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Peak Knee Flexion Angles During Stair Descent in TKA Patients

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ABSTRACT

Reduced peak knee flexion during stair descent (PKSD) is demonstrated in subjects with total knee arthroplasty (TKA), but the underlying factors are not well studied. 3D gait patterns during stair descent, peak passive knee flexion (PPKF), quadriceps strength, pain, proprioception, demographics, and anthropometrics were assessed in 23 unilateral TKA-subjects ~19 months post-operatively, and in 23 controls. PKSD, PPKF and quadriceps strength were reduced in the TKA-side, but also in the contralateral side. A multiple regression analysis identified PPKF as the only predictor (57%) to explain the relationship with PKSD. PPKF was, however sufficient for normal PKSD. Deficits in quadriceps strength in TKA-group suggest that strength is also contributing to smaller PKSD. Increased hip adduction at PKSD may indicate both compensatory strategy and reduced hip strength.

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Studies show that total knee arthroplasty (TKA) gives rise to various movement abnormalities, such as gait asymmetries including reduced knee flexion [1]. Functional performance following knee arthroplasty may be assessed in stair gait [2]. Descending stairs requires more knee flexion than ascending stairs [3], and there is evidence of reduced knee flexion in stair descent after TKA [4,5]. Although it is concluded that flexion in the prosthetic knee is reduced during stair descent [2,4–6], the underlying mechanisms are uncertain. Although knee flexion is restricted due to the mechanics of the prosthesis [7], stair studies show that the range of motion (ROM) leaves a margin of 6°–16°, theoretically permitting sufficient knee flexion for normal stair descent [4–6]. There are, however also other qualities with TKA that may be important to consider, such as reduced or increased sagittal laxity in the prosthetic knee joint [8–10]. In posterior cruciate retaining knee prostheses a paradoxical forward slide instead of a normal rollback of the tibio-femoral contact point occurs with increasing flexion. This suggests that TKA related factors other than reduced ROM, may be responsible for reduced knee flexion in stair descent [7]. Also factors outside joint mechanics may be considered, such as weakness of the knee and hip muscles [11–14], fear of movement [15], reduced proprioception [16], leg length discrepancy [17],

obesity [18–20], pain [7,21,22], time since TKA surgery [13,23], age [24], and habitual gait patterns [1].

Studies on level walking propose that asymmetrical gait after TKA-surgery is retained from the pre-surgery gait and characterized by a specific walking pattern, presumably for unloading the affected knee [1]. Asymmetrical gait patterns are shown to be retained up to 18 months after surgery in spite of little or no pain [21]. Similarly, subjects with osteoarthritis have demonstrated decreased knee flexion during stair descent compared to knee-healthy subjects [25]. Asymmetrical gait patterns after a TKA may predispose the individual to contralateral joint degeneration [1], and the risk of the contralateral knee to progress to TKA due to osteoarthritis has been estimated to be 37.2% within 10 years [26]. As the maximum knee flexion moments are 4 times higher during stair descent than in level walking [27], the underlying mechanisms for asymmetrical stair descent are important to study.

With the evidence of reduced knee flexion after TKA as a departure point, the purpose of this study was to investigate peak knee flexion during stair descent (PKSD) in the TKA-side compared to the contralateral side, and compared to age matched controls without knee problems. In addition, hip joint kinematics was registered to describe compensatory strategies during stair descent. If PKSD is reduced in the TKA knee, factors that may contribute to this reduction would be examined; peak passive knee flexion (PPKF), isokinetic quadriceps strength, anterior knee laxity (AKL), fear of movement, joint position sense (JPS), leg length, current pain, time since TKA, body mass index (BMI), and age.

