Compensatory Gait Mechanics in Patients with Unilateral Knee Arthritis

CHRIS A. McGIBBON and DAVID E. KREBS

ABSTRACT. ***Objective.*** Few studies exist on gait adaptation caused by knee osteoarthritis (OA), and those have only explored adaptations of the kinematics and kinetics of the knee joint itself. We characterize ankle, knee, hip, and low back mechanical energy expenditures (MEE) and compensations (MEC) during gait in patients with knee OA.

Methods. Thirteen elderly patients with unilateral knee OA and 10 matched healthy elderly controls were studied during preferred and paced speed gait. Gait speed, step length, and lower extremity and low back joint MEE and MEC were compared between groups.

Results. Patients with knee OA had lower, but not significantly different, walking speed and step length compared to the controls, and had significantly different joint kinetic profiles. Patients had reduced ankle power at terminal stance, lacked a second positive peak in knee power, and had increased power absorption at the hip. Abnormal knee kinematics were exaggerated when walking at a paced speed, but hip kinetics normalized among patients with OA.

Conclusion. Reduced ankle plantar-flexion power in patients with knee OA was probably due to disrupted transfer of energy through the knee. Lack of concentric knee power supports prior studies' conclusions that patients with knee OA avoid using their quadriceps to stabilize the knee, probably to reduce articular loads. Patients with knee OA increase eccentric hip power due to increased hip extension caused by abnormal knee kinematics, potentially increasing hip articular forces. This passive mechanism, however, may assist in the advancement of the leg into swing phase. (J Rheumatol 2002;29:2410–9)

Key Indexing Terms:

GAIT	ELDERLY	UNILATERAL ARTHRITIS
KNEE	POWER FLOW	ENERGY TRANSFER

Roughly 15% of elders have symptomatic osteoarthritis (OA)[1]. Pain and loss of joint mobility with OA is associated with muscle weakness and functional limitations[2], which eventually lead to disability. The growing acceptance of physical activity and exercise as a treatment option for OA is evidenced by its dominance in the literature[3-10]. There is a need to measure the mechanisms underlying changes in patient function, in addition to the more typical investigation of changes in impairments and symptoms.

Measures of functional performance include maximum walking distance or 6 minute walk test[4-6,11], walking time (generally over 50 ft)[3], walking velocity[2,8,10,11], cadence and/or stride length[6,8], chair rise and/or stair ascent/descent time[3,12], and postural sway[9,10]. These measures are useful for gauging improvement in function, but are not informative about how function has been improved, or their contribution to joint preservation and protection.

Fewer studies report lower extremity kinematics and kinetics in patients with OA during walking. Stauffer, *et al*[13] found that patients with knee OA had significantly less dynamic knee flexion range of motion during gait. Others have found relationships among knee adduction moment, disease severity, and knee varus/valgus alignment[14-16]. Recently, Kaufman, *et al*[17] reported reduced knee extensor moment in patients with knee OA, suggesting a mechanism for reducing articular forces of the painful and unstable knee joint. No study, however, examined the role played by other joints in facilitating compensatory mechanics of the knee.

McGibbon, *et al*[18] used mechanical energy analysis to identify compensations of the lower extremity joints for a sample of age matched healthy and disabled elders. The disabled subjects had a variety of impairments, but most had some lower limb degenerative joint disease. Disabled subjects transferred less energy with ankle plantar-flexors in late stance phase than did the healthy subjects, but increased low back and hip energy expenditures compared to healthy subjects. In another study, McGibbon, *et al*[19] examined compensatory mechanics in relation to strength and pathology in functionally limited elderly women. Weaker

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subjects expended less ankle and knee energy and more hip and back energy than stronger subjects. However, inverse correlations between ankle energy expenditure and hip and back energy expenditure for subjects with musculoskeletal impairments were independent of strength.

These studies suggest a potentially valuable means of examining the compensatory mechanics of patients with knee OA. Further, this approach may be very useful for understanding how locomotor function improves following intervention[20].

We examined lower extremity mechanical energy transfers and compensations using methods as described[18], but for patients having unilateral knee OA compared with age matched healthy elders. We hypothesized that: (1) consistent with their disability status, elders with knee OA would have lower ankle and knee mechanical energy expenditure (MEE)[18], and greater hip and low back MEE, than age matched healthy subjects; and (2) consistent with their primary pathology, elders with knee OA would compensate by reducing knee MEE to a greater extent than age matched healthy subjects, resulting in a higher mechanical energy compensation (MEC)[18] coefficient.

MATERIALS AND METHODS

Thirteen elderly patients (11 women, 2 men) with diagnosed unilateral knee OA participated in the study. Patients ranged in age from 50 to 83 years old, with mean age of 72.9 years (± 8.9 yrs). Subjects were recruited through outpatient physical therapy services at the Massachusetts General Hospital, Boston, Massachusetts. Inclusion criteria were that patients have diagnosed unilateral knee OA, be 50 years of age or older, and be able to ambulate without the use of walking aids. Subjects with other forms of arthritis (rheumatoid, septic, etc.) and comorbidities such as balance or other neurological impairment or cardiopulmonary or respiratory diseases were excluded. Four of the 13 subjects had been previously diagnosed with bilateral knee OA, but had undergone unilateral total knee replacement at least one year prior to the study. Knee Society scores for the operated leg were > 85 for all 4 subjects, and thus fit the inclusion of unilateral knee OA. Ten healthy elders (6 women, 4 men) also participated in the study. Healthy subjects ranged in age between 68 and 83 years (mean 73.3 ± 4.6) and had no orthopedic or neurological disorders, as determined prior to the study. All subjects were community dwelling, and all signed informed consent prior to gait analysis.

