Proximal Gait Adaptations in Medial Knee OA

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ABSTRACT: The purpose of this study was to examine interlimb differences in gait kinematics and kinetics in patients with symptomatic medial knee OA. The main objective was to identify hip joint movement strategies that might lower the knee adduction moment and also compensate for decreased knee flexion during weight acceptance. Gait analysis was performed on 32 patients with moderate medial compartment knee OA. Kinetic and kinematic data were calculated and side-to-side comparisons made. Radiographs were used to identify frontal plane alignment. No interlimb difference in the peak knee adduction moment was found $(p = 0.512)$, whereas a greatly reduced hip adduction moment was seen on the involved side $(p < 0.001)$ during the early part of stance. The involved limb flexed significantly less and hip and knee flexion moments were smaller compared to the uninvolved side. Gait adaptations involving a lateral sway of the trunk may successfully lead to relatively lower ipsilateral knee adduction moments, and would further be reflected by a lower adduction moment at the hip. Subjects did not compensate for less knee flexion by any dynamic means, and likely experience a resulting higher joint impact. These gait adaptations may have implications with respect to development of weakness of the ipsilateral hip musculature and progression of multiarticular OA. © 2008 Orthopaedic Research Society. Published by Wiley Periodicals, Inc. J Orthop Res

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Knee osteoarthritis (OA) is a common cause of functional disability, the medial compartment being the most affected.^{1,2} Medial compartment OA is associated with genu varum, medial joint space narrowing, medial joint laxity, quadriceps weakness, as well as sclerosis and attrition of subchondral bone. $1,3-5$ People with knee OA have altered gait patterns. $6-9$ Knee OA may contribute to altered gait patterns as efforts are made to avoid pain, to minimize joint forces through the deteriorating cartilage, 10 or due to feelings of joint instability.¹¹ The wear and tear process in OA may occur during normal ambulation, and dynamic loading is an important risk factor in knee OA.12 The vast majority of studies of gait in patients with knee OA report discrete single plane kinematic and/or kinetic events at the knee joint alone in attempts to answer questions relating to cause and effect, and to the progression of OA. Multiplanar and multijoint evaluation of joint kinematics and kinetics could provide additional information about compensatory strategies for pain, instability, and deformity during gait.

The adduction moment at the knee is generally considered to reflect the magnitude of medial joint loading.13,14 People with medial knee OA often have a large external knee adduction moment during stance. $8,14,15$ This may, in part, be due to anatomical alterations in the varus aligned knee and to the pathophysiology of OA disease progression.⁸ Over time, the high loading within the medial compartment may exacerbate joint pain and the progression of knee OA.6,8,16–19 Biomechanical and surgical interventions such as shoe wedges, unloader braces, and high tibial osteotomy, are aimed at reducing medial compartment loading. Other mechanisms, such as hip abductor strength, have been proposed to have a protective effect with regard to progression of medial knee $OA^{20,21}$ Chang et al.²⁰ hypothesize that because of their large cross-sectional area, hip muscles can generate significant forces that may be beneficial in regulating medial/lateral knee load distribution, thus providing frontal plane stability.

Although it is likely that deformity and alterations of loading at one lower extremity joint have a significant effect on loading of the other joints of the lower extremities, there have been few studies to evaluate such effects in OA predisposed joints.¹² Asymmetric loading may be a consequence of gait alterations in response to chronic pain and structural pathology. The adaptations patients with unilateral OA make to limit pain or improve function may place relatively greater loads on the contralateral extremity²² and/or other joints of the ipsilateral lower extremity.²¹ This may, in turn, have important consequences in the multi-articular evolution of lower extremity OA.

Mechanical alterations at adjacent joints can affect knee joint loading. Identifying compensatory strategies during gait can have important implications for rehabilitation of patients with medial knee OA, more specifically, with respect to joint range of motion and muscle strength. Therefore, the purpose of this paper was to examine frontal and sagittal plane kinematic and kinetic gait alterations at the hip and knee in patients with unilateral moderate, symptomatic medial knee OA. We hypothesized that frontal and sagittal plane interlimb kinematic and kinetic differences at the knee would be accompanied by alterations at the hip that could contribute to lower loads at the knee. Moreover, we hypothesized that a hip strategy, such as relatively greater hip flexion, would be noted in response to the lack of knee flexion on the involved side during weight acceptance in order to improve shock absorption.

