Do Older Adults With Knee Osteoarthritis Place Greater Loads on the Knee During Gait? A Preliminary Study

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ABSTRACT. Messier SP, DeVita P, Cowan RE, Seay J, Young HC, Marsh AP. Do older adults with knee osteoarthritis place greater loads on the knee during gait? A preliminary study. Arch Phys Med Rehabil 2005;86:703-9.

Objective: To compare the gait of older adults with knee osteoarthritis (OA) to an age-, sex-, and weight-matched healthy cohort that would provide preliminary data to examine the hypothesis that adults with knee OA have abnormal knee joint moments and place greater loads on the knee joint during walking compared with healthy adults.

Design: Nonrandomized, descriptive study of healthy and osteoarthritic older adults.

Setting: University clinical research laboratory.

Participants: Ten older adults with tibiofemoral and/or patellofemoral radiographic evidence and pain and disability attributed to knee OA and 10 age-, sex-, and weight-matched healthy adults.

Interventions: Not applicable.

Main Outcome Measures: Three-dimensional gait analysis to calculate knee joint forces and hip, knee, and ankle joint moments; an analysis of covariance adjusted for differences in walking speed between the groups; electromyographic data to verify our interpretation of the knee joint moment data.

Results: The joint forces and moments did not differ statistically between the OA and healthy groups. Nonsignificant differences in the OA group relative to the healthy group included between 7% and 8% greater knee joint compressive (OA group, 3.67±0.24 body weight [BW]; healthy group, 3.40±0.24 BW) and shear (OA group, 0.47±0.04 BW; healthy group, .44±.04 BW) forces, 33% higher knee extension moments (OA group, .32±.07Nm/kg; healthy group, .24±.07Nm/kg), and 24% lower knee internal abduction moments (OA group, .25±.06Nm/kg; healthy group, .33±.06Nm/kg).

Conclusions: Previous research suggests that mechanical overload may be associated with knee OA. Our results do not provide statistical evidence to support this hypothesis. Nevertheless, the trends in the data, along with previous results, suggest the need to investigate further the possible existence of a biomechanical pathway to knee OA.

Key Words: Gait; Joint diseases; Osteoarthritis; Rehabilitation.

RHEUMATIC CONDITIONS are the leading cause of disability in the United States.[1] An estimated 1 in 3 adults, or approximately 70 million Americans, are affected by these conditions.[2] Arthritis affects a disproportionate number of older adults, with 48% of adults aged 65 years and older reporting at least 1 arthritic condition.[3] These rheumatic diseases cost $65 billion in medical care and lost productivity annually and are associated with 744,000 hospitalizations.[3]

Osteoarthritis (OA) is the most common rheumatic disease and is characterized by degradation of the articular cartilage and an increase in subchondral bone density.[4] The knee is the most often affected weight-bearing joint and is second only to the hand as the most common site of this degenerative joint disease.[5] The symptoms include pain, tenderness, stiffness, crepitus, and possible inflammation in the affected joint.[6] A diagnosis of OA is based on these symptoms and radiographic joint changes.[7]

The pain, decreased muscle mass, muscle weakness, changes in proprioception, and compensation that result from knee OA decrease mobility and alter movement mechanics.[8,9] Although many of these changes occur naturally with age, the alterations are exaggerated in people afflicted with OA.[10] Whether these changes in movement mechanics lead to the development of the disease or develop as an adaptive mechanism to symptoms remains an important research question.

Previous research suggests that people with knee OA may differ from nonarthritic subjects in strength, flexibility, gait, and the distribution of loads to the lower extremity.[9] There is also evidence that abnormal external knee adduction moments may contribute to the severity of knee OA.[11] Several recent studies examined sagittal plane joint moments in knee OA patients but had conflicting results. In comparison with healthy subjects, Kaufman et al[12] found that OA subjects had lower internal knee extensor moments, Baliunas et al[13] showed identical knee moments between the groups, and Al-Zahrani and Bakheit[14] noted higher knee moments during gait in knee OA subjects.

