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Knee motion variability in patients with knee osteoarthritis: the effect of self-reported instability

Jonathan A. Gustafson, B.S.¹, Megan E. Robinson², G. Kelley Fitzgerald, PT, Ph.D., FAPTA³, Scott Tashman, Ph.D.⁴, and Shawn Farrokhi, PT, Ph.D., DPT⁵

¹Doctoral Student, Department of Bioengineering, University of Pittsburgh, Pittsburgh, PA, USA

²Research Assistant, Department of Physical Therapy, University of Pittsburgh, Pittsburgh, PA, USA

³Professor, Department of Physical Therapy, Director, Physical Therapy Clinical and Translational Research Center, University of Pittsburgh, Pittsburgh, PA, USA

⁴Associate Professor Department of Orthopaedic Surgery, Department of Bioengineering, Director, Biodynamics Laboratory, University of Pittsburgh, PA, USA

⁵Assistant Professor, Departments of Physical Therapy & Bioengineering, Co-director, Human Movement Research Laboratory, University of Pittsburgh, Pittsburgh, PA, USA

Abstract

Background—Knee osteoarthritis has been previously associated with a stereotypical knee-stiffening gait pattern and reduced knee joint motion variability due to increased antagonist muscle co-contractions and smaller utilized arc of motion during gait. However, episodic self-reported instability may be a sign of excessive motion variability for a large subgroup of patients with knee osteoarthritis. The objective of this work was to evaluate the differences in knee joint motion variability during gait in patients with knee osteoarthritis with and without self-reported instability compared to a control group of older adults with asymptomatic knees.

Methods—Forty-three subjects, 8 with knee osteoarthritis but no reports of instability (stable), 11 with knee osteoarthritis and self-reported instability (unstable), and 24 without knee osteoarthritis or instability (control) underwent Dynamic Stereo X-ray analysis during a decline gait task on a treadmill. Knee motion variability was assessed using parametric phase plots during the loading response phase of decline gait.

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Address correspondence to: Shawn Farrokhi, PT, Ph.D. Department of Physical Therapy, University of Pittsburgh, 6035 Forbes Tower, Pittsburgh, PA 15260, USA. Phone: (412) 648-1332, Farrokhi@pitt.edu.

This study was approved by the Health Sciences Institutional Review Board, University of Pittsburgh, Pittsburgh, PA, USA.

Author contributions

All authors were involved in drafting the article or revising it critically for important intellectual content, and all authors approved the final version to be published. Dr. Farrokhi and Mr. Gustafson had full access to all of the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis

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Findings—The stable group demonstrated decreased sagittal-plane motion variability compared to the control group ($p=0.04$), while the unstable group demonstrated increased sagittal-plane motion variability compared to the control ($p=0.003$) and stable groups ($p<0.001$). The unstable group also demonstrated increased anterior-posterior joint contact point motion variability for the medial tibiofemoral compartment compared to the control ($p=0.03$) and stable groups ($p=0.03$).

Interpretation—The finding of decreased knee motion variability in patients with knee osteoarthritis without self-reported instability supports previous research. However, presence of self-reported instability is associated with increased knee motion variability in patients with knee osteoarthritis and warrants further investigation.