MATERIALS AND METHODS

Thirty-two subjects (25 men, 7 women) with genu varum and moderate medial compartment knee OA were recruited into

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the study from a local orthopedic practice. They had been scheduled for either a high-tibial osteotomy (HTO) or a fitting for a medial "unloader" brace for their condition. All subjects signed informed consent forms approved by the Human Subjects Review Board at the University of Delaware prior to testing. Diagnosis was based on clinical history, a physical examination, and from standing posteroanterior radiographs with the knee flexed $30^{\circ}.^{23-25}$ All of the enrolled subjects had a Kellgren and Lawrence (KL) grade²⁶ of 2 or 3. Malalignment was confirmed from bilateral weight-bearing anteroposterior long cassette radiographs.²⁷ The weight-bearing line (WBL) ratio was calculated as the perpendicular distance from the WBL to the medial edge of the proximal tibia divided by the full width of the tibial plateau;²⁸ a ratio less than 50% denotes varus angulation (Fig. 1B). The mechanical axes angle (MAA) in the frontal plane was derived by the intersection of a line drawn from center of the femoral head to the tibial spines and another line from the tibial spines the center of the ankle mortise; angles less than 180° denote varus (Fig. 1C). Subjects' demographic and radiographic data are highlighted in Table 1. Previous ligament reconstruction, a history of ligament deficiency, cardiovascular disease, diabetes, neurological impairment, impaired balance, rheumatoid arthritis, total knee replacement in either knee, other orthopedic problems in the hips, ankles, or spine, concurrent symptoms from the contralateral knee, or a body mass index (BMI) ≥ 40.0 were exclusion criteria for this study.

Three-dimensional lower extremity gait analysis was performed on all subjects; kinematic data collected at 120 Hz using a six-camera optoelectric motion analysis system (VICON, Oxford Metrics, London, England) and kinetic data collected for consecutive steps onto two Bertec (Worthington, OH) force platforms at 1800 Hz. Markers were placed bilaterally on the iliac crests, trochanters, lateral knees, and malleoli, and on the fifth metatarsal head. Calipers were used to record individuals' anthropometric measures and joint centers were calculated from a static calibration trial. Rigid thermoplastic

mechanical axes angle (\tilde{C}) .

Table 1. Subject Characteristics (Mean \pm SD (Range), $Mean \pm SD$ of Involved/Uninvolved Limb)

Age (years)	53.3 ± 7.8 (40-73)
BMI (kg/m^2)	30.1 ± 3.6 (24.4-37.3)
KOS(%)	$60.5 \pm 17.1(29 - 94)$
WBL $(\%)$	$18.1 \pm 12.4/28.8 \pm 16.7$
MAA (\circ)	$173 \pm 4/176 \pm 4$

BMI, body mass index; KOS, knee outcome survey; WBL, weightbearing line; MAA, mechanical axes angle.

shells affixed with four tracking markers were attached to bilateral thighs and shanks with elastic wraps to minimize movement artifacts during dynamic trials. Marker triads were placed on the sacrum and on each foot. Ten walking trials were collected and averaged, where subjects contacted the force platforms without targeting. Subjects walked along a 10-m walkway at a self-selected pace until consistent velocity $(\pm 5\%)$ was achieved, determined from two photoelectric cells. Marker trajectories and ground reaction force data were low-pass filtered (Butterworth fourth order, phase lag) at 6 and 40 Hz, respectively. Data were analyzed using Visual 3D software (C-Motion, Inc, Rockville, MD). Lower limb kinematics were calculated using rigid body analysis. External joint moments were derived using inverse dynamics and normalized to body mass (kg) and height (m) at the point initial contact (IC) and that of the first peak knee adduction moment (PKAM1) during weight acceptance.

Pain and functional status were assessed using the selfreported Knee Outcome Survey—Activities of Daily Living Scale (KOS-ADLS).²⁹

Hierarchical regression analysis was used to evaluate whether increased hip joint excursion was used to compensate for decreased knee flexion excursion (dependent variable) during weight acceptance [from IC, as determined by a FP threshold of 20 Newtons, through peak knee flexion (PKF)]. Paired t-tests were used to determine interlimb differences in structure (MAA), kinematics, and kinetics (knee and hip frontal and sagittal plane angles and moments). Pearson Product-Moment Correlation Coefficients and linear regression analysis were used to determine associations between structural (MAA), kinematic, and kinetic variables. Significance was set at $\alpha = 0.05$.