Although these kinetic data provide insight into the causes of abnormal gait patterns, to our knowledge only 1 study has compared both joint forces and moments during gait in subjects with and without knee OA. More specifically, Schipplein and Andriacchi[15] compared 15 healthy men and women with 19 patients with knee varus deformity and radiographic evidence of knee OA. The patient group exerted greater muscle forces and higher joint loads than the healthy group when walking at similar speeds. It was not clear, however, how many patients experienced pain and disability due to knee OA. Moreover, the patient group was more overweight than the healthy group, with mean body mass indexes (BMIs) of 29 and 25kg/m^2, respectively. Previous work indicates that obese people have altered gait mechanics compared with people of normal

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weight[16-18]; therefore, not accounting for changes in movement mechanics due to excessive weight may mask osteoarthritic-related differences between healthy and OA groups.

The aim of this preliminary study was to compare the knee joint forces and moments in subjects with and without knee OA, independent of obesity, age, and sex and after adjusting for differences in walking speed. We hypothesized that older, nonobese adults with knee OA have abnormal knee joint moments and place greater loads on the knee joint during walking than age-, sex-, and weight-matched healthy adults.

METHODS

Participants

The osteoarthritic participants for this investigation were 10 older adults (74.1±1.49y) who had volunteered for a large cross-sectional study on disability due to knee OA.[19] They were matched with 10 healthy adults on age (73.0±1.61y), sex (9 women, 1 man), and body mass (OA group, 65.1±2.61kg; healthy group, 58.3±2.74kg). All participants had a BMI of less than 27kg/m^2 to eliminate the effects of obesity on gait.[20] Exclusion criteria for the control group included a history of arthritis or musculoskeletal injury to the lower extremities and any recurrent knee pain.

The symptoms associated with OA included pain in the affected knee plus one of the following: tenderness, mild swelling, or crepitus.[6] Criteria for radiographic evidence of OA included narrowing of the joint space, sclerosis of the subchondral bone, and osteophyte formation.[7] The degree of severity of OA was assessed using the Kellgren-Lawrence scale[21] that rates the level of disease on a scale of 0 to 4, with 0 equal to no symptoms, 1 equal to mild disease, 2 equal to moderate disease, 3 equal to severe disease, and 4 equal to very severe disease.

Procedures

Participants were required to complete 2 visits to the laboratory. During the first visit, participants completed an information worksheet, height and weight were recorded, freely chosen walking speeds were measured, and an informed consent form was explained and signed. The second visit consisted of a 3-dimensional gait analysis. For the purpose of evaluation, the tested leg of each control participant was matched to the affected leg of the respective OA participant.

Freely chosen walking speed was measured by a photoelectric control system[a] with model 65301 IR cells placed 7.6m apart and interfaced with a digital timer. The first 3 trials were used as accommodation trials, and 10 subsequent trials were averaged to produce a representative walking speed.

We obtained 3-dimensional temporospatial and kinematic gait data using a set of 25 passive reflective markers arranged in the Cleveland Clinic full body configuration[b] and a 4-camera motion analysis system[b] set to sample data at 60Hz. Temporal characteristics collected included walking speed, cadence, step width, stride length, support time, nonsupport time, and double-support time.

Kinematic data were collected, tracked, edited, and smoothed using EVA, version 5.0 software,[b] a Sparc[c] workstation, and a Butterworth low-pass filter with a cutoff frequency of 6Hz. The processed data were transferred to a personal computer where they were compiled using OrthoTrak 4.0 clinical gait analysis software[b] to generate lower-extremity kinematic and kinetic data.

A 6-channel force platform[d] was integrated with the motion capture system to allow simultaneous kinetic data collection at 500Hz. We collected 6 successful trials on each participant, and 3 were chosen for subsequent analysis. A successful trial was defined as a trial in which the participant's entire foot was placed on the surface area of the force platform while he/she walked within ±3.5% of his/her freely chosen walking speed. To control for the effects of footwear on gait, all participants wore the same model athletic shoes.

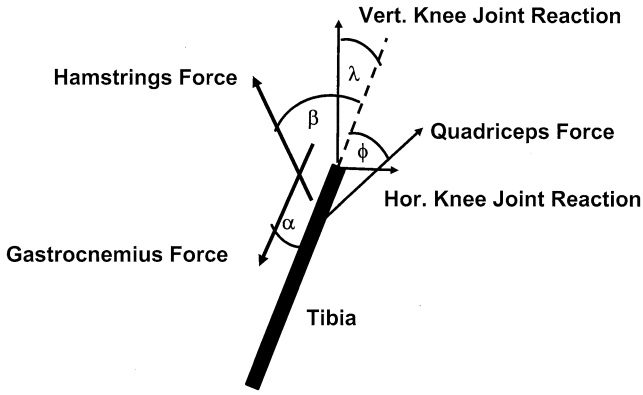

Fig 1. Planar view of knee model. Knee shear and compressive forces were calculated from the muscle and joint reaction forces and lateral knee ligaments (calculated from the frontal plane moments). The knee forces were directed perpendicular and parallel to the line of the tibia. Abbreviations: Hor, horizontal; Vert, vertical.

