this method, K_{vert} is calculated as:

$$Kvert = m(\frac{2\pi}{P})^2$$

Where *m* is the mass of the body, and P is the period of vertical vibration. This method assumes that running, trotting, and hopping are considered to be successions of vertical bounces of the body. The period of vertical vibration incorporates the vertical acceleration of the body and contact time with the ground, which describes the rebound behavior of the spring-mass system. This method appears to be limited to vertically oriented tasks, though the authors extrapolated its use to hopping and running tasks.

Leg Stiffness

The traditional, uniplanar model of leg stiffness uses Hooke's Law principles; however, the leg length change can be calculated with a larger number of components. Leg stiffness (K_{leg}) has been broadly expressed as the ratio of the maximum vertical ground reaction force (F_{max}) to change in leg length (ΔL):

$$Kleg = \frac{Fmax}{\Delta \mathrm{L}}$$

Where $\Delta L = \Delta y + L_0(1 - \cos\theta)$ and $\theta = \sin^{-1}(ut_c/2L_0)$; L_0 = standing leg length θ = half angle of the arc swept by the leg, u = horizontal velocity, and t_c = ground contact time. McMahon and Cheng[5] developed this equation to account for dynamic changes that occur while loading the limb. Moderate to good reliability has been reported during running tasks.[14, 133] Leg stiffness calculated in this manner considers several additional factors that appear to be more appropriate to human gait. As with K_{vert}, K_{leg} incorporates maximum vertical COM displacement, but adds the component of the leg and its change in length. Standing leg length, as well as the sweeping of the leg during the time of ground contact, are also integrated into the spring constant calculation. Incorporating these descriptors of the movement provides a richer, more in-depth characterization of the behavior of the limb during human gait.

Hsu et al [188] observed greater leg stiffness in older adults during stair descent; however, the lateral malleolus was used as the distal marker for leg length from the greater trochanter. Hortobagyi and DeVita[189] assessed concurrent muscle pre- and coactivity in elderly adults during a step-down task and found significantly greater leg stiffness associated with greater muscle coactivation; however, linear, 2D video analysis was conducted to assess the leg as a spring-mass system. Jones et al[190] also assessed leg stiffness during a step-down task in lower-limb amputees. The traditional leg stiffness equation was used (Fmax/ΔL) by using the COP and hip joint, and the max vertical GRF. The primary methodological concerns of this analysis were: 1) this remains a 2D assessment of leg stiffness and 2) the step-down activity does not mimic true stair negotiation. A critical point to observe is that all three used a different calculation for change in leg length, instead using linear displacement of the leg, rather than considering the arc of motion swept by the leg.

Multiplanar Leg Stiffness

Despite the advancement of leg stiffness calculation methods, a significant limitation is that these methods account only for uniplanar directions. The F_{max} refers to the vertical ground reaction force, which is a single component of the resultant force vector. Though it incorporates the behavior of the limb, K_{leg} only refers to sagittal plane motion. Coleman et al[7] and, subsequently Liew et al[10] have proposed models that incorporate multiplanar and triplanar ground reaction force vectors by integrating medial-lateral (M-L), anterior-posterior (A-P), and vertical components of the resultant ground reaction force.

Coleman et al[7] proposed a "gold standard" measure of leg stiffness using direct kinetic-kinematic analysis of leg stiffness that incorporates vertical and A-P forces to obtain the force vector in line with the leg. The braking phase of gait is of primary interest, particularly when considering the leg as a spring. The calculation tested in this analysis measured the "true change in leg length" by defining leg length as the distance from the greater trochanter to the center of pressure (COP) of the foot. Rather than using only the vertical ground reaction force, as described previously by McMahon and Cheng[5], the vertical and the A-P ground reaction force is used to generate a resultant ground reaction force that is in line with the leg in the direction of leg compression. This more advanced, multiplanar estimation of leg stiffness is examines the ratio of the maximum force in the line of the leg during compression (F_{leg}) and the change in true leg length (ΔL_{true}) expressed as:

$$K = maxF_{leg}/\Delta L_{true}$$

Several factors are included in the calculation of F_{leg} and ΔL_{true} when estimating leg stiffness in this manner. The force directed in line with the leg (F_{leg}) must be determined:

$$F_{leg} = \cos(\theta_{LEG})F_R$$

The angle between the horizontal axis and the line along which F_{leg} is directed (θ_{LEG}) and can be calculated as:

$$\theta_{LEG} = (90 - \theta_{Ltrue}) - \theta_R$$

The angle at which that resultant force is directed (θ_R) must also be known, which can be calculated using trigonometry:

$$\theta_R = \cos^{-1}(\frac{F_V}{F_R})$$

The angle between the true leg and the horizontal axis (θ_{Ltrue}) can also be calculated using trigonometry when the vertical distance from the greater trochanter (A) and horizontal distance from the greater trochanter to the COP (B) are known:

$$\theta_{Ltrue} = \tan^{-1}\left(\frac{A}{B}\right)$$

Determining F_{leg} requires calculating the resultant ground reaction force (F_R) of the leg using Pythagorean Theorem with the vertical (F_V) and horizontal (F_H) as:

$$F_R = \sqrt{(F_V^2 + F_H^2)}$$

Using a more advanced mathematical approach, leg stiffness can be estimated using trigonometric functions. Rather than just the vertical component of the ground reaction force being accounted for, the A-P force is also accounted for in this model. The authors suggest that this is a more appropriate measure of overall leg stiffness than previous models; however, a limitation arises when considering this method of calculation. In this model, the leg spring is defined as the distance between the greater trochanter and the COP. Using the COP creates a more versatile application allowing for assessments that include varying foot strike patterns. While it seems pertinent to consider the COP as the distal point of the leg spring, the greater trochanter may not be an appropriate estimation of the hip joint. Three commonly used methods for determining the hip joint center of rotation have been validated and seem to be viable options.[191-193] The limitation of using the greater trochanter to estimate the hip joint center is not exclusive to this method; however, the model assumes that the force directed through the leg travels through the greater trochanter itself, which may not appropriate.

3-Dimensional Leg Stiffness

The most recent approach to modeling leg stiffness has attempted to account for the limitation of previous methods that do not account for triplanar leg compression. With the evolution of leg stiffness estimation to include ground reaction force components, several authors[10, 194] have adopted the use of dimensionless scaling factors originally proposed by Blickhan.[195] By using a scaling factor that is a ratio of body weight (BW) and leg length (LL), relative dimensionless leg stiffness can be expressed. The method proposed by Liew et al[10] not only incorporates a third plane into the raw calculation of leg spring compression, but also expresses leg stiffness as a dimensionless unit.

Several factors are unique to the model proposed by Liew et al.[10] The hip joint center and COP are used, rather than the greater trochanter and COP, for determining leg length. Reliability of gait data has been improved when using the functional landmark of the hip to estimate the hip joint center.[132] The resultant ground reaction force is also derived from vertical, A-P, and M-L components of the GRF vector. The dot product of the resultant GRF vector and the unit vector of the leg is taken in order to calculate the proportion of the resultant GRF that is in line with leg. In doing so, a projected GRF vector is created as a scalar quantity at each frame. The projected GRF can then be compared to the leg vector frame by frame. The peak of this projection (the vector of greatest magnitude that is most in line with the leg vector) can then be used to estimate stiffness. The leg vector is taken as a line from the COP to the estimated hip joint center. Therefore, the peak projected GRF and leg length change are used when determining leg stiffness, and K_{leg} can be calculated as before:

$$Kleg = \frac{Peak\ Projected\ GRF}{\Delta \mathrm{L}}$$

Dimensionless K_{leg} is then expressed as:

$$DKleg = Kleg/(BW * LL^{-1})$$

Where the scaling factor ($BW*LL^{-1}$) is a ratio of body weight (BW) to standing leg length (LL).

This method has not been tested rigorously during dynamic tasks, as the authors examined it with running in healthy adults. More strenuous examination is warranted during other tasks.

KNEE OSTEOARTHRITIS AND KNEE ARTHROPLASTY PREVALANCE, SIGNIFICANCE, LIMB STRENGTH AND BIOMECHANICAL EVALUATION

Knee Osteoarthritis Etiology, Prevalence, and Significance

Over 50% of people show signs of OA by the age of 65, suggesting that it is the most common joint disorder worldwide.[196] Knee OA is characterized by degeneration of articular cartilage and subchondral bone of the tibia and/or femur. Upwards of 80% of individuals over age 75 show signs of OA, with knee OA being the primary manifestation.[196] Recent reports indicate a staggering increase in knee OA prevalence.[197] Matched sample propensity scoring controlled for age, body mass index, and others, indicated knee OA prevalence in post-industrial age (post-1940), was approximately twice as high compared to pre-industrial (pre-1940) cases (prevalence ratio: 1.9; 95% CI, 1.1–3.5; $P < 0.029$). Covariate balance demonstrated increases in prevalence by each covariate by 99% for age, 100% for BMI, 71% for sex, and 95% for ethnicity. The authors conclude that maintaining greater levels of activities of daily living and injuries, among other environmental considerations, have undoubtedly led to increases in knee OA. This may indicate that knee OA may be more preventable that is commonly thought. In order to decrease pain and limitations during activities of daily living for this population, knee arthroplasty is the gold-standard for treatment of end-stage knee OA.[198] The procedure is growing in the United States. Total knee arthroplasty increased in volume 161.5% from 1991 to 2010, and is expected to grow to nearly 3.5 million procedures by 2030, including revisions.[199] Available data suggests that 600,000 TKA procedures are performed in the United States each year costing approximately $15,000 per procedure making it a $9 billion industry.[198]

With growing demand and cost, measures should be taken to assess patient outcomes for limiting the necessity for both primary TKA procedures and revisions. Patient satisfaction is a typical outcome measure following TKA, often measured through patient pain and functional assessments.[93] Bourne et al.[200] examined 1703 TKAs that approximately one in five (19%) patients were not satisfied with the outcome. The strongest predictors of patient dissatisfaction after primary TKA were expectations not being met (10.79 greater risk), a low 1-year WOMAC score (2.59 greater risk), preoperative pain at rest (2.49 greater risk) and postoperative complication(s) requiring readmission (1.99 greater risk). Specifically related to functional goals, patients were least satisfied in getting in or out of a bus/car (30% unsatisfied) and ascending stairs (27% unsatisfied). Patient dissatisfaction appears to decrease with advancing age, residual symptoms, expectations not being met and less functional improvement.[201] This may suggest that successful TKA procedures, as measured by patient satisfaction, may be a result of age related decreases in expectations and lower activity levels.

Biomechanical Foundations of Knee Osteoarthritis

The Kellgren-Lawrence (K-L) scale is often used when assessing radiographs to quantify the severity of disease progression.[202] The K-L scale ranges from 0 to 4, with 0 indicating no OA presence and 4 indicating severe OA. The presence of osteophytes, periarticular ossicles, joint cartilage narrowing and sclerosis within the bone are characteristics of knee OA grading criteria.[202] Although symptoms are widely reported and can be debilitating, symptomology is not considered when using this grading scale. These criteria for grading of knee OA severity are often present, but alignment considerations that often dictate movement patterns should also be considered. Furthermore, the biomechanical manifestation of these severity scores is important to explore in determining a successful course of treatment and/or prevention. Restoration of knee alignment is a goal of TKA in order to restore function to the knee and lower extremity. Jeffery et al[203] suggested that the knee should be aligned to 0° ± 3° of the mechanical axis. Stickley et al[204] further posited that the tibiofemoral angle was insufficient in determining the mechanical axis, measured in 788 consecutive cases. The mechanical axis goal of 0° ± 3° has been verified in a similar sample by Andrews et al.[100] Proper knee alignment appears to mediate other predictors that are described further in the following passages, though each biomechanical predictor of knee OA has been examined to some extent.

Perhaps the most widely studied predictor of knee OA progression and development via biomechanical evaluation is the external knee adduction moment (KAM) that is experienced during the stance phase of gait. Many studies have analyzed gait biomechanics during ambulation and knee adduction moment has become increasingly relevant.[60, 67, 117, 205-209] Although peak KAM represents the instantaneous peak moment in the frontal plane, it is also accepted to be a surrogate for loading in the medial compartment of the knee.[60, 61]

Studies examining peak KAM changes over time are therefore important to understand the effectiveness of TKA and implications for implant survivorship. At three weeks postoperatively, Shimada et al[209] reported a 30% peak KAM decrease, while at 12-months post-

TKA subjects showed only a 16% decrease. Orishimo et al[208] reported similar findings that peak KAM was reduced to 85% of the preoperative level at 6 months while increasing to 94% of the preoperative level at 1 year. Hatfield et al[117] found that changes between pre TKA and post TKA trending with symptoms. Patients demonstrated lower KAM but also in conjunction with significant differences between the first peak of KAM and KAM at midstance. An important consideration for the examination of KAM is the influence of walking velocity on it and other kinetic and kinematic variables. Walking velocity increases post-TKA with the magnitude of peak KAM during early stance.[210] This is in conjunction with Robbins[211] who found a 7% increase in peak KAM with a 15% increase in walking velocity. Given these results, the walking velocity should be considered when interpretting changes in KAM, and other variables of interest.

Total exposure to an external KAM, or impulse, has been reported, though less frequently than KAM itself.[62, 205, 206, 208, 212] The moment impulse incorportates both the magnitude of KAM and the duration of stance.[212] Dynamic medial knee loading measured in this manner has been identified as a potential risk factor for loss of tibial cartilage volume and was more sensitive for discriminating between OA severity than KAM peak measurements alone.[57, 213] As with KAM, KAM impulse is susceptible to changes in walking velocity, and the velocity should be considered when interpreting any changes. Robbins[211] considered this at three ambulation speeds: self-selcted, slow and fast. The slow and fast paces were 15% slower and faster, respectively than their self-selected pace. At higher walking velocities, KAM impulse was lower than at slow walking velocities; whereas peak KAM increased with higher walking velocities. Slowed gait speeds increase loading exposure on the medial knee tissues as evidenced by the KAM impulse over the period of stance. Debbi et al[206] studied fifty patients with end-stage knee OA with gait analysis, a Timed-Up-Go test, a six-minute walk test, and WOMAC scores. First and second peak KAM decreased to 74% and 79%, respectively, of preoperative levels, while KAM impulse decreased to 73% of preoperative levels. Notably, KAM impulse did not change while peak of KAM decreased. This provides greater insight into total medial loading of the knee and that, while exmaining peak KAM may be beneficial for understanding the maximal load experienced, it may at least in some instances be inadequate to explain medial load distribution throughout the entire period of stance.

Orishimo et al[208] studied KAM impulse in addition to peak KAM and found that KAM impulse during the braking phase decreased from pre-TKA to six months post-TKA but then saw a rise at the 1-year mark. The pattern was similar to peak KAM, though KAM impulse remained lower than pre-operative levels. In the propulsive phase, the KAM impulse decreased at six months and remained low at 1-year. A key consideration with this study was the model used for biomechanical gait analysis. Further study is required to validate the return of KAM values to pre-operative levels, and this may also be a consequence of sampling. Morgenroth et al[62] demonstrated inconsistent findings to Bennell et al,[57] in that KAM impulse was not associated with medial tibial cartilage defects on MRI images, though the authors acknowledge a low sample size as a limitation to interpretation. Paterson et al[205] also examined KAM impulse from pre-TKA to post-TKA and found a significant decrease in total moment exposure. Differences were noted between sexes, but not between obesity categories, which may suggest that alignment differences mediate total loading of the medial compartment. There was a significant reduction in KAM impulse for men while women did not show changes post-TKA. It is important to note that men have been shown to have higher peak KAM and KAM impulse than women,[205] and therefore may have a greater likelihood of demonstrating decreases post-operatively.