Keywords

Instability; Variability; Kinematics; Gait

1. INTRODUCTION

Knee osteoarthritis (OA) is a prevalent degenerative joint disease marked by progressive erosion of the articular cartilage and loss of joint space, often leading to slackening of the knee joint ligaments and increased joint laxity (Lewek et al., 2004; Sharma et al., 1999). It is well documented that patients with knee OA often utilize a stereotypical knee-stiffening gait pattern as an adaptive strategy to overcome joint laxity by enhancing dynamic knee joint stability through increased antagonist muscle co-contractions and reduced knee joint motion (Astefian et al., 2008; Childs et al., 2004; Lewek et al., 2004; Zeni and Higginson, 2009). Recently, significant reductions in knee motion variability, most likely as a result of adopting a knee-stiffening gait strategy and increased muscle co-contractions were reported for patients with knee OA who were waitlisted for joint replacement surgery (Yakhdani et al., 2010) and those with more severe structural disease (Kiss, 2011). Similar reductions in knee joint motion variability due to increased muscle co-contractions have also been previously reported for patients with knee joint laxity due to anterior cruciate ligament (ACL) deficiency who are at high risk for developing knee OA (Georgoulis et al., 2006; Moraiti et al., 2007). Although potentially effective in reducing the effects of laxity, a knee-stiffening gait strategy and the associated decrease in knee joint motion variability could facilitate progression of knee OA due to repetitive loading of the same areas of joint contact which can cause microtrauma to finite regions of the articular cartilage (Messier et al., 1992; Radin et al., 1991).

Self-reported knee instability, described as buckling, shifting, or giving way of the knee joint is a common complaint in as high as 60%–80% of patients with knee OA (Fitzgerald et al., 2004; Knoop et al., 2012; Ramsey et al., 2007; van der Esch et al., 2012). While it has been evidenced that self-reports of instability may not be related to passive joint laxity in patients with knee OA (Schmitt et al., 2008; Schmitt and Rudolph, 2007; van der Esch et al., 2012), it was recently reported that presence of self-reported instability may be a sign of inadequate dynamic knee joint stability and diminished knee joint control in this patient population (Farrokhi et al., 2014). It has also been suggested that presence of knee instability is associated with an increased fear of falling in patients with knee OA (Nguyen et al., 2014). To this end, higher risk of falls in older adults has been previously associated with

increased gait variability due potentially to deficits in knee extension strength and lower limb proprioception (Callisaya et al., 2010; Callisaya et al., 2011; Hausdorff et al., 2001; Lord et al., 2003), which have also been regularly hypothesized as important causal factors for self-reported knee instability in patients with knee OA (Collins et al., 2011; Fitzgerald et al., 2004; Knoop et al., 2012). Deficits in muscular strength and proprioceptive sensory input, which are thought to alter the neural control of the muscles around the knee joint, have also been previously associated with greater knee flexion/extension motion variability after ACL reconstruction (Moraiti et al., 2009).

Increased variability of the of joint contact point motion at the level of joint surfaces may also be a sign of mechanical instability and increased risk of cartilage damage due to greater joint contact shear forces. Additionally, increased joint contact point motion variability could potentially lead to deleterious loading of the regions of the articular cartilage unaccustomed to normal loading (Andriacchi et al., 2004) in this subgroup of patients with knee OA and self-reports of instability. Although current evidence suggests that self-reported instability in patients with knee OA may be a sign of inadequate knee joint control and excessive knee motion variability, formal evaluation of knee joint motion variability has not been performed in this patient population.

The main objective of the current study was to compare variability of knee joint kinematics and contact point motion during the loading response phase of decline gait in three groups of older adults: 1) patients with knee OA without self-reports of instability (OA stable); 2) patients with knee OA with self-reported instability (OA unstable); and 3) healthy subjects without knee OA or reports of instability (control). We hypothesized that the OA stable group would have reduced knee motion variability compared to the control and the OA unstable groups. Additionally, we hypothesized that compared to the control and the OA stable groups, the OA unstable group would demonstrate increased knee motion variability.

2. METHODS

2.1. Participants

This research was conducted in compliance with the Declaration of Helsinki, and informed consent approved by the University of Pittsburgh's institutional review board was obtained from all subjects. A total of 43 older adults participated in this study. Subjects were divided into one of following three groups: 1) an "OA stable" group consisting of volunteers with knee OA but no reports of knee instability; 2) an "OA unstable" group consisting of volunteers with knee OA and concurrent self-reports of episodic knee instability; and 3) a "control" group consisting of volunteers with neither knee OA nor self-reported knee instability. Participants were included in the OA stable and OA unstable groups if they: 1) met the American College of Rheumatology classification criteria for knee OA (Altman et al., 1986), and 2) demonstrated primary medial compartment radiographic knee OA of at least grade II or higher according to the Kellgren and Lawrence (KL) radiographic severity rating scale (Kellgren and Lawrence, 1957). Subjects rated the severity of their knee instability on a six-point numeric scale (Table 1) adapted from the Knee Outcome Survey-Activities of Daily Living Scale (Irrgang et al., 1998). The test-retest reliability of this tool has previously been determined to be adequate (intraclass correlation coefficient = 0.76) in a