The smoothed coordinate data, ground reaction, and gravitational and inertial forces served as input to an inverse dynamics model to calculate selected 3-dimensional hip, knee, and ankle internal joint moments. The moments of interest included hip flexion and extension; knee flexion, extension, abduction, internal rotation, and external rotation; and ankle plantarflexion. Additionally, knee joint forces were calculated using a model developed by DeVita and Hortobágyi[22] (fig 1). The joint moments and joint reaction forces were used to determine hamstrings, quadriceps, and gastrocnemius muscle forces. Subsequently, we used this information to determine knee compressive forces and anteroposterior (AP) shear forces.

The moments represent the internal moments produced by the skeletal muscles and other tissues crossing the joints. Positive moments represent net extensor or plantarflexor, internal rotation, and abduction moments.

Surface electromyographic activity was recorded for the vastus lateralis, rectus femoris, and biceps femoris. Muscle bellies were marked with indelible marker while the participant was contracting during manual muscle testing. After appropriate skin preparation, 2 Meditrace[e] disposable electrodes were placed 2.5cm apart and orientated longitudinally over the muscle belly. A common ground lead was placed on the fibular head, and footswitches were placed under the heel and toe of the running shoe to mark foot contact and toe-off. To minimize noise artifact, electromyography leads were secured to the leg using the same method that was used to secure the Cleveland Clinic triads.

The myoelectric signal was telemetered with a BioPac M100 system.[f] Data were collected at 900Hz with BioPac MA-100 software, stored on a personal computer, and later analyzed with custom LabView software.[g] During analysis, data were band-pass filtered (40–400Hz), full-wave rectified, and subsequently smoothed at 5Hz using a low-pass fourth-order Butterworth digital filter. Three trials were averaged to yield representative values of muscle activation for each participant.

Statistical Analyses

Previous research has shown that walking speed directly affects the magnitude of the ground reaction forces during walking.[23] An independent *t* test showed a significant between-group difference in walking speed; therefore, an analysis of covariance (ANCOVA; SPSS, version 10.0 software[h]) was used when comparing force and moment data. Consequently, these analyses examine the differences between the OA and healthy groups after adjusting for differences in walking speed. Statistical significance was set at *P* less than or equal to .05. The dependent variables included peak knee compressive, shear, and resultant joint forces; peak knee vertical loading rate (ie, peak vertical force/time); and peak knee flexion and extension, abduction and adduction, and internal and external rotation moments. Peak hip flexion and extension and peak ankle plantarflexion moments were analyzed descriptively to identify possible compensatory strategies implemented by the OA participants.

In addition, knee joint range of motion (ROM), temporal characteristics, electromyographic data, and unadjusted knee force and moment data are presented descriptively (mean ± standard error [SE]). These data highlight the adjustments that older adults with knee OA make in their gait mechanics.

RESULTS

For the 10 participants in the OA group, OA was distributed among the medial (60%), lateral (30%), and patellofemoral (90%) compartments of the knee. By using the Kellgren-Lawrence scale, radiographic severity was rated as mild in 3 participants, moderate in 5 participants, and severe in 2 participants. This cohort also reported mild to moderate knee pain attributed to knee OA (mean, 2.48±0.70; scale range, 1 [no pain] to 6 [excruciating pain]).[19,24] Parenthetically, data from the Framingham cohort indicate that only 40% of people with radiographic knee OA are symptomatic, underscoring the poor association between radiographic evidence of knee OA and pain.[25,26]

Kinetic Variables

We examined our data both before and after adjusting for differences in walking speed. Examining the unadjusted kinetic data provides a picture of the adjustments older adults with knee OA make in their gait mechanics. Examining the data using walking speed as a covariate provides insight into the differences between groups independent of a major consequence of OA, a decline in walking speed. From a clinical viewpoint, physicians and therapists must deal with the adjustments patients have made as a consequence of their disease. From a research perspective, controlling for as many confounding variables as possible may reveal the underlying mechanisms of the disease.

