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Research Article (Member)

Title: Frontal plane hip joint loading according to pain severity in people with hip osteoarthritis[1]

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ABSTRACT

The primary objective was to examine the hip adduction moment during walking in people with hip osteoarthritis (OA) according to pain severity. Sixty-eight participants with unilateral symptomatic hip OA were included. Pain during walking was assessed on a 5-point Likert item within the Western Ontario and McMaster Universities Index (no pain = 12; mild pain n = 37; moderate pain n = 19). Measures of the external hip adduction moment (peaks, Nm/BW×BH (%) and impulse, Nm.s/BW×BH (%)) were determined. Other measures included frontal plane hip, pelvis and trunk kinematics, walking speed and peak isometric hip abductor strength. Variables were compared according to pain severity using linear models and biomechanical variables were examined. Participants with moderate pain had a significantly higher second peak hip adduction moment and impulse compared to those with less pain. There was no difference in any measure of hip adduction moment between those with mild pain and no pain. There were no differences in kinematics across pain severity categories. Participants with moderate pain had a significantly slower walking speed compared to participants with mild and no pain. Participants with moderate pain had weaker peak isometric hip abductor strength compared to those with mild pain and no pain. The hip adduction moment during walking, hip abduction strength and walking speed differs according to pain severity during walking in people with hip OA.

Keywords: hip osteoarthritis; pain; walking

1. Introduction

Hip osteoarthritis (OA) is a major public health problem[1]. Although modifiable treatment targets for hip OA remain somewhat elusive, recent evidence suggests that higher cumulative frontal plane hip jont loading could be important[2]. Patients with hip OA are heterogenous and factors such as severity of hip pain is likely to influence hip joint loading. Understanding whether modifiable factors, such as hip joint loading, differ according to hip pain severity could help to better target conservative interventions for people with hip OA.

Hip joint contact forces cannot be easily quantified and indirect measures from gait analysis are often used to infer hip joint load[3,4]. In particular, the external hip adduction moment is highly correlated with hip joint contact force[2]. Recent data suggest that higher cumulative loading in the frontal plane predicts joint space narrowing in women with hip OA[4]. Interestingly however, people with radiographic and symptomatic mild-to-moderate hip OA exhibit a lower external hip adduction moment during late stance compared to healthy controls[3]. Pain can influence movement strategies[5,6], and can thereby influence hip joint loading irrespective of structure. Higher pain intensity has been associated with reduced walking speed and consequently reduced frontal plane knee joint loading in people with knee OA[7]. However, research into pain severity and hip joint loading is scant in people with hip OA. Correlational studies in people with end-stage hip OA scheduled for total hip arthroplasty have failed to observe an assocation between pain and frontal plane hip joint moments[8,9,10]. Understanding whether the external hip adduction moment differs according to hip pain severity during walking in people with less severe hip OA could help better target interventions aiming to preseve integrity of the joint struture.

The primary purpose of this study was to determine if the external external hip adduction moment differs according to hip pain severity in people with mild-to-moderate symptomatic hip OA. We hypothesised that the external hip adduction moment would be lower as pain severity increased. The secondary aim was to investigate possible explanatory variables including hip abductor strength and frontal plane hip, pelvis and trunk kinematics, according to hip pain severity.

2. Methods

2.1 Participants

Forty-eight participants from a cross-sectional study[11] and baseline data from 25 participants enrolled into a randomised controlled trial (RCT)[12] with unilateral symptomatic hip OA were available. Both studies were conducted at the University of Melbourne: between November 2006 and May 2008 for the cross-sectional study and between May 2010 to April 2012 for the RCT. Participants were recruited from the community via advertisements in newspapers and on radio. Ethics approval was obtained from the University of Melbourne Human Research Ethics Committee and all participants provided their written informed consent.

Eligible participants had i) unilateral hip OA according to the American College of Rheumatology classification criteria of pain and radiographic changes[13] and femoral or acetabular osteophytes along with joint space narrowing and Kellgren-Lawrence[14] grade greater than or equal to 2 on a standing x-ray; and ii) hip or groin pain on most days of the past month. Exclusion criteria common to both studies were: i) presence of neurologic, cardiac or other medical conditions that would compromise lower limb function; ii) back pain or other joint pain greater than hip pain; iii) lower extremity joint replacement; iv) systemic arthritic conditions such as rheumatoid arthritis; v) additional previous pathology such as fracture; iv) inability to walk unaided; vii) inadequate ability to understand English.