Subjects walked barefoot along a 10 m walkway, first at their preferred walking speed and then at a controlled cadence of 120 steps·min^{-1} set by a metronome. One or 2 practice trials were allowed prior to data collection. Gait data were captured using a 4 camera Selspot II optoelectric light emitting diode (LED) tracking system (Selective Electronics, Partille, Sweden) and 2 Kistler piezoelectric force platforms (Kistler Instruments, Winterthur, Switzerland). Arrays of LED were placed on the midsections of 11 body segments (feet, shanks, thighs, pelvis, trunk, arms, and head), enabling globally referenced 6 degree-of-freedom motion (6 DOF) to be captured for each body segment. Details of the gait analysis protocol are published[21,22].

Subjects' anatomical data were then used to transform the global 6 DOF kinematics into 6 DOF body segment kinematics[23]. Body segment mass, center of mass, and mass moment of inertia were computed from regression equations using subject-specific anatomical measurements[24,25]. Segment angular and linear velocities and accelerations were computed by numerical differentiation of segment position data, and used with segment mass-inertial data to compute the net joint torques based on the Newtonian inverse dynamic approach[26].

Force plate data are critical for calculating the lower extremity torques required for the mechanical energy analysis. Thus a constraint was placed on all subjects' gait trials for inclusion in the study. First, subjects had to strike one or both force plates cleanly with the entire foot. If any portion of the foot (heel or toe) was not in contact with the force plate, or questionably so, the trial was discarded. Second, if the subject's contralateral foot came in contact with the same force plate of the ipsilateral side, the trial was discarded. All 10 subjects had at least one usable gait trial for both preferred speed and paced speed gait. However, 10 of the 13 OA subjects had only preferred speed gait trials, and 9 of the 13 subjects only had usable data for the paced speed trials. The majority of subjects' data were averaged over 2 or 3 repeated trials.

The mechanical energy approach used was originally described by Aleshinsky[27-31], and we have described the analysis technique in detail[18,19,32]. Briefly, mechanical power was computed at the proximal and distal end of each body segment, and then combined to arrive at net joint powers for each joint. The signs and relative magnitudes of the segmental and net joint powers determine the "mode" of energy transfer: proximal transfer, distal transfer, or no transfer, with each having concentric or eccentric conditions. Each mode represents a unique form of MEE. Further, calculating the ratio of the net joint work to the total absolute energy at the joint gives the MEC at the joint. The MEC represents the degree of muscular compensation and has values between 0 and 1, where the extreme value 0 means the joint dynamics, nominally from the surrounding joints' muscles, are totally "uncompensated," i.e., no energy is transferred from one segment to another requiring the joint muscles to generate/absorb all energy added to/removed from the adjoining segments (no-transfer condition). The extreme value 1 means muscles spanning the joint are "totally compensated," i.e., all energy entering one segment is delivered from an adjoining segment, requiring no muscle assistance (transfer condition).

Joint MEE variables were reduced to 3 transfer conditions as previously described[18]: concentric energy transfer, $MEE_{(+)}$; eccentric energy transfer, $MEE_{(-)}$; and no energy transfer, $MEE_{(0)}$. MEC were calculated separately for concentric energy transfer, $MEC_{(+)}$ and eccentric energy transfer, $MEC_{(-)}$. Note that MEC is always zero for no-transfer intervals, because by definition there is no compensation.

Multivariate analysis of variance (Wilk's Λ) were used to assess between-groups differences in $MEE_{(+)}$, $MEE_{(-)}$ and $MEE_{(0)}$, and $MEC_{(+)}$ and $MEC_{(-)}$, for ankle, knee, hip, and low back. Height normalized gait velocity (calculated from averaging the forward velocity of the whole body center of gravity during stance phase and dividing by height) was used as a covariate for each multivariate test. Step length was also acquired. Due to the small sample size and large number of comparisons, and the conservative nature of Wilk's Λ, the α level for the multivariate Wilk's Λ was set 0.10. Univariate statistics for individual comparisons were further investigated using a Bonferonni α correction ($\alpha = 0.10/3 = 0.033$ for MEE and $\alpha = 0.10/2 = 0.05$ for MEC). We relaxed the interpretation of any nonsignificant Wilk's Λ tests when it was clear that a significant univariate test was outweighed by nonsignificant tests. All statistical analyses were performed using SPSS for Windows (v. 8.0; SPSS Inc., Chicago, IL, USA).

RESULTS

Figure 1 shows knee and hip flexion profiles for a representative healthy subject and patient with knee OA. In addition, 3 dimensional android figures representing the actual subjects are shown above the plots. Clearly, the patient with OA fully extends the knee in mid-to-late stance, flexes the knee less in swing phase, and flexes and extends the hip more than the healthy subject. The ground reaction force line of action (arrow) suggests, in this patient with OA, greater hip torque in combination with higher rate of change of hip flexion/extension results in an increase in hip power. Reduced knee torque and diminished knee flexion velocity