Table 1: Adjusted Mean Peak Knee Joint Forces* During Walking for the Osteoarthritic and Healthy Groups

Variable	OA Group	Healthy Group	% Difference†	*P*	Effect Size
Shear force (BW)	0.47±0.04	0.44±0.04	6.8	.74	.25
Compressive force (BW)	3.67±0.24	3.40±0.24	7.9	.49	.38
Resultant force (BW)	3.70±0.26	3.42±0.26	8.2	.49	.36
Loading rate (BW/s)	20.61±2.34	20.13±2.48	2.6	.90	.06

NOTE. Values are mean ± SE.
*Means adjusted for the effect of walking speed using ANCOVA. Means are for most affected leg of the OA group and matched side of the healthy group.
†Positive percentage difference: OA group > healthy group.

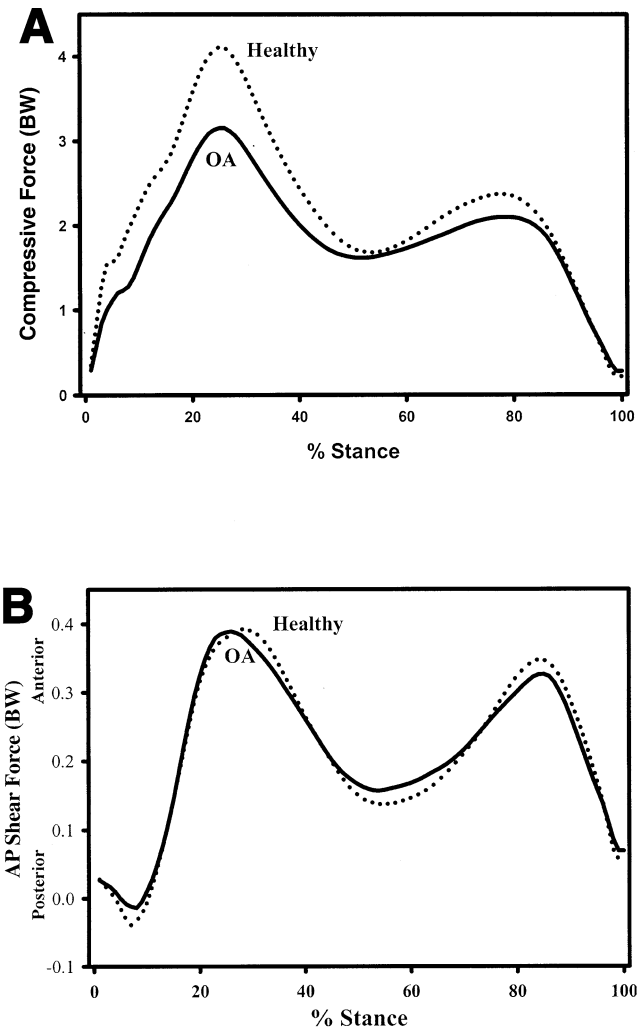

Fig 2. Unadjusted mean (A) compressive and (B) AP shear forces in units of body weight during stance for the OA and healthy groups.

Knee Joint Forces

After adjusting for differences in walking speed, the OA group had between 6.8% and 8.2% greater knee joint forces. There were, however, no significant differences ($P>.05$) between the groups. For the compressive and resultant forces, the effect sizes were moderate, 0.38 and 0.36, respectively (table 1).

The unadjusted mean ± SE peak compressive knee forces were 3.21 × body weight (BW) ± 0.23 and 3.86 BW ± 0.23 for the OA and healthy groups, respectively. Mean peak AP shear forces at the knee were .45±.04 BW for both the OA and healthy groups (figs 2A, B). Resultant knee forces were lower in the OA group (3.24±0.23 BW) than in the healthy group (3.88±0.44 BW). Knee vertical loading rates were attenuated in the OA group (OA group, 17.60±1.57 BW/s; healthy group, 23.47±3.85 BW/s).