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Materials and Methods

Participants

This cross-sectional study took place in the period of October 2010 to December 2010. It was conducted according to the guidelines of the Helsinki declaration, and informed consent with detailed information about the study was obtained from all participants. Ethical approval was granted by the Regional Committees for Medical and Health Research Ethics (REC). Via the orthopedic department 139 enquiries were sent to subjects who had surgery \geq one year to $<$ three years ago with unilateral TKA with a cemented Duracon prosthesis from Stryker Orthopedics (Howmedica, Rutherford, NJ); a posterior cruciate ligament-retaining prosthesis with fixed-bearing platform and without a patella component. The timeframe was selected to avoid the post-surgical early symptoms such as acute pain and swelling, and assure resolution of post-surgical symptoms that may interfere with gait [28]. Fifty-two TKA patients volunteered to participate and 23 of them filled the criteria and were included in the study. Twenty-three age and gender matched control subjects without knee problems were recruited from the university and hospital staff. For all participants, inclusion criteria were $<$ 65 years of age to exclude age-related physical limitations, BMI $<$ 35, no diagnosed neurological or orthopedic conditions (other than TKA). The characteristics of the participants are presented in Table 1.

Laboratory Procedure

The stair descent was assessed with a custom-made free standing staircase module without handrail, three steps up and three steps down, dimensioned according to normal stair standards; step 18 cm and tread 26 cm. The subjects were asked to descend the stairs in a step-over-step pattern in a self-selected, normal and comfortable pace with a total of six trials. All subjects performed the first three trials with the right foot on the first step and the three last trials with the left foot on the first step. The PPKF measurements were performed with the subjects resting relaxed in supine position on a therapy bench while the therapist moved each knee toward flexion with the foot in contact with the support surface, until a tight end feel. JPS was assessed with the subjects blindfolded and seated at the edge of the bench with feet hanging freely. The therapist actively moved each leg to a random knee angle, held for 3 s and then returned back to the starting position. The subject was then asked to reposition the leg into the same knee angle.

Whole body kinematics to assess PKSD, ipsilateral hip adduction at PKSD, PPKF, and JPS was recorded at 100 Hz with an eight-camera system (Oqus, Qualisys, Sweden) and compiled into three dimensional animations (Qualisys Track Manager, 2.6 682, Qualisys, Sweden). The camera system was calibrated prior to each session and the global coordinate system was defined as; medio-lateral X, antero-posterior Y, and vertical Z. System accuracy was measured with an average residual of \approx 0.5 mm. Infrared light emitted from the cameras was reflected from 34 spherical 19 mm reflex markers attached to defined anatomical landmarks denoting proximal and distal ends of body segments. Additional 4-marker clusters were

Table 1
Mean (\pm SD) Subject Demographics.

Variable	TKA-Group (N = 23)	Control Group (N = 23)	P
Male/female	11/12	10/13	.767
Age (years)	57.6 \pm 5.8	54.7 \pm 7.4	.151
Body weight (kg)	88.0 \pm 15.1	74.3 \pm 15.5	.004
Height (cm)	172 \pm 0.1	173 \pm 0.1	.556
BMI (kg/m ²)	29.9 \pm 5.0	24.5 \pm 3.1	.000

BMI = body mass index.

placed on thighs and shanks. For the kinematic data analysis, a model based on 50 markers was built (Visual 3D Basic/RT, 4.00.20, C-Motion Maryland, USA). Subjects were barefoot and wore tight clothing to ensure that the reflective markers were firmly secured to anatomical landmarks.

Kinematic 3D analyses were performed to identify PKSD (x-axis) and adduction in the ipsilateral hip (y-axis) at PKSD. The maximum flexion angle (x-axis) was obtained for PPKF. For JPS, the x-axis of both the assisted and actively repositioned knee angle was obtained to determine the angular error for the reposition accuracy. Leg length measurements were performed in supine with a regular tape measure from the center of the superior anterior iliac spine to the top of the lateral malleolus. Maximal quadriceps strength was measured in an isokinetic dynamometer (Biodex) following a 10 min warm-up period with low intensity on a stationary bike. Three sub-maximal voluntary isokinetic contractions were performed for familiarization and to potentiate the muscle. Quadriceps peak torque (Nm) was obtained by concentric isokinetic extensions performed in a standardized range of motion ranging from 0° to 90° at 60°/s for a total of 5 repetitions for each knee.

A Norwegian adjusted version of TSK was used to quantify fear of re-injury due to movement and physical activity [29]. Each statement was scored on a scale from 1 to 4; strongly disagree to strongly agree. The total score ranges from 13 to 52, a higher score indicating a higher degree of kinesiophobia.