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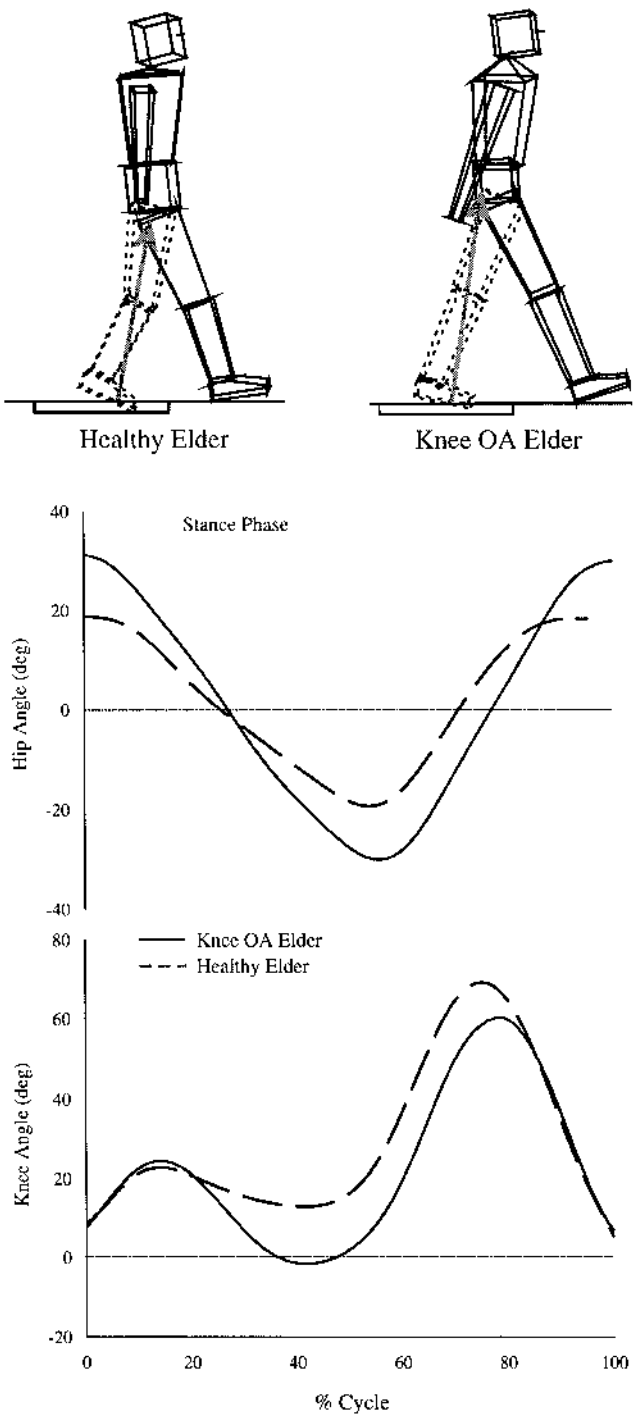

Figure 1. Knee and hip flexion/extension angles during gait for a representative healthy elder and elder with knee OA. The figures represent each subject's measured body posture near the end of stance phase of gait. The arrow represents the line of action of the ground force.

(due to locking the knee at full extension) results in lower knee torque.

Figure 2 summarizes ankle, knee, and hip power across all subjects in each group, during the stance phase of preferred speed gait. The obvious differences between healthy and OA subjects are decreased magnitude of the peak positive ankle power in late stance, lack of the second positive peak in knee power, and increased magnitude of the peak negative hip power in mid-stance (Figure 2 a, b, and c, respectively) for OA subjects. Ankle and hip energy expenditures were significantly different between healthy and OA subjects for preferred gait (Table 2). When controlling for gait speed, OA subjects transferred less concentric ankle energy to the foot ($p = 0.018$), and absorbed more eccentric hip energy transferred proximally from the thigh into the pelvis ($p = 0.038$) and away from the pelvis ($p = 0.012$). Differences in knee energy expenditure between healthy and knee OA subjects were not significant either before or after controlling for gait velocity. There were no significant differences in low back energy expended by healthy and OA subjects.

Figures 3 a, b, and c show ankle, knee, and hip power, respectively, across all subjects in each group, during the stance phase of paced speed gait. As with preferred speed gait, OA subjects had lower peak positive ankle energy (Figure 3a), and no second peak in positive knee power (Figure 3b). The hip power profiles, however, were very similar for healthy and OA groups during paced gait (Figure 3c). Ankle and low back energy expenditures were significantly different between healthy and OA subjects for paced gait (Table 3). When controlling for gait speed, OA subjects transferred less concentric ankle energy to the foot ($p = 0.026$) and absorbed more eccentric low back energy transferred proximally from the pelvis into the trunk ($p = 0.022$). Patients with OA expended less concentric energy at the knee ($p = 0.031$), but this difference became nonsignificant when controlling for gait velocity. There were no significant differences in hip energy expended by healthy and OA subjects.

Compensation coefficients (Figures 4 and 5) indicate that OA patients compensated more knee ($p = 0.015$) and low back ($p = 0.020$) muscle energy compared to the healthy subjects during paced gait. Comparison of compensation coefficients for other joints were nonsignificant.

Comparison of the healthy and OA groups' demographics (Table 1) showed no significant difference in age ($p = 0.772$), height ($p = 0.550$), or weight ($p = 0.137$). Although OA subjects' preferred gait speed (1.03 ± 0.26 m/s) was slower than for healthy subjects (1.19 ± 0.22 m/s), the difference was not statistically significant ($p = 0.152$). When paced at 120 steps per minute, however, the healthy subjects walked significantly faster than the patients with OA ($p = 0.017$). The healthy elders were able to increase their gait speed (1.27 ± 0.17 m/s) to match the faster pace; however, the OA subjects were unable or unwilling to increase their speed (1.02 ± 0.25 m/s), and their average speed actually decreased by a small, insignificant margin.