The RCT[12] had the following additional inclusion criteria: i) 50 years or older; ii) average pain intensity in the past week of 40 or higher on a visual analogue scale (0-100mm); iii) at least moderate difficulty with daily activities. Additional exclusion criteria for the RCT were: i) hip surgery within the past 6 months; ii) planned lower limb surgery; iii) physiotherapy, chiropractic treatment or prescribed exercises from the hip, lumbar spine or both in the past 6 months; iv) walking continuously for more than 30 minutes daily and regular structured exercise more than once weekly; v) uncontrolled hypertension, or morbid obesity (body mass index >36kg/m^2); vi) unable to comply with study protocol; vii) 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 McMasters Universities Osteoarthritis Index (WOMAC) subscale ranging from 0-20[15] within the past seven days. Higher score 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 Gait analysis

Reflective markers according to the standard Plug-in-Gait configuration (Vicon, Oxford, UK) were used. 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 120Hz and 1200Hz, respectively. Marker trajectory data were filtered using a Woltring quintic spline filter (mean square error 15 mm^2). The peak hip adduction angle was calculated during stance. Pelvic angles were determined using a rotation-obliquity-tilt Cardan angle sequence[16], and the maximum value of contralateral pelvic drop (obliquity) was extracted. Using reflective markers attached to the manubrium, 2nd and 10th thoracic spinous processes, a trunk segment was defined with the sagittal and frontal plane angle of the trunk expressed in relation to the laboratory coordinate system[17]. Positive frontal plane trunk lean with respect to the lab coordinate system indicated lean towards the stance limb. Kinematic values, step length, walking speed and stance time were calculated for at least five trials and averaged.

The external frontal plane hip joint moment was calculated using inverse dynamics (Vicon Plug-In-Gait v1.9) and expressed in the thigh coordinate system. The first and second peaks of the hip adduction moment and the positive hip adduction moment impulse were extracted; the hip adduction moment impulse reflects the moment magnitude and duration during stance. Both a non-normalised moment (Nm), and moment normalised to body size (body weight (BW) multiplied by body height (HT), expressed as a percentage ((Nm/(BW×HT)%))[18]), are presented. The joint moment was normalised to body size to determine if differences in the joint moment were due to factors other than differences in body size[18]. The joint moment was also reported non-normalised given that body mass can contribute to joint contact forces[19]. However, for interpretation we refer to the body-size normalised hip adduction moment.

2.4 Peak isometric hip abduction strength assessment

Peak isometric hip abduction strength was measured in the study hip. A hand-held dynamometer (Lafayette Instrument Company, Indiana, USA) was stabilised by a belt above the lateral femoral condyle in a gravity-eliminated supine position, with both hips in neutral abduction-adduction. The lever arm was recorded as the distance from the most prominent aspect of the greater trochanter to the point of dynamometer attachment within 5cm proximal to the femoral condyle. Participants performed a single submaximal and maximal contraction for familiarisation. Participants then performed two maximal trials for approximately three seconds. Each participant received standardised instruction to exert maximum effort. The average peak force (N) of the two maximal trials was multiplied by lever length to calculate torque (Nm) and normalised to body mass (Nm/kg). We have previously reported test-retest reliability of hip abductor strength assessment in patients with hip OA (intraclass correlation coefficient 0.84; standard error of measurement 12.1Nm)[20].

2.5 Other 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[15]. Anterior-posterior x-ray images were acquired in standing position. Kellgren-Lawrence grading scale was used to determine radiographic disease severity[14].

2.6 Statistical analysis

Stata version 14.2 (Statacorp, College Station, TX, USA) was used to perform statistical analyses and statistical significance was set at 0.05. One-way analysis of variance and chi-square tests were used to compare participant characteristics according to pain severity. Separate linear regression models were used to determine differences for each gait parameter (dependent variable) according to level of pain severity (independent variable). Gait parameters were entered as continuous variables and pain severity was entered as a categorical variable. The coefficient corresponding to the magnitude of each gait-related parameter was extracted for each level of pain severity, along with the corresponding p-value. Residuals of each linear model conformed to appropriate assumptions such that scatter plots of residuals satisfied the assumptions of normality and constant variance.