Anterior knee laxity (AKL) was measured with KT1000 (MED-metric Knee Ligaments Arthrometer) in accordance with manufacturer instructions [30]; muscle relaxation was assured, and knee flexion angle 30°, limb orientation, and arthrometer placement on the leg with respect to the joint line and rotation in relation to the patella were standardized. Consistent application of the patella pad pressure and testing reference position was retained and a pulling force of 20 lb was applied. Translation was expressed in millimeters (mm) and calculated as the mean of two consecutive trials with stable values. To assess the TKA-participant's general level of discomfort/pain on the day of the testing, a Numeric Rating Scale (NRS) ranging from 0 to 10 (0=no pain, 10=worst pain) was used.

Statistics

Normality was inferred by Kolmogorov–Smirnov. PKSD and ipsilateral hip adduction at PKSD, PPKF, JPS, AKL, quadriceps peak torque/body weight, and leg-length were compared between legs within subjects with paired sample t-test and between subjects with one way ANOVA. Correlations (Pearson) between PKSD, PPKF, JPS, leg-length and quadriceps peak torque (not normalized) were made across and within groups and knees. Partial correlations controlled for leg-length were made for PKSD related to isokinetic quadriceps strength and to hip adduction at PKSD. Multiple regression analysis was used to explore factors (PPKF, JPS, AKL, quadriceps peak torque, TSK, leg length,, current pain (NRS), post-operative time, body mass index (BMI), and age) potentially explaining reduced PKSD in the TKA-leg. The significance level was set at $P < 0.05$. All statistics were performed using Statistical Package for the Social Sciences (SPSS Inc., Chicago, IL, USA), version 20.

Results

Between and within group differences are found in Table 2. TKA-subjects displayed significantly smaller PKSD in their prosthetic knee compared to the contralateral knee and controls. At PKSD, hip adduction was larger in the TKA-side compared to the contralateral side and controls. Quadriceps peak torque was lower in the TKA-side compared to the contralateral side and controls. No significant differences were found between the prosthetic knee and contralateral knee, or between groups with regard to leg length, JPS, and AKL.

Table 2Mean (\pm SD) Knee Function, Side-to-Side and Group Differences.

Variable	TKA-Group (N = 23)			Control Group (N = 23)		Prosthesis Vs. Both Legs Comb.		Contralateral Vs. Both Legs Comb.	
	Prosthesis	Contralateral	P	Both Legs Combined		P		P	
PKSD x ($^{\circ}$)	88.9 \pm 4.8	96.0 \pm 6.1	.000	101.0 \pm 6.3		.000		.010	
PPKF x ($^{\circ}$)	110.5 \pm 13.0	138.0 \pm 13.9	.000	147.8 \pm 9.7		.000		.008	
Hip y at PKSD (adduction) ($^{\circ}$)	7.3 \pm 6.0	2.1 \pm 6.0	.018	1.3 \pm 4.2		.000		.680	
Leg length (cm)	89.4 \pm 4.2	89.9 \pm 3.8	.224	90.8 \pm 6.6		.396		.557	
Quadriceps peak torque (Nm/bw)	1.24 \pm 0.40	1.67 \pm 0.43	.000	2.07 \pm 0.33		.000		.001	
JPS error ($^{\circ}$)	5.3 \pm 5.0	5.7 \pm 3.5	.649	4.4 \pm 3.1		.502		.212	
Anterior knee laxity (mm)	5.2 \pm 1.6	5.5 \pm 1.9	.535	5.6 \pm 2.5		.530		.935	
Fear of movement (TSK)	27.8 \pm 7.7		n/a	n/a		n/a		n/a	
Pain (NRS)	1.4 \pm 1.8		n/a	n/a		n/a		n/a	
Time since TKA (months)	19.3 \pm 8.2		n/a	n/a		n/a		n/a	

PKSD = peak knee angle descending stairs, PPKF = peak passive knee flexion, JPS = joint position sense (positive indicates overshoot), TSK = Tampa Scale of Kinesiophobia, NRS = numeric rating scale, TKA = total knee arthroplasty, n/a = not applicable.