population including individuals with knee OA ((Fitzgerald et al., 2004). Participants in the OA unstable group had to have a self-reported knee instability rating of 3 on the knee stability scale indicating that the patient perceived the symptom of instability to be affecting their ability to perform activities of daily living (Fitzgerald et al., 2004). To be included in the control group, participants had to have no history of knee pain and a KL radiographic severity rating of grade 1.

Participants were excluded, regardless of their group designation, if they had a past history of traumatic knee injury, lower extremity total joint arthroplasty, cardiovascular disease, uncontrolled hypertension, or neurological disorders that affect lower extremity function. To ensure safe participation in the study, individual patients were also excluded if they required use of an assistive device for ambulation, reported a history of two or more falls within the previous year, or if they reported lack of confidence in ambulating a distance of 30.5 m (100 ft.) without an assistive device or need of a rest period.

2.2. Experimental Setup

Dynamic Stereo X-ray (DSX) methods were used to quantify tibiofemoral joint kinematics and contact mechanics from biplane radiographic images acquired during the loading response phase of decline gait. Loading response was chosen as the critical time period that is associated with high demands on the knee and reports of dynamic alignment change in the presence of knee OA (Astefhen and Deluzio, 2005; Schipplein and Andriacchi, 1991). Participants were placed on an instrumented treadmill (Berotec Corp., Columbus, OH) within the biplane X-ray system so that the knee of interest could be seen throughout the loading response phase. For participants with knee OA, the knee in which patients reported symptoms or episodes of instability was designated as the knee of interest. In cases where both knees experienced symptoms or episodes of instability, the more problematic knee was chosen as the knee of interest. For control participants, the knee from the dominant lower limb was designated as the knee of interest.

The biplane X-ray system contained two X-ray gantries that were configured with their beam paths intersecting at 60° in a plane parallel to the floor. Each gantry contained a 100 kW pulsed X-ray generator (CPX 3100CV; EMD Technologies, Quebec, Canada), a 40 cm image intensifier (Thales, Neuilly-sur-Seine, France), and a high-speed 4 megapixel digital video camera (Phantom v10, Vision Research, Wayne, New Jersey, USA). The X-ray generators were customized to provide short-duration pulses at high repetition rates. For the current study, radiographs were generated with a 1ms pulse width at 100 Hz, with a maximum radiographic protocol of 90 kVp/200 mA and a 100 ms total x-ray exposure per trial.

Participants' knees were imaged during a moderately declined walking condition (7% grade, 0.75 m/s) due to the frequent patient reports of knee instability during this task. To this end, previous reports indicate that decline walking is more demanding on the knee joint as it leads to significant increases in knee flexion angle, vertical ground reaction force and knee joint moments compared to level walking (Kuster et al., 1995; Lay et al., 2006; McIntosh et al., 2006; Redfern, 1997). Data for three separate steps were collected and averaged for statistical analysis. For each step, subjects walked on the treadmill for at least 1 minute to

reach a steady state. The X-ray system was then manually triggered to record a time period starting just before the instance of initial heel contact to at least 200 ms thereafter. Ground reaction forces were then used to determine the loading response phase of each step, which was defined as the first 20% of the stance phase of gait for each subject (Ho et al., 2012; Neville et al., 2010). For safety, participants were attached to an overhead harness system during all treadmill walking trials.