The rate of moment development in the frontal plane is less well understood, and less researched as it relates to knee OA development and progression. Morgenroth et al[62] studied KAM rate in conjunction with peak KAM and impulse variables in their MRI study of cartilage thickness. They assessed KAM loading rate as the maximum instantaneous slope of the KAM curve from initial foot contact to the first peak of the KAM during stance. Results showed significant correlation between medial tibiofemoral joint degeneration and peak KAM. The relationship between the MRI score and KAM loading rate were significant after adjusting for peak KAM. However, the relationship between the MRI score and peak KAM was not significant after adjusting for the KAM loading rate. Their results indicate a strong association between KAM rate and medial tibiofemoral joint space narrowing, cartilage thickness, and subchondral bone marrow lesions. High KAM rates, even when adjusting for peak KAM, demonstrate strong relationships with medial knee OA progression. Most recently, Doyle et al[212] examined KAM rate development in transtibial amputees and the influence on the contralateral limb. Most notably, this study found that the down slope limb demonstrated greater KAM rate on all surfaces. This was positively associated with degenerative changes of the knee in the contralateral limb in prosthesis users, but not in healthy controls that did not alter loading mechanics.

The kinematic equivalent of frontal plane moment is varus velocity and is commonly referred to as varus thrust. Varus thrust was initially associated with knee osteoarthritis development and observed by Chang et al.[65] Frontal plane angular velocity of the knee is now recognized as a predictor of knee OA onset and progression,[74] though it has also been associated with the presence of iliotibial band friction syndrome.[214] Chang et al[65] found that in varus aligned knees, varus thrust increased the odds of OA progression 3-fold. Subsequent analysis by Chang et al[60, 74] attributed varus thrust/angular velocity and peak KAM to knee OA onset and progression. Farrokhi et al[215] considered varus and valgus motion related to muscle strength in patients with knee OA. Knee OA patients were found to have greater knee varus motion excursions compared to the control group. Subjects with diminished quadriceps strength caused by knee OA symptoms demonstrated greater excursions into a varus position.

In the sagittal plane, the external knee flexion moment (KFM) is widely researched, and is considered to indicate load attenuation, overall health, post-operative recovery, quality of life, and quadriceps muscle strength.[59, 61, 117, 216] Chehab et al[61] found that KFM was significantly associated with medial, lateral, and posterior tibial cartilage changes across five years. This was important to consider, such that KAM was a primary target for intervention; however, KFM was significantly associated with medial-lateral tibial cartilage thickness distribution, suggesting that KFM also influences medial load distribution during stance. Hatfield et al[117] examined 42 patients with knee OA, pre- and post-TKA. Principal components analysis indicated significant increases in external knee flexion moment following TKA, in conjunction with late stance external knee extension moment, indicating load attenuation and improved propulsion. Bimodal knee flexion moments during early to mid-stance indicate quadriceps function and increased willingness to load the limb. Landry et al[210] obtained similar findings, in addition to knee flexion moment having an inverse relationship with severity of knee OA disease presence. Increased severity was associated with decreased knee flexion moment through stance, a quadriceps avoidance gait pattern with diminished knee flexion excursion. Knee OA patients exhibited smaller magnitudes of KFM at both self-selected walking pace and 150% of subject's self-selected pace. Kaufman et al[217] studied knee kinematics and kinetics in 139 patients with Grade 2 knee OA, ranging in age from 30-82 years. Rather than differences in KFM between OA patients and controls, knee flexion angles experienced during loading were different; however, the authors concluded that subjects attempt to minimize pain by reducing the quadriceps muscle group activation which reduces KFM during stance, but not knee flexion excursions. Chang et al and Chehab et al[60, 61] agree that external knee flexion moment contributes to medial knee loading and is also predictive of knee OA progression and development.

Rate of torque development has been researched (considered the same as rate of moment development). Kline et al[59] recently concluded that rate of torque development in the quadriceps muscle group is a significant contributor to knee flexion excursion following TKA. Quadriceps avoidance gait is typically observed as limited knee flexion, and the authors conclude that decreased rate of torque development of the quadriceps predicts quadriceps avoidance gait (adjusted R^2=0.436). It is therefore desirable to maximize rate of torque development following surgery to facilitate resumption of normal gait. Quadriceps function has been examined in its ability to modulate knee kinetics and kinematics. Davis et al[218] examined 53 knee OA patients (47% female, 62.3 +/- 7.1 years, BMI = 28.5 +/- 3.9 kg/m^2) and were enrolled in a 28-day strengthening protocol for the quadriceps femoris muscle group. Maximum isometric quadriceps strength and walking gait biomechanics were collected on the involved limb at baseline and 4-weeks following the strengthening intervention. 2 x 2 ANOVAs were used to evaluate the effects of group (responders and non-responders) and time (baseline and 4-weeks) on time-normalized waveforms for knee flexion angle, vertical ground reaction force, and internal knee extension moment. A significant group x time interaction for KFA demonstrated greater KFA in the first half of stance at baseline and greater knee extension in the second half of stance at 4-weeks in responders to the strengthening. There was no significant group x time interaction for vGRF or internal KEM. The authors conclude that quadriceps strengthening may stimulate small changes in KFA in individuals with knee OA.

Luc-Harkey et al [101] found small associations between quadriceps rate of torque development and walking velocity. Greater involved-side late rate of torque development and greater early rate of torque development were associated with faster walking. Greater bilateral average late rate of torque development was associated with faster walking and faster stair climbing. No quadriceps torque development variable was significantly associated with WOMAC function score. Involved-limb quadriceps rate of torque development showed weak associations with walking velocity, but not self-reported patient outcome measures, in individuals with knee OA. Bilateral average quadriceps rate of torque development was moderately associated with walking speed, suggesting that as patients recover and gain more confidence in walking, they also gain more quadriceps function. Conversely, one could argue that subjects gain increased walking velocity because of improved quadriceps rate of torque development.

Stair Negotiation Biomechanics in Knee OA and Arthroplasty Patients

The first complaint of patients receiving TKA is often the inability to negotiate stairs.[219]. Standifird et al[220] completed a systematic review examining stair ambulation biomechanical studies for post-TKA patients, with only 13 studies available for review. During stair ascent, patients showed reductions in knee flexion angle at contact, peak knee flexion, total flexion excursion ROM and ascent velocity compared to healthy controls. During stair descent, TKA patients either regained or exceeded frontal and sagittal plane peak moments, or results were uninterpretable due to confounding factors. Such factors include pain, psychological fear-avoidance, or the speed at which patients ascend or descend stairs. Hicks-Little et al.[221] found that their knee OA group demonstrated smaller peak knee flexion angles during the swing phase, as well as smaller knee flexion angles at initial contact and during the support phase. McClelland et al[222] studied 40 patients with a modern TKA implant and compared them to 40 matched control subjects during stair ascent and descent. Interestingly, not all subjects were able to complete stair negotiation unassisted. The knee OA group was subdivided into Cluster 1 (normal) and Cluster 2 (abnormal), which demonstrated differing KFM during both stair ascent and descent; during stair ascent, the control group showed peak KFM during loading of 3.8%BW*HT, whereas most of the Cluster 1 group showed peak KFM during loading of 2.7%BW*HT. During stair descent, the control group saw a maximum knee flexion moment during loading of 3.2%BW*HT, whereas most of the Cluster 1 group a maximum knee flexion moment during loading of 2.6%BW*HT. This study reported a distinct change in pattern of the KFM waveform during stair ascent and descent.

Bjerke et al[223] examined 23 unilateral TKA subjects against and equal size sample of controls during stair ascent. The study protocol included EMG and assessed knee muscle strength by maximal voluntary concentric contractions of the vastus lateralis and semitendinosus. They found quadriceps peak torque on the TKA-involved limb to be significantly lower compared to the contralateral limb. The stronger, contralateral limb demonstrated lower peak torque than the control group. The hamstring strength of the TKA-involved limb was significantly lower than bilateral limbs of the control group. Bjerke[46] also examined knee flexion angles in TKA patients descending stairs. Multiple regression analysis identified peak passive knee flexion as a significant predictor explaining stair descent knee flexion, whereas pain, quadriceps strength, proprioception, and anthropometric features were not.

It is useful to examine normative data for biomechanics of stair negotiation. Protopapadaki et al[63] examined 33 young, healthy subjects, (16 M, 17 F, range 18-39 years). Kinematic data were recorded using 3D motion analysis. Kinetic data were standardized to body mass and height. Paired-samples t tests showed significantly greater hip and knee angles (mean difference: hip 28.10 degrees (SD 4.08), knee 3.39 degrees (SD 7.20)) and hip and knee moments (hip 0.25 Nm/kg (SD 0.18), knee 0.17 Nm/kg (SD 0.15)) during stair ascent compared to descent. Significantly greater ankle dorsiflexion angles (9.90 degrees (SD 3.80)) and plantarflexion angles (8.78 degrees (SD 4.80)) were found during stair descent compared to ascent. The authors determined that stair ascent was a more demanding task when compared to stair descent for healthy young subjects. Novak and Brouwer[64] described age-related differences in sagittal and frontal plane joint moments. Twenty-three young and 32 older adults (>55 years) were examined during stair negotiation. Age-related differences were found in the magnitudes of the moment contributions during event transitions for stair ascent and descent. Within groups, the moment profiles were generally consistent. Ankle and knee moments predominantly contributed to extensor support in the sagittal plane. Younger subjects generally showed less variation in intra-subject joint moments during stair negotiation, while older subjects varied considerably more between stair negotiation trials.

DISTINCTIONS BETWEEN MULTI- AND SINGLE-RADIUS TOTAL KNEE ARTHROPALSTY

The treatment for end-stage knee OA is complex and intricate. A decision must be made regarding the appropriate implant. Knee arthroplasty, either total (TKA) or unicompartmental (UKA) remains the gold standard for the treatment of this condition.[224] As previously noted regarding alignment, the ultimate goal of knee arthroplasty is to restore normal function of the tibiofemoral joint and, in the process, eliminate pain for patients experiencing knee OA. Restoration of the mechanical axis to near-neutral is considered a primary goal to reduce failures associated with poor alignment.[100, 204] Another goal, perhaps the most important, is longevity of the implant. All implants contain a tibial component, a femoral component, and in many cases a patellar component that are designed to meet alignment goals. Each component has theoretical advantages and disadvantages associated with them.

Although there is considerable variation in TKA implant design, all attempt to restore the normal center of rotation of the tibiofemoral joint. Contrasting viewpoints regarding the axis of rotation of the tibiofemoral joint have been developed, and each implant design is tailored to suit either the migrating joint center[225] or the trans epicondylar joint center.[226] The femoral component primarily meets these needs through various designs, though the tibial component also contributes. Femoral components have been developed as multiradius (MR) and single radius (SR) implants and are typically made of cobalt-chrome or titanium. Each is designed to treat the knee very differently with regards to how the center of rotation behaves.

The theoretical advantage of MR implants is that they accommodate for a migrating center of rotation, as described by Hollister.[225] Single radius implants are designed to limit the center of rotation to a single point on the insert, at the trans epicondylar axis, as described by Churchill.[226] The theoretical advantage to this design is that it provides a smoother trajectory and more uniform motion during flexion and extension of the knee. The basis for this theoretical advantage is that the relatively stationary axis of rotation creates a longer moment arm for the extensor mechanism during weight bearing flexion of the knee, thereby maintaining the mechanical advantage of the extensor mechanism.[125] Both designs have theoretical merit; however, despite some conflicting results, evidence seems to support the SR design. It should be noted that few studies have examined MR and SR designs in tasks beyond walking gait, patient reported outcomes, and strength evaluation. More difficult tasks than walking may be required to assess the proposed advantages of each implant design.[227]

Functional and patient reported outcomes have largely shown positive results. Regardless of implant design, between 90% and 98% satisfaction rates in quality of life, pain, and functional domains have been reported for TKA, with revision rates between 0% and 6%.[79] Jones et al[119] reviewed patient reported outcomes among TKA surgeries and found positive overall satisfaction rates, regardless of the surgery performed. Oliviu[120] also found no differences between MR and SR patients functional scores and 5+ year survivorship of the implants. With good patient-reported outcomes for each implant, rehabilitation should be considered as a contributing factor to TKA success. Rossi et al[83] brought into question the potential of regaining strength and function of the operated limb compared to the non-operated limb in unilateral TKA patients. Regaining quadriceps function should be the primary goal of rehabilitation post-TKA. Yoshida[86] demonstrated that TKA patients may exhibit between-limb deficits in quadriceps strength as long as three years post-surgery, in addition to altered gait patterns. It is important to understand that knee function may never return to “normal” levels. It is suggested that knee extensor strength training be an integral facet of rehabilitation to improve functional capabilities by maximizing rate of torque development.[59] Making improvements in these regards can help diminish quadriceps avoidance gait through early rehabilitation interventions. Furthermore, it should be implemented for as long as 6 months post-surgery, suggested by Vahtrik et al.[228] They examined isometric maximal voluntary contraction of the extensor muscles and the relationship to knee loading during gait in 13 females receiving primary TKA. Six-months after TKA, the maximal voluntary contraction of the non-operative limb was similar to pre-operative levels, but the operated limb was markedly lower than pre-operative levels.

In vivo kinematics and functional studies have been conducted to evaluate MR and SR component performance. Several studies have found no differences in kinematics between MR and SR implants.[82, 125, 229, 230] Hall et al[125] determined that MR and SR implants retained comparable knee extensor mechanism function; however, these were posterior cruciate retaining implants. Wolterbeek et al[229] drew similar conclusions between MR and SR implants with cruciate retaining, cruciate sacrificing, and mobile and fixed-bearing TKA designs. Fifty-two knees were examined in vivo. Jo et al[230] found better intraoperative varus-valgus stability of SR designs at 30° of flexion, but no differences at 0°, 60°, and 90° of flexion; however, Kessler[231] found the helical axis of the SR implants to be more uniform than MR implants in vivo. Video fluoroscopy showed that the angular and spatial localization of the MR implant was larger than the SR design, indicating a greater degree of available motion and potentially less stability during load bearing.

Evidence leans towards the SR implant design facilitating early quadriceps recovery and contributing to satisfaction rates. Several authors have reported improved functional outcomes of SR over MR designs to support this, evidenced by early post-operative functional gains,[116] lower anterior knee pain,[232] and decreased quadriceps avoidance gait patterns.[227] Collados-Maestre et al[116] examined 237 patients randomly receiving MR and SR TKA. The SR group, upon 5-year follow-up as a minimum, demonstrated better KSS scores, range of motion, decreased extension lag, greater quadriceps strength, improved ability to rise from a chair, and decreased WOMAC pain scale scores. Implants demonstrated similar revision rates and implant survivorship. Luo et al[232] conducted 10 year clinical follow up of TKA patients receiving MR and SR prostheses retrospectively and found that MR and SR posterior-stabilized prostheses both demonstrate good outcomes. They conclude further that the SR design demonstrated fewer incidences of anterior knee pain upon follow-up, but overall, each performed well and were similar across clinical outcome measures. Larsen et al[227] examined SR and MR implants during gait at pre-op and 1 year post-TKA. The MR knees remained more extended and demonstrated diminished power absorption during the loading response, whereas SR knees were similar to controls. The authors suggest that in SR knees, the patellofemoral moment arm facilitates early quadriceps muscle function. Furthermore, isokinetic extension torque accompanied by improved functional outcomes,[122] as well as improved performance accompanied by less hamstring coactivation required to stabilize the knee during a sit-to-stand task.[124] Gomez-Barrena et al[122] observed higher functional Knee Society scores, fewer physiotherapy sessions, and less time with two crutches for patients receiving the SR design. Isokinetic evaluation showed decreased flexion peak torque, increased extension peak torque, and lower flexor/extensor ratio in patients with the single-radius design. Wang et al[124] is in agreement, demonstrating that patients receiving MR implants require greater coactivation of the hamstrings and quadriceps to achieve stability during a stand-up-and-go test, in addition to having longer testing times.