As PKSD and quadriceps peak torque differed between groups as well as between legs and as leg length may predict PKSD, correlations were performed to analyze the relationships between these factors (Table 3). Smaller PKSD correlated with longer legs across groups and within the control group. Smaller PKSD correlated also with higher quadriceps peak torque within the control group, whereas higher quadriceps peak torque correlated with larger PKSD across groups. When controlled for leg-length, PKSD and quadriceps torque correlated only across groups. None of these factors correlated within the TKA-group. Hip adduction was positively correlated with PKSD across groups when corrected for leg length. There were no within groups correlations between PKSD and hip adduction.

PPKF was significantly reduced in the prosthetic knee compared to the contralateral knee and to the control group (Table 2). PPKF correlated with PKSD across and within groups and legs. Multiple regression analysis within the TKA-leg (Table 4) showed that only PPKF explained (57%) the variance in PKSD. Notably, quadriceps peak torque did not explain PKSD within the TKA-group.

Discussion

The results showed smaller PKSD in the TKA-leg compared to controls, corroborating earlier studies [2,4-6]. Additional within subject comparisons in the present study showed that PKSD was smaller in the TKA-leg compared to the contralateral leg. PKSD in the contralateral leg was also smaller than in controls. The reason for the reduced PKSD in the contralateral knee could be due to influence of the limited PKSD in the prosthetic knee. A further utilization of flexion in the contralateral knee would lead to a very asymmetrical and dysfunctional gait. As expected from earlier studies [13], PPKF was

strongly reduced in the prosthetic knee compared to controls, but also the contralateral knee displayed smaller PPKF than controls. Although the present study did not have access to x-rays of the contralateral knees, it could still be proposed that there exists asymptomatic gonarthrosis limiting knee flexion [31]. As shown in other studies [4-7], TKA-subjects walked with smaller PKSD, although PPKF was sufficient both in the prosthetic knee and in the contralateral knee in order to descend stairs with similar knee flexion as controls. PPKF correlated across groups with PKSD and was the only factor that explained the variance in PKSD in the TKA-leg.

Quadriceps peak torque was considerably lower in the TKA-group compared to controls and, the TKA-leg was weaker than the contralateral leg. In the control group smaller PKSD correlated with longer legs as well as with greater quadriceps torque. This shows that although individuals with longer legs were stronger, they were also able to descend stairs with less knee flexion, as shown when controlled for leg-length the correlation between PKSD and quadriceps peak torque disappeared. In the TKA-group there were no such correlations, indicating that other factors such as reduced PPKF had greater impact on PKSD. Results across groups, however, indicate that quadriceps peak torque may be an important factor for normal stair descent. Studies on elderly have shown that stair negotiation requires knee extensor moments in excess of the maximum isometric muscle strength available and that stair decent exceeds maximal isometric capacity [32]. No comparable studies have explored the relationship between strength and stair gait in TKA-subjects, but findings for flat surface gait show correlations between asymmetric peak knee flexion and quadriceps strength three months after surgery [13,23]. Gait asymmetry seems to disappear over time while strength asymmetry still persists, suggesting that there may be a strength threshold when gait symmetry is no longer affected [13]. Reduced PPKF and

Table 3

Correlations for Variables Across and Within Groups and Sides.

Correlated Variables	Across Groups and Sides df = 79		Control Group Both Legs Combined df = 19		TKA-Group Prosthesis df = 18		TKA-Group Contralateral df = 18	
	R ²	P	R ²	P	R ²	P	R ²	P
Partial correlations, zero order								
PKSD*leg-length	-.219	.049	-.634	.002	.023	.923	.315	.177
PKSD*quadriceps peak torque	.229	.040	-.647	.002	.268	.254	-.107	.655
PKSD*hip adduction at PKSD	-.259	.020	.115	.620	.207	.382	.034	.886
Leg-length*quadriceps peak torque	.176	.116	.752	<.001	.247	.294	.356	.123
Partial correlations, controlled for leg length								
PKSD*quadriceps peak torque	.279	.012	-.405	.085	.281	.259	-.281	.258
PKSD*hip adduction at PKSD	-.281	.011	-.086	.725	.218	.385	.081	.748
Bivariate correlations								
PKSD*PPKF	.762	<.001	.428	.003	.674	.001	.688	<.001

PKSD = peak knee angle descending stairs, PPKF = peak passive knee flexion.