Table 2: Adjusted Mean Peak Internal Joint Moments* During Walking for the Osteoarthritic and Healthy Groups

Joint Moment (Nm/kg)	OA Group	Healthy Group	% Difference†	*P*	Effect Size
Knee extension	0.32±0.07	0.24±0.07	33.3	.45	.38
Knee flexion	0.46±0.03	0.45±0.03	2.2	.90	.11
Knee abduction	0.25±0.06	0.33±0.06	–24.2	.35	.44
Knee adduction	0.15±0.03	0.10±0.03	50.0	.30	.56
Knee internal rotation	0.22±0.02	0.25±0.02	–12.0	.36	.50
Knee external rotation	0.16±0.03	0.18±0.03	–11.1	.75	.22
Hip extension	0.98±0.07	0.90±0.07	8.8	.49	.38
Hip flexion	0.48±0.06	0.57±0.06	–15.7	.32	.50
Ankle plantarflexion	1.26±0.12	1.38±0.12	–8.7	.51	.33

NOTE. Values are mean ± SE.
*Means adjusted for the effect of walking speed using ANCOVA. Means are for most affected leg of the OA group and matched side of the healthy group.
†Positive percentage difference: OA group > healthy group.

Knee Joint Moments

The OA group had a 33% greater peak extension moment with a moderate effect size of .38 and a 24% lower peak abduction moment relative to the healthy group (effect size =.44). However, ANCOVA showed no significant differences in any of the adjusted knee joint moments between the groups (table 2). Trends in the OA group's unadjusted knee moment data included decreased extension and internal rotation and similar abduction peak moments relative to the healthy group (figs 3, 4).

Hip and Ankle Joint Moments

Hip and ankle moments were analyzed descriptively to identify possible compensatory strategies implemented by the OA participants. After adjusting for differences in walking speed, the hip extension moment was 9% greater and hip flexion and ankle plantarflexion moments were attenuated in the OA group (table 3). Effect sizes ranged from .33 for ankle plantarflexion to .50 for hip flexion (table 2). Examination of the unadjusted hip and ankle joint moment data indicated reduced peak moments in the OA group relative to the healthy group.

Electromyographic Data

The electromyographic patterns for both groups were consistent with our joint moment data and were similar to previously reported results[27] (figs 3E, F).

Temporospatial Characteristics and Knee ROM

Temporospatial characteristics of the 2 groups are in table 4. The OA group tended to have a shorter stride length, lower cadence, shorter nonsupport time, greater step width, longer support time, and longer double-support time than the healthy group. Knee flexion and extension ROMs were similar between the groups. One notable exception, however, was the tendency of the OA group to hyperextend the knee at heel strike (figs 3A, B).

DISCUSSION

Analysis of the knee joint force and moment data provide insight into the possible mechanisms associated with the development of knee OA. The lower extremity's first shock-absorbing reaction to floor impact is ankle plantarflexion.[27] At approximately 7% of the gait cycle, knee flexion, restrained by the quadriceps, constitutes the second shock-absorbing reaction. Consequently, internal knee extension moments, in response to external knee flexion moments, increase and may result in increased knee joint loading. Not surprisingly, the slower walking speed of the OA group resulted in lower joint forces and moments during this period of stance. We suggest that OA participants reduce knee extension moments and thus knee compressive forces in response to pain by reducing walking speed.

In a previous study, we suggested that OA participants walking at similar speeds to healthy participants maintain walking speed by increasing hip ROM. Our results suggest that greater hip extensor moments may assist in the maintenance of walking speed, but that hip flexor and ankle plantarflexor moments do not compensate for the attenuated contribution of the affected knee extensor moment. This results in the slower walking speed typically observed in this disabled population.[12,28,29]

After adjusting for differences in walking speed, the peak knee extension moments were statistically similar between the groups, although the OA group showed a nonsignificant trend with 33% greater values. Schipplein and Andriacchi[15] found that when walking at the same speed, arthritic patients had peak extension moments that were twice the magnitude of those of a healthy control group. In addition, their knee OA patients had approximately a 16% greater knee peak compressive force. This is in agreement with our data, which found that greater extension moments were coupled with 8% greater knee compressive forces. The increased extension moments may be an adaptive mechanism that increases compressive forces to help maintain stability.[15]

Our results indicate that there are no significant differences in knee joint loads between OA and healthy older adults. Although no direct evidence links joint loads to knee OA, Clements[30] and Radin[31] and colleagues have associated excessive loading with chondrocyte death and the development of knee OA in animal models. More recently, Felson et al[32,33] suggested that bone marrow edema lesions in the femoral condyles and tibial plateau correlated highly with pain and progression of knee OA. These lesions are associated with microfractures and malalignment and are thus related to increased loading.[34] The trends in our data (eg, 33% greater knee extension moments and 8% greater knee joint loads in the OA group) and the results of others[15,30,31] imply that more definitive evidence is needed to determine whether higher extension moments increase knee joint forces. We speculate that these increased loads, applied over the course of many years, may cause microcracks and bone marrow edema resulting in a gradual degradation of the articular cartilage.