Stair negotiation has been less researched, though there is emerging evidence. Fenner[233] reported improvements in frontal plane knee moments during stair descent; however, no comparison was made between MR and SR designs, with all subjects receiving MR implants. Recently, Sumner[104, 234] evaluated kinematics and kinetics of the knee and found the SR design to be superior to the MR design when compared to healthy controls. Comparisons were made during stair ascent[104] and during stair descent.[234] Subjects receiving SR implants demonstrate greater kinematic stability in the sagittal plane during stair ascent and descent. Patients with SR implants showed normalization in external knee flexion moments, knee flexion angles, and spatiotemporal gait parameters, while MR patients maintained differences from controls. This evidence suggests that SR and MR designs have similar long-term results, but SR designs may improve early outcomes due to the proposed mechanism of facilitating quadriceps function. Further investigation may be needed to confirm MR vs SR implants during more challenging tasks, such as stair descent, that require greater quadriceps control and increased knee flexion angles.[46]

CONSIDERATIONS FOR UNICOMPARTMENTAL KNEE ARTHROPLASTY

Unicompartmental knee arthroplasty (UKA) serves the same purpose of TKA, in that the goal is to restore normal knee function. The indication for selecting this implant design is isolated cartilage degeneration to either the medial or lateral compartment.[105] Successful UKA procedures are dependent on rigorous protocol adherence, which generally involves a minimally invasive midvastus approach.[235] The benefits of a successful surgery, when UKA is indicated, are decreased post-operative pain, shorter recuperation time, fewer perioperative complications, and same-day discharge after surgery.[105]

Given these advantages, this minimally invasive procedure would theoretically be preferred. Annual revision rates have been observed as low as 1% in medial and lateral UKA procedures, with no differences between either procedure.[236] Lim et al[237] reported that TKA produced overall greater improvements in pain compared to UKA, though the effect may have been due to lower overall pain pre-operatively in UKA patients than TKA patients. They concluded that the only advantage to UKA is early functional gains after surgery, with no long term (3 years) differences. Wilson[80] reported better outcomes in pain, functional, and complication scales for UKA over TKA, while TKA showed lower overall revision rates. Unicompartmental arthroplasties have been established as safe outpatient procedures, with minimal risk of short-term complications.[105, 235]

Though the clinical outcomes show a clear early advantage, the ability of the implant to restore the naturally occurring anatomy of the tibiofemoral joint must be considered when deciding to use UKA vs TKA. Performance of UKA implants has been evaluated by assessing kinematics and load-bearing capacity during functional tasks.[54, 87, 88, 238, 239] Fu et al[54] examined UKA patients undergoing stair ascent to examine between-limb differences in knee moments in medial and lateral UKA. Twenty-six patients were recruited with single limb knee OA and found that the medial UKA group displayed greater peak extensor moments for the non-diseased limb. Reduced knee extensor moments are displayed by both medial and lateral UKA groups, and medial compartment loading is not necessarily isolated to the medial compartment in lateral compartment UKA patients. Jung et al[89] compared knee kinematics between simultaneous bilateral TKA patients and UKA patients during stair climbing. Patients received a UKA on one knee and TKA on the other and underwent 3D motion capture analysis. The UKA demonstrated greater degrees of rotation in the transverse plane but were otherwise similar to TKA. The authors concluded that given the similarities, with increased UKA rotation capacity, that UKA more closely replicates anatomy of the knee. Komnik et al[88] concluded that TKA and UKA demonstrated similar gait parameters, as evaluated by 3D motion capture. First-peak knee adduction moments were diminished by 27% (TKA group) and 22% (UKA group) compared with the CG during decline walking. The authors note that walking may not be a difficult enough task to reproduce any meaningful differences between the impant designs. Most recently, Shiwaku et al[239] observed less varus angulation at greater degrees of knee flexion to be associated with improved clinical outcomes in UKA patients. Data were retrospectively collected from 46 UKA for medial compartment knee OA with the goal of investigating the effects of navigation-based varus or axial rotational alignment. Patients were scored with the KOOS at final follow-up. No differences in KOOS were observed been alignment groups; however, the neutral at 90 degrees of flexion group demonstrated significantly greater outcome scores than the varus alignment at 90 degrees of flexion. The authors concluded that improved functional scores are associated with more neutral alignment throughout a total flexion ROM.

Given the lack of functional deficits compared to TKA, UKA seems the most viable alternative due to its early post-operative and peri-operative advantages. The UKA design appears to mitigate the problems associated with TKA complications while also restoring normal function of the knee. Early UKA intervention may be more appropriate; however, it is important to consider the degree of cartilage degeneration in both the medial and lateral compartments. UKA is not recommended in patients with bicompartmental narrowing, nor with joint space narrowing with differences greater than 20%.[105]

REFERENCES

1. Proske, U. and S.C. Gandevia, *The proprioceptive senses: their roles in signaling body shape, body position and movement, and muscle force.* Physiol Rev, 2012. **92**(4): p. 1651-97.
2. Han, J., et al., *Assessing proprioception: A critical review of methods.* J Sport Health Sci, 2016. **5**(1): p. 80-90.
3. Relph, N. and L. Herrington, *Criterion-related validity of knee joint-position-sense measurement using image capture and isokinetic dynamometry.* J Sport Rehabil, 2015. **24**(1).
4. Butler, R.J., H.P. Crowell, and I.M. Davis, *Lower extremity stiffness: implications for performance and injury.* Clinical Biomechanics, 2003. **18**(6): p. 511-517.
5. McMahon, T.A. and G.C. Cheng, *The mechanics of runing: how does stiffness couple with speed.* Journal of Biomechanics, 1990. **21**: p. 65-78.
6. Pearson, S.J. and J. McMahon, *Lower limb mechanical properties: determining factors and implications for performance.* Sports Med, 2012. **42**(11): p. 929-40.
7. Coleman, D.R., et al., *Leg stiffness in human running: Comparison of estimates derived from previously published models to direct kinematic-kinetic measures.* J Biomech, 2012. **45**(11): p. 1987-91.
8. Arampatzis, A., G.P. Bruggemann, and V. Metzler, *The effect of speed on leg stiffness and joint kinetics in human running.* Journal of Biomechanics, 1999. **32**: p. 1349-1353.
9. Brughelli, M. and J. Cronin, *Influence of running velocity on vertical, leg and joint stiffness : modelling and recommendations for future research.* Sports Med, 2008. **38**(8): p. 647-57.
10. Liew, B.X.W., et al., *A comparison and update of direct kinematic-kinetic models of leg stiffness in human running.* J Biomech, 2017. **64**: p. 253-257.
11. Lorimer, A.V., J.W.L. Keogh, and P.A. Hume, *Using stiffness to assess injury risk: comparison of methods for quantifying stiffness and their reliability in triathletes.* PeerJ, 2018. **6**: p. e5845.
12. Maloney, S.J. and I.M. Fletcher, *Lower limb stiffness testing in athletic performance: a critical review.* Sports Biomech, 2018: p. 1-22.
13. Beerse, M. and J. Wu, *Vertical stiffness and center-of-mass movement in children and adults during single-leg hopping.* J Biomech, 2016. **49**(14): p. 3306-3312.
14. Pappas, P., G. Dallas, and G. Paradisis, *Reliability of Leg and Vertical Stiffness During High Speed Treadmill Running.* J Appl Biomech, 2017. **33**(2): p. 160-165.
15. Liew, B., K. Netto, and S. Morris, *Increase in Leg Stiffness Reduces Joint Work During Backpack Carriage Running at Slow Velocities.* J Appl Biomech, 2017. **33**(5): p. 347-353.
16. Baert, I.A.C., et al., *Inter- and intrarater reliability of two proprioception tests using clinical applicable measurement tools in subjects with and without knee osteoarthritis.* Musculoskelet Sci Pract, 2018. **35**: p. 105-109.
17. Relph, N. and L. Herrington, *The effects of knee direction, physical activity and age on knee joint position sense.* Knee, 2016. **23**(3): p. 393-8.
18. Relph, N. and L. Herrington, *Knee joint position sense ability in elite athletes who have returned to international level play following ACL reconstruction: A cross-sectional study.* Knee, 2016. **23**(6): p. 1029-1034.
19. Bernier, J. and D. Perrin, *Effect of coordination training on proprioception of the functionally unstable ankle.* J Orthop Sports Phys Ther, 1998. **27**(4): p. 264-274.

20. Daman, M., et al., *The effect of combined exercise therapy on knee proprioception, pain intensity and quality of life in patients with hypermobility syndrome: A randomized clinical trial.* J Bodyw Mov Ther, 2019. **23**(1): p. 202-205.
21. Piriyaprasarth, P., et al., *Trials needed to assess knee proprioception following stroke.* Physiother Res Int, 2009. **14**(1): p. 6-16.
22. Sharma, L., et al., *IS KNEE JOINT PROPRIOCEPTION WORSE IN THE ARTHRITIC KNEE VERSUS THE UNAFFECTED KNEE IN UNILATERAL KNEE OSTEOARTHRITIS?* Arthritis Rheum, 1997. **40**(8): p. 1518-1525.
23. Wingert, J.R., et al., *Joint-position sense and kinesthesia in cerebral palsy.* Arch Phys Med Rehabil, 2009. **90**(3): p. 447-53.
24. Friemert, B., et al., *Group I afferent pathway contributes to functional knee stability.* J Neurophysiol, 2010. **103**(2): p. 616-22.
25. Marcora, S.M., *Role of feedback from Group III and IV muscle afferents in perception of effort, muscle pain, and discomfort.* J Appl Physiol (1985), 2011. **110**(5): p. 1499; author reply 1500.
26. Vallabhajosula, S., J.M. Yentes, and N. Stergiou, *Frontal joint dynamics when initiating stair ascent from a walk versus a stand.* Journal of Biomechanics, 2012. **45**(3): p. 609-613.
27. Kristianslund, E., T. Krosshaug, and A.J. van den Bogert, *Effect of low pass filtering on joint moments from inverse dynamics: implications for injury prevention.* J Biomech, 2012. **45**(4): p. 666-71.
28. Suner-Keklik, S., et al., *The Validity and Reliability of Knee Proprioception Measurement Performed With Inclinometer in Different Positions.* J Sport Rehabil, 2017. **26**(6).
29. Arvin, M., et al., *Reproducibility of a knee and hip proprioception test in healthy older adults.* Aging Clin Exp Res, 2015. **27**(2): p. 171-7.
30. Cohen, J., *Statistical power analysis for the behavioral sciences.* Academic Press, 1988.
31. Fritz, C.O., P.E. Morris, and J.J. Richler, *Effect size estimates: current use, calculations, and interpretation.* J Exp Psychol Gen, 2012. **141**(1): p. 2-18.
32. Hajizadeh, M., et al., *Knee Kinematics and Joint Moments During Stair Negotiation in Subjects With Anterior Cruciate Ligament Deficiency and Reconstruction: A Systematic Review and Meta-Analysis.* PM R, 2016. **8**(6): p. 563-579 e1.
33. Leppanen, M., et al., *Sagittal Plane Hip, Knee, and Ankle Biomechanics and the Risk of Anterior Cruciate Ligament Injury: A Prospective Study.* Orthop J Sports Med, 2017. **5**(12): p. 2325967117745487.
34. Relph, N., L. Herrington, and S. Tyson, *The effects of ACL injury on knee proprioception: a meta-analysis.* Physiotherapy, 2014. **100**(3): p. 187-95.
35. Bade, M.J., et al., *Predicting functional performance and range of motion outcomes after total knee arthroplasty.* Am J Phys Med Rehabil, 2014. **93**(7): p. 579-85.
36. Bennell, K.L., R.S. Hinman, and B.R. Metcalf, *Association of sensorimotor function with knee joint kinematics during locomotion in knee osteoarthritis.* Am J Phys Med Rehabil, 2004. **83**: p. 455-463.
37. Bennell, K.L., et al., *Relationship of knee joint proprioception to pain and disability in individuals with knee osteoarthritis.* J Orthop Res, 2003. **21**: p. 792-797.
38. Ferris, D.P. and C.T. Farley, *Interaction of leg stiffness and surface stiffness during human hopping.* J Appl Physiol, 1997. **82**(1): p. 15-22.
39. Hobara, H., K. Kanosue, and S. Suzuki, *Changes in muscle activity with increase in leg*

stiffness during hopping. Neurosci Lett, 2007. **418**(1): p. 55-9.

40. Promsri, A., et al., *Leg Dominance as a Risk Factor for Lower-Limb Injuries in Downhill Skiers-A Pilot Study into Possible Mechanisms.* Int J Environ Res Public Health, 2019. **16**(18).
41. Dos'Santos, T., et al., *The effect of limb dominance on change of direction biomechanics: A systematic review of its importance for injury risk.* Phys Ther Sport, 2019. **37**: p. 179-189.
42. Relph, N. and L. Herrington, *The effect of conservatively treated ACL injury on knee joint position sense.* Int J Sports Phys Ther, 2016. **11**(4): p. 536-543.
43. Cammarata, M.L. and Y.Y. Dhaher, *Associations between frontal plane joint stiffness and proprioceptive acuity in knee osteoarthritis.* Arthritis Care Res (Hoboken), 2012. **64**(5): p. 735-43.
44. Cammarata, M.L., T.J. Schnitzer, and Y.Y. Dhaher, *Does knee osteoarthritis differentially modulate proprioceptive acuity in the frontal and sagittal planes of the knee?* Arthritis Rheum, 2011. **63**(9): p. 2681-9.
45. van Tunen, J.A.C., et al., *Association of malalignment, muscular dysfunction, proprioception, laxity and abnormal joint loading with tibiofemoral knee osteoarthritis - a systematic review and meta-analysis.* BMC Musculoskelet Disord, 2018. **19**(1): p. 273.
46. Bjerke, J., et al., *Peak knee flexion angles during stair descent in TKA patients.* J Arthroplasty, 2014. **29**(4): p. 707-11.
47. Hillier, S., M. Immink, and D. Thewlis, *Assessing Proprioception: A Systematic Review of Possibilities.* Neurorehabil Neural Repair, 2015. **29**(10): p. 933-49.
48. Knoop, J., et al., *Proprioception in knee osteoarthritis: a narrative review.* Osteoarthritis Cartilage, 2011. **19**(4): p. 381-8.
49. Li, L., et al., *Correlation study of knee joint proprioception test results using common test methods.* J Phys Ther Sci, 2015. **28**.
50. Romero-Franco, N., J.A. Montano-Munuera, and P. Jimenez-Reyes, *Validity and Reliability of a Digital Inclinometer to Assess Knee Joint-Position Sense in a Closed Kinetic Chain.* J Sport Rehabil, 2017. **26**(1).
51. Laessoe, U., et al., *Fall risk in an active elderly population--can it be assessed?* J Negat Results Biomed, 2007. **6**: p. 2.
52. Petrella, R.J., P.J. Lattanzio, and M.G. Nelson, *EFFECT OF AGE AND ACTIVITY ON KNEE JOINT PROPRIOCEPTION1.* American Journal of Physical Medicine & Rehabilitation, 1997. **76**(3): p. 235-241.
53. Rasnick, R., et al., *Knee Joint Loads and Surrounding Muscle Forces during Stair Ascent in Patients with Total Knee Replacement.* PLoS One, 2016. **11**(6): p. e0156282.
54. Fu, Y.C., et al., *Knee moments after unicompartmental knee arthroplasty during stair ascent.* Clin Orthop Relat Res, 2014. **472**(1): p. 78-85.
55. Krajnc, Z., et al., *Increased risk of knee injuries and osteoarthritis in the non-dominant leg of former professional football players.* Wien Klin Wochenschr, 2010. **122 Suppl 2**: p. 40-3.
56. Kristianslund, E., T. Krosshaug, and A.J. van den Bogert, *Artefacts in measuring joint moments may lead to incorrect clinical conclusions: the nexus between science (biomechanics) and sports injury prevention!* Br J Sports Med, 2013. **47**(8): p. 470-3.
57. Bennell, K.L., et al., *Higher dynamic medial knee load predicts greater cartilage loss over 12 months in medial knee osteoarthritis.* Ann Rheum Dis, 2011. **70**(10): p. 1770-4.