Sensitivity analyses were conducted to determine the effect of walking speed and duration of symptoms on measures of the hip adduction moment according to pain severity. Walking speed and duration of symptoms were entered into the models as continuous variables. The coefficient corresponding to the magnitude of each hip adduction moment related parameter was extracted for each level of pain severity, along with the corresponding p-value. For the comparison of non-normalised hip adduction moments, absolute speed (i.e. non-normalised) was entered into each model. Walking speed normalised to leg length using Froude number to account for the influence of leg length on speed[22,23] was entered into the models investigating normalised non-normalised hip adduction moments. For sex and radiographic disease severity, the interactions terms (e.g. sex × pain severity; radiographic disease severity × pain severity) were evaluated in separate models to determine if the association between measures of the hip adduction moment and pain severity differ according to sex and radiographic disease severity. Sex and radiographic disease severity were entered into the models as categorical variables.

3. Results

Of the 73 participants available, 12 reported no pain, 37 reported mild pain and 19 reported moderate pain during walking. Only five participants recorded severe or extreme pain during walking and were therefore excluded from subsequent analysis. Participant characteristics and spatiotemporal measures according to pain severity during walking are presented in Table 1. Age, sex, height, body mass, body mass index, symptom duration, radiographic disease severity and presence of bilateral radiographic disease were comparable across levels of pain severity. Participants with moderate pain had a significantly slower walking speed compared to participants with mild pain and no pain (Table 1). Stride length was significantly shorter in participants with moderate pain compared to those with mild pain (Table 1). No other differences were observed for any other spatiotemporal measures assessed according to pain severity (Table 1).

3.1 Hip adduction moment

The hip adduction moment according to pain severity is depicted in Figure 1 and peak and impulse data presented in Table 2. There were no differences in the normalised first peak hip adduction moment according to pain severity. The normalised second peak hip adduction moment was 31% higher in those with moderate pain compared to those with no pain ($p=0.04$) and 32% higher in those with moderate pain compared to those with mild pain (p=0.01). The normalised hip adduction moment impulse was 33% higher in those with moderate pain compared to those with no pain (p=0.03) and 34% higher in those with moderate pain compared to those with mild pain (p<0.01). No other significant differences were observed in the hip adductor moment according to pain severity. Irrespective of whether the hip adduction joint moment was non-normalised or normalised to body size, the results remained largely unchanged (Table 2). Results also remained unchanged when including the five participants with severe or extreme pain into the moderate pain group (data not shown).

3.2 Sensitivity analyses

Sensitivity analyses indicated that results remained relatively unchanged when considering the effect of walking speed on measures of hip adduction moment according to pain severity (Table 2). However, statistical differences were no longer apparent when accounting for walking speed between those with moderate pain and no pain for the second peak hip adduction moment (normalised; p=0.01) and hip adduction moment impulse (non-normalised; p=0.09; normalised p=0.01). With respect to symptom duration, the difference in normalised impulse between those with moderate pain and no pain was no longer statistically significant (p=0.07; Supplementary Table 1). Duration of symptoms did not alter statistical differences for any other hip adduction moment comparison across pain severities. There was no statistical evidence to suggest that the association between measures of the hip adduction moment and pain severity differed according to sex or radiographic disease severity (interaction terms p≥0.18; Figure 2 and Figure 3).

3.3 Trunk and pelvis kinematics, hip abduction strength

Frontal plane trunk and pelvis kinematics were did not differ across the three levels of pain severity (Table 2). Peak hip adductor strength normalised to body mass was lower in participants with moderate pain compared to participants with no pain during walking (Table

1). No other differences in peak hip adduction strength were observed according to pain severity (Table 1).

4. Discussion

People with unilateral symptomatic hip OA who reported moderate hip pain during walking had greater frontal plane hip joint loading, as evidenced by an elevated magnitude of the second peak hip adduction moment and hip adduction moment impulse during stance, compared to those with either mild pain or no pain. Although there was no evidence of different frontal hip joint, pelvis or trunk kinematics during walking according to pain severity, participants with moderate hip pain had weaker peak isometric hip abductor strength compared to participants with no pain. Importantly, these observations suggest that the external hip adduction moment, a potential target to prevent structural hip OA progression[4], differs according to severity of hip pain during walking in people with unilateral symptomatic hip OA.

