Pelvic and Lower Limb Compensatory Actions of Subjects in an Early Stage of Hip Osteoarthritis

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Objective: To determine if compensatory actions take place at the pelvis and other joints of the affected lower limb in subjects who were in an early stage of hip osteoarthritis (OA).

Design: Nonrandomized, case-control study.

Setting: A gait laboratory.

Participants: Seventeen patients with OA of the hip (clinical group) matched with 17 healthy elderly subjects (nonclinical group).

Interventions: Video data obtained while subjects walked a 10-meter walkway twice and stepped across a forceplate.

Main Outcome Measures: Four phasic and temporal gait parameters (walking speed, stance phase relative duration, stride length, cadence) 10 pelvic (pelvic tilt, obliquity, rotation at push-off maximum range of motion for all 3) and hip (3 hip angles at push-off, maximum hip flexion) kinematic parameters, 3 hip moments, and twenty-seven 3-dimensional peak muscle powers (labeled by joint, peak power, plane) developed in the lower limb joints during the gait cycle.

Results: Subjects in the clinical group were characterized by a 12.4% slower walking speed. The pelvis was more upwardly tilted (2.5 times) at push-off in the clinical group than in the nonclinical group. Obliquity, measured in the frontal plane, revealed that the pelvis dropped more (2.4 times) on the unsupported limb of the clinical group at push-off. In the sagittal plane, subjects in the clinical group absorbed less energy in their second hip peak power for decelerating the thigh extension and generated less hip pull (third hip peak power) than the nonclinical group by 34% and 29%, respectively. In the sagittal plane, the clinical group had 57% lower second knee peak power to straighten the joint shortly after heel strike, and 43% less knee absorption (third peak power) at push-off. During the push-off phase, the clinical group developed more than twice their third peak knee power in the frontal plane and 5 times more their third peak knee power in the transversal plane than the peak knee power of the nonclinical group in an attempt to control knee adduction and to facilitate body-weight transfer by an internal rotation. At the end of the swing phase, the fourth peak power in the sagittal plane showed the absorption power required to decelerate the leg; it was reduced by 35% in the clinical group, representing a strategy to increase walking speed by lengthening the stride length.

Conclusions: Even at an early stage of hip OA, joint degeneration was compensated by an increase in pelvis motion and muscle power generation or absorption modifications in other lower limb joints.

Key Words: Gait; Kinematics; Leg; Osteoarthritis; Hip; Pelvis; Rehabilitation.

SEVERAL STUDIES INVOLVING SUBJECTS with osteoarthritis (OA) of the hip have dealt with temporal gait parameters,[1] lower limb kinematics,[2,3] ground reaction forces,[4] muscle moments,[5] or muscle powers.[6] These studies underlined specific aspects of advanced OA gait. It can be assumed that the observed locomotor perturbations caused by compensatory or adaptive mechanisms result from severe OA.

In previous studies, patients were often at an advanced stage of hip degeneration. Subjects were assessed before and after total hip replacement[5-9] or only after total hip replacement.[6,10,11] In some studies, the gait of subjects with OA of the hip was assisted by canes[12] or walking aides,[3] reducing the force acting on the affected joints. In these instances, subjects were severely affected with OA and were often scheduled for surgery within a month or 2. Subjects with severe OA constitute an ideal preoperative control group to study the results of total hip replacements. However, such subjects cannot be considered representative of the OA population because many do not undergo surgery. Furthermore, the consequence of severe OA on gait patterns is not expected to be similar at the initial and advanced stages of hip disease.

Crosbie and Vachalathiti[13] reported coordination between pelvis and hip during the gait cycle of able-bodied subjects. It can be assumed that perturbed hip kinematics caused by OA can affect the movement of the pelvis. Perturbed hip and pelvic motions can also influence the support and propulsion actions of the lower limb. Mechanical power, called *muscle power*, calculated at the joint, is the product of the net mechanical muscle moment by its corresponding joint angular velocity.[14,15] It has been recognized as a valuable gait descriptor because it combines both kinetic (moment) and kinematic (motion) information.[14] Muscle powers have been used to describe gait pattern in subjects without disability or functional limitation[16,17] and to analyze the gait of individuals with total hip replacement,[6] amputees,[18] or subjects fitted with an orthosis.[19]

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Muscle power will be considered in this study as a relevant indicator of joint compensation resulting from OA of the hip.