58. Bennell, K.L., et al., *Comparison of neuromuscular and quadriceps strengthening exercise in the treatment of varus malaligned knees with medial knee osteoarthritis: a randomised controlled trial protocol.* BMC Musculoskelet Disord, 2011. **12**: p. 276.
59. Kline, P.W., et al., *Rate of torque development is the primary contributor to quadriceps avoidance gait following total knee arthroplasty.* Gait Posture, 2019. **68**: p. 397-402.
60. Chang, A.H., et al., *External knee adduction and flexion moments during gait and medial tibiofemoral disease progression in knee osteoarthritis.* Osteoarthritis Cartilage, 2015. **23**(7): p. 1099-106.
61. Chehab, E.F., et al., *Baseline knee adduction and flexion moments during walking are both associated with 5 year cartilage changes in patients with medial knee osteoarthritis.* Osteoarthritis Cartilage, 2014. **22**(11): p. 1833-9.
62. Morgenroth, D.C., et al., *The relationship between knee joint loading rate during walking and degenerative changes on magnetic resonance imaging.* Clin Biomech (Bristol, Avon), 2014. **29**(6): p. 664-70.
63. Protopapadaki, A., et al., *Hip, knee, ankle kinematics and kinetics during stair ascent and descent in healthy young individuals.* Clin Biomech (Bristol, Avon), 2007. **22**(2): p. 203-10.
64. Novak, A.C. and B. Brouwer, *Sagittal and frontal lower limb joint moments during stair ascent and descent in young and older adults.* Gait Posture, 2011. **33**(1): p. 54-60.
65. Chang, A., et al., *Thrust during ambulation and the progression of knee osteoarthritis.* Arthritis Rheum, 2004. **50**(12): p. 3897-903.
66. Hall, M., et al., *The knee adduction moment and knee osteoarthritis symptoms: relationships according to radiographic disease severity.* Osteoarthritis Cartilage, 2017. **25**(1): p. 34-41.
67. Apostolopoulos, A.P., et al., *Kinematic and Kinetic Waveform Changes of the Knee Joint Following a Mobile Bearing Total Knee Arthroplasty-Gait Analysis and Single Step Ascent.* J Knee Surg, 2019.
68. Agarwal, A., et al., *Comparison of gait kinematics in total and unicondylar knee replacement surgery.* Ann R Coll Surg Engl, 2019. **101**(6): p. 391-398.
69. Miller, S., et al., *Comparison of gait kinetics in total and unicondylar knee replacement surgery.* Ann R Coll Surg Engl, 2018. **100**(4): p. 267-274.
70. Serpell, B.G., et al., *A review of models of vertical, leg, and knee stiffness in adults for running, jumping or hopping tasks.* J Sports Sci, 2012. **30**(13): p. 1347-63.
71. Becker, R., et al., *Expectation, satisfaction and clinical outcome of patients after total knee arthroplasty.* Knee Surg Sports Traumatol Arthrosc, 2011. **19**(9): p. 1433-41.
72. Eckstein, F., et al., *Side differences of knee joint cartilage volume, thickness, and surface area, and correlation with lower limb dominance--an MRI-based study.* Osteoarthritis Cartilage, 2002. **10**(12): p. 914-21.
73. Chang, A., et al., *Frequency of varus and valgus thrust and factors associated with thrust presence in persons with or at higher risk of developing knee osteoarthritis.* Arthritis Rheum, 2010. **62**(5): p. 1403-11.
74. Chang, A.H., et al., *Varus thrust and knee frontal plane dynamic motion in persons with knee osteoarthritis.* Osteoarthritis Cartilage, 2013. **21**(11): p. 1668-73.
75. Henao-Murillo, L., K. Ito, and C.C. van Donkelaar, *Collagen Damage Location in Articular Cartilage Differs if Damage is Caused by Excessive Loading Magnitude or Rate.* Ann Biomed Eng, 2018. **46**(4): p. 605-615.

76. Radzak, K.N., et al., *Asymmetry between lower limbs during rested and fatigued state running gait in healthy individuals.* Gait Posture, 2017. **51**: p. 268-274.
77. Hosaka, K., et al., *Asian-specific total knee system: 5-14 year follow-up study.* BMC Musculoskelet Disord, 2011. **12**: p. 251.
78. Mugnai, R., et al., *Can TKA design affect the clinical outcome? Comparison between two guided-motion systems.* Knee Surg Sports Traumatol Arthrosc, 2014. **22**(3): p. 581-9.
79. Shan, L., et al., *Intermediate and long-term quality of life after total knee replacement: a systematic review and meta-analysis.* J Bone Joint Surg Am, 2015. **97**(2): p. 156-68.
80. Wilson, H.A., et al., *Patient relevant outcomes of unicompartmental versus total knee replacement: systematic review and meta-analysis.* BMJ (Clinical research ed.), 2019. **364**: p. l352-l352.
81. Johal, S., et al., *Unicompartmental Knee Arthroplasty: The Past, Current Controversies, and Future Perspectives.* J Knee Surg, 2018. **31**(10): p. 992-998.
82. Hinarejos, P., et al., *No differences in functional results and quality of life after single-radius or multiradius TKA.* Knee Surg Sports Traumatol Arthrosc, 2016. **24**(8): p. 2634-40.
83. Rossi, M.D., L.E. Brown, and M. Whitehurst, *Knee extensor and flexor torque characteristics before and after unilateral total knee arthroplasty.* Am J Phys Med Rehabil, 2006. **85**(9): p. 737-46.
84. Kurowicki, J., et al., *Quadriceps and Hamstring Muscle Strength Improves After Unicompartmental Knee Arthroplasty.* Surgical technology international, 2017. **31**: p. 267.
85. Li, Y., et al., *Knee strength, power and stair performance of the elderly 5 years after unicompartmental knee arthroplasty.* Eur J Orthop Surg Traumatol, 2018. **28**(7): p. 1411-1416.
86. Yoshida, Y., J. Zeni, and L. Snyder-Mackler, *Do patients achieve normal gait patterns 3 years after total knee arthroplasty?* J Orthop Sports Phys Ther, 2012. **42**(12): p. 1039-49.
87. Nha, K.W., et al., *Gait comparison of unicompartmental knee arthroplasty and total knee arthroplasty during level walking.* PLoS One, 2018. **13**(8): p. e0203310.
88. Komnik, I., et al., *Non-Sagittal Knee Joint Kinematics and Kinetics during Gait on Level and Sloped Grounds with Unicompartmental and Total Knee Arthroplasty Patients.* PLoS One, 2016. **11**(12): p. e0168566.
89. Jung, M.C., et al., *Difference in knee rotation between total and unicompartmental knee arthroplasties during stair climbing.* Knee Surg Sports Traumatol Arthrosc, 2014. **22**(8): p. 1879-86.
90. Creaby, M.W., et al., *Dynamic knee loading is related to cartilage defects and tibial plateau bone area in medial knee osteoarthritis.* Osteoarthritis Cartilage, 2010. **18**(11): p. 1380-5.
91. Kim, W.K., et al., *Reliability and validity of isometric knee extensor strength test with hand-held dynamometer depending on its fixation: a pilot study.* Ann Rehabil Med, 2014. **38**(1): p. 84-93.
92. Martins, J., et al., *Reliability and Validity of the Belt-Stabilized Handheld Dynamometer in Hip- and Knee-Strength Tests.* J Athl Train, 2017. **52**(9): p. 809-819.
93. Harbourne, A.D., et al., *Predictors of return to desired activity 12 months following unicompartmental and total knee arthroplasty.* Acta Orthop, 2019. **90**(1): p. 74-80.
94. Bakeman, R., *Recommended effect size statistics for repeated measures designs.* Behavior

Research Methods, 2005. **37**(3): p. 379-384.

95. Berman, A.T., S.J. Bosacco, and C. Israelite, *Evaluation of Total Knee Arthroplasty Using Isokinetic Testing.* Clinical Orthopaedics and Related Research, 1991. **&NA;**(271): p. 106???113.
96. Walsh, M., et al., *Physical impairments and functional limitations: a comparison of individuals 1 year after total knee arthroplasty with control subjects.* Physical Therapy, 1998. **78**(3): p. 248.
97. Berth, A., et al., *Strength and voluntary activation of quadriceps femoris muscle in total knee arthroplasty with midvastus and subvastus approaches.* J Arthroplasty, 2007. **22**(1): p. 83-8.
98. Takubo, A., et al., *Comparison of Muscle Recovery Following Bi-cruciate Substituting versus Posterior Stabilized Total Knee Arthroplasty in the Asian Population.* 2017. **30**(7): p. 725-729.
99. Umrani, S.P., K.Y. Cho, and K.I. Kim, *Patellar eversion does not adversely affect quadriceps recovery following total knee arthroplasty.* J Arthroplasty, 2013. **28**(4): p. 591-4.
100. Andrews, S.N., et al., *Fixed Distal Femoral Cut of 6 degrees Valgus in Total Knee Arthroplasty: A Radiographic Review of 788 Consecutive Cases.* J Arthroplasty, 2018.
101. Luc-Harkey, B.A., et al., *Quadriceps Rate of Torque Development and Disability in Persons With Tibiofemoral Osteoarthritis.* J Orthop Sports Phys Ther, 2018. **48**(9): p. 694-703.
102. Mutsuzaki, H.T., R. Mataki, Y. Wadano, Y., *Target range of motion for rehabilitation after total knee arthroplasty.* J Rural Med, 2017. **12**(1): p. 33-37.
103. Kito, N., et al., *Contribution of knee adduction moment impulse to pain and disability in Japanese women with medial knee osteoarthritis.* Clin Biomech (Bristol, Avon), 2010. **25**(9): p. 914-9.
104. Sumner, B., et al., *Comparison of Knee Kinematics and Kinetics during Stair Ascent in Single-Radius and Multiradius Total Knee Arthroplasty.* J Knee Surg, 2019. **32**(9): p. 872-878.
105. Rodriguez-Merchan, E.C. and P. Gomez-Cardero, *Unicompartmental knee arthroplasty: Current indications, technical issues and results.* EFORT Open Rev, 2018. **3**(6): p. 363-373.
106. Wilson, C.T., A. Damarell, R. Krishnan, J. , *Knee instaiblity as the primary cause of failure following Total Knee Arthroplasty (TKA): A systematic review on patient, surgical and implant characteristics of revised TKA patients.* The Knee, 2017. **24**(6): p. 1271-1281.
107. Silva, M., et al., *Knee strength after total knee arthroplasty.* The Journal of Arthroplasty, 2003. **18**(5): p. 605-611.
108. Andriacchi, P.T., O.J. Galante, and W.R. Fermier, *The influence of total knee-replacement design on walking and stair-climbing.* The Journal of Bone & Joint Surgery, 1982. **64**(9): p. 1328-1335.
109. Andriacchi, T.P., et al., *Patellofemoral design influences function following total knee arthroplasty.* The Journal of arthroplasty, 1997. **12**(3): p. 243-249.
110. Iwaki, H.P., V. Freeman, MAR. , *Tibiofemoral movement 1: the shapes and relative movements of the fmur and tibia in the unloaded cadaver knee.* Journal of Bone and Joint Surgery (Br), 2000. **82**: p. 1189-95.

111. Browne, C., et al., *Patellofemoral forces after total knee arthroplasty: effect of extensor moment arm.* Knee, 2005. **12**(2): p. 81-8.
112. D'Lima, D.D., et al., *Quadriceps moment arm and quadriceps forces after total knee arthroplasty.* Clinical Orthopaedics And Related Research, 2001(392): p. 213-220.
113. Parratte, S. and M.W. Pagnano, *Instability after total knee arthroplasty.* The Journal of bone and joint surgery. American volume, 2008. **90**(1): p. 184-194.
114. Mahoney, O.M., et al., *The effect of total knee arthroplasty design on extensor mechanism function.* The Journal of arthroplasty, 2002. **17**(4): p. 416-421.
115. Kim, D.H., et al., *Is Single-Radius Design Better for Quadriceps Recovery in Total Knee Arthroplasty?* Knee Surg Relat Res, 2015. **27**(4): p. 240-6.
116. Collados-Maestre, I., et al., *Better functional outcome after single-radius TKA compared with multi-radius TKA.* Knee Surg Sports Traumatol Arthrosc, 2017. **25**(11): p. 3508-3514.
117. Hatfield, G.L., et al., *The effect of total knee arthroplasty on knee joint kinematics and kinetics during gait.* J Arthroplasty, 2011. **26**(2): p. 309-18.
118. Stoddard, J.E., et al., *The kinematics and stability of single-radius versus multi-radius femoral components related to mid-range instability after TKA.* J Orthop Res, 2013. **31**(1): p. 53-8.
119. Jones, E.L., et al., *A systematic review of patient reported outcomes and patient experience in enhanced recovery after orthopaedic surgery.* Ann R Coll Surg Engl, 2014. **96**(2): p. 89-94.
120. Oliviu, R.C., et al., *Mid-term results of total knee replacement with single-radius versus multi-radius posterior-stabilized implants.* Acta Orthop Traumatol Turc, 2016. **50**(2): p. 125-31.
121. Liu, S., et al., *Meta-Analysis of Outcomes of a Single-Radius Versus Multi-Radius Femoral Design in Total Knee Arthroplasty.* J Arthroplasty, 2016. **31**(3): p. 646-54.
122. Gomez-Barrena, E., et al., *Functional performance with a single-radius femoral design total knee arthroplasty.* Clin Orthop Relat Res, 2010. **468**(5): p. 1214-20.
123. Wang, H., et al., *A biomechanical comparison between the single-axis and multi-axis total knee arthroplasty systems for the stand-to-sit movement.* Clinical biomechanics (Bristol), 2005. **20**(4): p. 428-433.
124. Wang, H., et al., *Biomechanical differences exhibited during sit-to-stand between total knee arthroplasty designs of varying radii.* J Arthroplasty, 2006. **21**(8): p. 1193-9.
125. Hall, J., et al., *Extensor Mechanism Function in Single-Radius Vs Multiradius Femoral Components for Total Knee Arthroplasty.* The Journal of Arthroplasty, 2008. **23**(2): p. 216-219.
126. Creaby, M.W., et al., *Sagittal plane joint loading is related to knee flexion in osteoarthritic gait.* Clin Biomech (Bristol, Avon), 2013. **28**(8): p. 916-20.
127. Chandran, V.D., et al., *Knee muscle co-contractions are greater in old compared to young adults during walking and stair use.* Gait Posture, 2019. **73**: p. 315-322.
128. Astephen Wilson, J.L., et al., *The association between knee joint biomechanics and neuromuscular control and moderate knee osteoarthritis radiographic and pain severity.* Osteoarthritis Cartilage, 2011. **19**(2): p. 186-93.
129. Swanik, C.B., S.M. Lephart, and H.E. Rubash, *Proprioception, kinesthesia, and balance after TKA with cruciate-retaining and posterior stabilized prostheses.* Journal of Bone and Joint Surgery, 2004. **86**(2): p. 328-334.