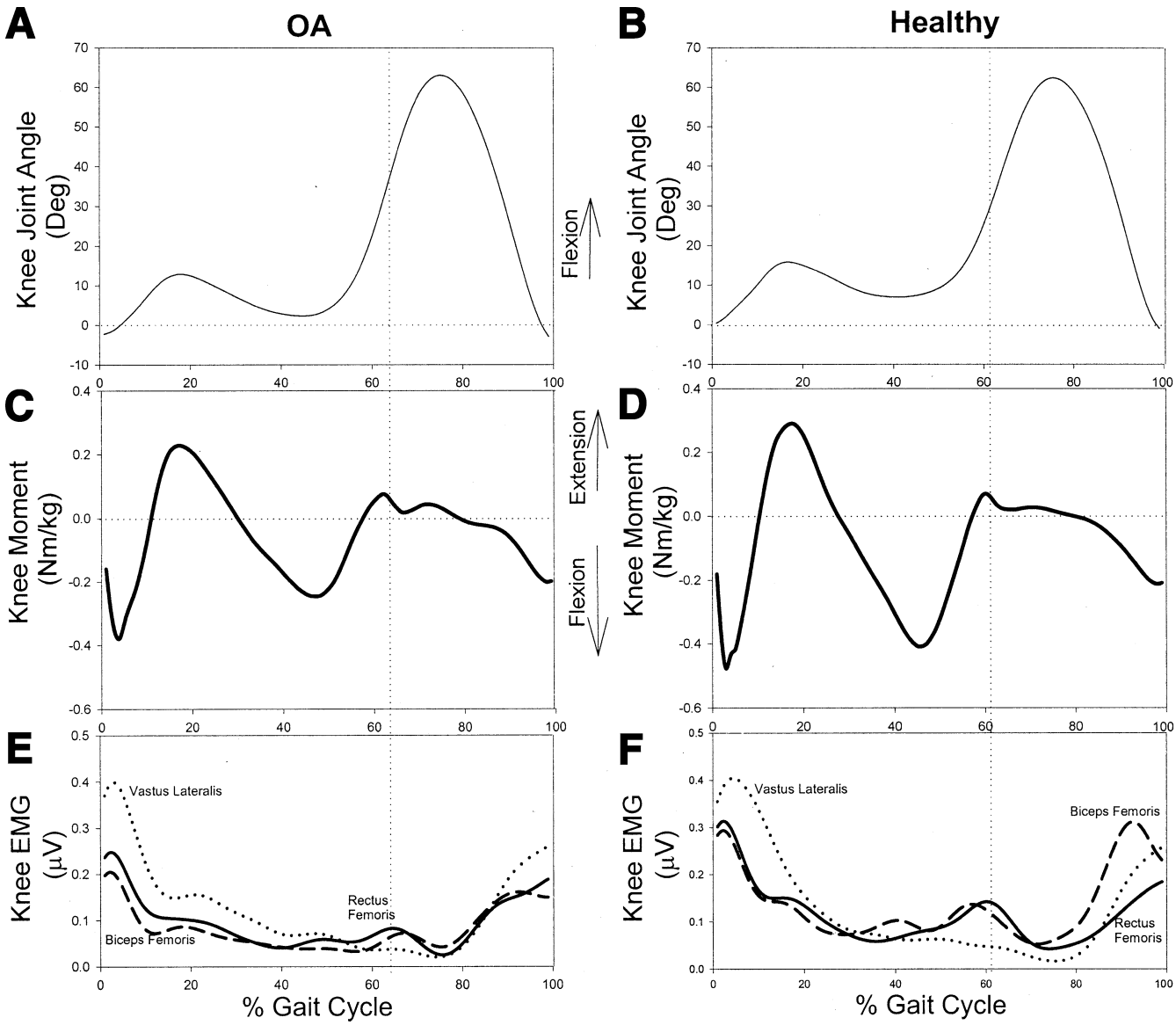

Fig 3. Knee joint data. Unadjusted mean (A, B) knee ROM, (C, D) knee extension and flexion joint moments, and (E, F) electromyographic patterns of the vastus lateralis, biceps femoris, and rectus femoris for the OA and healthy groups. Abbreviation: EMG, electromyographic activity.