This study sought to determine if compensatory actions occurred at the pelvis and other joints of the affected lower limb in subjects who were in an early stage of OA of the hip. Gait analyses were performed to test if pelvic motions increased because of a reduced hip range of motion (ROM) during gait and if muscle power developed in the lower limb was altered to compensate the osteoarthritic hip kinematics.

METHODS

Seventeen subjects (9 women, 8 men; average age, 58.9 ± 7.1yr; average height, 1.67 ± 0.12m; average weight, 76.6 ± 14.7kg) with unilateral OA of the hip (8 right, 9 left limbs) formed the clinical group. The early stage of OA was defined by clinical examination, a Lequesne Index[20] score lower than 11, and a Kellgren and Lawrence[21] index score lower than 3. The Lequesne Index is representative of the functional level of the OA patient and is based on a 24-point scale, which includes pain level, walking distance, and ability to perform certain daily activities. The test validity was commonly accepted and variability was above 1 point. The average score on the Lequesne Index for the clinical group was 7.65 ± 2.22; none had a score above 11, a value used to determine the need for total hip replacement surgery. The severity of hip OA was determined by the Kellgren and Lawrence[21] radiographic index, a 5-point scale. The Kellgren and Lawrence index was lower than 3 for all subjects. Thus, all clinical subjects were functionally independent, ambulated without assistive devices, and were not immediate candidates for hip surgery. Additionally, subjects had no history of recent trauma or inflammatory disease.

Seventeen subjects (9 women, 8 men) formed the nonclinical group. Other than OA of the hip for the clinical group, the exclusion criteria for all subjects were musculoskeletal ailments, scoliosis, joint replacement, use of medication, a history of heart disease, neurologic or locomotor disorder, and recent surgery and surgery scheduled within 6 months of the gait evaluation. The protocol was reviewed and approved by the local institutional review board. Before experimentation and after being informed of the entire protocol, each patient signed a consent form.

Gait analyses were performed by 2 examiners (a kinesiologist, a physician) in the Human Movement Laboratory of the Neurophysiologic Exploration Department at the Regional University Center in Lille, France. The kinematics data were collected with a Vicon 360 system.[a] Two 50-Hz cameras were located at 3.5 meters from the center of a 10-meter long walkway to cover the area required for 1 complete gait cycle. A third camera was placed at the end of the walkway and was aligned along the axis of progression to obtain an anterior view of the subject. An AMT1[b] forceplate (250Hz) located in the middle of the 10-meter walkway was synchronized with the cameras of the Vicon system. Before data collection, the cameras were calibrated by using 20 markers located within a calibration volume of 2.8 by 1.0 wide and 1.8 meters high.

Before the gait analysis, 15 reflective markers were placed over anatomic landmarks of each subject according the Vicon Clinical Manager[a] protocol as outlined by Kadaba et al[22] and Davis et al.[23] A reflective marker was placed over the sacrum, and on the anterosuperior iliac, middle thigh, knee, middle leg, ankle, heel, and toe of each lower limb. Video data of each subject was then obtained while the subject was standing in an upright position to establish the neutral position of the joints and lower limb segments. Afterward, subjects were asked to walk along the walkway at their natural speed without being told about the presence of the forceplate. The starting point of walking was defined by the examiners so that each subject was able to take a step in the middle of the forceplate. The right limb was arbitrarily chosen for assessment in the nonclinical group subjects whereas the affected limb was chosen for the clinical group. Two trials were selected within the 5 acquired on the criteria of speed similarity like that recommended by Vardaxis et al,[17] totaling 68 gait trials (2 trails for 2 groups of 17 subjects).

The 3-dimensional coordinates of the marker system were calculated and filtered by using the Vicon system software. Walking speed, stance phase relative duration, stride length, and cadence were obtained from video and forceplate data. Additionally, 10 kinematic parameters, that is, pelvic tilt, obliquity and rotation at push-off, maximum ROM during stance for the earlier 3 parameters, 3 hip angles at push-off, and maximum hip flexion, were calculated.