130. Cho, K.Y., et al., *Does Cruciate-Retaining Total Knee Arthroplasty Show Better Quadriceps Recovery than Posterior-Stabilized Total Knee Arthroplasty? - Objective Measurement with a Dynamometer in 102 Knees.* Clin Orthop Surg, 2016. **8**(4): p. 379-385.
131. Kean, C.O., et al., *Impact loading following quadriceps strength training in individuals with medial knee osteoarthritis and varus alignment.* Clin Biomech (Bristol, Avon), 2017. **42**: p. 20-24.
132. Besier, T.F., et al., *Repeatability of gait data using a functional hip joint centre and a mean helical knee axis.* Journal of Biomechanics, 2003. **36**(8): p. 1159-1168.
133. Joseph, C.W., et al., *The interday reliability of ankle, knee, leg, and vertical musculoskeletal stiffess during hopping and overground running.* Journal of Applied Biomechanics, 2013. **29**: p. 386-394.
134. Ruggiero, L., S. Dewhurst, and T.M. Bampouras, *Validity and Reliability of Two Field-Based Leg Stiffness Devices: Implications for Practical Use.* J Appl Biomech, 2016. **32**(4): p. 415-9.
135. Farley, C.T., J. Glasheen, and T.A. McMahon, *Running springs: speed and animal size.* J Exp Biol, 1993. **185**: p. 71-86.
136. Cavagna, G.A., et al., *The determinants of the step frequency in running, trotting, and hopping in man and other vertebrates.* Journal of Physiology, 1988. **399**: p. 81-92.
137. Stuart, D.G., *Integration of posture and movement: contributions of Sherrington, Hess, and Bernstein.* Hum Mov Sci, 2005. **24**(5-6): p. 621-43.
138. Leyton, A.S., C., *Observations on the excitable cortex of the chimpanzee, orangutan, and gorilla.* Experimental Physiology, 1916. **11**(2): p. 135-222.
139. Morecraft, R.J., et al., *Terminal distribution of the corticospinal projection from the hand/arm region of the primary motor cortex to the cervical enlargement in rhesus monkey.* J Comp Neurol, 2013. **521**(18): p. 4205-35.
140. Hazzaa, N.M., et al., *Somatotopic organization of corticospinal/corticobulbar motor tracts in controls and patients with tumours: A combined fMRI-DTI study.* Neuroimage Clin, 2019. **23**: p. 101910.
141. Moore, J.C., *The golgi tendon organ: a review and update.* The American Journal of Occupational Therapy, 1984. **38**(4): p. 227-236.
142. Paillard, J., *Body schema and body image - a double dissociation in deafferented patients.* Motor Control, Today and Tomorrow, 1999: p. 197-214.
143. Anema, H.A., et al., *A double dissociation between somatosensory processing for perception and action.* Neuropsychologia, 2009. **47**(6): p. 1615-20.
144. Cote, M.P., L.M. Murray, and M. Knikou, *Spinal Control of Locomotion: Individual Neurons, Their Circuits and Functions.* Front Physiol, 2018. **9**: p. 784.
145. Lance, J.W. and J.G. McLeod, *4 - Spinal reflexes*, in *A Physiological Approach to Clinical Neurology (Third Edition)*, J.W. Lance and J.G. McLeod, Editors. 1981, Butterworth-Heinemann. p. 73-100.
146. Schwenkreis, P., et al., *Repetitive training of a synchronised movement induces short-term plastic changes in the human primary somatosensory cortex.* Neurosci Lett, 2001. **312**: p. 99-102.
147. Houk, J. and E. Henneman, *Responses of Golgi tendon organs to active contractions of the soleus muscle of the cat.* J Neurophysiol, 1967. **30**(3): p. 466-81.
148. Grey, M.J., et al., *Group II muscle afferents probably contribute to the medium latency*

soleus stertch reflex during walking in humans. Journal of Physiology, 2001. **534**(3): p. 925-933.

149. Roujeau, T., P. Decq, and J.P. Lefaucheur, *Surface EMG recording of heteronymous reflex excitation of semitendinosus motoneurones by group II afferents.* Clin Neurophysiol, 2004. **115**(6): p. 1313-9.
150. Smith, S.A., J.H. Mitchell, and M.G. Garry, *The mammalian exercise pressor reflex in health and disease.* Exp Physiol, 2006. **91**(1): p. 89-102.
151. Amann, M., *Significance of Group III and IV muscle afferents for the endurance exercising human.* Clin Exp Pharmacol Physiol, 2012. **39**(9): p. 831-5.
152. Nobrega, A.C., et al., *Neural regulation of cardiovascular response to exercise: role of central command and peripheral afferents.* Biomed Res Int, 2014. **2014**: p. 478965.
153. Messlinger, K., . *Functional morphology of nociceptive and other fine sensory endings (free nerve endings) in different tissues*, in *Progress in brain research*. 1996, Elsevier. p. 273-298.
154. Porreca, F. and M.H.G. Ossipov, G. F., *Chronic pain and medullary descending facilitation.* TRENDS in Neurosciences, 2002. **25**(6): p. 319-325.
155. Bendtsen, T.F., et al., *The Optimal Analgesic Block for Total Knee Arthroplasty.* Reg Anesth Pain Med, 2016. **41**(6): p. 711-719.
156. Kaufman, M.P. and K.J. Rybicki, *Discharge properties of group III and IV muscle afferents: their responses to mechanical and metabolic stimuli.* Circ Res, 1987. **61**(4 Pt 2): p. I60-5.
157. Kaufman, M.P., et al., *Effects of static muscular contraction on impulse activity of groups III and IV afferents in cats.* Journal of Applied Physiology, 1983. **55**(1): p. 105-112.
158. Weerakkody, N.S., et al., *Local subcutaneous and muscle pain impairs detection of passive movements at the human thumb.* J Physiol, 2008. **586**(13): p. 3183-93.
159. Goble, D., *Proprioceptive Acuity Assessment Via Joint Position Matching: From Basic Science to General Practice.* Phys Ther, 2010. **90**(8): p. 1176-1184.
160. Craske, B., *Perception of impossible limb positions induced by tendon vibration.* Science, 1977. **196**(4285): p. 71-73.
161. Johansson, R.S. and J.R. Flanagan, *Coding and use of tactile signals from the fingertips in object manipulation tasks.* Nat Rev Neurosci, 2009. **10**(5): p. 345-59.
162. Blalock, D., et al., *Joint instability and osteoarthritis.* Clin Med Insights Arthritis Musculoskelet Disord, 2015. **8**: p. 15-23.
163. Moore, K.L., A.F. Dalley, and A.M.R. Agur, *Clinically Oriented Anatomy*. 7th ed. 2014: Wolters Kluwer; Lippincott Williams & Wilkins.
164. Blakemore, S.J., D. Wolpert, and C. Frith, *Why can't you tickle yourself?* Neuroreport, 2000. **11**(11): p. R11-6.
165. Piriyaprasarth, P., et al., *The reliability of knee joint position testing using electrogoniometry.* BMC Musculoskelet Disord, 2008. **9**: p. 6.
166. Torres, R., et al., *Impact of patellar tendinopathy on knee proprioception: a cross-sectional study.* Clin J Sport Med, 2017. **27**(1): p. 31-36.
167. Zavieh, M.K., et al., *Measurement of force sense reproduction in the knee joint: application of a new dynamometric device.* J Phys Ther Sci, 2016. **28**: p. 2311-2315.
168. Niespodzinski, B., et al., *Relationship between Joint Position Sense, Force Sense, and Muscle Strength and the Impact of Gymnastic Training on Proprioception.* Biomed Res Int, 2018. **2018**: p. 5353242.

169. Relph, N. and L. Herrington, *Interexaminer, intraexaminer and test-retest reliability of clinical knee joint-position-sense measurements using an image-capture technique.* J Sport Rehabil, 2015. **24**(2).

170. Roudaut, Y., et al., *Touch sense: functional organization and molecular determinants of mechanosensitive receptors.* Channels (Austin), 2012. **6**(4): p. 234-45.

171. Elangoven, N., A. Herrmann, and J. Konczak, *Assessing Proprioceptive Function: Evaluating Joint Position Matching Methods Against Psychophysical Thresholds.* Phys Ther, 2014. **94**: p. 553-561.

172. Pai, Y., et al., *Effect of age and osteoarthritis on knee proprioception.* Arthritis & Rheumatism, 1997. **40**(12): p. 2260-2265.

173. Brindle, T.J., et al., *The influence of ankle joint movement on knee joint kinesthesia at various movement velocities.* Scandinavian Journal of Medicine & Science in Sports, 2010. **20**(2): p. 262-267.

174. Moon, S. and Y. Kim, *Effect of blocked vision treadmill training on knee joint proprioception of patients with chronic stroke.* J Phys Ther Sci, 2015. **27**: p. 897-900.

175. Han, J., et al., *Does elastic resistance affect finger pinch discrimination?* Human factors, 2013. **55**(5): p. 976-984.

176. Kobayashi, H., W. Kakihana, and T. Kimura, *Combined effects of age and gender on gait symmetry and regularity assessed by autocorrelation of trunk acceleration.* Journal of NeuroEngineering and Rehabilitation, 2014. **11**.

177. Kaplan, F.S., et al., *Age-related changes in proprioception and sensation of joint position.* Acta Orthop Scand, 1985. **56**(1): p. 72-4.

178. Wingert, J.R., C. Welder, and P. Foo, *Age-related hip proprioception declines: effects on postural sway and dynamic balance.* Arch Phys Med Rehabil, 2014. **95**(2): p. 253-61.

179. Kim, G.H., S. Suzuki, and K. Kanda, *Age-related physiological and morphological changes of muscle spindles in rats.* J Physiol, 2007. **582**(Pt 2): p. 525-38.

180. Narici, M.V. and C. Maganaris, N., *Plasticity of the muscle-tendon complex with disuse and aging.* Exerc Sport Sci Res, 2007. **35**(3): p. 126-134.

181. Shaffer, S.W. and A.L. Harrison, *Aging of the somatosensory system: A translational perspective.* Phys Ther, 2007. **87**(2): p. 193-207.

182. Gunther, M. and R. Blickhan, *Joint stiffness of the ankle and the knee in running.* Journal of Biomechanics, 2002. **35**: p. 1459-1474.

183. Mauroy, G., B. Schepens, and P.A. Willems, *Leg stiffness and joint stiffness while running to and jumping over an obstacle.* J Biomech, 2014. **47**(2): p. 526-35.

184. Hebert-Losier, K. and A. Eriksson, *Leg stiffness measures depend on computational method.* J Biomech, 2014. **47**(1): p. 115-21.

185. McMahon, T.A., G. Valiant, and E.C. Frederick, *Groucho running.* J Appl Physiol (1985), 1987. **62**(6): p. 2326-37.

186. Dalleau, G., et al., *A simple method for field measurements of leg stiffness in hopping.* Int J Sports Med, 2004. **25**(3): p. 170-6.

187. Lloyd, R.S., et al., *Reliability and validity of field-based measures of leg stiffness and reactive strength index in youths.* J Sports Sci, 2009. **27**(14): p. 1565-73.

188. Hsu, M.-J., et al., *Leg stiffness and electromyography of knee extensors/flexors: Comparison between older and younger adults during stair descent.* The Journal of Rehabilitation Research and Development, 2007. **44**(3).

189. Hortobagyi, T. and P. DeVita, *Muscle pre- and coactivity during downward stepping are*

associated with leg stiffness in aging. Journal of Electromyography and Kinesiology, 2000. **10**: p. 117-126.

190. Jones, S.F., et al., *The mechanics of landing when stepping down in unilateral lower-limb amputees.* Clinical Biomechanics, 2006. **21**(2): p. 184-193.
191. Bennett, H.J., et al., *Validation of the greater trochanter method with radiographic measurements of frontal plane hip joint centers and knee mechanical axis angles and two other hip joint center methods.* J Biomech, 2016. **49**(13): p. 3047-3051.
192. Weinhandl, J.T. and K.M. O'Connor, *Assessment of a greater trochanter-based method of locating the hip joint center.* J Biomech, 2010. **43**(13): p. 2633-6.
193. Kirkwood, R.N., E.G. Culham, and P. Costigan, *Radiographic and non-invasive determination of the hip joint center location: effect on hip joint moments.* Clinical Biomechanics, 1998. **14**: p. 227-235.
194. Silder, A., T. Besier, and S.L. Delp, *Running with a load increases leg stiffness.* J Biomech, 2015. **48**(6): p. 1003-8.
195. Blickhan, R. and R.J. Full, *Similiarity in multi-legged locomotion: Bouncing like a monopode.* Journal of Comparative Physiology, 1993. **173**: p. 509-517.
196. Arden, N. and M.C. Nevitt, *Osteoarthritis: epidemiology.* Best Pract Res Clin Rheumatol, 2006. **20**(1): p. 3-25.
197. Wallace, I.J., et al., *Knee osteoarthritis has doubled in prevalence since the mid-20th century.* Proc Natl Acad Sci U S A, 2017. **114**(35): p. 9332-9336.
198. Cram, P., et al., *Total knee arthroplasty volume, utilization, and outcomes among Medicare beneficiaries, 1991-2010.* JAMA, 2012. **308**(12): p. 1227-36.
199. Kurtz, S., et al., *Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030.* J Bone Joint Surg Am, 2007. **89**(4): p. 780-5.
200. Bourne, R.B., et al., *Patient satisfaction after total knee arthroplasty: who is satisfied and who is not?* Clin Orthop Relat Res, 2010. **468**(1): p. 57-63.
201. Noble, P.C., et al., *The John Insall Award: Patient expectations affect satisfaction with total knee arthroplasty.* Clin Orthop Relat Res, 2006. **452**: p. 35-43.
202. Kellgren, J.H. and J.S. Lawrence, *Radiological assessment of osteoarthrosis.* Ann Rheum Dis, 1957. **16**.
203. Jeffery, R.M., RW. Denham RA., *Coronal alignment after total knee replacement.* Journal of Bone and Joint Surgery (Br), 1991. **73**: p. 709-14.
204. Stickley, C.D., et al., *Standard Radiographs Are Not Sufficient for Assessing Knee Mechanical Axis in Patients With Advanced Osteoarthritis.* J Arthroplasty, 2017. **32**(3): p. 1013-1017.
205. Paterson, K.L., et al., *The influence of sex and obesity on gait biomechanics in people with severe knee osteoarthritis scheduled for arthroplasty.* Clin Biomech (Bristol, Avon), 2017. **49**: p. 72-77.
206. Debbi, E.M., et al., *Frontal plane biomechanics of the operated and non-operated knees before and after unilateral total knee arthroplasty.* Clin Biomech (Bristol, Avon), 2015. **30**(9): p. 889-94.
207. Niki, Y., et al., *Kinematically aligned total knee arthroplasty reduces knee adduction moment more than mechanically aligned total knee arthroplasty.* Knee Surgery, Sports Traumatology, Arthroscopy, 2018. **26**(6): p. 1629-1635.
208. Orishimo, K.F., et al., *Does total knee arthroplasty change frontal plane knee biomechanics during gait?* Clin Orthop Relat Res, 2012. **470**(4): p. 1171-6.