Table 4
Standard Linear Regression Analysis of TKA Side Using PKSD as the Dependent Variable.

Predictor Variable	β	P
Age (y)	.202	.228
Fear of movement (TSK)	.026	.905
PPKF (°)	.794	.001
Quadriceps peak torque (Nm)	.223	.267
Anterior knee laxity (mm)	−.189	.364
Pain (NRS)	.043	.862
Time since TKA (months)	.357	.069
Leg length (cm)	.108	.588
JPS error	−.239	.306
BMI	.005	.978

β is the standardized partial-regression coefficient. Model with all independent variables (adjusted $R^2 = 0.568$), PKSD = peak knee angle descending stairs, PPKF = peak passive knee flexion, TSK = Tampa Scale of Kinesiophobia, NRS = numeric rating scale, TKA = total knee arthroplasty, JPS = joint position sense, BMI = body mass index.

insufficient quadriceps torque may, in spite of no direct correlation between PKSD and the latter in the TKA-group, contribute to explain smaller PKSD in the present study, as reduced PPKF may affect the length–tension relationship of the quadriceps with considerable weakening of muscle force near full flexion. With reduced PPKF follows reduced muscle length, and optimal myofibril cross binding is likely reduced to a smaller part of ROM. This is based on the fact that PPKF is larger in healthy knees. Thus healthy knees will exhibit a higher force generating capacity especially near PKSD due to a more optimal length–tension relationship [33].

Hip adduction was considerably larger in the TKA-side compared to the controls as well as to the contralateral side. Across groups, smaller PKSD was related to greater hip adduction. This suggests that greater hip adduction compensated for reduced PKSD, or possibly that increased hip adduction depends on weakness of the hip muscles in general and abductors in particular as shown by Piva et al [14]. There were however no correlations between PKSD and hip adduction within groups. The effect may have been attenuated, at least in part, due to leg-length, as the correlation becomes stronger when controlled for leg-length. In stair decent, hip adduction may compensate for short legs when normal knee flexion does not suffice.

The TSK-score indicated a medium degree of kinesiophobia, similar to other studies that found TSK [34] to correlate with function assessed by WOMAC. This suggests that medium fear of movement did not impact the physical challenge of stair descent in the TKA-subjects. Similar to TSK-scores, the level of pain reported in the present study was low and with little variance, and thus did not contribute to explain the observed smaller PKSD. Low pain was also in line with findings in other studies [7,22]. Nor did AKL explain reduced PKSD. The TKA-subjects in the present study displayed optimal AKL-range, 5–10 mm, in the prosthetic knee in agreement with the Knee Society Score [9]. Furthermore, there were no differences in leg-length within or between groups that potentially could explain differences in PKSD between or within groups. Leg-length did however, correlate with PKSD in controls but not in the TKA group, suggesting that other factors were more important, such as PPKF. In agreement with earlier studies [35], JPS revealed no significant differences in reposition accuracy between the legs or groups, and did not contribute to explain PKSD. In spite of a significantly heavier TKA group, BMI did not contribute to explain PKSD.

In conclusion, only PPKF, although sufficient to allow uncompensated stair descent, explained smaller PKSD in the TKA-group. Reduced quadriceps peak torque may contribute to PKSD. In spite of not being correlated to PKSD within the TKA-group it was correlated across groups, as PKSD was considerably smaller and quadriceps peak torque lower in the prosthetic knee compared to the contralateral knee and controls. A combination of reduced PPKF and quadriceps

peak torque may affect the length–tension relationship in the muscle with considerable weakness close to full flexion and explain why TKA-subjects did not use the sufficient PPKF to descend stairs without compensation. Increased hip adduction in the TKA-group indicated a compensation for reduced PKSD or reduced hip abductor strength or possibly both.

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