The 3-dimensional joint reaction forces and net muscle moments were calculated by using the Vicon Clinical Manager software.[22,23] All kinematic and kinetic data were normalized with respect to 100% of the gait cycle duration, defined by 2 consecutive heel strikes of the right limb for the nonclinical group. The gait events (beginning, foot off, end) of each gait cycle were identified manually by the 2 examiners by using kinematic and forceplate data. Results for the clinical group were normalized with respect to the limb corresponding to the affected side.

The 3-dimensional muscle powers in each plane were estimated by the product of the net muscle moment developed at each joint of the lower limb by the angular velocity. When the muscle moments and the angular velocities have the same polarity, the power is positive and is considered to generate energy during a concentric muscle contraction. When the polarities are different, the power is negative and it is assumed that energy is being absorbed in an eccentric muscle contraction.[15] The powers were normalized with respect to the individual body mass and labeled according to the proposition of Eng and Winter.[16] The first letter corresponded to the joint (Hip, Knee, Ankle). The number was related to the order of the peak powers (2–4, according to the joint and plane) and the second letter referred to the planes (Sagittal, Frontal, Transverse). For example, K3F corresponds to the third peak muscle power of the knee developed in the frontal plane. Each peak power was identified manually by the 2 examiners.

Data analysis for the clinical and nonclinical groups were performed with Student's *t* tests for the 4 phasic and temporal gait parameters, the 10 kinematic parameters (6 for the pelvis, 4 for the hip), the 3 hip moments, and the 27 peak power values. Parameters having a p less than .05 were considered to be statistically different. For the significantly different parameters, effect size[24] was given (effect size^2 = variance of the difference between the 2 groups/variance of the pooled data).

RESULTS

The 17 subjects who formed the nonclinical group were slightly older than the clinical group (63.6 ± 5.2yr), but their average height (1.69 ± .06m) and weight (71.1 ± 14.0kg) were similar. All subjects were in good health and did not display a limb-length discrepancy larger than 1cm.

Table 1 presents the temporal and phasic gait parameters for clinical and nonclinical groups as well as statistical differences and effect size for p less than .05. Subjects in the clinical group were characterized by a 12.4% slower walking speed resulting from a 7.0% shorter stride length and a 5.2% reduced cadence, though no significant differences were found in the relative

Table 1: Temporal and Phasic Gait Parameters

	Clinical Group*	Nonclinical Group*	Effect Size
Speed (m/s)	1.05 ± .12*	1.18 ± .12*	1.09
Stance (% of gait cycle)	63.37 ± 7.56	61.68 ± 2.01	—
Stride length (m)	1.16 ± .12*	1.24 ± .10*	.70
Cadence (step/min)	108.62 ± 10.03*	114.27 ± 7.31*	.65

NOTE. Values are mean ± standard deviation (SD).
* $p < .05$.

duration of the stance phase. The effect size was very high for all parameters, which were significantly different.

Pelvic angles are presented in figure 1 for the clinical and nonclinical groups. For the frontal plane, the curves for both groups were similar, but in the transverse and even more in the sagittal plane the shape of the curves differed. The selected hip and pelvic parameters are those widely used in gait evaluation[3,5,12,13] and are easy to visualize. Parameters that combine kinematic and kinetic information appear to be more sensitive to gait modifications,[16] as in initial unilateral hip OA.

Of the 6 kinematic parameters used to describe pelvic motion, 4 were statistically different and are shown in table 2. At push-off, the pelvis, viewed in the sagittal plane, was tilted upwardly in both groups. However, for the clinical group, the upward tilt of the pelvis was 2.5 times more (11.72 ± 5.37 vs 4.61 ± 5.36) than that of the nonclinical subjects. An upwardly tilted pelvis is indicative of a decrease lumbar lordosis or anterior trunk flexion.[25,26] Obliquity measured in the frontal plane revealed that the pelvis in the clinical group dropped more (2.4 times: −3.09 ± 1.23 vs −1.28 ± 1.78) on the unsupported limb at push-off. ROM of the pelvic tilt and

