RESEARCH ARTICLE

Hip joint kinematics and segment coordination variability according to pain and structural disease severity in hip osteoarthritis

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Abstract

This study aimed to evaluate hip joint kinematic variability and segment coordination variability during walking according to pain and radiographic disease severity in people with hip osteoarthritis. Fifty-five participants with hip osteoarthritis had pain severity assessed during walking using an item on the Western Ontario and McMasters Universities Osteoarthritis Index (no pain = 10; mild pain = 28; moderate pain = 17). Radiographic disease severity was graded by Kellgren and Lawrence scale (KL2 = 29; KL3 = 21; KL4 = 5). Hip kinematics variability was estimated as the curve coefficient of variation. Vector coding was used to calculate coordination variability for select joint couplings. One-way analysis of variances with planned adjusted post hoc comparisons were used to compare hip kinematics variability and coordination variability of select segment couplings (pelvis sagittal vs thigh sagittal; pelvis frontal vs thigh frontal; pelvis transverse vs thigh transverse; thigh sagittal vs shank sagittal; thigh frontal vs shank sagittal; thigh transverse vs shank sagittal) according to pain and radiographic disease severity. No main effect of pain severity was observed for sagittal or transverse plane hip kinematic variability ($P \geq .266$), and although there was a main effect for frontal plane hip kinematic variability ($P = .035$), there were no significant differences when comparing between levels of pain severity ($P > .006$). There was no main effect of radiographic disease severity on hip kinematic variability in the sagittal ($P = .539$) or frontal ($P = .307$) plane. No significant differences in coordination of variability of segment couplings were observed (all $P \geq .229$). Movement variability as assessed in this study did not differ according to pain severity during walking or radiographic disease severity.

KEYWORDS
biomechanics, gait variability, hip osteoarthritis, hip pain, imaging, walking

1 | INTRODUCTION

Hip osteoarthritis (OA) is a chronic disease and a major public health problem.[1] There is no known cure for hip OA and clinical guidelines recommend that total hip arthroplasty is reserved for end-stage disease.[2,3] Rates of total hip arthroplasty are spiraling upwards in many countries,[4] highlighting the urgent need to improve nonsurgical treatment options. Pain with activity, and to a lesser extent, structural disease severity are drivers in determining whether a patient with hip OA undergoes a total hip replacement.[5] Although evidence

implicates abnormal movement variability in various musculoskeletal conditions,[6] little is known about measures of movement variability in people with hip OA. Understanding whether movement variability during walking differs according to pain severity and structural disease severity may help inform treatment strategies.

Movement variability can be assessed through various techniques and there is no consensus on the most appropriate approach.[6] Hip joint kinematic variability and segment coordination variability assess different aspects of movement variability and are potentially relevant in the context of hip OA. Hip joint kinematic variability can be considered an output of gait (ie, end-point variability) isolated to the affected joint. It is characterized by the fluctuation in kinematic values during gait, quantified by a coefficient of variation over the gait cycle (Figure 1). Studies on hip OA report limited hip range of motion,[7–10] limited hip extension,[7,10,11] as well as alterations in the frontal and transverse[7,11] plane during walking. Thus, despite the lack of research on movement variability in hip OA, it is reasonable to consider that hip joint kinematic variability alterations also exist. From a traditional view of end-point variability,[12,13] people with more severe hip OA could be expected to have greater hip joint kinematic variability to avoid concentrating repetitive loads on the structurally compromised, painful joint. End-point variability assessed as stride-time variability, reduced following total hip arthroplasty, and correlated with reduced pain.[14] This indirectly supports the influence of hip OA severity on hip joint kinematic variability.

Segment coordination variability extends beyond the osteoarthritic joint (ie, hip joint kinematic variability) and is considered a marker of motor system health or adaptability.[12,15] Segment coordination variability quantifies the variety of segment movement pattern by assessing the interaction one segment has with another[12] (Figure 2). Variability of the segment coordination patterns which contribute to joint kinematics may differ according to health status.[15] Segment coordination variability can discriminate between those with and without musculoskeletal injury, such that coordination variability between selected segments is reduced in people with a history of iliotibial band syndrome[16] and patellofemoral pain.[17] Higher segment coordination variability may indicate poorly controlled motion while lower segment coordination variability may indicate motion that is overly constrained.[12] Hip pain and structural hip pathology are likely to impose greater constraints on the motor system and lower segment coordination variability.

The purpose of this exploratory cross-sectional study was to test the hypotheses that (a) greater hip joint kinematic variability and (b) lower segment coordination variability (pelvis-thigh and thigh-shank couplings) would be associated with greater pain and radiographic disease severity compared with less severe pain and radiographic disease in people with unilateral symptomatic hip OA.