Many researchers have calculated knee joint forces during walking, using primarily healthy young adults as subjects and various inverse-dynamics and forward-dynamics models. Compressive and resultant peak forces ranged from 2.8 to 6 BW across studies.[35-38] Our unadjusted peak mean compressive force for the healthy older cohort was 3.9 BW with a 95% confidence interval that ranged from 3.5 to 4.4 BW. Hence, our healthy older cohort exerted forces that fell in the midrange of previously reported results. Schipplein and Andriacchi[15] reported a total joint reaction force (medial and lateral compartment loads summed) of 3.7 BW for 19 OA patients who walked at similar speeds to an age-matched healthy cohort. This is in agreement with our data, which also indicate a mean peak compressive force of 3.7 BW adjusted for differences in walking speed.

The external adduction moment adducts the knee during stance and is counteracted by the internal abduction moment.[11] Several researchers[11,39] have linked increases in this moment to the development and progression of medial tibiofemoral OA. Both passive (eg, iliotibial band) and, to a lesser extent, active (eg, biceps femoris) structures resist the external adduction moment. Increases in this frontal plane moment are thought to lead to an increase in the compressive load transmitted across the knee, predominately to the medial compartment. We found, however, no significant differences in the peak internal abduction moment between the groups. Evidence of knee OA in our participants was distributed among the various knee compartments and ranged from mild to severe. Our abduction moment data agree with the results of a similar study[12] of 139 patients with mild to moderate knee OA and 20 healthy controls. In contrast, the patients in the study by Sharma et al[11] had joint degeneration confined to the medial compartment. Hence, it appears the link between the external knee adduction moment (or internal abduction moment) and knee OA may be strongest

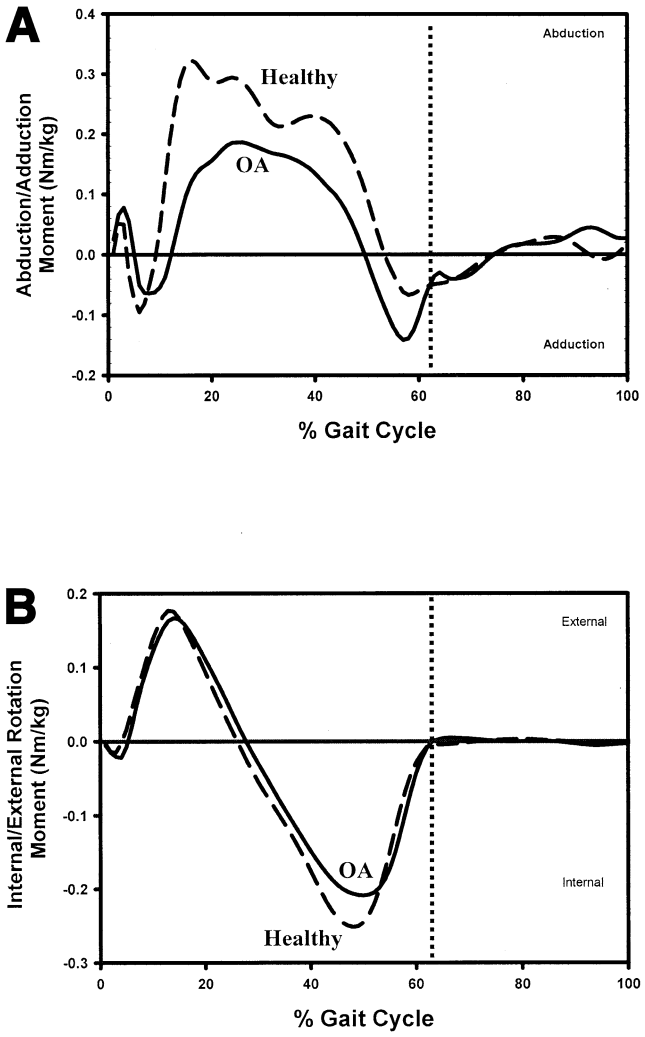

Fig 4. Unadjusted (A) internal mean abduction and adduction and (B) internal and external rotation moments for the OA and healthy groups.

in patients whose arthritis is confined to the medial compartment. In our OA patients, the distribution of joint disease among the 3 compartments of the knee and similar abduction moments to the healthy group may have accounted for, at least in part, the lack of significantly greater knee compressive and shear forces.