RESULTS

MAA measurements showed the involved knee to be in significantly greater varus than the uninvolved side $(p = 0.001;$ Table 1), and this structural interlimb difference was also measured during stance, as a greater adduction angle of the involved knee ($p < 0.001$; Table 2). Frontal plane static knee alignment (MAA) was related to peak knee flexion angle with more varus predicting less flexion during weight acceptance for all knees $(p < 0.001; r = -0.463)$. Post hoc testing revealed that the relationship was significant on the involved $(p =$ 0.013; $r = -0.450$) but not the uninvolved side ($p = 0.78$; $r = -0.327$.

Hierarchical regression analysis showed that ipsilateral hip excursion from flexion toward extension explained half of the variance knee flexion excursion of the involved side during weight acceptance $(p < 0.001$; **Figure 1.** Neutral alignment (A), weight-bearing line (B), and the involved side during weight acceptance $p < 0.001$, mechanical axes angle (C).

	Mean INV-UN (SD)	95% CI	<i>p</i> -Value
Knee adduction angle at IC $(°)$	3.5(3.0)	2.4; 4.5	${<}0.001$
Hip adduction angle at IC $(°)$	$-2.5(5.0)$	$-4.3; -7$	0.009
Knee adduction angle at PKAM1 (°)	4.9(4.2)	3.4; 6.4	${<}0.001$
Hip adduction angle at PKAM1 $(°)$	$-2.5(5.6)$	$-4.6:-.5$	0.015
Knee add.moment at PKAM1	0.015(0.126)	$-.031;0.060$	0.512
Hip add.moment at PKAM1	$-0.101(0.142)$	$-.152:-.050$	${<}0.001$
Knee flexion angle at IC $(°)$	0.4(4.1)	$-1.1; 1.8$	0.615
Hip flexion angle at IC $(°)$	0.7(4.6)	$-1.0; 2.4$	0.395
Knee flexion angle at PKF $(°)$	$-4.4(6.8)$	$-6.8; -1.9$	0.001
Hip flexion angle at PKF $(°)$	$-2.7(5.1)$	$-4.5:-0.8$	0.006

Table 2. Mean Difference (SD) of Kinematic and Kinetic Variables between Involved (INV) and Uninvolved (UN) Knee and Hip

Moments normalized to body mass and height.

significantly added to the model $(p < 0.002)$ resulting in a stronger relationship ($p < 0.001$; $r^2 = 0.625$). This was not the case for the uninvolved limb where hip extension excursion alone explained a portion of the ipsilateral variance $(p=0.001; r^2=0.266)$. Sagittal plane angles were similar at heel-strike. Conversely, the hip and knee on the involved side had a significantly smaller flexion angle than on the uninvolved side during weight acceptance (mean difference of 2.7° $(p = 0.006)$, 4.4° $(p = 0.001)$) for the hip and knee, respectively, at PKF) (Table 2). The truncated flexion of the involved knee therefore resulted in a smaller knee joint excursion $(13.9 \text{ vs. } 18.6^\circ; p < 0.001)$ and a greater relative hip extension excursion $(9.6 \text{ vs. } 7.6^{\circ};$ $p = 0.004$) as the trunk advanced during stance (Fig. 2). In the frontal plane, the involved knee demonstrated a larger knee adduction angle than the uninvolved, by 3.4° at

Figure 2. Hip flexion and adduction angle and knee flexion angle curves during stance expressed in degrees.

IC ($p < 0.001$), the difference reaching 4.9° at PKAM1 (Table 2; $p < 0.001$). The ipsilateral hip on the other hand was more abducted at heel-strike $(4.5 \text{ vs. } 2.1^{\circ} \text{ abduction})$; Table 2; $p = 0.009$ and remained relatively more abducted than the uninvolved side at PKAM1 (mean difference 2.5°; Table 2; $p = 0.015$ (Fig. 2).

There was no side to side difference in the peak knee adduction moment $(p = 0.512)$ whereas the hip adduction moment was 25% smaller than that of the uninvolved side $(p < 0.001)$ (Table 2 and Fig. 3). The adduction moment at PKAM1 was significantly greater at the knee than the hip on the involved side $(p = 0.043)$, whereas the uninvolved limb demonstrated the typical pattern of greater adduction moment at the hip than the knee $(p = 0.007)$.