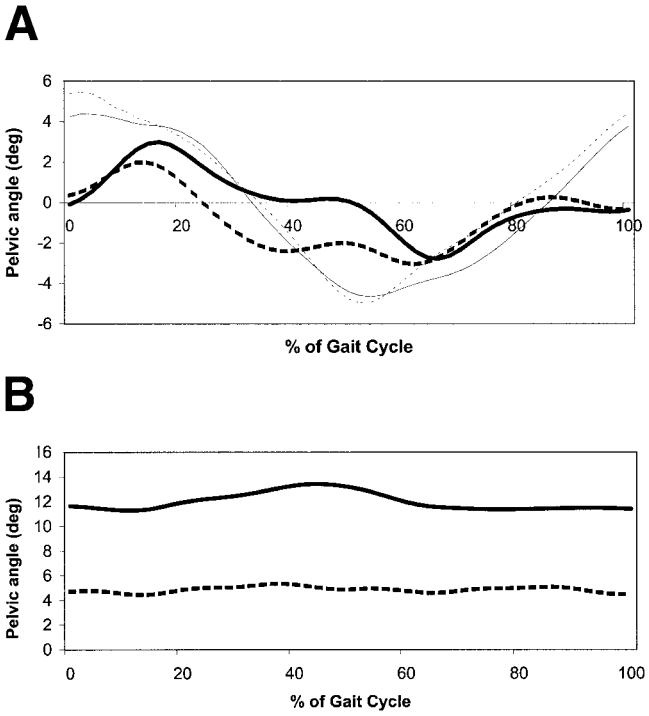

Fig 1. Pelvic angle curves for clinical group (dashed line) and nonclinical group (solid line) in (A) for obliquity (bold line) and rotation (thin line) and in (B) for pelvic tilt.

Table 2: Values of Hip Motion, Pelvis Motion, Hip Moment, and Effect Size

Motion (deg)	Clinical Group	Nonclinical Group	Effect Size
Hip			
Maximum extension	1.77 ± 10.3*	−9.31 ± 6.27*	1.34
Flexion at push-off	9.44 ± 9.13*	−2.45 ± 8.77*	1.33
Abduction at push-off	−.34 ± 7.10*	3.02 ± 4.10*	.60
Rotation at push-off	6.89 ± 13.81*	−7.31 ± 13.70*	1.03
Moment flexion	−0.8 ± .37*	−1.22 ± .31*	1.22
Moment abduction	.73 ± .42	.75 ± .21	—
Moment internal rotation	.01 ± .08	.02 ± .05	—
Pelvis			
Tilt at push-off	11.72 ± 5.37*	4.61 ± 5.36*	1.37
Obliquity at push-off	−3.09 ± 1.23*	−1.28 ± 1.78*	1.20
Rotation at push-off	−3.68 ± 2.89	−3.51 ± 3.81	—
ROM of tilt	2.71 ± 2.75*	1.28 ± 1.97*	.61
ROM of obliquity	6.14 ± 1.66*	5.78 ± 1.68*	—
ROM of rotation	11.28 ± 3.86	11.18 ± 4.39	—

NOTE. Values are meant ± SD.
* $p < .05$.

obliquity were also much higher in the clinical group during stance phase. Thus, not only was the pelvic position more pronounced in the clinical group, but also there was evidence of greater mobility. Three of the 4 effect sizes of pelvic parameters were above .60.

Hip motion in the clinical and nonclinical groups was statistically different in all ranges assessed (table 2). At push-off, subjects of the clinical group had about 4 times more hip flexion (9.44 ± 9.13 vs −2.45 ± 8.77), 2 times greater internal rotation (6.89 ± 13.81 vs −7.31 ± 13.70), and about 11 times less abduction (−.34 ± 7.10 vs 3.02 ± 4.10). In the clinical group, the hip was in extension, whereas in the nonclinical group it was in flexion. All effect sizes was above .60.

The muscle power curves of the clinical group generally followed those in the nonclinical group (fig 2). There were some slight discrepancies between the mean muscle power curves and the mean peak powers (table 3). This stems from the fact that the individual peak values do not necessarily occur at the same time in each individual trial and for each individual, but rather within 1% to 4% time range.

Nine of the 27 peak powers taken from the individual trials and not from the mean curve were different. Five of those peak muscle powers were in the sagittal plane, 3 in the frontal plane, and 1 in the transverse plane (table 3). Thus, the muscle powers in the clinical group were modified in the plane of progression (sagittal) as well as in the other planes. The differences in peak muscle powers occurred throughout the gait cycle, though 5 of them were related to the push-off period.

The clinical group absorbed less H2S mechanical energy in decelerating the thigh extension and generated less hip pull (H3S) than the nonclinical group by 34% and 29%, respectively. The lack of hip action at push-off was attributed to a 52% reduction in hip flexion moment.