2 | METHODS

2.1 | Participants

Thirty-four participants from a cross-sectional study[18] and baseline data from 21 participants enrolled in a clinical trial[19] were available for
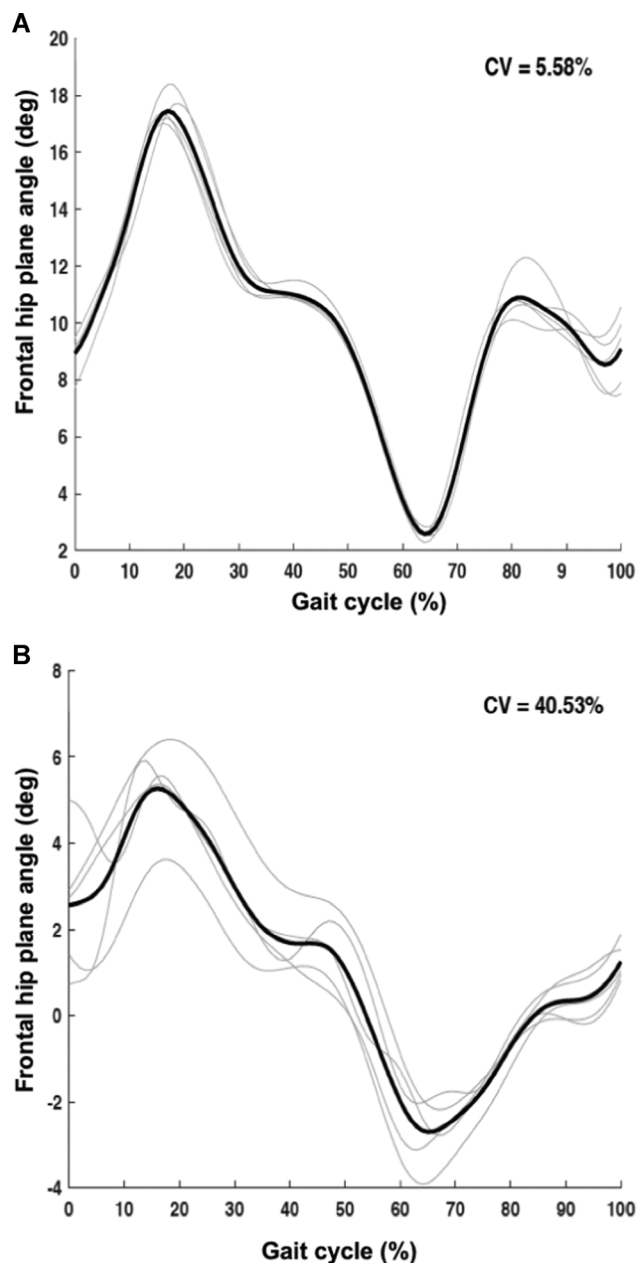

FIGURE 1 Representation of (a) low and (b) high curve CV for hip joint angle in the frontal plane. Single trials are in gray with the mean of the trials in black. CV, coefficient of variation

analysis. Participants were recruited from the community via advertisements in newspapers and on radio. Ethical approval was obtained from the University of Melbourne Human Research Ethics Committee and all participants provided their written informed consent.

Eligible participants had (a) hip OA according to the American College of Rheumatology classification criteria of pain and radiographic changes,[20] femoral or acetabular osteophytes along with joint space narrowing and Kellgren-Lawrence (KL)[21] grade ≥ 2 on a standing X-ray; and (b) hip or groin pain on most days of the past month. Exclusion criteria common to both studies were: (a) presence of neurologic, cardiac or other medical conditions that would compromise lower limb function;
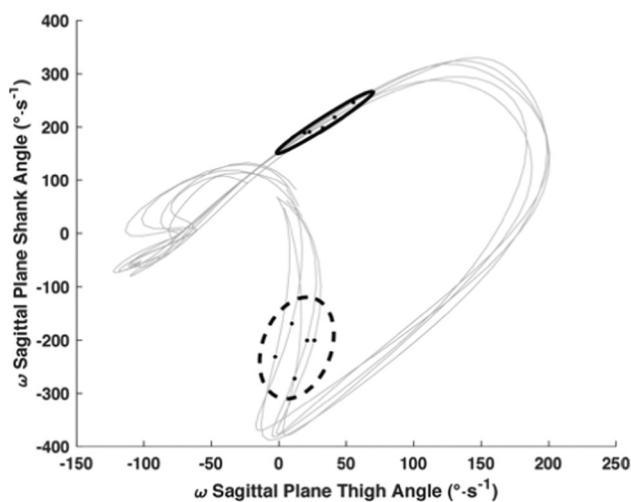
FIGURE 2 Sagittal plane hip vs knee angular velocity with fitted ellipses and illustration for high (dashed line) and low (solid line) points of variability across the gait cycle