DISCUSSION

Frontal and sagittal plane interlimb kinematic and kinetic differences at the knee were accompanied by specific movement patterns at the hip. The hypotheses were partially supported in that a frontal plane hip strategy was observed on the involved side that resulted in maintaining medial loading of the affected knee to a level similar to that of the uninvolved side. Subjects

Figure 3. Frontal plane moments at the hip (left) and knee (right) during stance phase of gait expressed as Nm, normalized by $mass \times height$.

did not, however, use any dynamic hip strategy to compensate for the limited flexion of the involved knee during weight acceptance; in fact, the movement pattern at the ipsilateral hip was the opposite of what we expected and theoretically would increase load as the limb demonstrated less dynamic movement through the lower extremity in comparison to the uninvolved side.

People with medial knee OA have higher knee adduction moments compared to controls, $11,30$ and Hunt et al.31 reported a side-to-side difference in adduction moment in their study of patients with KL grade 4 medial OA. Our subjects, however, had no interlimb differences in the peak knee adduction moment. Healthy individuals have a greater hip than knee adduction moment during stance,³² as was demonstrated on the uninvolved side throughout stance in our subjects. However, the hip adduction moment on the involved side was not only significantly smaller than that of the contralateral hip, but also smaller than the involved knee adduction moment during weight acceptance. The ipsilateral hip was relatively abducted throughout much of stance compared to the other side. With the foot on the ground, hip abduction is accomplished by an ipsilateral frontal plane rotation of the pelvis relative to the femoral head. Thus, the most parsimonious explanation for the low hip adduction moment is a lateral trunk lean during early stance, with the secondary effect of the observed greater abduction of the involved hip (Fig. 4). Compensatory gait maneuvers that reduce the peak adduction moment have been described in the literature, and have been hypothesized to be utilized to varying degrees by patients.¹⁴ Mündermann et al.^{21,33} demonstrated lowered knee adduction moments in healthy subjects during trials where they laterally shifted their trunks compared to their normal gait patterns and suggested that trunk lean may successfully reduce the knee adduction moment in patents with mild to moderate knee $OA^{21,33}$ Evidence from the present study supports this concept. Although this strategy may lessen medial forces at the knee during gait, it unfortunately also reduces demand on the hip abductor muscles, leading to subsequent hip abductor weakness.³²

Figure 4. Resultant ground reaction force vector during gait of the involved (top left) versus uninvolved (bottom left) limb and a schematic proposed lateral lean of the trunk (right).

adaptations on the side of the more symptomatic knee. That knee then went on to greater degree of progression of knee OA, possibly due to the lack of excursion of movement through the joint on that side and greater cumulative loading. Furthermore, as the muscles grow weaker, the hip abductors may develop a lack of eccentric

During the earliest phases of the gait cycle the intensity of the loading during impact is reduced by shock-absorbing eccentric muscle reactions at the ankle, knee, and hip. Ankle plantarflexion is controlled by the pretibial muscles, knee flexion by the quadriceps, and contralateral pelvic drop by the hip abductors. All three motion patterns occur during the loading response phase of gait and also help limit vertical oscillations of the body's center of mass.³² As expected, the subjects in this study demonstrated less knee flexion during weight acceptance on the involved side in comparison to the uninvolved side. The relationship seen between amount of knee varus (MAA) and PKF angles may reflect greater symptoms from the more varus aligned knees that results in less willingness to move into knee flexion during weight acceptance. No effective compensatory strategy for shock absorption was measured during this phase of stance in any plane of the hip joint of the involved side. To the contrary; the joint was more abducted and relatively more extended than the uninvolved hip through weight acceptance. Reduced knee flexion excursion during the loading response phase of stance may represent an attempt to avoid pain and/or stabilize the knee and may be coupled with increased muscle coactivation. 6 Childs et al. 6 suggest the combination of greater muscle coactivation and limited joint excursion may lead to increased compressive loading and reduce the potential femoral contact area over which the force can be distributed, which may, in turn, contribute to increased cumulative loading in localized areas. Maintaining the ipsilateral hip joint relatively more extended and abducted does not effectively dissipate the loading force and as a result the knee may be even more susceptible to development and progression of OA. Although the strategy adopted by the subjects in the