2.3. Quantification of Knee Joint Kinematics

All participants also underwent computed tomography (CT) imaging of their knee using a GE LightSpeed CT Scanner (LightSpeed Pro 16, GE Medical Systems, Fairfield, Connecticut, USA). The CT field of view was approximately 28×28 cm, slice thickness ranged from 0.6 to 1.25 mm, and in-plane resolution was approximately 0.55 mm per pixel. The CT images were reconstructed to create 3D bone models of the distal femur and proximal tibia (Tashman and Anderst, 2003). The tibia and femur were manually segmented and feature-based interpolation was performed to create 3D bone models using MIMICS (Materialise Inc, Ann Arbor, Michigan, USA). A model-based tracking algorithm was then employed to determine 3D joint motion by matching the radiographic images with projections through the 3D volumetric bone models (Anderst et al., 2009). This experimental approach has shown to have excellent accuracy in terms of measurement bias of better than 1.0 degree for rotations and 0.7mm for translations and measurement precision of better than 0.9 degrees for rotations and 0.7mm for translations (Anderst et al., 2009).

Subject-specific local anatomical coordinate systems were created for both the femur and tibia as previously described (Tashman and Anderst, 2003). The origin of the femoral coordinate system was defined as the point halfway between the center of the medial and lateral femoral condyles. Similarly, the origin of the tibial coordinate system was defined to be the point located halfway between the most medial and lateral aspects of the tibial plateau. Rotations of the tibia relative to the femur were defined with respect to the bone-fixed coordinate systems, calculated using body-fixed axes in the order of flexion/extension, adduction/abduction, and internal/external rotation (Tashman and Anderst, 2003). Neutral rotations (zero values) were defined as the position where the tibia and femoral coordinate systems were aligned. The location of the joint contact point was estimated using the distance-weighted centroid of the region of closest proximity between the bony surfaces in both the medial and lateral tibiofemoral joint compartments (Anderst et al., 2005). Joint contact points for each frame were described for the tibia using a Cartesian coordinate system aligned with the tibial plateau.

2.4. Quantification of Knee Joint Variability Index

Knee joint motion variability was calculated using a previously described methodology (Lewek et al., 2006) from the phase plots of knee joint rotations or translations versus angular or linear velocities of the joint during the loading response phase of decline gait (Figure 1). Phase plots were used to determine knee motion variability by considering joint angular or linear velocity as a function of joint angle or linear contact point position, thus allowing us to detect changes in variability due to alterations in either joint angle/position or angular/linear velocity. Phase plots also provide a more detailed estimating of motion

variability across multiple time points during the movement cycle as compared to more traditional variability analyses of isolated discrete time points.

Knee joint rotations or translations were normalized according to the following equation:

$$\theta_i^{norm} = \frac{2(\theta_i - \min(\theta_i))}{\max(\theta_i) - \min(\theta_i)} - 1 \quad (1)$$

where θ represents the joint angle or position, and i is each data point during the loading response phase of gait. Similarly, knee joint angular or linear velocities were normalized as:

$$\omega_i^{norm} = \frac{\omega_i}{\max(|\omega_i|)} \quad (2)$$

where ω is the first derivative of the joint's angular or linear position at each data point i during the loading response phase of gait. Phase angles (ϕ) were calculated as the angle between the positive horizontal axis (joint rotation/translation) and the line connecting the origin of each graph to the data point of interest for each successive data point during the loading response phase of gait. A knee joint "variability index" was then defined as the average of the standard deviations between phase angles for identical time points during the loading response phase across the three individual steps for each subject. The variability of angular knee motion was calculated in the sagittal, frontal and transverse planes. The variability of linear joint contact mechanics was calculated for anterior-posterior (AP) and medial-lateral (ML) motions of the medial and lateral tibiofemoral compartments for each subject.