A similar trend was noted for step length. During preferred gait speed, there was no significant difference ($p =$

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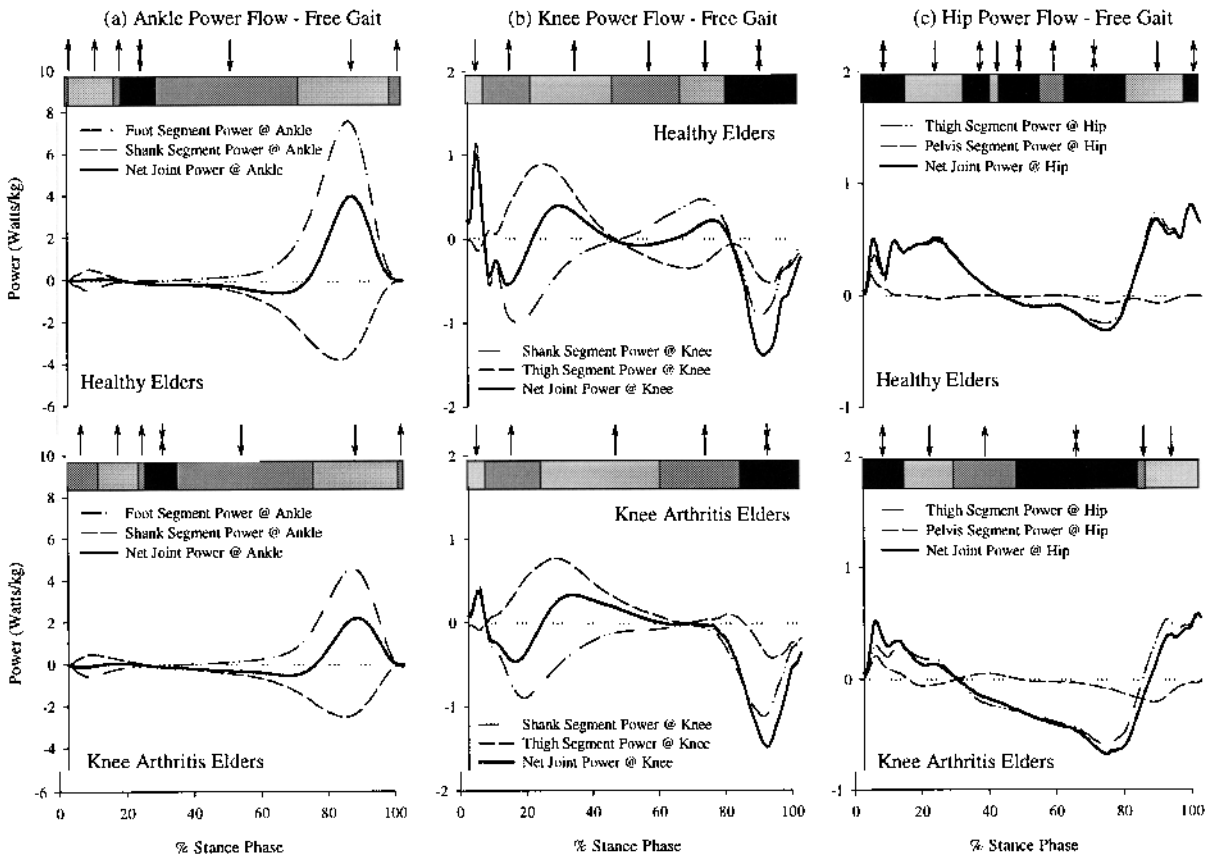

Figure 2. Ankle (a), knee (b), and hip (c) power flow during preferred speed gait, averaged for healthy elders and for elders with knee OA (standard deviations not shown for clarity). Shaded bars indicate the energy transfer conditions (light gray = concentric, dark gray = eccentric, black = no transfer) and arrows indicate the direction of energy transfer (up arrow = proximal, down arrow = distal, outward double arrow = concentric no-transfer, inward double arrow = eccentric no-transfer).

0.142) in step length between healthy elders (0.62 ± 0.10 m) and elders with knee OA (0.55 ± 0.09 m). However, during paced gait, healthy elders increased their step length (0.63 ± 0.08 m), while the OA subjects decreased their step length (0.53 ± 0.12 m), the resulting group difference being significant ($p = 0.03$). Because of these differences in gait speed and step length, and their close association, all between-groups comparisons of MEE above were controlled for gait speed.

Finally, to ensure the OA patients with a unilateral knee replacement were not different from other OA patients without knee replacement, all variables (gait speed, step length, and all mechanical energy measures) were compared between those 2 subgroups of the OA group. No statistically significant difference was found for any variable. Because the age range of OA patients was greater than that of healthy subjects, a similar analysis was run comparing older and younger OA patients (mean cutoff). Again, no significant difference in any variable was found between older and younger patients with OA. Small sample sizes for these subgroups tests, however, are not generalizable due to the likelihood of type II errors.

DISCUSSION

Despite the overwhelming evidence that various therapeutic interventions can improve the symptoms of OA, there are relatively few reports that directly measure functional benefits, in part because most investigators are content to indirectly measure function with questionnaires. That some reports find modest functional improvements following intervention[2,5,6,8], but others report no significant improvements in whole body function[10,33], may be due, in part, to insensitivity of questionnaires and timed gait variables, or because strengthening and isometric exercises do not neces-

Table 1. Subject characteristics.

	Group	
	Healthy Elders	Knee Arthritis Elders
Age, yrs, mean (SD)	73.72 (4.63)	72.80 (8.92)
Height, m, mean (SD)	1.67 (0.11)	1.65 (0.10)
Weight, kg, mean (SD)	72.74 (8.57)	81.78 (16.69)
Sex, n		
Male	4	2
Female	6	11

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Table 2. Preferred speed gait mechanical energy expenditures (MEE) at the ankle, knee, hip, and low back joints for the 3 energy transfer conditions (concentric, eccentric, and no-transfer). Means, standard deviations (SD) and adjusted means (for covariate) are presented for healthy elderly and disabled elderly groups. Between-groups statistics are the multivariate (Wilk's λ) p values followed by univariate p values for each joint, each consisting of the main effect (no covariate) adjusted main effect (with covariate), and corresponding covariate regression. Mechanical energy units are expressed in Joules percent body mass.