present study may have resulted in lower than expected knee adduction moments of the involved knee, it has potential negative sequelae as well. Displacing the trunk laterally shifts the center of gravity and allows the body to be balanced over the stance leg with minimal muscular support at the hip joint.³⁴ The hip joint's abductor muscles are thus put at a mechanical advantage that may result in muscle weakness over time. Persistence of a Trendelenberg gait may ensue, even after a successful intervention such as osteotomy or bracing, because the hip abductors are now weak. Chang et aL^{20} associated low hip adduction moments with worsening of radiographic knee OA over an 18-month period and concluded that stronger hip abductors that could successfully counter greater hip moments, might protect against progression of knee OA. Given that the data from both lower extremities were pooled, our results would indicate that the lower hip moments were related to gait muscle control during stance that can load the contralateral limb abruptly as transition is made from single to double stance. This may be of consequence in the development of multiarticular OA as increased dynamic loading in the contralateral knee of subjects with established unilateral knee OA has been reported and suggested to favor the development and progression of OA in the contralateral knee.^{$12,22$}

Notably, most of our subjects were male. OA is predominant in women older than 55, but gender differences are not as strong for younger people.³⁵ Our subjects were mostly relatively younger and/or very active people trying to delay the need for joint replacement by using an ''unloading'' brace or scheduled for HTO. Very few of the subjects in our practice who agree to undergo HTO are women. Bracing also seems to be used by a greater number of men.

In summary, significant asymmetries across the lower extremity joints were seen on side-to-side comparison of hip and knee kinematics during stance for subjects with medial knee OA. The patients had adduction moments of the involved knee of similar levels as the uninvolved one did, likely accomplished by using a lateral sway of the trunk, as evident by a relatively more abducted hip and the much smaller hip adduction moments on the involved side. Subjects did not seem to use any effective dynamic means to compensate for less knee flexion during weight acceptance, and likely experience a resulting higher joint impact, which may impact OA progression in the involved knee.