209. Shimada, N., et al., *Courses of change in knee adduction moment and lateral thrust differ up to 1 year after TKA.* Knee Surg Sports Traumatol Arthrosc, 2016. **24**(8): p. 2506-11.
210. Landry, S.C., et al., *Knee biomechanics of moderate OA patients measured during gait at a self-selected and fast walking speed.* J Biomech, 2007. **40**(8): p. 1754-61.
211. Robbins, S.M. and M.R. Maly, *The effect of gait speed on the knee adduction moment depends on waveform summary measures.* Gait Posture, 2009. **30**(4): p. 543-6.
212. Doyle, S.S., et al., *The effect of surface inclination and limb on knee loading measures in transtibial prosthesis users.* J Neuroeng Rehabil, 2019. **16**(1): p. 37.
213. Kean, C.O., et al., *Comparison of peak knee adduction moment and knee adduction moment impulse in distinguishing between severities of knee osteoarthritis.* Clin Biomech (Bristol, Avon), 2012. **27**(5): p. 520-3.
214. Stickley, C.D., et al., *Dynamic Varus and the Development of Iliotibial Band Syndrome.* J Athl Train, 2018. **53**(2): p. 128-134.
215. Farrokhi, S., et al., *Knee joint contact mechanics during downhill gait and its relationship with varus/valgus motion and muscle strength in patients with knee osteoarthritis.* Knee, 2016. **23**(1): p. 49-56.
216. Sosdian, L., et al., *Longitudinal changes in knee kinematics and moments following knee arthroplasty: a systematic review.* Knee, 2014. **21**(6): p. 994-1008.
217. Kaufman, K.H., C. Morrey, B. Morrey, M. An, K., *Gait characteristics of patients with knee osteoarthritis.* Journal of Biomechanics, 2001. **34**: p. 907-915.
218. Davis, H.C., et al., *Sagittal plane walking biomechanics in individuals with knee osteoarthritis after quadriceps strengthening.* Osteoarthritis Cartilage, 2019. **27**(5): p. 771-780.
219. Costigan, P.D., KJ. Wyss, UP., *Knee and hip kinetics during normal stair climbing.* Gait & Posture, 2002. **16**: p. 31-37.
220. Standifird, T.W., H.E. Cates, and S. Zhang, *Stair ambulation biomechanics following total knee arthroplasty: a systematic review.* J Arthroplasty, 2014. **29**(9): p. 1857-62.
221. Hicks-Little, C.A., et al., *Lower extremity joint kinematics during stair climbing in knee osteoarthritis.* Med Sci Sports Exerc, 2011. **43**(3): p. 516-24.
222. McClelland, J.A., et al., *Patterns in the knee flexion-extension moment profile during stair ascent and descent in patients with total knee arthroplasty.* J Biomech, 2014. **47**(8): p. 1816-21.
223. Bjerke, J., et al., *Compensatory strategies for muscle weakness during stair ascent in subjects with total knee arthroplasty.* J Arthroplasty, 2014. **29**(7): p. 1499-502.
224. Allen, K.D. and Y.M. Golightly, *State of the evidence.* Curr Opin Rheumatol, 2015. **27**(3): p. 276-83.
225. Hollister, A., et al., *THE AXES OF ROTATION OF THE KNEE.* Clinical Orthopaedics And Related Research, 1993(290): p. 259-268.
226. Churchill, D.L., et al., *The transepicondylar axis approximates the optimal flexion axis of the knee.* Clinical Orthopaedics And Related Research, 1998(356): p. 111-118.
227. Larsen, B., M.C. Jacofsky, and D.J. Jacofsky, *Quantitative, Comparative Assessment of Gait Between Single-Radius and Multi-Radius Total Knee Arthroplasty Designs.* J Arthroplasty, 2015. **30**(6): p. 1062-7.
228. Vahtrik, D., et al., *Relationship between leg extensor muscle strength and knee joint loading during gait before and after total knee arthroplasty.* Knee, 2014. **21**(1): p. 216-20.

229. Wolterbeek, N., R.G. Nelissen, and E.R. Valstar, *No differences in in vivo kinematics between six different types of knee prostheses.* Knee Surg Sports Traumatol Arthrosc, 2012. **20**(3): p. 559-64.
230. Jo, A.R., et al., *A comparison of stability and clinical outcomes in single-radius versus multi-radius femoral design for total knee arthroplasty.* J Arthroplasty, 2014. **29**(12): p. 2402-6.
231. Kessler, O., et al., *Sagittal curvature of total knee replacements predicts in vivo kinematics.* Clin Biomech (Bristol, Avon), 2007. **22**(1): p. 52-8.
232. Luo, Z., et al., *Long-term results of total knee arthroplasty with single-radius versus multi-radius posterior-stabilized prostheses.* J Orthop Surg Res, 2019. **14**(1): p. 139.
233. Fenner, V.U., H. Behrend, and M.S. Kuster, *Joint Mechanics After Total Knee Arthroplasty While Descending Stairs.* J Arthroplasty, 2017. **32**(2): p. 575-580.
234. Sumner, B., et al., *Comparison of Knee Kinematics and Kinetics during Stair Descent in Single- and Multi-Radius Total Knee Arthroplasty.* 2019.
235. Cross, M.B. and R. Berger, *Feasibility and safety of performing outpatient unicompartmental knee arthroplasty.* Int Orthop, 2014. **38**(2): p. 443-7.
236. Chawla, H., et al., *Annual revision rates of partial versus total knee arthroplasty: A comparative meta-analysis.* Knee, 2017. **24**(2): p. 179-190.
237. Lim, J.W., et al., *Oxford unicompartmental knee arthroplasty versus age and gender matched total knee arthroplasty - functional outcome and survivorship analysis.* J Arthroplasty, 2014. **29**(9): p. 1779-83.
238. Komnik, I., et al., *Compromised knee internal rotation in total knee arthroplasty patients during stair climbing.* PLoS One, 2018. **13**(10): p. e0205492.
239. Shiwaku, K., et al., *Varus kinematics at knee flexion affect clinical outcomes of unicompartmental knee arthroplasty: Intraoperative navigation-based kinematics evaluation.* Asia Pac J Sports Med Arthrosc Rehabil Technol, 2020. **20**: p. 6-11.

PART III

APPENDICES

APPENDIX A

RETROREFLECTIVE MARKER LOCATIONS FOR 3D BIOMECHANICAL ANALYSIS

Bilateral:

- 1st MP Joint (remove after calibration)
- 2nd MP Joint
- 5th MP Joint
- Base of 5th Metatarsal
- Medial Malleolus (remove after calibration)
- Lateral Malleolus
- Posterior Calcaneus (insertion of Achilles tendon)
- Medial Femoral Epicondyle (remove after calibration)
- Lateral Femoral Epicondyle
- Anterior Superior Iliac Spine (ASIS)
- Posterior Superior Iliac Spine (PSIS)
- Acromioclavicular (AC) Joint

Unilateral:

- Jugular (suprasternal) Notch
- Xiphoid Process
- C7 Spinous Process
- T10 Spinous Process
- Inferior Angle of Right Scapula

(Xiphoid Process and T10 Spinous Process attached to inactive heart-rate monitor)

Four Arrays (exact position does not matter):

- Bilateral Femur – Thigh
- Bilateral Shank – Lower Leg

APPENDIX B

LAB EQUIPMENT AND KNEE IMPLANT SPECIFICATIONS

Scale: Cardinal Detecto certified scale, model no. 442
Webb City, MO USA

Height: Seca, model no. 67032, Country Technology, Inc.
Gays Mills, WI USA

Vicon: Nexus 2.5.0
Vicon Motion Systems, Vicon LA, Culver City, CA USA

Cameras: 18 Total
7x MX T40-S
6x MX 13
5x MX 3+
All: Vicon Motion Systems, Vicon LA, Culver City, CA USA

Force plates: 2x OR6 Series, model no. MCA 6
1 in floor, 1 in stairs
Advanced Mechanical Technology, Inc., Phoenix, AZ USA

Visual 3Dv4: Software from C-Motion, Inc.
Germantown, MD USA

Timers: Brower Timing System, x4 infrared TC-Photographic timers
Power Systems, Inc., Knoxville, TN USA

Dynamometer: 2x MicroFET2 Wireless Dynamometer
Hoggan Scientific, LLC, Salt Lake City, UT USA

Stair Dimensions – each step is within ADA compliance:
→ 18.5 cm tall x 46.5cm wide (force plate width) x 28cm deep (tread)

Digital Camera: Basler A602FC
2x; 1 operational, 1 not operating

Lamps: 2x Smith-Victor DYH 600W; both operational

Knee Implants – all FDA approved

Multiradius TKA: “Balanced Knee System” Ortho Development Corp (Draper, UT USA)

Single radius TKA: “Get Around Knee” Stryker Orthopedics (Mahwah, NJ USA)

Unicompartmental: “Oxford – Medial” Zimmer Biomet Orthopedics (Warsaw, IN USA)

APPENDIX C

APPROVED DATA COLLECTION FORMS FOR ALL INCLUDED STUDIES

Activity Assessment Survey

Subject ID#: _______________ Data Collection Period 0 1 2 3 4 5 6 7

Please circle the number that best describes current activity level.

1. Wholly inactive, dependent on others, and cannot leave residence
2. Mostly inactive or restricted to minimum activities of daily living
3. Sometimes participates in mild activities, such as walking, limited housework and limited shopping
4. Regularly participates in mild activities
5. Sometimes participates in moderate activities such as swimming or could do unlimited housework or shopping
6. Regularly participates in moderate activities
7. Regularly participates in active events such as bicycling
8. Regularly participates in active events, such as golf or bowling
9. Sometimes participates in impact sports such as jogging, tennis, skiing, acrobatics, ballet, heavy labor, or backpacking
10. Regularly participates in impact sports

Please circle the number that best answers the following question. “How does your knee affect your ability to rise form a chair?”:

1. “Because of my knee I cannot rise from a chair.”
2. “Because of my knee, I can only rise from a chair if I use my hands and arms to assist.”
3. “I have pain when rising from the seated position, but it does not affect my ability to rise from the seated position.”
4. “My knee does not affect my ability to rise from a chair.”

Are you satisfied with your implant?
Yes No

Manual Muscle Testing Data Collection

Subject ID#: _______________ Data Collection Period 0 1 2 3 4 5 6 7

Patient's Operated leg: L / R Dominant Leg: L / R

Tester: ______________________

	Left Leg						Right Leg					
	Trial 1 Score (ft-lbf)	Pain Score (HHD/ Jt)	Trail 2 Score (ft-lbf)	Pain Score (HHD/ Jt)	Trial 3 Score (ft-lbf)	Pain Score (HHD/ Jt)	Trial 1 Score (ft-lbf)	Pain Score (HHD/ Jt)	Trial 2 Score (ft-lbf)	Pain Score (HHD/ Jt)	Trial 3 Score (ft-lbf)	Pain Score (HHD/ Jt)
Hip abduction		/		/		/		/		/		/
Knee extension		/		/		/		/		/		/

Anthropometric Data

Subject ID#: _______________ Date_________

Age________________ Gender: F / M

Data Collection Period 0 1 2 3 4 5

Patient's Operated leg: L / R Dominant Leg: L / R

Date of Surgery_________________

Weeks after Surgery________________

Vicon/Nexus Measurements

Weight (kg)	
Height (mm)	
Age (yrs)	
Left leg length (mm)	
Left knee width (mm)	
Left ankle width (mm)	
Right leg length (mm)	
Right knee width (mm)	
Right ankle width (mm)	

Data Collection Form

Subject ID#: _______________

Data Collection Period   0 1 2 3 4 5

Patient's Operated leg: L / R   Dominant leg: L / R

Total Trials: 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20

Walking Trials

Trial	Which foot hit the plate	Walking Pace (s)
1	R / L	
2	R / L	
3	R / L	

Stair Ascent

Trial	Which foot hit the plate	Walking Pace (s)
1	R / L	
2	R / L	
3	R / L	

Stair Decent

Trial	Which foot hit the plate	Walking Pace (s)
1	R / L	
2	R / L	
3	R / L	

Anthropometric Data

Subject ID#: ________________ Date__________

Age_________________

Gender: F / M Dominant Leg: L / R

Measurements

Weight (kg)	
Height (m)	

Stair Negotiation Data Collection Form

Stair Ascent		Stair Descent	
Trial	Which foot hit the plate	Trial	Which foot hit the plate
1	R / L	1	R / L
2	R / L	2	R / L
3	R / L	3	R / L
4	R / L	4	R / L
5	R / L	5	R / L
6	R / L	6	R / L
7	R / L	7	R / L
8	R / L	8	R / L
9	R / L	9	R / L
10	R / L	10	R / L

ID #: ____________________________________ **DATE**: ____________________

Participant Health Questionnaire:

1	Has your doctor ever said that you have a heart condition and that you should only perform physical activity recommended by a doctor?	**YES**	**NO**
2	In the past month, have you had chest pain?	**YES**	**NO**
3	Do you lose your balance because of dizziness?	**YES**	**NO**
4	Have you ever been diagnosed with Parkinson's Disease?	**YES**	**NO**
5	Do you have a history of fainting?	**YES**	**NO**
6	Have you ever been diagnosed with a neurological disorder?	**YES**	**NO**
7	Do you have diabetes mellitus?	**YES**	**NO**
8	Do you have a bone or joint problem that could be made worse by physical activity?	**YES**	**NO**
9	Has a doctor ever diagnosed you with rheumatoid arthritis or osteoarthritis?	**YES**	**NO**
10	Within the six months, have you experienced an injury to your knee or any severe knee pain?	**YES**	**NO**
11	Have you had a previous hip, knee, ankle, or foot surgery?	**YES**	**NO**

M / F **AGE:** ____________________

ANTHROPOMETRICS

(to be filled out by the research team)

WEIGHT: ____________________ **HEIGHT:** _____________________

APPENDIX D

APPROVED INFORMED CONSENT DOCUMENTS FOR ALL INCLUDED STUDIES

RESEARCH SUBJECT INFORMATION AND CONSENT FORM

TITLE: Biomechanical Comparison of Multi- and Single Radius Implant Designs During Level Walking and Stair Climbing Tasks

PROTOCOL NO.: 2014-018
WIRB® Protocol #20141194

SPONSOR: Cris Stickley, PhD, ATC

INVESTIGATOR: Cass Nakasone, MD
888 South King Street
Honolulu, Hawaii 96813
United States

SITE(S): University of Hawaii at Manoa
PE/A Complex Room 231, Lower Campus Road
Honolulu, Hawaii 96822
United States

Straub Clinic & Hospital
888 S. King Street
Honolulu, Hawaii 96813
United States

STUDY-RELATED PHONE NUMBER(S): Cass Nakasone, M.D.
808-522-4232

Cris Stickley PhD, ATC
808-956-3798

This consent form may contain words that you do not understand. Please ask the study doctor or the study staff to explain any words or information that you do not clearly understand. You may take home an unsigned copy of this consent form to think about or discuss with family or friends before making your decision.

SUMMARY

You are being asked to be in a research study. The purpose of this consent form is to help you decide if you want to be in the research study. Please read this consent form carefully. To be in a research study you must give your informed consent. "Informed consent" includes:

- Reading this consent form
- Having the study doctor or study staff explain the research study to you
- Asking questions about anything that is not clear, and

- Taking home an unsigned copy of this consent form. This gives you time to think about it and to talk to family or friends before you make your decision.

You should not join this research study until all of your questions are answered.