Contrary to our hypothesis, frontal plane loading was considerably higher (31-34%) in people with moderate hip pain compared to those with lower levels of pain. This was despite the fact that those with greater pain walked slower. Although direct comparison of our findings is precluded, the magnitude of the second peak hip adduction moment in our hip OA cohort is comparable to previous research in people with mild-to-moderate hip OA, irrespective of pain severity[3]. However, people with mild-to-moderate hip OA have reportedly lower frontal plane loading during walking compared to healthy controls[3] and thus participants in the current study may also have lower frontal plane loading compared to healthy controls. Nevertheless, given that higher daily cumulative hip moment impulse in the frontal plane is associated with the progression of structural hip OA[4], our findings in people with moderate hip pain warrant further clinical consideration.

The external hip adduction moment is counteracted by an internal hip abductor moment contributed by active tension within the hip abductor muscles and also by tension within the passive structures of the hip joint[24]. Our data suggest that in participants with moderate pain there is a greater demand on the hip abductor muscles to balance the larger external hip adduction moment compared to those with less hip pain, however with potentially less strength to meet this augmented requirement. We observed that individuals with moderate pain were weaker compared to those with no pain. Hip abductor muscle weakness is likely in part attributable to reduced cross-sectional muscle area of the gluteus medius as previously reported in people with more severe hip OA compared to those with less severe hip OA[25]. Resistance training may be particularly warranted in patients with moderate pain to induce hip abductor muscle hypertrophy and thereby increase hip abductor strength. However, the implication of increasing hip abductor muscle strength on the external hip adduction moment magnitude during walking is unclear.

Previous research has observed inconsistent relationships between peak hip abductor strength and the external hip adduction moment in people with knee OA[26] and in healthy controls[27]. In the current study, there was no association between peak hip abduction strength and the second peak hip adduction moment using normalised data when accounting for pain severity during walking (p=0.998). The hip adduction moment can be altered by changing the magnitude of the frontal plane ground reaction force magnitude and/or the lever arm (perpendicular distance between hip joint centre and ground reaction force)[28]. In turn, these two parameters are largely determined by the position of the hip joint centre as well as the centre of pressure under the foot, body mass, and the body mass centre of mass position and acceleration[28]. These are potentially controlled by submaximal activations of multiple lower extremity, pelvic and upper body muscles. Humans do not exhibit maximal contraction of the hip abductors muscle during walking and use an unknown individual-specific proportion of their maximum strength. Therefore, measuring maximal isometric hip abductor strength may not be the most sensitive approach when examining the relationship between hip abductor muscle strength and the external hip adduction moment during walking. Other factors such as muscle activation patterns and/or upper body control that may influence frontal plane hip joint loading should be investigated.

The hip adduction moment can be potentially modulated by factors such as pelvis, hip and trunk kinematics[9,29], and walking speed[27]. However, we found no evidence to indicate that pelvis, hip and trunk kinematics in the frontal plane as measured in this study differed according to pain severity (Table 2). Further to this, there were no correlations between contralateral pelvic drop and measures of the hip adduction moment (r=0.13-0.16; p≥0.19), but greater peak lateral trunk lean was correlated with lower hip adduction moment (r=0.35-0.40; p≤0.01). Overall, it is possible that individuals adjust their kinematics in different ways such that consistent kinematic alterations in this study were not evident according to pain severity. Despite slower walking speeds in participants with moderate pain compared to participants with less pain, our observations with respect to frontal hip plane moment parameters remained largely unchanged when adjusting for walking speed. This indicates that walking speed is unlikely to account for the higher measures of frontal hip joint loading observed in the people with greater pain during walking. Moreover, slower walking speed in participants with greater pain severity would be expected to reduce, rather than increase joint loading as we observed.