Energy Transfer Condition (100* Joules/kg)	Healthy Elders†, n = 10		Knee Arthritis Elders, n = 10		Multivariate/ Univariate p		
	Mean (SD)	Adjusted Mean*	Mean (SD)	Adjusted Mean *	Effect, No Covariate	Effect w/ Covariate	Co-variate Regression
Ankle MEE					0.052	0.086	0.019
Concentric	38.14 (15.62)	36.70	21.99 (5.98)	23.43	0.007	0.018	0.024
Eccentric	11.06 (5.82)	11.21	9.74 (3.68)	9.58	0.551	0.493	0.217
No Transfer	1.94 (2.49)	2.02	1.24 (1.58)	1.16	0.460	0.397	0.157
Knee MEE					0.519	0.535	0.002
Concentric	8.64 (3.57)	8.12	6.73 (5.01)	7.25	0.341	0.647	0.063
Eccentric	5.52 (3.48)	5.02	6.63 (3.36)	7.13	0.478	0.143	0.017
No Transfer	13.22 (7.00)	12.01	11.34 (6.52)	12.55	0.542	0.828	0.002
Hip MEE					0.279	0.070	0.032
Concentric	8.90 (4.66)	8.71	7.66 (6.26)	7.85	0.621	0.748	0.599
Eccentric	2.29 (2.41)	1.91	7.69 (7.95)	8.07	0.055	0.038	0.332
No Transfer	11.76 (6.58)	10.04	18.02 (11.93)	19.74	0.163	0.012	0.002
Back MEE					0.512	0.580	0.879
Concentric	0.50 (0.63)	0.53	0.56 (0.39)	0.53	0.801	0.993	0.406
Eccentric	0.37 (0.42)	0.37	0.70 (0.52)	0.70	0.129	0.159	0.959
No Transfer	0.91 (0.96)	0.93	1.23 (1.68)	1.21	0.609	0.676	0.824

* Adjusted means and effect evaluated for gait velocity/height covariance (0.67 s^{-1}).
† Healthy subjects data are from McGibbon, *et al*[18].

sarily translate into improved locomotor function[20,22,34]. Subtle yet important changes in gait function may not be identifiable, nor satisfactorily understood, without examining the mechanistic source of such changes: studies suggest kinetic analyses (joint power and mechanical energy transfer in particular) of functional movements can provide this level of detail. Several studies have examined gait kinematic and kinetic variables in OA, most examining only the knee[13-17,35,36]. One exception is a study by Weidenhielm, *et al*[35] that reported higher contralateral hip moments in patients with unilateral knee OA compared to healthy controls, which could be corrected through surgical intervention of the involved knee. Unfortunately, however, few conclusive data are available to make nonsurgical treatment recommendations to improve gait function in patients with OA.

Our goal was to characterize gait function in patients with unilateral knee OA by examining the whole kinematic chain — from the foot to the trunk — during the load-bearing (stance phase) portion of gait. Current disability concepts[37-39] support the notion that gaining a better understanding of whole-body functional limitations, and how they are influenced by impairments, is more useful in designing therapeutic interventions than concentrating on eliminating the impairment only. To better ameliorate gait function, one must consider how the entire system is affected. Understanding gait compensations for knee OA may provide arthritis clinicians and scientists with useful data for designing functional training interventions. For example, as we show, the limited dynamic mobility of the knee during the stance phase may require compensation at other lower limb joints; a clinical intervention to not only strengthen the knee but to alleviate the compensatory kinetics might be effective for people with knee OA. An obvious example of the latter is a limp developed to avoid loading a painful knee: once the knee is nonpainful, and surrounding muscles stronger, gait training to more normally load the knee — and thus relieve the surrounding and contralateral joints from excessive stress — should be therapeutic.

Analyzing the causes and effects of interrelated movements of several body segments and joints, however, can be an onerous data analysis task. We simplified the burden associated with analysis of kinematic (displacement and velocity) and kinetic (force and torque) data of 4 joints by examining the combination of these quantities (power and energy). The mechanical power/energy methods used here can identify compensations in disabled elders' gait[18,19], and provide a systematic approach to evaluating *how* function is improved following intervention[20]. In this report, we concentrate on characterizing the mechanical compensations present in patients with unilateral knee OA.

Several characteristics of the patients studied here are similar to those of disabled elders we have previously studied[18,19]. Other characteristics, however, appear to differentiate patients with OA from a broader category of elders with functional limitations.