(b) back pain or other joint pain greater than hip pain; (c) lower extremity joint replacement; (d) systemic arthritic conditions such as rheumatoid arthritis; (e) additional previous pathology such as fracture; (f) inability to walk unaided; and (g) inadequate ability to understand English. The clinical trial[19] had the following additional inclusion criteria: (a) 50 years or older; (b) average pain intensity in the past week of 40 or higher on a visual analogue scale (0-100 mm); and (c) at least moderate difficulty with daily activities. Additional exclusion criteria for the clinical trial were: (a) hip surgery within the past 6 months; (b) planned lower limb surgery; (c) physiotherapy, chiropractic treatment or prescribed exercises for the hip, lumbar spine or both in the past 6 months; (d) walking continuously for more than 30 minutes daily and regular structured exercise more than once weekly; (e) uncontrolled hypertension, or morbid obesity (body mass index > 36 kg/m^2); (f) unable to comply with study protocol; and (g) current or within the past 3 months oral or intra-articular corticosteroid use.

2.2 | Pain

Pain was assessed using the 5-item Western Ontario and McMasters Universities Osteoarthritis Index (WOMAC) subscale with scores ranging from 0 to 20.[22] Higher scores indicates greater pain. Pain specific to walking was assessed using an item ("walking on a flat surface") within the pain subscale. Participants were classified according to pain severity during walking: "no pain" (score = 0), "mild pain" (score = 1), "moderate pain" (score = 2), "severe pain" (score = 3), and "extreme pain" (score = 4).

2.3 | Radiographic disease severity

A standard anteroposterior pelvic X-ray was acquired in a standing position with 15 degrees internal foot rotation,[20] unless

participants provided their own films from weight-bearing X-ray within the previous 12 months. The KL grading system[21] was used to determine radiographic disease severity. In the current study, KL grade ≥ 2 was used to determine eligibility, thus participants were graded as either "KL2" (definitive osteophytes with possible narrowing of joint space); "KL3" (moderate multiple osteophytes, definite narrowing of joint space and some sclerosis and possible deformity of bone ends); or "KL4" (large osteophytes, marked narrowing of joint space, severe sclerosis, and definite deformity of bone ends).

2.4 | Gait analysis

Reflective markers were placed atop landmarks for each participant according to the standard plug-in-gait configuration (Vicon, Oxford, UK). Medial knee and ankle markers were applied for the static calibration to assist in determining knee and ankle joint centres. Kinematic data were acquired using a 12 MX-camera motion capture system (Vicon, Oxford, UK). Ground reaction force data were recorded using AMTI force plates. Kinematic and force platform data were sampled at 120 and 1200 Hz, respectively. Marker trajectory data were filtered using a Woltring quintic spline filter (mean square error 15 mm^2). Hip angles were calculated throughout the gait cycle. Pelvic angles were determined using a rotation-obliquity-tilt Cardan angle sequence. Participants were required to complete five walking trials at a self-selected walking speed, and walking speed was recorded for the five trials and averaged.

A custom script written in MATLAB version 2017a (Mathworks, Natick, MA) was used to perform the variability analysis. Hip kinematic variability was time-normalized and assessed by calculating the curve coefficient of variation[23,24] for sagittal, frontal and transverse plane kinematics across trials (ie, gait cycles). The curve coefficient of variation is expressed as a percentage and represents the root mean square of standard deviation of hip joint angles over the collected gait cycles, divided by the mean of the hip joint angles collected over gait cycles.[24] Curve coefficient of variation values range between 0 and 1 (ie, 0%-100%) and values closer to 1 indicate higher variability. Figure 1a,b illustrates an example of high and low curve coefficient of variation of the hip joint angle in the frontal plane, respectively.

Coordination variability between selected segments was assessed using a modified version of vector coding.[25] The angular velocities of two segments for the select segment couplings were plotted across five strides relative to each other on the XY plot. An ellipse was fitted to these points using the equations previously described,[26] with the size scaling adjusted according to the χ^2 value.[27] The area of the ellipse at each data point represented a bivariate measure of coordination variability, whereby a larger ellipse area indicated greater coordination variability. Ellipse area was calculated at each point across time-normalized (ie, 0%-100% of the gait cycle) gait data. The mean coordination variability

across the gait cycle was estimated from this curve and calculated for each of the five trials and averaged. Figure 2 provides an example of sagittal plane thigh angular velocity vs shank angular velocity, with ellipses fitted illustrating points of high (dashed line) and low (solid line) variability over the gait cycle. The modified vector coding using angular velocity was used in the current study based on recent data suggesting that vector coding approaches using angular velocity data are less prone to statistical artefact.[25] However, interpretation from vector coding using joint angular velocity or joint angles are comparable.[28] Coordination variability was calculated for the following segment couplings: (a) pelvis sagittal vs thigh sagittal; (b) pelvis frontal vs thigh frontal; (c) pelvis transverse vs thigh transverse; (d) thigh sagittal vs shank sagittal; (e) thigh frontal vs shank sagittal; (f) thigh transverse vs shank sagittal. These couplings were selected based on previous variability studies in people with hip pathology[29] and alterations in pelvis kinematics between people with hip OA compared with controls.[11]