At the knee, 5 statistically different peak muscle powers were found. The clinical group developed 57% less K2S to straighten the knee shortly after heel strike, and 43% less knee absorption (K3S), facilitating push-off by the hip and ankle. While still in the push-off phase, the clinical group developed more than triple the K3F peak power (−.420 ± .421 vs −.124 ± .195) and 5 times more K3T (.050 ± .083 vs .010 ± .049)

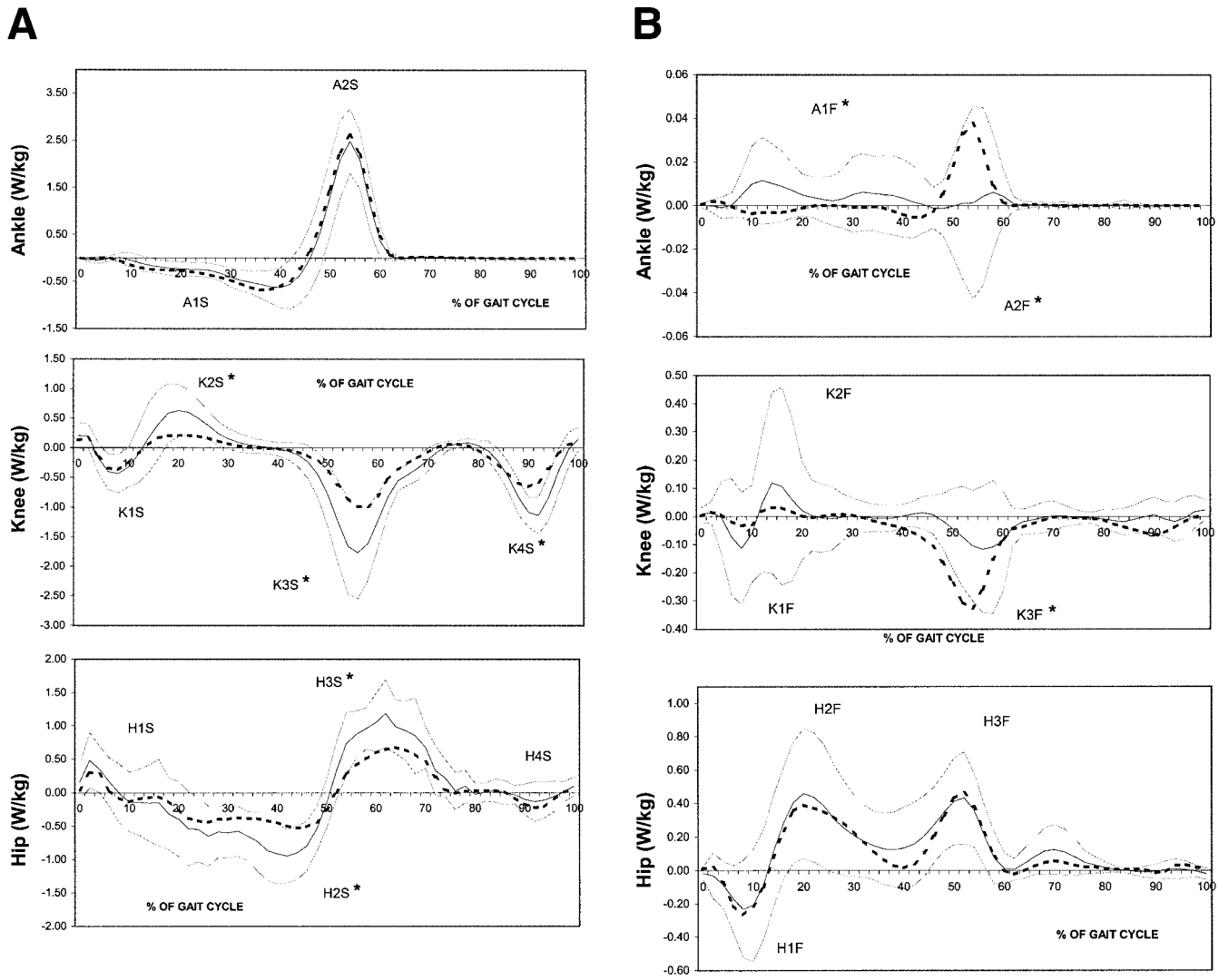

Fig 2. Ankle, knee, and hip muscle power curves developed at the hip, knee, and ankle in (A) the sagittal and (B) the frontal and (C) the transverse planes. For the nonclinical group, muscle power is presented as a dark line with its corresponding standard deviation, the light line. The dashed line represents the muscle power values for the clinical group. Peak muscle powers were labeled according to the proposition of Eng and Winter.[16] *$p < .05$.