2.5. Statistical Analysis

Analysis of variance (ANOVA) and chi-square or Fisher's exact tests were used to determine group differences in demographics and radiographic knee OA severity. Differences in the variability index of joint kinematics and tibiofemoral contact motion between the three groups was evaluated using Analysis of Covariance (ANCOVA), adjusting for group differences in body mass index (BMI) and radiographic severity of knee OA. All ANCOVA tests were performed after a square root transformation was implemented to improve normality of data distributions tested using the Shapiro-Wilk test. Post-hoc analyses consisting of paired t-tests with Tukey corrections for multiple comparisons were carried out when a significant ANCOVA test was identified. All statistical analyses were performed using STATA (version 11.0, STATA Corp. LP College Station, TX, USA).

3. RESULTS

3.1. Demographics

Compared to the control, the OA unstable group had higher body weight (mean difference 18.3 Kg; $P=0.005$) and BMI (mean difference 6.8 Kg/m²; $P<0.01$; Table 2). Radiographic disease severity was also significantly different between groups ($P<0.01$) with median and interquartile (IQL) ranges of KL scores for each group as follows: control = 0 (0,0); OA stable = 3 (3,3), and OA unstable = 4 (3,4).

3.2. Variability of Knee Kinematics and Contact Mechanics

Variability index in the sagittal plane was significantly lower in the OA stable group compared to the control group ($P=0.04$; Table 3). Additionally, variability index in the sagittal plane during the loading response phase of decline gait was significantly higher in the OA unstable group compared to the control ($P=0.003$) and the OA stable groups ($P<0.001$; Table 3). Greater AP contact point motion variability index was also observed for the medial tibiofemoral compartment in the OA unstable group compared to the control ($P=0.03$) and the OA stable groups ($P=0.03$; Table 4).

4. DISCUSSION

The findings from the current study support our initial hypothesis that the OA stable group would demonstrate reduced knee joint motion variability. More specifically, the OA stable group demonstrated a 57–93% decrease in their flexion motion variability compared to the control and the OA unstable groups. This finding is consistent with previous reports of decreased sagittal-plane knee motion variability in patients with knee OA (Kiss, 2011; Yakhani et al., 2010). One potential mechanism underlying the observed reductions in the sagittal plane motion variability in our OA stable group is the increased antagonist muscle co-contractions and a knee-stiffening gait strategy previously reported in the literature for patients with knee OA (Childs et al., 2004; Lewek et al., 2004; Schmitt and Rudolph, 2007). To this end, repetitions of loading patterns through a narrow range of knee joint motion variability can expose the same finite regions of the articular cartilage to excessive loading, which could in turn lead to cartilage lesions and deterioration of joint space (Messier et al., 1992; Radin et al., 1991).

Our group has previously published detailed kinematics measurements for the participants included in the current study (Farrokhi et al., 2014). In general, the knee OA stable group moved through nearly 4 degrees (i.e. 35%) less knee flexion excursion during the loading response phase of decline gait compared to the control group, which is consistent with previous reports of reduced knee flexion excursion during level gait in patients with knee OA compared to OA-free control subjects (Childs et al., 2004; Schmitt and Rudolph, 2007). However, the differences in knee flexion excursion for the current cohort did not reach statistical significance most likely due to the study's lack of adequate statistical power. Nevertheless, such reductions in the utilized knee flexion range of motion could theoretically reduce knee motion variability as the knee moves through a smaller arc of motion. As such, the observed finding of decreased knee flexion motion variability in the current study provides further evidence for rigidity of knee joint motion in patients with knee OA and demonstrates the utility of variability analysis in gaining additional insight into the pathomechanics of the arthritic knee joint despite the lack of statistically significant group differences in traditional kinematics measurements. Therefore, the information gained from variability analysis provides insight that goes beyond that of discrete time-point analyses which are customary in many biomechanical studies of knee OA.