Things to know before deciding to take part in a research study:

- The main goal of a research study is to learn things to help patients in the future.
- The main goal of regular medical care is to help each patient.
- No one can promise that a research study will help you.
- Taking part in a research study is entirely voluntary. No one can make you take part.
- If you decide to take part, you can change your mind later on and withdraw from the research study.
- The decision to join or not join the research study will not cause you to lose any medical benefits. If you decide not to take part in this study, your doctor will continue to treat you.
- Parts of this study may involve standard medical care. Standard care is the treatment normally given for a certain condition or illness.
- After reading the consent form and having a discussion with the research staff, you should know which parts of the study are experimental (investigational) and which are standard medical care.
- Your medical records may become part of the research record. If that happens, your medical records may be looked at and/or copied by the sponsor of this study and government agencies or other groups associated with the study.

After reading and discussing the information in this consent form you should know:

- Why this research study is being done;
- What will happen during the research;
- Any possible benefits to you;
- The possible risks to you;
- How problems will be treated during the study and after the study is over.

If you take part in this research study, you will be given a copy of this signed and dated consent form.

PURPOSE OF THE STUDY

The purpose of this study is to compare the function of patients, implanted with either a multi-radius or a single radius total knee arthroplasty design, during level walking and stair climbing tasks. You are being asked to participate in this study because you are undergoing total knee arthroplasty. About 100 subjects are expected to participate.

PROCEDURES

If you decide to participate in this study, you will be randomly assigned (by chance) to one of four possible groups and receive either a single radius knee implant or one of three multiple radii knee implants. You have an equal chance of being assigned to any one of the four implant groups. The implants that will be used in this study are:

- GetAroundKnee™, Stryker Orthopedics (single radius)
- Balanced Knee® System, Ortho Development (multiple radii),
- Persona ™ Total Knee, Zimmer (multiple radii)
- NexGen®, Zimmer (multiple radii)

These types of implants are approved by the FDA for the type of surgery you are having and will be used according to their approved indication.

You will be asked to report to the University of Hawaii at Manoa, Kinesiology and Rehabilitation Science Laboratory (Gait Lab) (Sherriff 100) for all testing visits before and after your knee surgery.

Upon arrival to the Gait Lab, you will be asked to fill out one survey in reference to your current pain and activity level. Measurements about your body will be taken and you will be asked to perform the following tasks:

(1) walk for 6 meters at a comfortable speed 6-10 times (Gait Analysis),

(2) walking up and down stairs at a comfortable speed 3-4 times, and

(3) push into stationary objects (fixed dynamometer) with your leg for three seconds for two different leg movements (Isometric Strength).

You will also be asked some questions about your daily activities. The entire visit will take approximately 60 minutes.

You will be asked to go to the Gait Lab for your first study visit before your surgery. You will be asked to return to the Gait Lab 5 more times over the next two years to repeat the procedures listed above (please see Table 1 below for visit schedule). Each visit to the Gait lab will take approximately 60 minutes.

Table 1. Visit Timeline

	Before Surgery	6 Weeks After Surgery	3 Months After Surgery	6 Months After Surgery	1 Year After Surgery	2 Years After Surgery
Gait Analysis (test)	X	X	X	X	X	X
Isometric	X	X	X	X	X	X

Strength						
Paper/Pencil Survey	X	X	X	X	X	X

RISKS AND DISCOMFORTS

Being randomized to one type of knee implant instead of the others, may lead to greater or lesser stability of the knee post-surgery.

There are risks associated with your knee replacement surgery, whether or not you participate in this study. These include:

- Blood clots that can, in rare cases, be life threatening
- Complications after a blood transfusion
- Allergic reaction to the medications or materials used
- Infection
- Injury to arteries or nerves in your leg
- Surgery may not reduce your pain and stiffness, possibly requiring more treatment
- Surgery may cause more pain
- Risks of anesthesia

You will be asked to review and sign a separate consent form for your knee surgery, and your surgeon will explain the risks of the procedure in more detail.

Gait analysis risks

Due to the level of physical activity involved during the testing procedures, there is a risk of injury. You may have pain in your affected joint during testing. You may also have some discomfort, muscle cramping or soreness during or after test sessions. Although we have people to assist you and handrails in place, there is a chance of falling during the test. There is a very remote chance of cardiac arrest and/or death. These risks are comparable to your routine rehabilitation and activities of daily living and will not affect your recovery from the surgery.

You cannot participate in this study if you are pregnant because the information collected during the walking test may not accurately represent your normal walking characteristics. If you are unaware that you are pregnant, participation in this study will result in no more danger to the mother or fetus than normal activities of daily living. However, if you become pregnant or think you might be pregnant during the course of this study, you must inform the researchers, and you will be removed from study participation.

NEW INFORMATION

You will be told about anything new that might change your decision to be in this study. You may be asked to sign a revised consent form if this occurs.

BENEFITS

You may not receive direct/immediate benefits from study participation. However, you will obtain information regarding your walking gait, functional activity capacity, hip muscular strength, and behavioral characteristics. Results of this study may assist physicians, physical therapists, and athletic trainers to ensure the optimal clinical outcomes to maintain the beneficial effects of total knee replacement.

PAYMENT FOR PARTICIPATION

You will not be paid for your participation in the study.

You will be given $5 that can be applied towards parking and/or transportation to the University of Hawaii Gait Laboratory each time you come for a visit. The money will be given to you after you arrive at the facility with a receipt, so it is a reimbursement. You will be reimbursed only for the visits that you attend.

COSTS

You are not expected to have additional costs related to the procedures and visits that may result from your participation in this research study.

Any additional costs associated with parking/transportation over and above the $5 provided will be your responsibility. The fee for parking at the University of Hawaii parking structure is $5 during the week and $6 on the weekends.

ALTERNATIVE TREATMENT

If you decide not to participate in this study, you will receive your knee replacement surgery with the type of implant that your doctor feels is best for you. Your follow-up care will be the same whether or not you are in this study.

USE AND DISCLOSURE OF YOUR HEALTH INFORMATION:

By signing this form, you are authorizing the use and disclosure of individually identifiable information. Your information will only be used/disclosed as described in this consent form and as permitted by state and federal laws. If you refuse to give permission, you will not be able to be in this research.

This consent covers all information about you that is used or collected for this study. It includes

- Past and present medical records
- Research records
- Records about your study visits.
- Information gathered for this research about:
    - Physical exams

    - Laboratory, x-ray, and other test results

    - Questionnaires

- Records about the implanted medical device.

Your authorization to use your identifiable health information will not expire even if you terminate your participation in this study or you are removed from this study by the study doctor. However, you may revoke your authorization to use your identifiable information at any time by submitting a written notification to the principal investigator, Cass Nakasone, MD at 888 S. King Street, Honolulu, HI 96813. If you decide to revoke (withdraw or "take back") your authorization, your identifiable health information collected or created for this study shall not be used or disclosed by the study doctor after the date of receipt of the written revocation except to the extent that the law allows us to continue using your information. The investigators in this study are not required to destroy or retrieve any of your health information that was created, used, or disclosed for this study prior to receiving your written revocation.

By signing this consent form, you authorize the following parties to use and or disclose your identifiable health information collected or created for this study:

- Cass Nakasone, MD, and his research staff for the purposes of conducting this research study.
- Straub Clinic & Hospital and Hawai'i Pacific Health

Your medical records may contain information about AIDS or HIV infection, venereal disease, treatment for alcohol and/or drug abuse, or mental health or psychiatric services. By signing this consent form, you authorize access to this information if it is in the records used by members of the research team.

The individuals named above may disclose your medical records, this consent form, and the information about you created by this study to:

- The sponsor of this study and their designees (if applicable)
- Federal, state, and local agencies having oversight over this research, such as the Office for Human Research Protections in the U.S. Department of Health and Human Services, Food and Drug Administration, the National Institutes of Health, etc.
- The University of Hawai'i
- Hawaii Pacific Health (HPH) Officials, the Western Institutional Review Board, and the HPH Office of Compliance for purposes of overseeing the research study and making sure that your ethical rights are being protected.

Some of the persons or groups that receive your study information may not be required to comply with federal privacy regulations, and your information may lose its federal privacy protection and your information may be disclosed without your permission.

COMPENSATION FOR INJURY

In the event of any physical injury from the research, only immediate and essential medical treatment is available. First Aid/CPR and a referral to a medical emergency room will be provided. In the event of any emergency incidence outside the lab as a result of this research, contact your regular medical doctor and inform the study coordinator: Cris Stickley Ph.D., ATC, at 808-956-3798. You should understand that, if you are injured in the course of this research process, you or your medical insurance will be billed for the costs of treating your injuries.

VOLUNTARY PARTICIPATION AND WITHDRAWAL

Your participation in this study is voluntary. You may decide not to participate, or you may leave the study at any time. Your decision will not result in any penalty or loss of benefits to which you are entitled.

Your participation in this study may be stopped at any time by the study doctor or the sponsor without your consent for any of the following reasons:

- it is in your best interest;
- you do not consent to continue in the study after being told of changes in the research that may affect you;
- you become pregnant;
- or for any other reason.

If you leave the study before the planned final visit, you may be asked by the study doctor to have some of the end of study procedures done.

SOURCE OF FUNDING FOR THE STUDY

This research study is sponsored by the University of Hawaii, Manoa.

QUESTIONS

Contact Cris Stickley Ph.D., ATC at 808-956-3798 or Dr. Cass Nakasone at 808-522-4232 for any of the following reasons:

- if you have any questions about this study or your part in it
- if you feel you have had a research-related injury or
- if you have questions, concerns, or complaints about the research

If you have questions about your rights as a research subject or if you have questions, concerns, input, or complaints about the research, you may contact:

Western Institutional Review Board® (WIRB®)
1019 39th Avenue SE Suite 120
Puyallup, Washington 98374-2115
Telephone: 1-800-562-4789 or 360-252-2500
E-mail: Help@wirb.com.

WIRB is a group of people who perform independent review of research.

WIRB will not be able to answer some study-specific questions, such as questions about appointment times. However, you may contact WIRB if the research staff cannot be reached or if you wish to talk to someone other than the research staff.

Do not sign this consent form unless you have had a chance to ask questions and have gotten satisfactory answers. If you agree to be in this study, you will receive a signed and dated copy of this consent form for your records.

CONSENT

I have read this consent form. All my questions about the study and my part in it have been answered. I freely consent to be in this research study.

I authorize the use and disclosure of my health information to the parties listed in the authorization section of this consent for the purposes described above.

By signing this consent form, I have not given up any of my legal rights.

____

Subject Name (printed)

CONSENT SIGNATURE:

____

Signature of Subject Date

____

Signature of Person Conducting Informed Consent Discussion Date

RESEARCH SUBJECT INFORMATION AND CONSENT FORM

TITLE: Biomechanical Analysis of the Oxford® Unicompartmental Knee Implant Design During Level Walking and Stair Negotiation

PROTOCOL NO.: 2016-007

SPONSOR: Cris Stickley, PhD, ATC
Honolulu, Hawaii
United States

INVESTIGATOR: Cass Nakasone, M.D.
888 South King Street
Honolulu, Hawaii 96813
United States

STUDY-RELATED PHONE NUMBER(S): Cass Nakasone, M.D.
808-522-4000

Cris Stickley PhD, ATC
808-956-3798

This consent form may contain words that you do not understand. Please ask the study doctor or the study staff to explain any words or information that you do not clearly understand. You may take home an unsigned copy of this consent form to think about or discuss with family or friends before making your decision.

SUMMARY

You are being asked to be a participant in a research study. The purpose of this consent form is to help you decide if you want to be in the research study. Please read this consent form carefully. To be in a research study you must give your informed consent. "Informed consent" includes:

- Reading this consent form
- Having the study doctor or study staff explain the research study to you
- Asking questions about anything that is not clear, and
- Taking home an unsigned copy of this consent form. This gives you time to think about it and to talk to family or friends before you make your decision.

You should not join this research study until all of your questions are answered.

Things to know before deciding to take part in a research study:

- The main goal of a research study is to learn things to help patients in the future.
- The main goal of regular medical care is to help each patient.
- No one can promise that a research study will help you.
- Taking part in a research study is entirely voluntary. No one can make you take part.
- If you decide to take part, you can change your mind later on and withdraw from the research study.
- The decision to join or not join the research study will not cause you to lose any medical benefits. If you decide not to take part in this study, your doctor will continue to treat you.
- Parts of this study may involve standard medical care. Standard care is the treatment normally given for a certain condition or illness.
- After reading the consent form and having a discussion with the research staff, you should know which parts of the study are experimental (investigational) and which are standard medical care.
- Your medical records may become part of the research record. If that happens, your medical records may be looked at and/or copied by the sponsor of this study and government agencies or other groups associated with the study.

After reading and discussing the information in this consent form you should know:

- Why this research study is being done;
- What will happen during the research;
- Any possible benefits to you;
- The possible risks to you;
- How problems will be treated during the study and after the study is over.

If you take part in this research study, you will be given a copy of this signed and dated consent form.

PURPOSE OF THE STUDY

The purpose of this study is to compare the function of patients with the Oxford partial knee implant design during level walking and stair negotiation tasks.

Approximately 20 people will participate in this study.

PROCEDURES

If you decide to participate in this study, you will be receiving per the physician's protocol the Oxford partial knee implant which is approved by the FDA for the type of surgery you are having and will be used according to their approved indication.

You will be asked to report to the University of Hawaii at Manoa, Kinesiology and Rehabilitation Science Laboratory (Gait Lab) (Sherriff 100) for all testing before and after your knee surgery.

Upon arrival to the Gait Lab, you will be asked to fill out one survey in reference to your current pain and activity level.

When you arrive at the Gait Lab measurements about your body will be taken and you will be asked to perform the following tasks:
(1) walk for 6 meters at a comfortable speed 6-10 times (Gait Analysis),
(2) walking up and down stairs at a comfortable speed 3-4 times, and
(3) push into stationary objects (fixed dynamometer) with your leg for three seconds for two different leg movements (Isometric Strength).
You will also be asked some questions about your daily activities. The entire visit will take approximately 60 minutes.

You will be asked to go to the Gait Lab for your first study visit before your surgery. Each visit to the Gait lab will take approximately 60 minutes. You will be asked to return to the Gait Lab four more times over the next one year to repeat the procedures listed above (please see Table 1 below for visit schedule).

Table 1. Visit Timeline

	Before Surgery	6 Weeks After Surgery	3 Months After Surgery	6 Months After Surgery	1 Year After Surgery
Gait Analysis (test)	X	X	X	X	X
Isometric Strength	X	X	X	X	X
Survey	X	X	X	X	X

RISKS AND DISCOMFORTS

There are risks associated with your knee replacement surgery. These include:
- Blood clots that can, in rare cases, be life threatening
- Complications after a blood transfusion
- Allergic reaction to the medications or materials used
- Injury to arteries in your leg
- Surgery may not reduce your pain and stiffness, possibly requiring more treatment
- Surgery may cause more pain

Due to the level of physical activity involved, there is a risk of injury. You may have pain in your affected joint during testing. You may also have some discomfort, muscle cramping or soreness during or after test sessions. Although we have people to assist you and handrails in place, there is a chance of falling during the test. There is a very remote chance of cardiac arrest and/or death. These risks are comparable to your routine rehabilitation and activities of daily living and will not affect your recovery from the surgery.

You cannot participate in this study if you are pregnant because the information collected during the walking test may not accurately represent your normal walking characteristics. If you are unaware that you are pregnant, participation in this study will result in no more danger to the mother or fetus than normal activities of daily living. However, if you become pregnant or think you might be pregnant during the course of this study, you must inform the researchers, and you will be excluded from study participation.

NEW INFORMATION

You will be told about anything new that might change your decision to be in this study. You may be asked to sign a revised consent form if this occurs.