than that of the control group. This was considered to be an attempt to control the knee adduction caused by the hip and to facilitate body-weight transfer by an internal rotation. At the end of the swing phase, the K4S peak absorption power was reduced by 35%, representing less leg deceleration. This suggests that these subjects developed a strategy to increase walking speed by lengthening stride length.

Though the ankle push-off peak power (A2S) was 10% higher in the clinical group, this difference was not statistically significant. However, in the clinical group-ankle actions were enhanced in the frontal plane. There was almost 6 times more A1F ($-.023 \pm .039$ vs $-.004 \pm .034$) to control body-weight transfer from the contralateral limb to the affected limb whereas the A2F developed 7.9 times ($.079 \pm .161$ vs $.010 \pm .038$) more lateral push-off. The effect sizes were generally well above .60 for all parameters and half of them were over 1. The most important effect size values were observed in the hip motion and in the knee sagittal muscle powers.

DISCUSSION

The objective of this study was to determine if subjects in early stage of unilateral OA of the hip developed compensatory actions at the pelvis and other joints of the affected lower limb. Hip motions were modified in the clinical group, particularly in the sagittal and frontal planes. A decrease in hip extension was observed in the clinical group at push-off with an increase in hip flexion during the stance phase. These kinematic modifications in subjects with OA were also observed by Hurwitz et al[5] in a group of 19 patients who underwent total hip replacement surgery within the 25 days of gait evaluation. In the early stage of the disease, as in severe unilateral hip OA, hip motion modifications were similar with different amplitudes. These modifications were reduced, but perceptible in early stage of OA.

The reduction in hip extension was associated with kinematic changes occurring at the pelvis. As reported by Thurston,[26] a greater pelvic tilt was observed at push-off and an

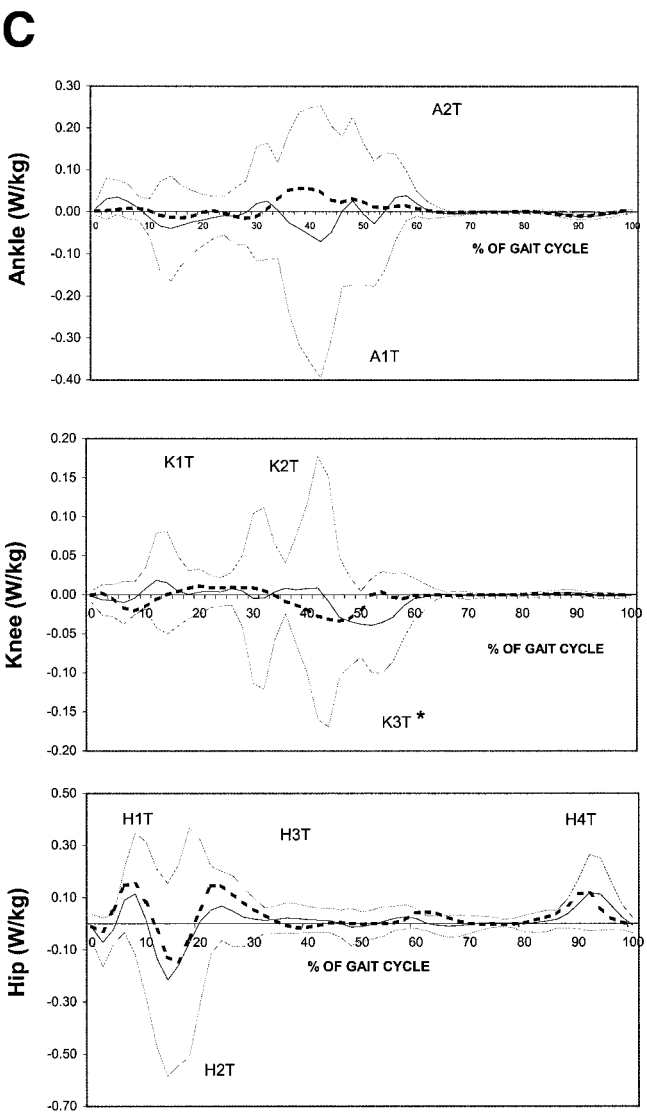

Fig 2. (*Continued*)