Our findings also support the hypothesis that the OA unstable group would demonstrate increased variability in their knee joint motion compared to the OA stable and the control groups. The observed increase in knee joint motion variability signifies a compromised knee

joint in terms of mechanical stability, potentially altering the integrity of the soft tissues (e.g. cartilage), which would also be detrimental to the long-term health of the knee joint. More specifically, the OA unstable group walked with a 2–3 fold increase in their sagittal-plane knee motion variability compared to the control and the OA stable groups. Additionally, the OA unstable group also demonstrated a 71–94% increase in their AP joint contact motion variability for the medial tibiofemoral compartment compared to their control and OA stable counterparts.

Considering that the AP motion of the knee joint contact position is somewhat dependent on the sagittal plane motion of the knee joint, it is plausible that the greater knee flexion motion variability observed in the OA unstable group may also be responsible for the increased variability of the medial compartment AP contact motion. To this end, a post-hoc analysis of the association between variability of knee flexion motion and the AP contact point motion of the medial compartment revealed a strong level of correlation between the two factors ($r = 0.819$, $p = 0.004$) in our OA unstable group. Regardless of the underlying mechanism, the increased variability of joint contact motion in the AP direction during a high-impact portion of the gait cycle indicates a mechanically unstable joint that could lead to undesired loading of joint areas unaccustomed to regular loading and higher damaging shear stresses at the joint surfaces (Andriacchi et al., 2004). Furthermore, the medial compartment localization of increased contact point motion variability combined with the previously reported increased medial compartment contact point excursions and velocities (Farrokhi et al., 2014) may place the medial tibiofemoral joint in this patient population at high risk for OA progression.

Decreased sagittal plane knee motion variability has been previously linked with significant reductions in fall risk in patients with knee OA (Yakhdani et al., 2010). To this end, presence of knee instability has been associated with an increased fear of falling that can lead to severe activity limitations and poor physical function in patients with knee OA (Nguyen et al., 2014). Therefore finding ways to address instability and the associated increase in motion variability in patients with knee OA has important clinical value. Strength and neuromuscular training interventions have shown to be effective in reducing self-reported knee instability in patients with knee OA (Fitzgerald et al., 2011; Knoop et al., 2013) as well as decreasing fall risk and improving gait stability in older adults (Faber et al., 2006; Krebs et al., 1998; Liu-Ambrose et al., 2004). Bracing the knee joint has also shown to improve functional knee stability along with pain relief and functional improvements in patients with knee OA and self-reports of instability as a result of the brace mechanically stabilizing the knee joint (Ramsey et al., 2007). Weight loss and the associated decrease in knee demands may also be effective in reducing episodes of instability, given the significantly higher BMI in our OA unstable group compared to the OA stable and control groups and the previous reports of increased prevalence of knee buckling with greater BMI in patients with knee OA (Felson et al., 2007). Although it stands to reason that knee motion variability should also be modified by these interventions, this hypothesis has not been formally tested and should be evaluated in future clinical studies. In doing so, variability could be utilized as an additional outcome measure of knee joint function to provide objective evidence that various rehabilitation programs for treatment of knee instability in patients with knee OA can lead to appropriate mechanical effects.

The findings of this study should be interpreted in light of its limitations. First, we fully acknowledge that the small sample size of the current study limits our ability to formulate definitive conclusions. However, the small effect sizes along with low power of our non-significant findings (tables 3 & 4) suggest that a larger sample size may still not have allowed for identification of additional group differences. Another potential limitation of this study is that subjects walked on a motorized treadmill which may have artificially reduce the natural variability of knee motion compared to over ground walking. However, given the comparative nature of our study, any systematic errors introduced by treadmill walking would be similar between groups, and in turn, would allow our observed group differences to be preserved.

5. CONCLUSION

Overall, the findings of this study provide evidence of altered knee joint motion variability in patients with knee OA. Whereas patients with knee OA who did not have episodes of instability experienced decreased motion variability, those with episodic knee instability walked with increased knee joint motion variability compared to their control counterparts. Given that both decreased and increased knee motion variability could place the joint at risk for greater symptoms and further damage over time, interventions to modify knee joint variability may be warranted. Additionally, considering variability analysis of kinematic data sets in order to gain additional insight into the pathomechanics of knee OA and to evaluate future clinical studies of patients with knee OA is recommended.