2.5 | Other descriptive measures

Physical function was assessed using the 17-item WOMAC index physical function subscale. The physical function scale ranges from 0 indicating no difficulty to 68 indicating extreme difficulty.[22] Peak isometric hip abductor strength (Nm/kg) was assessed according to a previously described technique.[30]

2.6 | Statistical analysis

Stata version 16.0 (Statacorp, College Station, TX) was used to determine differences in participant descriptive characteristics and significance was set at $P < .05$. For pain and radiographic disease severity, one-way analysis of variance and Pearson χ^2 tests were used to compare participant characteristics for continuous and categorical data, respectively. In the event of statistical significance, a pairwise comparison of means test was used to determine which level of pain or radiographic disease severity differed. Two one-way analysis of variance performed in MATLAB version 2017a (Mathworks) were used to compare coordination variability hip kinematic curve coefficient of variation and according to pain and radiographic disease severity. A type 1 family-wise error rate (alpha) of 0.05 was used for post hoc analyses when a main effect of pain severity or radiographic disease severity was observed by calculating a Sidak-corrected threshold determined by the number of comparisons (ie, 10 variability measures), resulting in an alpha of 0.005.

3 | RESULTS

Participant characteristics and walking speed according to pain severity and structural disease severity are presented in Table 1. Age, height, body mass, body mass index, symptom duration, walking speed, and hip abductor strength did not differ across levels of pain severity and structural disease severity. The proportion of females and presence of bilateral radiographic disease differed according to

TABLE 1 Descriptive characteristics

		Pain severity during walking			KL grade		
	Total group, n = 55	No pain, n = 10	Mild pain, n = 28	Moderate pain, n = 17	KL2, n = 29	KL3, n = 21	KL4, n = 5
Age, y	60.8 ± 7.8	61.2 ± 7.1	61.1 ± 7.8	60.3 ± 8.5	60.7 ± 7.9	61.1 ± 8.1	60.6 ± 7.1
Female, n (%)	32 (58)	6 (60)	18 (64)	8 (47)	22 (76)	9 (43)	1 (20)***
Height, m	1.67 ± 0.10	1.66 ± 0.11	1.67 ± 0.10	1.68 ± 0.1	1.65 ± 0.09	1.69 ± 0.10	1.74 ± 0.07
Weight, kg	77.2 ± 13.8	75.0 ± 14.3	76.4 ± 14.1	79.9 ± 13.5	72.1 ± 12.9	82.0 ± 13.1***	86.8 ± 11.1***
Body mass index, kg/m²	27.58 ± 3.87	27.24 ± 4.56	27.28 ± 4.20	28.26 ± 2.91	26.55 ± 3.98	28.70 ± 3.65	28.85 ± 2.93
Symptom duration, y	4.87 ± 3.80	6.25 ± 4.08	4.62 ± 4.11	4.48 ± 3.06	4.7 ± 3.3	5.1 ± 4.8	4.9 ± 1.6
Bilateral radiographic disease, yes (%)	25 (45)	4 (40)	10 (36)	11 (65)	8 (28)	13 (62)	4 (80)***
WOMAC pain, (0-20)[a]	6.3 ± 2.6	2.9 ± 2.0	6.0 ± 1.8*	8.6 ± 1.5*,**	5.9 ± 2.6	6.5 ± 2.8	7.2 ± 1.3
WOMAC function, (0-68)[a]	22.9 ± 11.3	12.2 ± 7.3	21.3 ± 10.4*	31.9 ± 7.5*,**	20.2 ± 11.8	25.2 ± 10.4	28.6 ± 9.7
Walking speed, m/s	1.23 ± 0.15	1.19 ± 0.14	1.25 ± 0.16	1.22 ± 0.13	1.21 ± 0.15	1.27 ± 0.12	1.14 ± 0.20
Hip abductor strength (Nm/kg)	1.23 ± 0.37	1.29 ± 0.49	1.26 ± 0.35	1.15 ± 0.34	1.29 ± 0.37	1.17 ± 0.40	1.19 ± 0.26