increase in ROM of the pelvis occurred during stance phase. Without visual gait alteration consecutive with severe OA of the hip, the clinical group of this study presented similar modification of hip and pelvis motion at push-off. These pelvic actions may be interpreted as compensation mechanisms for the hip limitations. The pelvic adaptations enabled the clinical group to maintain an effective extension of their lower limb at push-off and to preserve an overall mobility in the sagittal plane, minimizing the shorter stride length resulting from reduced hip ROM.[27]

An increase in the pelvic obliquity was also observed in the frontal plane as reported by Thurston.[26] Pelvic obliquity differed in the clinical group compared with the nonclinical group in the early stage and advanced stage of OA. However, these differences were less important in the early stage of OA. Wadsworth et al[3] and Murray et al[12] consider pelvic compensatory mechanisms as strategies to reduce hip pain in OA subjects. The frontal pelvic obliquity on the weight-bearing limb corresponding to a Trendelenburg sign is related to a painful hip.[28] According to Pauwels,[29] subjects with a painful hip caused by OA will reduce the load on the hip by decreasing the gluteus medius activity. Increasing pelvic tilt and inclining the trunk on the side of the supporting limb allows OA subjects to shorten the moment arm between hip and center of mass of the upper body. This gait adaptation corresponds to the lumbar movements of subjects with more severe OA of the hip as reported by Thurston.[26] Even at an early stage of OA, subjects in the clinical group developed a gait strategy to minimize the load on their painful hip.

In subjects with OA of the hip, pelvic mobility during gait was increased in the sagittal plane probably by a compensatory mechanism, and in the frontal plane by adopting an antalgic mechanism.[28] These pelvic adaptations should affect the natural mobility of the lumbar spine because of their kinematic interactions,[11,25,28,30] though Thurston[26] did not report any changes in spinal mobility. Increasing pelvic motion without lower spine involvement could increase the load acting on the lumbar spine and lead to lumbar dysfunction. The interaction between the modified pelvic kinematics and the lumbar spine is still not well understood, but it could explain in part why pain or lumbar spine arthrosis are often noticed in subjects with OA of the hip.[26] We hypothesize that a limited ROM at the hip may be partially compensated by an increase in pelvic motion to reduce gait perturbations and to minimize walking speed reduction.

Walking speed of the nonclinical group was slightly faster than the speeds reported in the literature.[1,5,6] Both a short stride

Table 3: Values of the 27 Muscle Powers (W/kg) and Effect Size

Peak Muscle Powers	Clinical Group	Nonclinical Group	Effect Size
Hip			
H1S	.441 ± .255	.578 ± .321	—
H2S	−.764 ± .643*	−1.166 ± .443*	.74
H3S	1.000 ± .509*	1.409 ± .429*	.87
H4S	−.352 ± .234	−.329 ± .280	—
H1F	−.277 ± .184	−.221 ± .321	—
H2F	.606 ± .359	.552 ± .338	—
H3F	.413 ± .231	.427 ± .292	—
H1T	.204 ± .271	.188 ± .319	—
H2T	−.241 ± .311	−.349 ± .423	—
H3T	.213 ± .399	.156 ± .227	—
H4T	.162 ± .134	.172 ± .129	—
Knee			
K1S	−.418 ± .347	−.521 ± .414	—
K2S	.336 ± .248*	.774 ± .433*	1.28
K3S	−1.134 ± .796*	−1.984 ± .816*	1.05
K4S	−.779 ± .326*	−1.202 ± .249*	1.47
K1F	−.166 ± .127	−.136 ± .143	—
K2F	.153 ± .121	.192 ± .345	—
K3F	−.420 ± .421*	−.124 ± .195*	.96
K1T	.029 ± .032	.024 ± .038	—
K2T	−.069 ± .060	−.060 ± .073	—
K3T	.050 ± .083*	.010 ± .049*	.60
Ankle			
A1S	−.836 ± .272	−.881 ± .369	—
A2S	2.931 ± .827	2.664 ± .638	—
A1F	−.023 ± .039*	−.004 ± .034*	.50
A2F	.079 ± .161*	.010 ± .038*	.69
A1T	−.017 ± .136	−.005 ± .184	—
A2T	.080 ± .247	−.009 ± .200	—