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REFERENCES

- 1. Felson DT, Lawrence RC, Dieppe PA, et al. 2000. Osteoarthritis: new insights. Part 1: the disease and its risk factors. Ann Intern Med 133:635–646.
- 2. Teichtahl A, Wluka A, Cicuttini FM. 2003. Abnormal biomechanics: a precursor or result of knee osteoarthritis? Br J Sports Med 37:289–290.
- 3. Hayes CW, Jamadar DA, Welch GW, et al. 2005. Osteoarthritis of the knee: comparison of MR imaging findings with radiographic severity measurements and pain in middle-aged women. Radiology 237:998–1007.
- 4. Sharma L, Lou C, Felson DT, et al. 1999. Laxity in healthy and osteoarthritic knees. Arthritis Rheum 42:861–870.
- 5. van der Esch M, Steultjens M, Wieringa H, Dinant H, Dekker J. 2005. Structural joint changes, malalignment, and laxity in osteoarthritis of the knee. Scand J Rheumatol 34:298–301.
- 6. Childs JD, Sparto PJ, Fitzgerald GK, et al. 2004. Alterations in lower extremity movement and muscle activation patterns in individuals with knee osteoarthritis. Clin Biomech (Bristol, Avon) 19:44–49.
- 7. Deluzio KJ, Astephen JL. 2007. Biomechanical features of gait waveform data associated with knee osteoarthritis: an application of principal component analysis. Gait Posture 25: 86–93.
- 8. Baliunas AJ, Hurwitz DE, Ryals AB, et al. 2002. Increased knee joint loads during walking are present in subjects with knee osteoarthritis. Osteoarthritis Cartilage 10:573–579.
- 9. Hubley-Kozey CL, Deluzio KJ, Landry SC, et al. 2006. Neuromuscular alterations during walking in persons with moderate knee osteoarthritis. J Electromyogr Kinesiol 16: 365–378.
- 10. Winter DA, Eng P. 1995. Kinetics: our window into the goals and strategies of the central nervous system. Behav Brain Res $67.111 - 120$
- 11. Lewek MD, Rudolph KS, Snyder-Mackler L. 2004. Control of frontal plane knee laxity during gait in patients with medial compartment knee osteoarthritis. Osteoarthritis Cartilage 12: 745–751.
- 12. Shakoor N, Hurwitz DE, Block JA, et al. 2003. Asymmetric knee loading in advanced unilateral hip osteoarthritis. Arthritis Rheum 48:1556–1561.
- 13. Hurwitz DE, Sumner DR, Andriacchi TP, et al. 1998. Dynamic knee loads during gait predict proximal tibial bone distribution. J Biomech 31:423–430.
- 14. Prodromos CC, Andriacchi TP, Galante JO. 1985. A relationship between gait and clinical changes following high tibial osteotomy. J Bone Joint Surg Am 67:1188– 1194.
- 15. Sharma L, Hurwitz DE, Thonar EJ, et al. 1998. Knee adduction moment, serum hyaluronan level, and disease severity in medial tibiofemoral osteoarthritis. Arthritis Rheum 41:1233–1240.
- 16. Birmingham TB, Kramer JF, Kirkley A, et al. 2001. Association among neuromuscular and anatomic measures for patients with knee osteoarthritis. Arch Phys Med Rehabil 82:1115–1118.
- 17. Hodge WA, Fijan RS, Carlson KL, et al. 1986. Contact pressures in the human hip joint measured in vivo. Proc Natl Acad Sci USA 83:2879–2883.
- 18. Hurwitz DE, Sharma L, Andriacchi TP. 1999. Effect of knee pain on joint loading in patients with osteoarthritis. Curr Opin Rheumatol 11:422–426.
- 19. Kirkley A, Webster-Bogaert S, Litchfield R, et al. 1999. The effect of bracing on varus gonarthrosis. J Bone Joint Surg Am 81:539–548.
- 20. Chang A, Hayes K, Dunlop D, et al. 2005. Hip abduction moment and protection against medial tibiofemoral osteoarthritis progression. Arthritis Rheum 52:3515–3519.
- 21. Mundermann A, Dyrby CO, Andriacchi TP. 2005. Secondary gait changes in patients with medial compartment knee osteoarthritis: increased load at the ankle, knee, and hip during walking. Arthritis Rheum 52:2835–2844.
- 22. Shakoor N, Block JA, Shott S, et al. 2002. Nonrandom evolution of end-stage osteoarthritis of the lower limbs. Arthritis Rheum 46:3185–3189.
- 23. Mason RB, Horne JG. 1995. The posteroanterior 45 degrees flexion weight-bearing radiograph of the knee. J Arthroplasty 10:790–792.
- 24. Messieh SS, Fowler PJ, Munro T. 1990. Anteroposterior radiographs of the osteoarthritic knee. J Bone Joint Surg Br 72:639–640.
- 25. Piperno M, Hellio Le Graverand MP, Conrozier T, et al. 1998. Quantitative evaluation of joint space width in femorotibial osteoarthritis: comparison of three radiographic views. Osteoarthritis Cartilage 6:252–259.
- 26. Kellgren JH, Lawrence JS. 1957. Radiological assessment of osteo-arthrosis. Ann Rheum Dis 16:494–502.
- 27. Swanson KE, Stocks GW, Warren PD, et al. 2000. Does axial limb rotation affect the alignment measurements in deformed limbs? Clin Orthop Relat Res 371:246–252.
- 28. Moore TM, Meyers MH, Harvey JP Jr. 1976. Collateral ligament laxity of the knee. Long-term comparison between plateau fractures and normal. J Bone Joint Surg Am 58:594– 598.
- 29. Irrgang JJ, Snyder-Mackler L, Wainner RS, et al. 1998. Development of a patient-reported measure of function of the knee. J Bone Joint Surg Am 80:1132–1145.
- 30. Hurwitz DE, Ryals AB, Case JP, et al. 2002. The knee adduction moment during gait in subjects with knee osteoarthritis is more closely correlated with static alignment than radiographic disease severity, toe out angle and pain. J Orthop Res 20:101–107.
- 31. Hunt MA, Birmingham TB, Giffin JR, et al. 2006. Associations among knee adduction moment, frontal plane ground reaction force, and lever arm during walking in patients with knee osteoarthritis. J Biomech 39:2213–2220.
- 32. Perry J. 1992. Gait analysis, normal, pathological function. Thorofare: SLACK, Inc.
- 33. Mundermann A, Asay JL, Mundermann L, et al. 2008. Implications of increased medio-lateral trunk sway for ambulatory mechanics. J Biomech 41:165–170.
- 34. Kendall F, McCreary E, Provance P. 1993. Muscles, testing and function. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins.
- 35. Srikanth VK, Fryer JL, Zhai G, et al. 2005. A metaanalysis of sex differences prevalence, incidence and severity of osteoarthritis. Osteoarthritis Cartilage 13:769– 781.