Abbreviations: KL: Kellgren and Lawrence grading system; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index.
[a]Higher values indicate greater severity.
*Significantly different to no pain ($P < .05$).
**Significantly different to mild pain ($P < .05$).
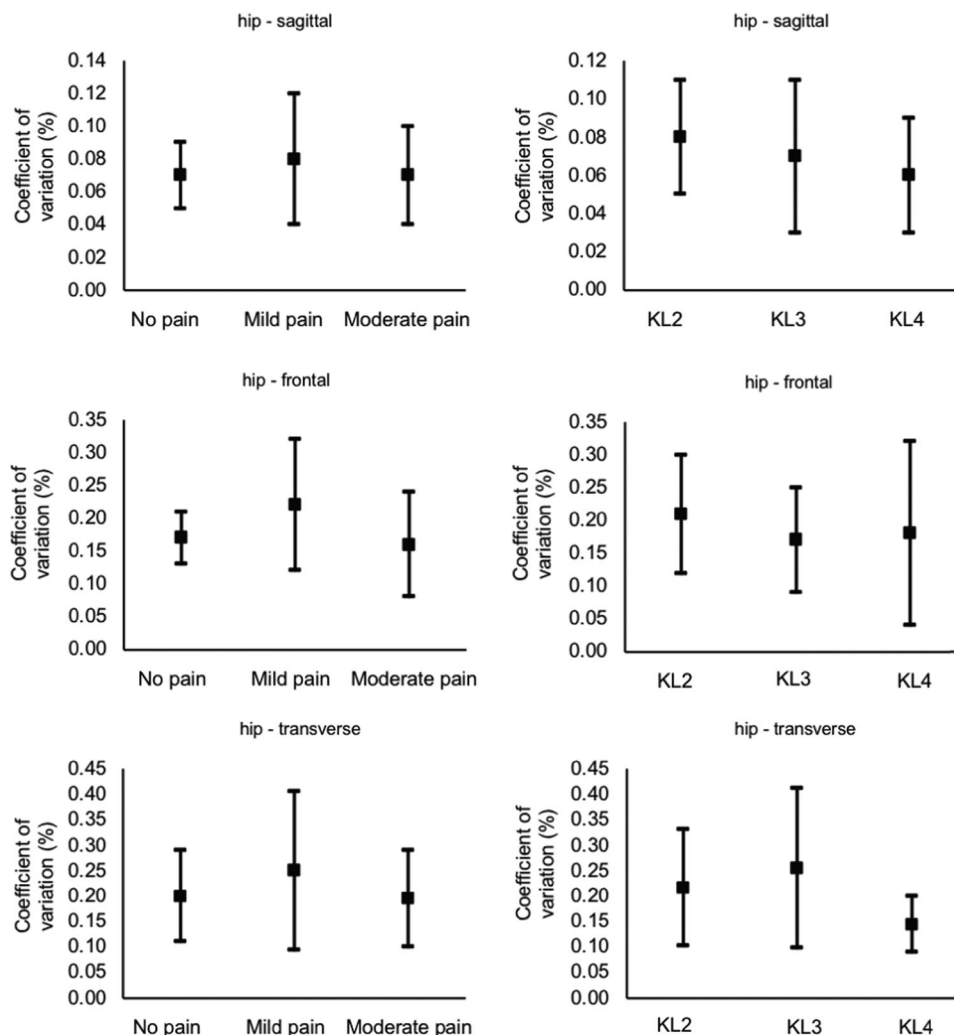***Significantly different to KL2 ($P < .05$).

FIGURE 3 Mean ± standard deviations of hip joint kinematic variability (coefficient of variation, %) in sagittal, frontal, and transverse plane according to pain severity during walking and radiographic disease severity assessed using Kellgren-Lawrence (KL) grading system. Higher curve coefficient of variation values represents higher variability (range between 0 and 1)

radiographic disease severity but were comparable when stratified according to pain severity (Table 1).

3.1 | Hip joint kinematic variability

Figure 3 illustrates curve coefficient of variation for hip kinematics in the sagittal, frontal, and transverse planes. There was a statistically significant main effect of pain severity in frontal plane curve coefficient of variability ($P = .035$), but no statistical difference in post hoc comparisons between levels of pain severity for frontal plane curve coefficient of variability (all $P \geq .006$). There was no main effect of pain severity on sagittal ($P = .453$) or transverse ($P = .266$) plane curve coefficient of variability and no main effect of radiographic disease severity for either sagittal ($P = .539$), frontal ($P = .307$), or transverse ($P = .206$) plane curve coefficient of variability.