NOTE. Values are meant ± SD.
* $p < .05$.

length and a decreased cadence characterized slow walking speed in the clinical group. Similar observation was made in OA subjects with[1-3,5,7,9] or without hip surgery.[1,6,7,9] According to Crowinshield et al,[31] a less dynamic gait lowers the resultant force acting on the hip and keeps this force in the vertical direction.[32] The reduced cadence diminishes the frequency of loading, though more steps would be required to reach a given destination because of a reduction in stride length. This strategy is different from previously reported gait studies of subjects over 63 years of age, in whom the stride length[33,34] or the cadence was reduced.[35,36]

The muscle power developed in the joints of the lower limb was also examined in this study. To our knowledge, only Loizeau et al[6] reported muscle power in a limited sample of OA subjects of whom had undergone hip surgery. The subjects in our clinical group were unable to extend their hip and had a reduced mobility and ROM at push-off. This resulted in a decreased hip flexion moment and less muscle power generation (H3S) to pull the thigh at push-off. There was a reduction in the peak H2S absorption power associated with the control of the backward rotation of the thigh under the hip's flexors actions.[16] A reduced H2S could be explained in part by a more upwardly titled pelvis in the clinical group.

Hip muscle power modifications may correspond to an antalgic gait pattern[28] that limits the mechanical constraint on the osteoarthritic joint. An antalgic position is characterized by external rotation of the lower limb, flexion of the knee, an upwardly tilted pelvis, and a Trendelenburg sign, leading to an asymmetric gait.[28] Because our clinical group was in an early state of the pathology, these signs were slightly perceptible and were observed only in 2 subjects. Muscle power may be more sensitive for identified gait modification than visual observation. Limitation in internal rotation after abduction, as well as flexion and adduction with decreasing of hip strength,[2,12,37,38] were considered early signs of the OA of the hip.

Knee and ankle power were also modified in the clinical group. The K2S peak generation is responsible for extending the knee after heel strike. Low K2S values are not characteristic of elderly subjects.[33,34] In our clinical group, a low K2S value may be related to a compensatory action for lack of hip extension. This allowed the OA subjects to maintain the pelvis in its upwardly tilted position or to maintain their lower limb alignment.

At the knee, there was a greater power knee absorption (K3F) in the frontal plane and slightly greater power generation (K3T) in the transverse plane. These could be associated with hip protection mechanisms designed to limit hip power generation and absorption and to control thigh abduction (K3F) to maintain lower limb alignment. K3F and K3T presented no differences between older elderly subjects compared with younger subjects.[39] These modifications observed in clinical group must be only consecutive of hip arthrosis.

The increased action of the ankle frontal power absorption (A1F) was not associated with advanced age,[33] but with controling the body-weight transfer onto the affected limb. This strategy may counteract the trunk's lateral sway and reduce the load on the hip. There was less knee power absorption by the clinical group to facilitate the combined action of an ankle push-off (A2S) with a reduced hip pull (H3S).[39] The A2S peak generation was reported to be lower in elderly subjects.[33,39] This reduction was lower in OA subjects and must be in relation with A1S. The slight reduction of power absorption (A1S) and an increase of power generation (A2S) were not statistically significant, but they appear to reduce hip load and to minimize gait perturbations. Indeed, the reduction of absorption and the increase of power generation allowed maintaining walking speed by more pronounced propulsion action of the ankle. Still in push-off phase, the ankle frontal power generation (A2F) propelled the foot medially to compensate for the lack of hip pull and to help with body-weight transfer.

CONCLUSION

Even at an early stage of OA and without visual gait alteration, hip degeneration was compensated for in part by the pelvis and other joints in the lower limb. Reduced ROM in the hip leads to an increased pelvic motion in both sagittal and frontal planes. Perturbed muscle power reflected a reduced propulsion, compensated for by some underlying adaptations. Among these, the knee and ankle attempted to maintain limb alignment that resulted in perceptible gait perturbations mainly at push-off.

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