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Highlights

- Knee joint motion variability was studied in patients with knee osteoarthritis.
- Comparisons were made with a group of older adults without knee osteoarthritis.
- Patients without self-reported instability had smaller flexion motion variability.
- Self-reported instability was associated with higher flexion motion variability.
- Self-reported instability was associated with more variable joint contact motion.

Figure 1.

A typical representation of the phase angle (ϕ) created from the phase plot of normalized spatial angle (θ) and normalized angular velocity (ω) during the loading response phase of decline gait.

Table 1

Self-reported knee instability rating scale

To what degree does giving way, buckling or shifting of the knee affect your level of daily activity?
0 = The symptom prevents me from all daily activity
1 = The symptom affects my activity severely
2 = The symptom affects my activity moderately
3 = The symptom affects my activity slightly
4 = I have the symptom but it does not affect my activity
5 = I do not have giving way, buckling, or shifting of the knee

Table 2

Comparisons of patient characteristics.

	Control (n = 24)	OA stable (n = 8)	OA unstable (n = 11)	p-value
Age (years)	69.6 (7.6)	69.4 (8.4)	69.9 (8.2)	0.99
Female (%)	13 (54.2)	7 (87.5)	6 (54.6)	0.26
Height (cm)	174.2 (12.1)	171.5 (14.6)	172.4 (10.4)	0.83
Weight (kg)	75.2 (15.7)	80.7 (12.2)	93.4 (15.9)*	< 0.01
BMI (kg/m²)	24.7 (3.8)	27.7 (5.3)	31.5 (4.8)*	< 0.01
Radiographic Severity				
Grade 0	22 (91.7%)	0*	0*	
Grade I	2 (8.3%)	0*	0*	
Grade II	0	1 (12.5)*	2 (18.2)*	< 0.01
Grade III	0	5 (62.5)*	3 (27.3)*	
Grade IV	0	2 (25.0)*	6 (54.5)*	

* Statistically significant differences compared to the “control” group

† Statistically significant differences compared to the “unstable” group

Table 3

Variability index for knee joint kinematics during the loading response phase of decline gait. The effect size, partial eta², is interpreted in the same manner as an R² statistic.

	Control (n=24)	Stable (n=8)	Unstable (n=11)	Power (1-β)	Effect Size (Partial Eta ²)
Sagittal Plane	17.1 (10.9)	7.5 (4.1) ^{†*}	33.9 (19.9) [*]	0.99	0.38
Frontal Plane	37.1 (20.2)	23.2 (17.1)	35.2 (17.6)	0.45	0.10
Transverse Plane	26.0 (15.2)	36.4 (19.1)	33.3 (18.1)	0.15	0.03

^{*} Statistically significant differences compared to the "control" group

[†] Statistically significant differences compared to the "unstable" group

Table 4

Anterior-posterior (AP) and medial-lateral (ML) variability index for medial and lateral tibiofemoral compartment joint contact point translations during the loading response phase of decline gait. The effect size, partial eta², is interpreted in the same manner as an R² statistic.

	Control (n=24)	Stable (n=8)	Unstable (n=11)	Power (1-β)	Effect Size (Partial Eta ²)
Medial Tibia AP Translation	28.3 (16.7) [†]	24.9 (19.1) [†]	48.3 (19.4)	0.81	0.20
Medial Tibia ML Translation	43.5 (17.8)	33.9 (13.5)	41.2 (16.9)	0.12	0.02
Lateral Tibia AP Translation	25.8 (18.7)	21.6 (15.9)	36.2 (13.8)	0.19	0.04
Lateral Tibia ML Translation	34.6 (17.4)	33.22 (12.1)	43.1 (16.7)	0.15	0.03

[†] Statistically significant differences compared to the "unstable" group

* Statistically significant differences compared to the "control" group