3.2 | Coordination variability

Figures 4 and 5 illustrate coordination variability for the select couplings investigated according to pain severity and radiographic disease severity, respectively. There was no main effect of pain severity for any of the segment couplings: pelvis sagittal vs thigh sagittal ($P = .229$); pelvis frontal vs thigh frontal ($P = .327$); pelvis transverse vs thigh transverse ($P = .463$); thigh sagittal vs shank sagittal ($P = .248$); thigh frontal vs shank sagittal ($P = .256$); thigh transverse vs shank sagittal ($P = .665$). Similarly, there was no main effect of radiographic disease severity on any of the segment couplings: pelvis sagittal vs thigh sagittal ($P = .402$); pelvis frontal vs thigh frontal ($P = .489$); pelvis transverse vs thigh transverse ($P = .598$); thigh sagittal vs shank sagittal ($P = .472$); thigh frontal vs shank sagittal ($P = .373$); thigh transverse vs shank sagittal ($P = .893$).
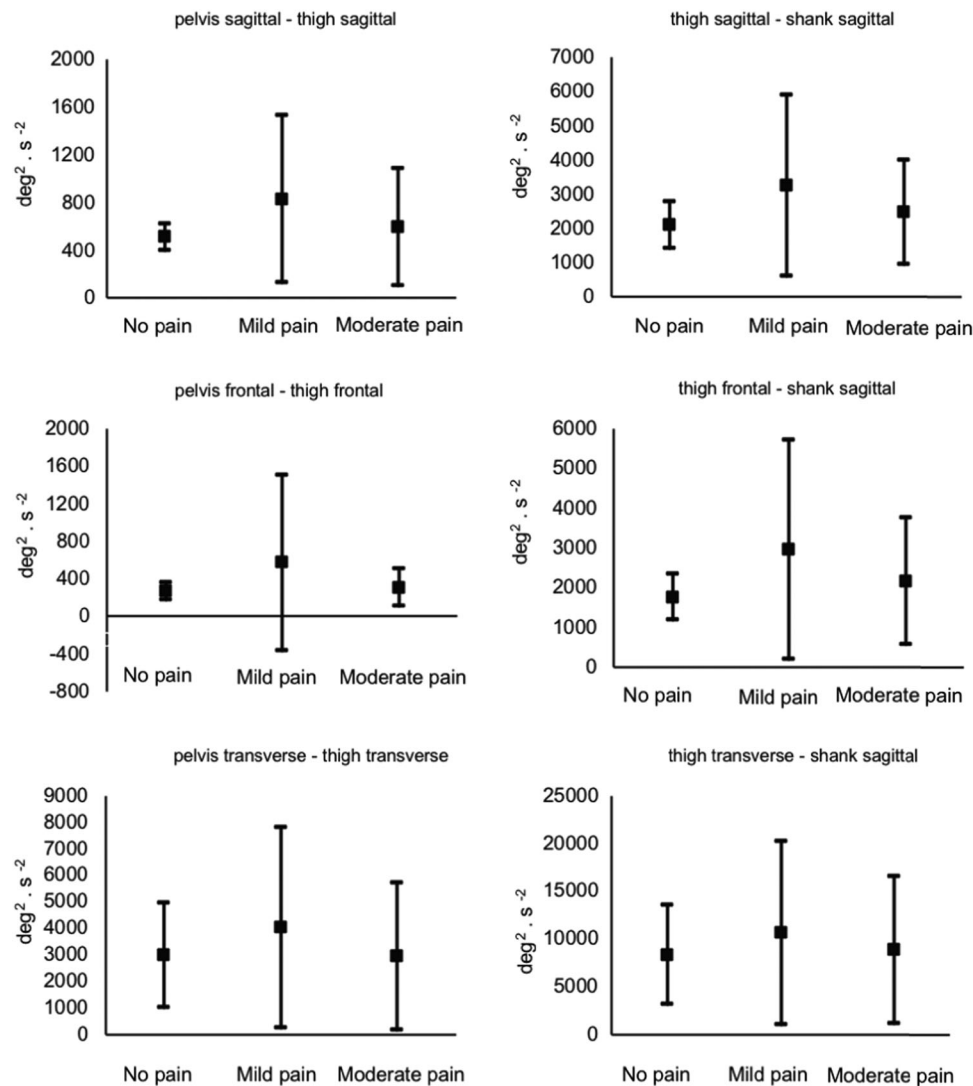
FIGURE 4 Mean ± standard deviations of segment coordination variability according to pain severity during walking assessed using the Western Ontario and McMasters Universities Osteoarthritis Index. Higher values indicate greater segment coordination variability

4 | DISCUSSION

The aim of this exploratory cross-sectional study was to evaluate measures of variability during walking according to pain severity and radiographic disease severity in people with hip OA. Contrary to our hypotheses, we found no robust evidence of greater hip joint kinematic variability or lower segment coordination variability during walking with greater pain or radiographic disease severity. These findings indicate that pain and radiographic disease do not influence hip joint kinematic variability and coordination of segments (pelvis-thigh and thigh-shank) in those with hip OA.