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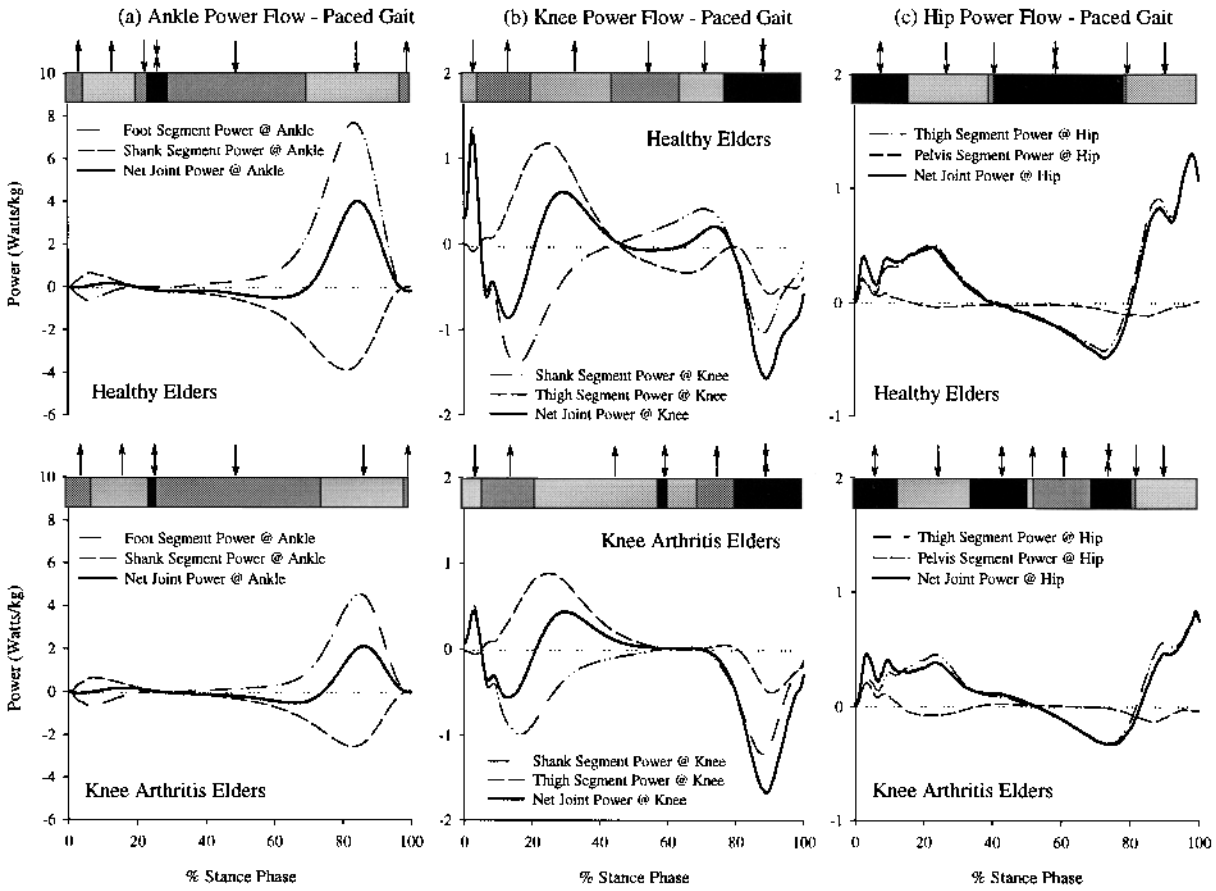

Figure 3. Ankle (a), knee (b), and hip (c) power flow during paced speed gait, averaged for healthy elders and for elders with knee OA (standard deviations not shown for clarity). Shaded bars indicate the energy transfer conditions (light gray = concentric, dark gray = eccentric, black = no transfer) and arrows indicate the direction of energy transfer (up arrow = proximal, down arrow = distal, outward double arrow = concentric no-transfer, inward double arrow = eccentric no-transfer).

Diminished ankle power at push-off (late stance phase) is one characteristic that appears to be a generalized characteristic of disablement, and perhaps of aging alone. Both Judge, *et al*[40] and Winter, *et al*[41] reported diminished ankle power in healthy elders compared to healthy young adults; however, Judge, *et al*[40] reported this difference could be explained by differences in step length. In a prior study[21], we reported no significant difference in ankle push-off power between healthy young and elderly females, but did find a significant difference between the healthy elderly women and functionally limited elderly women. We then compared healthy elderly men and women to a sex, age, height, and weight matched sample of frail elders with a combination of impairments (including arthritis but not limited to lower extremity impairments)[18]. Concentric ankle MEE in late stance was significantly lower for the disabled elders during both preferred speed and paced speed gait, but could be explained by differences in gait speed[18].

In this study, we found that differences in concentric ankle MEE between healthy subjects[18] and patients with unilateral knee OA could not be fully explained by differences in gait speed (Tables 2 and 3). Therefore, patients with knee arthritis reduce ankle plantar-flexion push-off power for reasons other than diminished walking speed. This conclusion is strengthened by the fact that the OA patients' gait speed was only significantly less than that of the healthy subjects during paced gait; therefore, despite their primary impairment, the patients with knee OA could walk at a relatively normal speed.

Figures 2b and 3b (knee power during preferred and paced gait, respectively) offer a possible explanation for the reduced ankle push-off power. Prior to push-off, the knee normally undergoes a second positive power peak, as shown for healthy subjects in Figures 2b and 3b. Energy of the thigh at the knee is negative and energy of the shank at the knee is positive, and greater (in absolute terms) than that of the thigh. Thus, energy is transferred distally during this portion of stance phase, and may contribute to the energy that is ultimately delivered to the foot at push-off. Clearly, the OA subjects lacked this second peak (due to locking the knee at or near full extension) and in addition transferred little or no energy distally (due to minimizing knee torque).

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Table 3. Paced (120 steps·min^{-1}) speed gait mechanical energy expenditures (MEE) at the ankle, knee, hip, and low back joints for the 3 energy transfer conditions (concentric, eccentric, and no-transfer). Means, standard deviations (SD), and adjusted means (for covariate) are presented for healthy elderly and disabled elderly groups. Between-groups statistics are the multivariate (Wilk's λ) p values followed by univariate p values for each joint, each consisting of the main effect (no covariate), adjusted main effect (with covariate), and corresponding covariate regression. Mechanical energy units are expressed in Joules percent body mass.

Energy Transfer Condition (100* Joules/kg)	Healthy Elders†, n = 10		Knee Arthritis Elders, n = 9		Multivariate/ Univariate p		
	Mean (SD)	Adjusted Mean*	Mean (SD)	Adjusted Mean*	Effect, No Covariate	Effect With Covariate	Co-variate Regression
Ankle MEE					0.011	0.197	0.001
Concentric	39.41 (12.00)	34.71	20.12 (6.85)	25.34	0.001	0.026	< 0.001
Eccentric	10.19 (4.01)	10.07	9.36 (5.32)	9.50	0.704	0.836	0.871
No transfer	1.44 (0.97)	1.60	1.20 (0.94)	1.02	0.596	0.292	0.280
Knee MEE					0.215	0.628	0.522
Concentric	9.48 (3.62)	8.97	5.71 (3.31)	6.27	0.031	0.187	0.362
Eccentric	7.55 (5.67)	6.55	5.33 (2.77)	6.45	0.302	0.968	0.156
No transfer	15.05 (7.53)	13.88	13.12 (7.08)	14.43	0.573	0.895	0.311
Hip MEE					0.360	0.375	0.153
Concentric	11.00 (5.57)	10.82	7.32 (6.86)	7.53	0.215	0.373	0.856
Eccentric	1.73 (1.51)	2.00	4.66 (4.45)	4.36	0.066	0.224	0.604
No transfer	14.12 (9.91)	11.48	15.93 (7.64)	18.87	0.665	0.125	0.050
Back MEE					0.225	0.051	0.236
Concentric	0.25 (0.18)	0.12	1.00 (1.35)	1.15	0.099	0.070	0.381
Eccentric	0.36 (0.32)	0.18	0.87 (0.92)	1.07	0.121	0.022	0.076
No transfer	1.28 (1.09)	1.13	1.27 (1.61)	1.43	0.989	0.705	0.501