Pain fluctuates in many people with hip OA[30] and mechanisms underpinning hip pain during walking are unclear. Although experimental hip pain reduced frontal hip plane loading during walking in healthy individuals[31], it is plausible that higher frontal plane loading during stance increases hip joint compression, and thereby pain[32] in people with hip OA. In this scenario, pain would be clinically prioritised over modification of joint loading as a treatment target. It is also important to consider that a reduction in pain may not result in a return to typical movement patterns[6] and it is possible that the osteoarthritic hip may continue to experience potentially suboptimal loads during walking, irrespective of alterations in pain intensity. Future research is needed to better understand the effect of treating symptoms on measures of hip joint loading.

Strengths of the study include the relatively homogenous group of patients with established hip OA and inclusion of those with unilateral symptoms only. There are several considerations of this study. First, participant selection is a potential source of bias, such that participants from the clinical trial were more symptomatic than participants from the cross-sectional study (Supplementary Table 2). Additional between-study differences included: duration of stance, hip abductor strength and hip adduction angle (Supplementary Table 2). Sensitivity analyses were performed to assess the effect of hip abduction muscle strength, hip adduction angle and stance duration. Results remained unchanged when accounting for hip adduction angle (Supplementary Table 3). Statistical differences persisted such that the normalised and non-normalised second peak hip adduction moment and hip adduction moment impulse remained higher in those with moderate pain compared to mild pain when accounting for hip abduction strength and stance duration (Supplementary Table 3). Second, interpretation of our findings is limited by the cross-sectional design which prevents us determining whether higher frontal plane loading observed in patients with pain precedes or follows development of pain. Third, although patients with unilateral symptoms were used in the current study, bilateral radiographic hip OA was present in some patients (n=34) and further research is required to determine the influence of bilateral radiographic disease on hip joint loading in people with hip OA. Fourth, walking pain was assessed from an item on the WOMAC that covered pain during walking over the past two[11] to seven[12] days rather than being measured during the walking task measured biomechanically. Fifth, hip joint loading remains unclear for hip OA patients with more severe pain than moderate hip pain during walking. Last, hip joint contact force cannot be readily assessed in vivo and we cannot conclusively determine that our findings would be similar if evaluating hip joint contact force.

In summary, we found cross-sectional evidence to suggest that people with hip OA who report moderate levels of hip pain during walking exhibit higher frontal plane loading during walking and have weaker hip abductor muscle strength compared to those with less pain during walking. Future research is needed to determine the effect of addressing hip abductor muscle function and/or pain on frontal plane loading in people with hip OA who report moderate levels of walking pain.

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Figure Captions

Figure 1 Ensemble average (± standard deviation) external hip adduction moment pattern (body-size normalized) over the gait cycle for hip osteoarthritis participants with no pain (black), mild pain (red) and moderate pain (blue).

Figure 2 Average (± standard deviation) external hip adduction moment (body-size normalized) according pain severity during walking for female (pink) and male (blue) hip osteoarthritis participants. Interaction term: sex × pain severity; radiographic disease severity × pain severity. BW: body weight; BH: body height

Figure 3 Average (± standard deviation) external hip adduction moment (body-size normalized) according pain severity during walking for hip osteoarthritis participants with Kellgren and Lawrence grade 2 (blue), Kellgren and Lawrence grade 3 (pink), Kellgren and Lawrence grade 4 (grey). Interaction term: radiographic disease severity × pain severity. KL: Kellgren and Lawrence; BW: body weight; BH: body height

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Table 1 Participant characteristics, hip strength and spatiotemporal measures. Data are presented as mean (SD), unless otherwise stated