Restricted hip range of motion,[7–10] limited hip extension,[7,10,11] and hip abduction,[11] are reported during walking in people with hip OA compared with healthy controls.[25,28–30] This study extends previous hip OA research by investigating hip joint kinematic variability. Our data hints that frontal plane hip variability may be dependent on pain severity, collectively suggesting that those with hip OA may

adjust both movement pattern and variability of movement in the frontal plane. Specifically, we found that frontal plane hip variability may be lower in those with less pain compared to those with more severe pain during walking in people with hip OA. This observation is consistent with the view that output variability is lower in healthy individuals and greater in less healthy individuals.[13] Greater hip joint kinematic variability may reflect an adaptive strategy to avoid isolating the location of repetitive loads on the compromised, painful hip joint. However, as hip joint kinematic variability was not associated with radiographic disease severity, the speculation that greater hip joint kinematic variability may be associated with less severe radiographic disease joint remains questionable. Taken together, greater hip joint kinematic variability in the frontal plane may be associated with pain during walking, but not associated with radiographic disease. This is unsurprising, given the discordance between symptoms and radiographic disease in people with hip OA.[31] Future research is required as our post hoc analysis of the main effect of pain severity
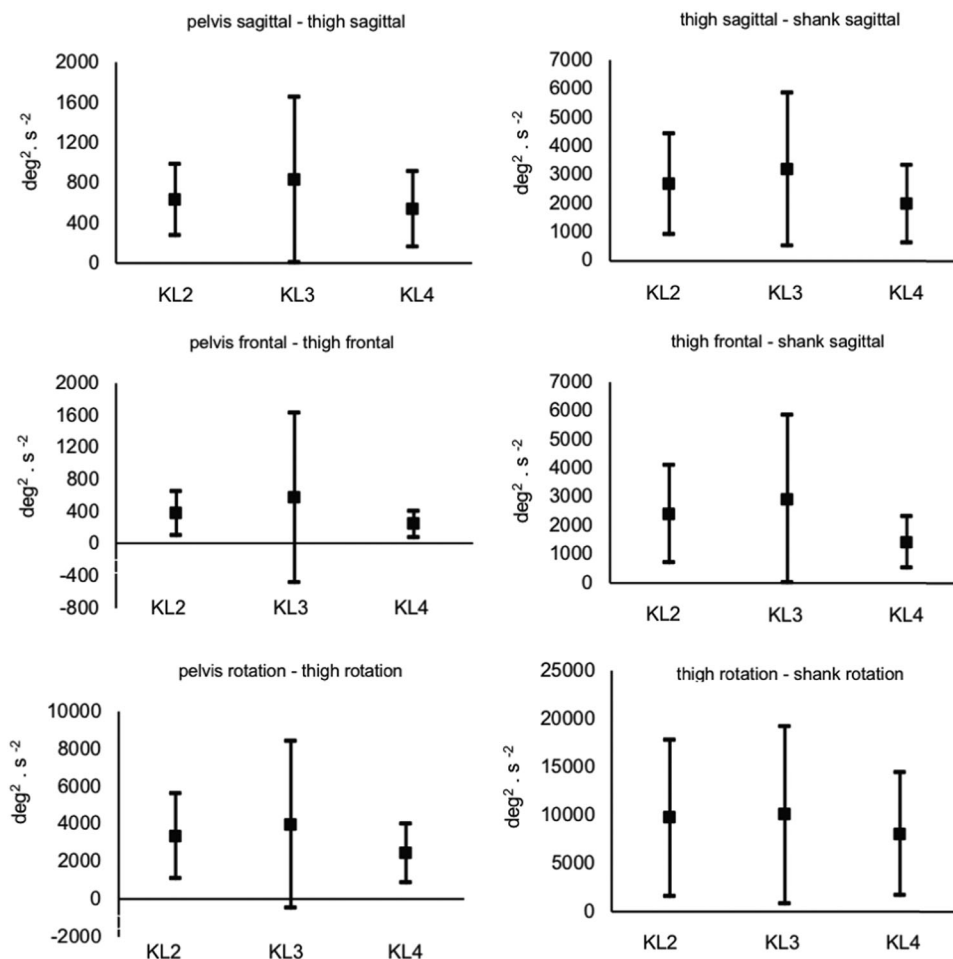
FIGURE 5 Mean ± standard deviations of segment coordination variability according to radiographic disease severity assessed using Kellgren-Lawrence (KL) grading system. Higher scores indicate greater segment coordination variability

on frontal plane hip joint kinematic variability did not reach statistical significance.