* Adjusted means and effect evaluated for gait velocity/height covariance (0.69 s^{-1}).
† Healthy subjects data are from McGibbon, *et al*[18].

This effect was particularly pronounced during paced gait: concentric knee MEE was significantly lower for OA patients compared to healthy subjects during paced gait (Table 3) despite the nonsignificant MANOVA test for all 3 knee MEE variables (this being an indication of the strength of the null findings for eccentric and no-transfer MEE).

Patients with OA also had significantly higher concentric compensation coefficients at the knee during paced gait compared to healthy subjects (Figure 4). This suggests patients with OA reduce knee motion and agonist muscle contractions (but do not necessarily reduce co-contractions) to limit the loads experienced by the joint. This finding agrees with Kaufman, *et al*[17] and Baliunas, *et al*[36], who reported reduced knee extensor moments in gait of patients with OA, and is consistent with the association between quadriceps weakness and knee OA[42]. It is unclear, however, if patients with OA in our study had weak quadriceps, or just avoided using them during gait, as we did not directly measure knee muscle strength.

The energy transfer patterns of the hip (Figure 2c) suggest it plays a significant role in compensating for the knee's dysfunction. During preferred speed gait, elders with knee OA expended significantly more eccentric hip energy than healthy subjects (Table 2). Figure 2c shows this excessive energy is absorbed by the hip in mid-stance, extending into late-stance phase. The lack of knee flexion in mid-stance for the OA patients (Figure 1), coupled with continuing forward progression of the pelvis, results in higher hip extension toward the latter portion of stance (Figure 1), generating higher angular velocities, and thus higher power. The mechanism was used to advance the leg into swing, and therefore may be a stretch reflex of the rectus femoris and other quadriceps muscles. Increased passive hip muscle forces could potentially arise because of this compensatory style, thus increasing the articular cartilage loads at the hip. Although knee locking is much more apparent during paced gait, the hip power profiles during paced gait were similar for healthy and OA groups. This was probably due to OA subjects decreasing step length, thus not requiring as much hip extension in mid-to-late stance phase.

Low back powers for patients with OA were markedly different than those of healthy subjects for paced gait, a difference not apparent for preferred speed gait. OA subjects' low back eccentric MEE was significantly greater than for healthy subjects (Table 3), and OA subjects low back concentric MEC was significantly greater than for healthy subjects (Figure 4). This suggests a difference in proximal segment coordination when OA subjects walked at a paced speed, even though their gait speed did not change appreciably. Considering the magnitudes of low back power, however, the additional power probably has little functional benefit, although the increased trunk motion may be potentially destabilizing. Unfortunately, to our knowledge, there are no other studies that have examined the

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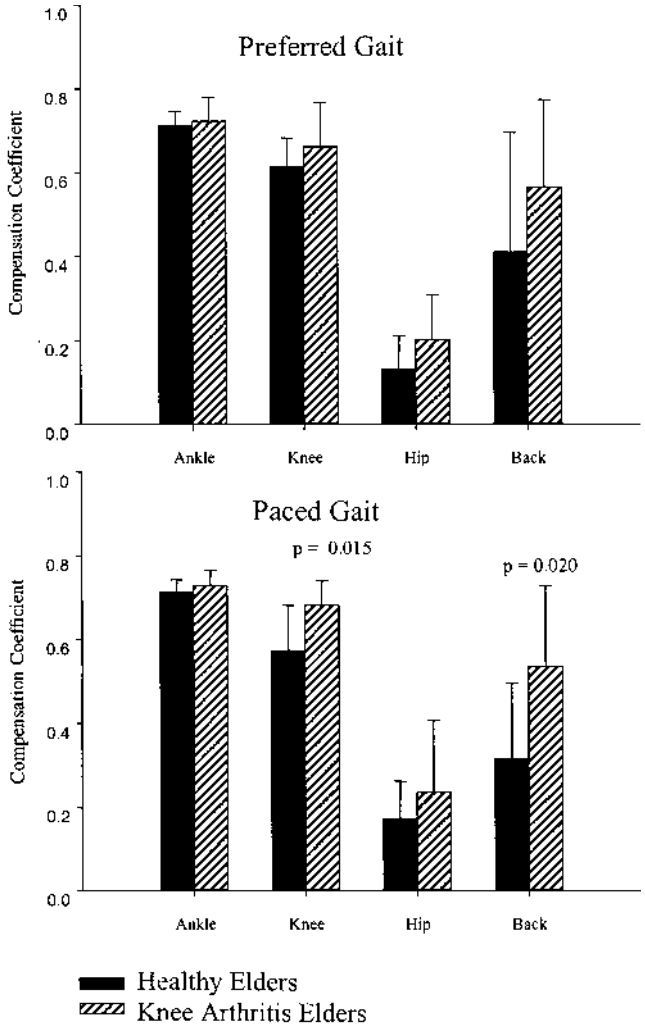

Figure 4. Concentric compensation coefficients for ankle, knee, hip, and low back for healthy elders and elders with knee OA during preferred (top) and paced (bottom) gait. Bars represent ± 1 SD.