	No pain (n = 12)	Mild pain (n = 37)	Moderate pain (n = 19)
Participant characteristics			
Age (year)	61.5 (6.9)	60.8 (8.3)	61.8 (9.3)
Females, n(%)	6 (50%)	17 (52%)	10 (45%)
Height (m)	1.69 (0.12)	1.68 (0.09)	1.68 (0.10)
Body mass (kg)	75.0 (13.1)	80.51 (17.5)	78.89 (13.1)
Body mass index (kg/m^2)	26.6 (4.8)	28.4 (5.0)	28.0 (2.9)
Test hip (left:right)	3:9	19:18	10:9
WOMAC Pain (0-20)	2.8 (2.1)	5.8 (1.9)[a]	8.5 (1.6) [a,b]
WOMAC Function (0-68)	11.3 (7.1)	20.3 (9.9)[a]	31.6 (7.3) [a,b]
Symptom duration (years)	6.0 (3.8)	4.5 (3.8)	4.3 (2.9)
Bilateral radiographic disease (yes:no)	5:11	16:17	12:10
Radiographic disease severity of symptomatic hip †			
Grade 2, n(%)	8 (%)	22 (%)	7 (%)
Grade 3, n(%)	4 (%)	11 (%)	9 (%)
Grade 4, n(%)	0	4 (%)	3 (%)
Isometric strength			
Peak hip abductor (Nm/kg)	2.37 (0.92)	1.91 (0.60)	1.73 (0.64) [a]
Spatiotemporal			
Speed (m/s)	1.29 (0.11)	1.25 (0.17)	1.12 (0.21) [a,b]
Normalised speed	0.44 (0.04)	0.44 (0.06)	0.38 (0.07) [a,b]
Stance duration (s)	0.61 (0.05)	0.62 (0.06)	0.65 (0.09)
Stride length (m)	0.65 (0.07)	0.64 (0.07)	0.59 (0.10) [a]

† Kellgren and Lawrence grading system

WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index

[a] Different to No pain group ($p<0.05$)

[b] Different to Mild pain group ($p<0.05$)

Table 2 Mean (SD) for biomechanical parameters and mean differences (95% confidence interval) between pain severity categories with and without adjustment for walking spe

	No pain (n = 12)	Mild pain (n = 37)	Moderate pain (n = 19)	**Unadjusted differences between pain severity categories** Mild pain minus No pain	Moderate minus No pain	Moderate pain minus Mild pain
Hip adduction moment						
Non-normalised						
First peak (Nm)	57.58 (22.54)	56.91 (17.77)	65.01 (24.78)	-0.67 (-14.44, 13.11)	7.43 (-7.86, 22.72)	8.10 (-3.61, 19.80)
Second peak (Nm)	43.97 (14.73)	47.39 (18.87)	62.00 (27.73)	3.42 (-10.61, 17.44)	**18.03 (2.45, 33.59)**[†]	**14.61 (2.69, 26.52)**[†]
Impulse (Nm.s)	20.03 (8.30)	21.09 (8.36)	28.01 (11.97)	1.06 (-5.22, 7.36)	**7.98 (0.99, 14.96)**[†]	**6.92 (1.57, 12.26)**[†]
Normalised[†]						
First peak (Nm/BW×BH (%))	4.67 (1.63)	4.34 (1.13)	4.97 (1.56)	-0.33 (-1.22, 0.57)	0.30 (-0.70, 1.30)	0.63 (-0.14, 1.39)
Second peak (Nm/BW×BH (%))	3.63 (1.27)	3.60 (1.32)	4.75 (1.82)	-0.03 (-0.94, 1.00)	**1.12 (0.04, 2.20)**[†]	**1.15 (0.32, 1.97)**[†]
Impulse (Nm.s/BW×BH (%))	1.62 (0.57)	1.61 (0.58)	2.16 (0.84)	-0.01 (-0.45, 0.43)	**0.54 (0.06, 1.03)**[†]	**0.55 (0.18, 0.92)**[†]
Pelvis kinematics (°)						
Peak contralateral pelvic drop (stance)	3.1 (1.9)	2.6 (2.4)	2.7 (2.5)	-0.5 (-2.0, 1.1)	-0.4 (-2.1, 1.4)	0.1 (-1.2, 1.4)
Trunk kinematics (°)						
Peak ipsilateral trunk lean (stance)[*]	2.9 (2.4)	3.0 (2.5)	3.1 (2.5)	0.1 (-1.5, 1.8)	0.2 (-1.6, 2.1)	0.1 (-1.3, 1.5)
Hip kinematics (°)						
Peak adduction angle (stance)	3.8 (3.8)	3.3 (4.9)	4.7 (5.3)	-0.5 (-3.7, 2.7)	0.9 (-2.7, 4.5)	1.4 (-1.4, 4.1)

[*] One participant removed from analysis as extreme outlier with 30 degree lateral trunk lean; results remain unchanged when including this outlier

[†] bold denotes statistical significance at 0.05 level

[a] Non-normalised hip adduction moment analysis adjusted for absolute walking speed