Table 3: Unadjusted Mean Peak Knee, Hip, and Ankle Sagittal Plane Internal Joint Moments During Walking for the Osteoarthritic and Healthy Groups

Joint Moment (Nm/kg)	OA Group	Healthy Group	% Difference*
Knee extension	0.26±0.12	0.30±0.12	–13.3
Knee flexion	0.40±0.03	0.51±0.05	–21.6
Hip extension	0.85±0.07	1.03±0.13	–17.5
Hip flexion	0.51±0.07	0.53±0.04	–3.8
Ankle plantarflexion	1.27±0.14	1.36±0.07	–6.6

NOTE. Values are mean ± SE.
*Positive percentage difference: OA group > healthy group.

Table 4: Mean Temporal and Spatial Gait Characteristics of the Osteoarthritic and Healthy Groups

Variable	OA Group	Healthy Group	% Difference*
Step width (cm)	13.48±0.66	13.07±0.74	3.1
Stride length (cm)	119.6±2.51	130.8±4.97	–8.6
Walking velocity (cm/s)	109.7±3.59	129.6±8.42	–15.4
Cadence (steps/min)	108.7±2.74	118.2±3.81	–8.0
Support time (%)	64.01±0.43	61.14±0.65	4.7
Nonsupport time (%)	35.98±0.43	38.36±0.65	–6.2
Double-support time (%)	13.65±0.37	11.63±0.92	17.4

NOTE. Values are mean ± SE.
*Positive percentage difference: OA group > healthy group.

Knee external and internal rotational moments are important in causing the medial and lateral rotations that are synchronized with similar movements at the subtalar joint (ie, inversion and eversion). The synchronization of these movements assists in proper weight transfer during the transition from double- to single-limb support. After adjusting for differences in walking speed, the OA group exhibited 8% to 15% lower values. These data suggest that lower transverse moments at the knee may help distinguish healthy from OA gait and may disrupt the normal transfer of weight during the stance phase of gait.

Several researchers[9,10] suggest that poor strength and proprioception in knee OA patients results in impaired balance. Indeed, stability is an important factor in the performance of activities of daily living. One possible stabilizing mechanism occurred at heel strike, where the OA group exhibited knee hyperextension (fig 3A). This action is common in patients with knee pain and weak quadriceps and helps preserve weight-bearing stability as the foot drops to the floor at initial contact.[24,40] Additional stabilizing mechanisms were apparent in the temporal characteristics of the OA group. Increased step width and double-support time and reduced walking speed, stride length, and cadence are consistent with the results of previous studies that compared knee OA participants with healthy controls.[9,23,28,29]

Electromyography was used to verify our joint moment data. At initial contact, the quadriceps and hamstrings (represented in figs 1E and F by the vastus lateralis and biceps femoris) work to stabilize the knee. As the participant's body weight rolls over the foot, a knee flexor moment is created and the vastus lateralis acts eccentrically to control knee flexion. During mid and terminal stance, there is little muscular action as an ankle plantarflexor torque controls forward acceleration of the tibia over the foot-ankle complex. During preswing (toe-off), the ground reaction force vector moves posterior to the knee creating an external flexor moment that is controlled by a small internal extensor moment. As a result, the rectus femoris restrains passive flexion. At the onset of the swing phase, the biceps femoris initiates knee flexion, which is augmented by forward thigh momentum.[27] Both groups exhibited these patterns.

CONCLUSIONS

This study sought to determine the feasibility of designing a study with sufficient power to examine whether older adults with knee OA have abnormal knee joint moments and place greater loads on the knee joint during walking relative to healthy older adults. Our results, which were limited by the small sample size (N=20) and low statistical power (β=.12), did not provide evidence that patients with knee OA exert greater loads on the knee than healthy adults. Nevertheless, the

trends in the data, along with previous results,[15] suggest the need to investigate further the possible existence of a biomechanical pathway to knee OA.

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