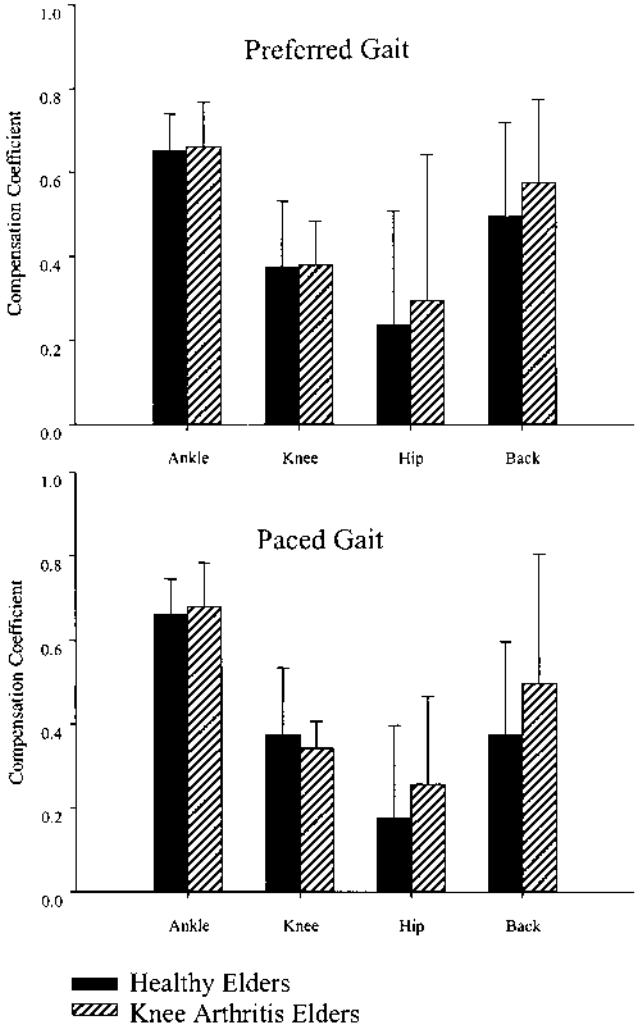

Figure 5. Eccentric compensation coefficients for ankle, knee, hip, and low back for healthy elders and elders with knee OA during preferred (top) and paced (bottom) gait. Bars represent ± 1 SD.

mechanical energy of the low back and hips for patients with only OA of the knee, so there are no data available to compare these mechanical power and energy findings.

Our study has several limitations that may reduce the generalizability of the findings. First, the data capture system only allowed a single stride to be captured during any single gait trial. As a result, averaging repeated trials was used to eliminate within-subject variability. Second, we had only a small sample of subjects with a somewhat disproportionate sex distribution. The small sample size may have reduced the statistical power to detect differences in more variables, such as gait speed and step length measures during preferred speed gait. It is unlikely that the sex disproportion had any appreciable effect on the group comparisons; however, it would be recommended to make such comparisons in a larger study. Third, the age range of the patients (50–83 yrs) was somewhat greater than that of the healthy subjects (68–83 yrs). Because the means of the 2 groups were statistically equivalent we expect this had a minimal effect on the group comparisons. Again, we recommend a larger study to test for confounding age effects. Finally, the inclusion of 4 patients with a unilateral knee replacement may be of some concern. Due to the high Knee Society scores these patients achieved on their operated side (> 85, considered good to excellent[43]), it is likely that patients' "healthy" side behaved similarly to the "healthy" side of the remaining patients without knee replacement. Further, statistical comparison on the operated and non-operated subjects revealed no statistical differences. The nonsignificant findings of these 2 additional tests (young vs old and operated vs non-operated) simply suggest these sources of variance did not confound the differences between healthy and OA subjects in this study. However, the small number of subjects in these comparisons could result in type II errors, and hence these findings are not generalizable.

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Our data suggest that examination of the kinetics of all the joints involved in locomotion (ankle, knee, hip, and low back) may be important for understanding how knee OA affects patient function. We offer the following conclusions: (1) Patients with knee OA have dramatically reduced ankle plantar-flexion power, probably due in part to disrupted transfer of energy across the knee prior to foot push-off and compensation provided by the hip. (2) Lack of concentric knee power in patients with knee OA supports prior conclusions that patients with OA avoid using their quadriceps to stabilize the knee, probably to reduce articular loads. (3) Patients with knee OA increase eccentric hip power due to increased hip extension caused by their abnormal knee kinematics. This passive mechanism may assist in the advancement of the leg into swing phase.

These data may also be important for designing appropriate rehabilitation programs aimed at improving patients' function as well as their symptoms. In this context we make the following recommendations, based on our findings: (1) High eccentric expenditures at the hip may be particularly important to address; tight hip flexors are a common age related impairment, often reducing hip extension in healthy elders[44]. Patients with knee OA may in fact use this impairment to their advantage, but possibly increase hip articular forces and risk destabilizing the pelvis[45]. Therapies such as gait training and strengthening exercise aimed at reducing hip flexion contracture and knee stiffness, and increasing hip, knee, and plantar-flexor muscle concentric strength, might help reduce maladaptive joint kinetics and improve gait function[20]. (2) Although our data suggest that the presence of knee OA alone is sufficient to engender differences in joint kinetics from matched, healthy controls, future studies should consider examining the compensatory characteristics of OA patients in terms of specific impairments and symptoms. Compensations adopted by individuals may vary according to severity of muscle weakness, knee stiffness, and hip flexion contracture. It may well be that varus or valgus deformity, or ligamentous laxity or stiffness, have idiosyncratic effects upon the particular compensation chosen[16]. This would allow functional and strength training programs to be better tailored to meet the needs of the individual.

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