[b] Normalised hip adduction moment analysis adjusted for walking speed normalised to leg length

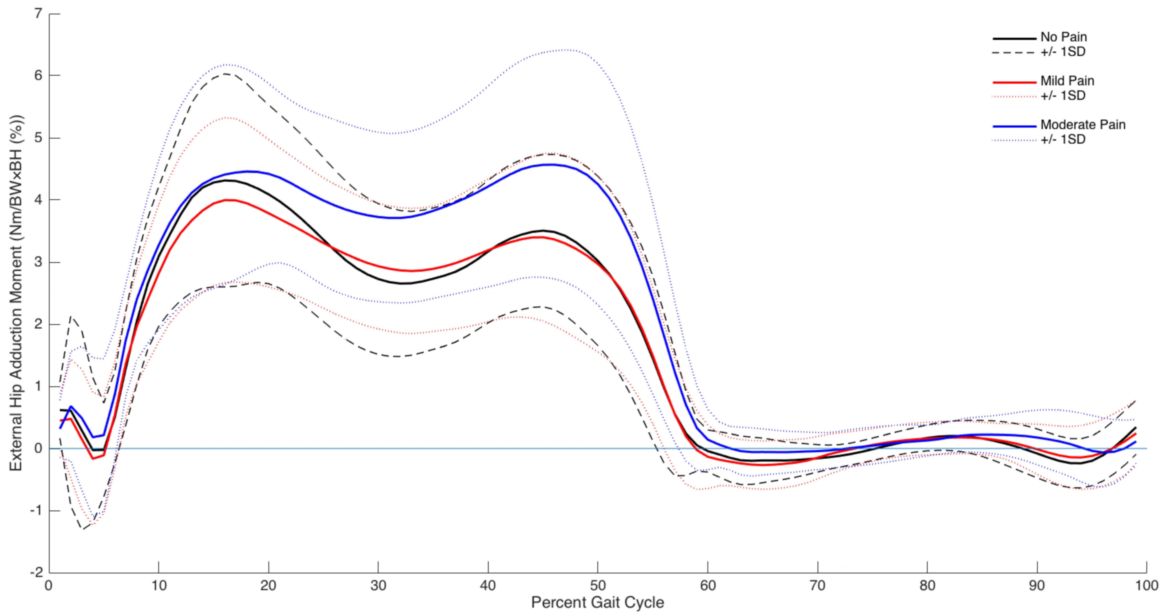

Figure 1

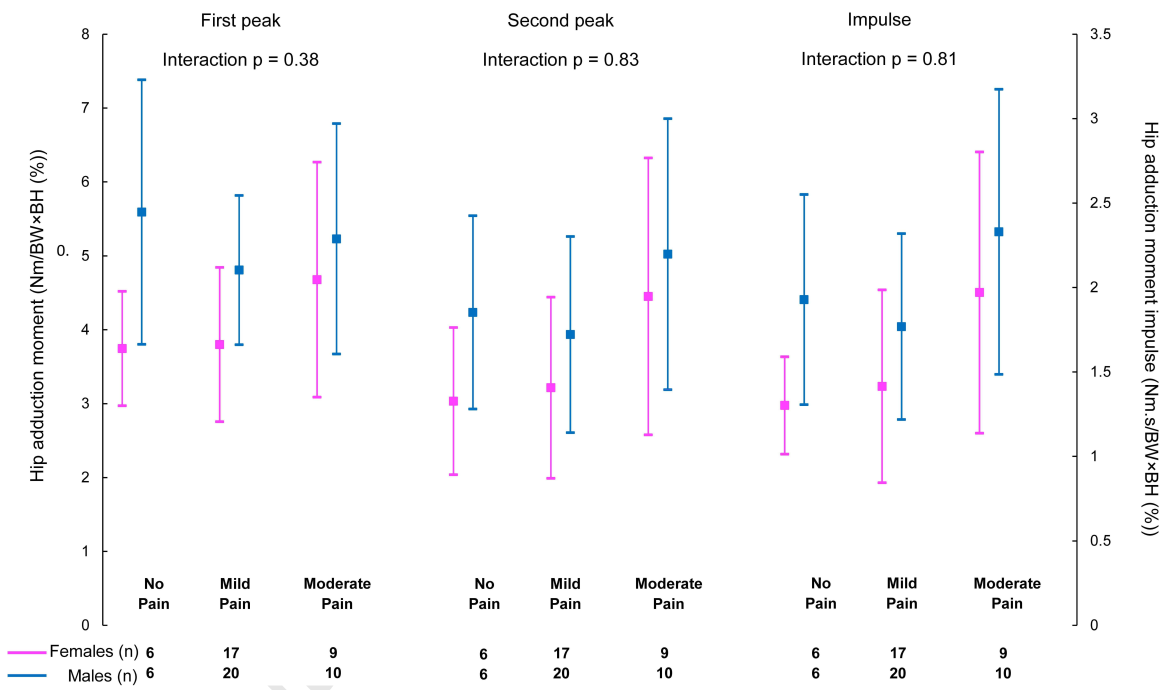

Figure 2

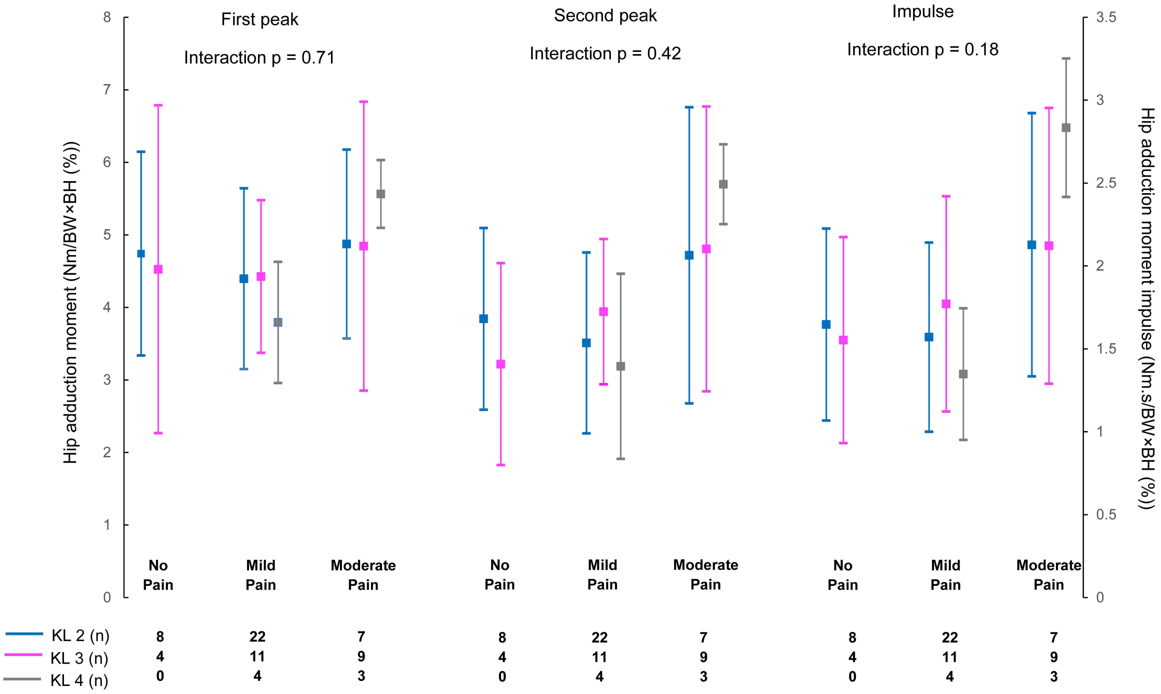
First peak
Interaction p = 0.71
Second peak
Interaction p = 0.42
Impulse
Interaction p = 0.18
Hip adduction moment (Nm/BW×BH (%))
Hip adduction moment impulse (Nm.s/BW×BH (%))
8
7
6
5
4
3
2
1
0
3.5
3
2.5
2
1.5
1
0.5
0
No Pain
Mild Pain
Moderate Pain
No Pain
Mild Pain
Moderate Pain
No Pain
Mild Pain
Moderate Pain
KL 2 (n) 8 22 7 8 22 7 8 22 7
KL 3 (n) 4 11 9 4 11 9 4 11 9
KL 4 (n) 0 4 3 0 4 3 0 4 3

Figure 3