Segment coordination variability was investigated to provide insight into motor system health. We anticipated that segment coordination variability would be lower with greater pain and radiographic disease due to greater constraints imposed and less scope to vary motor strategy, in the presence of greater pain and radiographic disease. Failure to observe differences in segment coordination variability might be explained by a motor control system with the flexibility to adapt to constraints of pain and structural disease, irrespective of severity. Notably, we found no differences in peak hip abductor muscle strength according to pain or radiographic disease severity. Peak muscle strength is a crude indicator of motor system health, and assessment of muscle activation patterns would provide better insight into muscle coordination.[32,33] Another possibility as to why no differences were observed is that participants who report no pain and minimal structural degeneration actually do have the capacity to use higher levels of segment coordination variability. Perhaps these individuals did not to utilize the strategies available to them because walking was not sufficiently challenging to require the use of various segment coordination strategies. For example, stair climbing may be more sensitive than walking to detect alterations in segment coordination variability. Stair use is a particularly relevant task for assessment, as difficulty with stair climbing is also one of the driving factors for people with advanced hip OA to undergo a hip joint replacement.[5]

It is important to acknowledge that this study did not include a healthy control group, thus it is still plausible that segment coordination variability differentiates between those with and without hip OA. Indeed at the joint level, lower hip-knee joint coordination variability has been reported in those with acetabular lesions and concurrent pain.[29] Visual inspection of Figures 3 and 4 highlight that very large variation around the means of coordination variability between select segments were evident, particularly for those with pain and moderate to severe structural disease severity. This highlights the complexity of assessing coordination variability, suggesting that factors other than pain severity and/or structural disease severity may play an important role. The use of radiographs to assess joint structure has limited sensitivity[34,35] and other more sensitive imaging techniques (eg, magnetic resonance imaging) may influence our observations. For example, the presence of acetabular cartilage lesions was associated with lower hip-knee joint

coordination variability compared with no acetabular cartilage lesions in a cohort with various levels of radiographic disease severity in the hip joint.[12] Nevertheless, given the main effect of pain severity on frontal plane hip kinematic variability, and that no main effect of radiographic disease severity was observed highlights that symptoms should be considered irrespective of structural pathology.

Strengths of this study include a relatively homogenous group of participants with clinical and radiographic hip OA and inclusion of participants with unilateral symptoms only. In the exploratory nature of this study, we conducted multiple comparisons. However, to enhance confidence in our observations we applied a statistical correction to reduce the risk of type 1 error. There are several limitations to this study. First, although our sample had unilateral symptomatic hip OA, a proportion of our sample (45%) were diagnosed with bilateral radiographic hip OA which may influence measures of variability acquired from the most symptomatic side. To our knowledge, the effect of bilateral symptoms or bilateral disease on movement variability has not been previously assessed. Second, pain was assessed from a single item on the WOMAC questionnaire that covered pain during walking over the past two and seven days for participants enrolled in the cross-sectional study[18] and clinical trial,[19] respectively, rather than being assessed when three-dimensional analysis was acquired. Although we observed no statistical difference in duration of symptoms across the groups (Table 1), it is highly plausible that duration of pain and perception of pain influence movement strategies,[36] and thereby movement variability. Third, the number of participants with severe radiographic disease in our study was very small (n = 5), which limits confidence in our observations comparing those with severe radiographic disease to those with less severe radiographic disease. Fourth, we did not evaluate coordination variability between the trunk and pelvis. Increased trunk lean toward the symptomatic side and pelvic drop toward the contralateral side has been observed in people with hip OA,[10] suggesting that investigation into coordination variability between these two segments may be warranted. Lastly, although the number of strides (n = 5) used in the current study is comparable with other literature,[16,37] it is likely that more strides (ie, between 10 and 20) could provide a more accurate estimate of movement variability during walking.[15,38]

5 | CONCLUSION

In this cross-sectional study of people with hip OA, we found no evidence that measures of movement variability as assessed in this study differed according to pain severity during walking or radiographic disease severity. Motor control strategies utilized during walking as evaluated through coordination variability of selected segments appeared not to change in the presence of varying levels of pain or radiographic disease. These findings question the clinical relevance of movement variability as assessed in this study on pain severity and integrity of joint structure in those with hip OA.

AUTHOR CONTRIBUTIONS

MH, AF, JB conceived the study concept. BRM and YHP acquired the data. BRM, YHP, AF, and MH analyzed the data. MH, AF, JB, LED, KA, TVW, YHP, and KLB interpreted the data. MH and AF drafted the manuscript. All authors revised the manuscript for intellectual content and approved the final version.

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