BENEFITS

You may not receive direct/immediate benefits. However, you will obtain information regarding your walking gait, functional activity capacity, hip muscular strength, and behavioral characteristics. Results of this study may assist physicians, physical therapists, and athletic trainers to ensure the optimal clinical outcomes to maintain the beneficial effects of knee replacement.

PAYMENT FOR PARTICIPATION

You will be given $5 that can be applied towards parking and/or transportation to the University of Hawaii Gait Laboratory each time you come for a visit. The money will be given to you after you arrive to the facility, so it is a reimbursement. If you do not finish the study, you will be paid only for the visits you have completed.

COSTS

There are no additional costs related to the procedures and visits that may result from your participation in this research study.

Any costs associated with parking/transportation over and above the $5 provided will be your responsibility. The fee for parking at the University of Hawaii parking structure is $5 during the week and $6 on the weekends.

ALTERNATIVE TREATMENT

Your alternative is not to participate in this study.

USE AND DISCLOSURE OF YOUR HEALTH INFORMATION:

By signing this form, you are authorizing the use and disclosure of individually identifiable information. Your information will only be used/disclosed as described in this consent form and as permitted by state and federal laws. If you refuse to give permission, you will not be able to be in this research.

This consent covers all information about you that is used or collected for this study. It includes

- Past and present medical records
- Research records
- Records about your study visits.
- Information gathered for this research about:
    - Physical exams
    - Laboratory, x-ray, and other test results
    - Questionnaires
- Records about the implanted medical device.

Your authorization to use your identifiable health information will not expire even if you terminate your participation in this study or you are removed from this study by the study doctor. However, you may revoke your authorization to use your identifiable information at any time by submitting a written notification to the principal investigator, Cass Nakasone, MD at 888 S. King Street, Honolulu, HI 96813. If you decide to revoke (withdraw or "take back") your authorization, your identifiable health information collected or created for this study shall not be used or disclosed by the study doctor after the date of receipt of the written revocation except to the extent that the law allows us to continue using your information. The investigators in this study are not required to destroy or retrieve any of your health information that was created, used, or disclosed for this study prior to receiving your written revocation.

By signing this consent form, you authorize the following parties to use and or disclose your identifiable health information collected or created for this study:

- Cass Nakasone, MD, and his research staff for the purposes of conducting this research study.
- Straub Medical Center and Hawaiʻi Pacific Health
- The University of Hawaiʻi

Your medical records may contain information about AIDS or HIV infection, venereal disease, treatment for alcohol and/or drug abuse, or mental health or psychiatric services. By signing this consent form, you authorize access to this information if it is in the records used by members of the research team.

The individuals named above may disclose your medical records, this consent form, and the information about you created by this study to:

- The sponsor of this study and their designees (if applicable)
- Federal, state, and local agencies having oversight over this research, such as the Office for Human Research Protections in the U.S. Department of Health and Human Services, Food and Drug Administration, the National Institutes of Health, etc.
- The University of Hawaiʻi
- Hawaii Pacific Health (HPH) Officials, the Western Institutional Review Board, and the HPH Office of Compliance for purposes of overseeing the research study and making sure that your ethical rights are being protected.

Some of the persons or groups that receive your study information may not be required to comply with federal privacy regulations, and your information may lose its federal privacy protection and your information may be disclosed without your permission.

COMPENSATION FOR INJURY

In the event of any physical injury from the research, only immediate and essential medical treatment is available. First Aid/CPR and a referral to a medical emergency room will be provided. In the event of any emergency incidence outside the lab as a result of this research, contact your medical doctor and inform the study coordinator: Cris Stickley Ph.D., ATC, at 808-956-3798. You should understand that if you are injured in the course of this research process that you or your medical insurance will be billed for the costs of treating your injuries.

VOLUNTARY PARTICIPATION AND WITHDRAWAL

Your participation in this study is voluntary. You may decide not to participate, or you may leave the study at any time. Your decision will not result in any penalty or loss of benefits to which you are entitled.

Your participation in this study may be stopped at any time by the study doctor or the sponsor without your consent for any of the following reasons:

- it is in your best interest;
- you do not consent to continue in the study after being told of changes in the research that may affect you;
- you become pregnant;
- or for any other reason.

If you leave the study before the planned final visit, you may be asked by the study doctor to have some of the end of study procedures done.

SOURCE OF FUNDING FOR THE STUDY

This research study is sponsored by the University of Hawaiʻi at Manoa.

QUESTIONS

Contact Cris Stickley Ph.D., ATC at 808-956-3798 or Dr. Cass Nakasone at 808-522-4232 for any of the following reasons:

- if you have any questions about this study or your part in it
- if you feel you have had a research-related injury or
- if you have questions, concerns, or complaints about the research

If you have questions about your rights as a research subject or if you have questions, concerns, or complaints about the research, you may contact:

Western Institutional Review Board® (WIRB®)
1019 39th Avenue SE Suite 120
Puyallup, WA 98374-2115
Telephone: 1-800-562-4789 or 360-252-2500
E-mail: Help@wirb.com.

WIRB is a group of people who perform independent review of research.

WIRB will not be able to answer some study-specific questions, such as questions about appointment times. However, you may contact WIRB if the research staff cannot be reached or if you wish to talk to someone other than the research staff.

Do not sign this consent form unless you have had a chance to ask questions and have gotten satisfactory answers.

If you agree to be in this study, you will receive a signed and dated copy of this consent form for your records.

CONSENT

I have read this consent form. All my questions about the study and my part in it have been answered. I freely consent to be in this research study.

I authorize the use and disclosure of my health information to the parties listed in the authorization section of this consent for the purposes described above.

By signing this consent form, I have not given up any of my legal rights.

____
Subject Name (printed)

CONSENT SIGNATURE:

Signature of Subject Date

Signature of Person Conducting Informed Consent Discussion Date

CONSENT

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Subject Name (printed)

CONSENT SIGNATURE:

Signature of Subject Date

Signature of Person Conducting Informed Consent Discussion Date

RESEARCH SUBJECT INFORMATION AND CONSENT FORM

TITLE: Comparison of Open vs. Closed Kinetic Chain Knee Joint Position Sense to Stair Negotiation Biomechanics in a Control Population

PROTOCOL NO.: 2018-00568

PRIMARY INVESTIGATOR:
Cris Stickley, PhD, ATC
1337 Lower Campus Rd
Honolulu, Hawaii 96822
United States

STUDENT INVESTIGATOR:
Derek Beeler, MS, ATC
1337 Lower Campus Rd
Honolulu, Hawaii 96822
United States

SITE(S):
University of Hawaii at Manoa
Biomechanics and Gait Laboratory
Stan Sheriff Center Room 100
Honolulu, Hawaii 96822
United States

STUDY-RELATED PHONE NUMBER(S):
Derek Beeler, MS, ATC
219-241-9822

Cris Stickley PhD, ATC
808-956-3798

This consent form may contain words that you do not understand. Please ask the study doctor or the study staff to explain any words or information that you do not clearly understand. You may take home an unsigned copy of this consent form to think about or discuss with family or friends before making your decision.

SUMMARY

You are being asked to be a participant in a research pilot study. The purpose of this consent form is to help you decide if you want to be in the research study. Please read this consent form carefully. To be in a research study you must give your informed consent. “Informed consent” includes:

- Reading this consent form
- Having the study doctor or study staff explain the research study to you
- Asking questions about anything that is not clear, and
- Taking home an unsigned copy of this consent form. This gives you time to think about it and to talk to family or friends before you make your decision.

You should not join this research study until all of your questions are answered.

Things to know before deciding to take part in a research study:

- The main goal of a <u>research study</u> is to learn things to help patients in the future.
- The main goal of <u>regular medical care</u> is to help each patient.
- No one can promise that a research study will help you.
- Taking part in a research study is entirely voluntary. No one can make you take part.
- If you decide to take part, you can change your mind later on and withdraw from the research study.
- The decision to join or not join the research study will not cause you to lose any medical benefits. If you decide not to take part in this study, your doctor will continue to treat you.
- Parts of this study may involve standard medical care. Standard care is the treatment normally given for a certain condition or illness.
- After reading the consent form and having a discussion with the research staff, you should know which parts of the study are experimental (investigational) and which are standard medical care.
- Your medical records may become part of the research record. If that happens, your medical records may be looked at and/or copied by the sponsor of this study and government agencies or other groups associated with the study.

After reading and discussing the information in this consent form you should know:

- Why this research study is being done;
- What will happen during the research;
- Any possible benefits to you;
- The possible risks to you;
- How problems will be treated during the study and after the study is over.

If you take part in this research study, you will be given a copy of this signed and dated consent form.

PURPOSE OF THE STUDY

The purpose of this study is to compare knee extension and double leg squatting techniques for measuring knee and lower limb joint position sense. Subject stair negotiation (ascent and descent) biomechanics will be assessed to compare any relationship between either of the joint position sense tests and biomechanical variables during stair negotiation.

PROCEDURES

If you decide to participate in this study, you will be asked to report to the University of Hawaii at Manoa, Kinesiology and Rehabilitation Science Laboratory (Biomechanics and Gait Lab) (Stan Sheriff 100) for a one-time data collection procedure.

Upon arrival to the Biomechanics and Gait Lab, you will be asked to fill out one brief survey in reference to your current health. When you arrive at the Biomechanics and Gait Lab, measurements about your height and body weight will be taken. After 31 reflective markers are placed on your legs, hips, and upper body, you will be asked to perform the following tasks, in a randomized order:

(1) A familiarization trial to determine the test angle to be reproduced, followed by ten (10) double leg squat repetitions to the test angle of 30 degrees of knee flexion. Each repetition will be held for 5 seconds prior to returning to the starting position

(2) A familiarization trial for each limb to determine the test angle to be reproduced, followed by ten (10) single leg squat repetitions to the test angle of 30 degrees of knee flexion on each limb (total of 20 single leg squat repetitions). Each repetition will be held for 5 seconds prior to returning to the starting position

(3) A familiarization trial for each limb, followed by ten (10) single leg, seated knee extension repetitions to the test angle of 30 degrees of knee flexion, to be completed on the right and left limbs (total of 20 knee extension repetitions). Each repetition will be held for 5 seconds prior to returning to the starting position

(4)10 consecutive stair ascent and descent trials utilizing 3 steps for each limb. 5 trials will be completed for the right limb, followed by 5 trials to be completed for the left limb

The entire visit will take approximately 60 minutes or less.

RISKS AND DISCOMFORTS

There are minimal risks associated with your participation in this study. These include but are not limited to:

- Soreness during and/or after participation
- Lower leg injury
- Stiffness after participation
- Falling or tripping on stairs

Due to the level of physical activity involved, there is a risk of injury. You may have pain in your legs during testing. You may also have some discomfort, muscle cramping or soreness during or after test sessions. Although we have people to assist you and a chair for balance in place, there is a chance of falling during the test. There is a very remote chance of cardiac arrest and/or death. These risks are comparable to your activities of daily living.

NEW INFORMATION

You will be told about anything new that might change your decision to be in this study. You may be asked to sign a revised consent form if this occurs.

BENEFITS

You may not receive direct/immediate benefits. However, you may obtain information regarding your joint position sense tests, as well as information for the biomechanics of stair negotiation. Results of this study may assist physicians, physical therapists, strength and conditioning specialists, and athletic trainers to ensure the optimal clinical outcomes when considering the relationship between stair negotiation and joint position sense.

PAYMENT FOR PARTICIPATION

There is no compensation provided for your participation in this one-time data collection. No medical insurance is collected. There is no charge to your insurance company for your participation in this study.

COSTS

There are no additional costs related to the procedures and visit. Any costs for transportation to/from the UH Biomechanics and Gait Lab are your responsibility.

USE AND DISCLOSURE OF YOUR HEALTH INFORMATION:

By signing this form, you are authorizing the use and disclosure of individually identifiable information. Your information will only be used/disclosed as described in this consent form and as permitted by state and federal laws. If you refuse to give permission, you will not be able to be in this research.

This consent covers all information about you that is used or collected for this study. It includes

- Research records
- Records about your study visit
- Self-reported medical questionnaire documentation

Your authorization to use your identifiable health information will not expire even if you terminate your participation in this study or you are removed from this study by the study staff. However, you may revoke your authorization to use your identifiable information at any time by submitting a written notification to the principal investigator, Dr. Cris Stickley, University of Hawaii at Manoa, Honolulu, HI 96822. If you decide to revoke (withdraw or "take back") your authorization, your identifiable health information collected or created for this study shall not be used or disclosed by the study staff after the date of receipt of the written revocation except to the extent that the law allows us to continue using your information. The investigators in this study are not required to destroy or retrieve any of your health information that was created, used, or disclosed for this study prior to receiving your written revocation.

By signing this consent form, you authorize the following parties to use and or disclose your identifiable health information collected or created for this study:

- Cris Stickley and his research staff for the purposes of conducting this research study.
- University of Hawaii at Manoa.

The individuals named above may disclose this consent form and the information about you created by this study to:

- The sponsor of this study and their designees (if applicable)
- Federal, state, and local agencies having oversight over this research, such as the Office for Human Research Protections in the U.S. Department of Health and Human Services, Food and Drug Administration, the National Institutes of Health, etc.
- The University of Hawaiʻi for purposes of overseeing the research study and making sure that your ethical rights are being protected.

Some of the persons or groups that receive your study information may not be required to comply with federal privacy regulations, and your information may lose its federal privacy protection and your information may be disclosed without your permission

COMPENSATION FOR INJURY

In the event of any physical injury from the research, only immediate and essential medical treatment is available. First Aid/CPR and a referral to a medical emergency room will be provided. In the event of any emergency incidence outside the lab as a result of this research, contact your medical doctor and inform the study coordinator: Cris Stickley Ph.D., ATC, at 808-956-3798. You should understand that if you are injured in the course of this research process that you or your medical insurance will be billed for the costs of treating your injuries.

VOLUNTARY PARTICIPATION AND WITHDRAWAL

Your participation in this study is voluntary. You may decide not to participate, or you may leave the study at any time. Your decision will not result in any penalty or loss of benefits to which you are entitled.

Your participation in this study may be stopped at any time by the study doctor or the sponsor without your consent for any of the following reasons:

- it is in your best interest;
- you do not consent to continue in the study after being told of changes in the research that may affect you;
- or for any other reason.

If you leave the study before the planned final visit, you may be asked by the study doctor to have some of the end of study procedures done.

SOURCE OF FUNDING FOR THE STUDY

This research study is sponsored by the University of Hawaii, Manoa.

QUESTIONS

Contact Cris Stickley Ph.D., ATC at 808-956-3798 or Derek Beeler MS, ATC at 219-241-9822 for any of the following reasons:

- if you have any questions about this study or your part in it
- if you feel you have had a research-related injury or
- if you have questions, concerns, or complaints about the research

If you have questions about your rights as a research subject or if you have questions, concerns, or complaints about the research, you may contact:

UH Human Studies Program at (808) 956-5007 or uhirb@hawaii.edu to discuss problems, concerns, and questions; obtain information; or offer input with an informed individual who is unaffiliated with the specific research protocol.

Please visit http://go.hawaii.edu/jRd for more information on your rights as a research participant. The Human Studies Program will not be able to answer some study-specific questions, such as questions about appointment times. However, you may contact them if the research staff cannot be reached or if you wish to talk to someone other than the research staff.

Do not sign this consent form unless you have had a chance to ask questions and have gotten satisfactory answers. If you agree to be in this study, you will receive a signed and dated copy of this consent form for your records.

CONSENT

I have read this consent form. All my questions about the study and my part in it have been answered. I freely consent to be in this research study.

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____

Subject Name (printed)

CONSENT SIGNATURE:

____ ________________

Signature of Subject Date

____ ________________

Signature of Person Conducting Informed Consent Discussion Date