EXTENDED REPORT

Investigation of hip abductor activation in subjects with clinical unilateral hip osteoarthritis

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Objectives: (*a*) To compare the magnitude of gluteus medius and tensor fascia lata activation between a group of subjects with clinical unilateral hip osteoarthritis and a group of healthy older adults. (*b*) To compare the magnitude of activation of the gluteus medius and tensor fascia lata between sides in a group of subjects with clinical unilateral hip osteoarthritis and a group of healthy older adults.

Methods: 19 subjects with clinical unilateral hip osteoarthritis and 19 healthy controls were investigated. The subjects performed a stepping task during which recordings were obtained using surface electromyograms from the hip abductors, and kinetic data were obtained from a dual force platform.

Results: Subjects with clinical hip osteoarthritis had higher gluteus medius activation than the healthy older adults (p=0.037). In addition, there were no differences in the magnitude of gluteus medius activation between the sides (p=0.733). There was no difference in the force platform data between the groups (p=0.078).

Conclusions: The increased magnitude of gluteus medius activation in the group with hip osteoarthritis is evidence of a muscular dysfunction associated with hip disease. This has implications for the progressive nature of the disease and for its conservative management.

A healthy joint is dependent on the neuromuscular system to provide movement, joint stability, shock absorption, and proprioception.[1] These multiple functions are so important that a joint is said to survive at the pleasure of the neuromuscular system.[2] Impairments in the neuromuscular system would therefore have the potential to contribute to joint damage. Not surprisingly there has been considerable interest in the role of a neuromuscular disturbance in the aetiology of osteoarthritis (OA).[1 3–9]

Most of the evidence linking neuromuscular deficits to joint damage in humans has involved the knee. Several studies have shown proprioceptive impairments in the affected[10 11] and unaffected limb[11] in subjects with OA. A gait study found changes in quadriceps control of knee flexion in subjects with knee pain caused by activity,[12] and recent evidence supports a relationship between quadriceps weakness and the radiographic presence[8] but not progression[13] of knee OA. Using electromyography (EMG) another study found a higher level of activation in the quadriceps during a knee extension task of women with knee OA.[14]

Although fewer studies have explored the relationship between hip joint disease and neuromuscular dysfunction, a link has been reported between abnormal muscular activity and hip OA. In all cases a dysfunction has been found in the tensor fascia lata (TFL) or gluteus medius (GMD), which work together to maintain the level of the pelvis in single leg stance.[15 16] The GMD also provides joint stability[15] and is active when postural stability is challenged in the mediolateral (ML) direction.[17 18] One gait study found continuous activity in the TFL and inhibition of the GMD of some subjects with hip OA.[19] In contrast, another found continuous activity in the GMD of subjects with severe hip OA.[20] Higher levels of GMD and TFL activation relative to maximum voluntary contraction have also been demonstrated during gait in subjects with hip OA after osteotomy.[21]

This research is consistent with theories on the TFL, stating that it is prone to tightness and overactivity in the presence of joint disease.[22] However, as seen above, reports of GMD activation changes with hip joint disease are inconsistent, with some researchers supporting[19] and others refuting[19 21] traditional clinical observations, which suggests that GMD is a muscle prone to weakness and inhibition.[22] Potential reasons for this include variations in the severity of OA studied, the task performed, and analysis techniques. Further clarification of possible dysfunction in the hip abductors, particularly GMD, is needed as it is unclear whether there is an increase or decrease in the level of activation of these muscle groups in hip disease. Further quantification of any change in the level of activation would also be of clinical and academic interest. In addition, it is also unknown whether there are changes in EMG activation in the unaffected limb of subjects with hip OA, similar to the proprioceptive impairments demonstrated in the unaffected side of subjects with knee OA.[11] Information of this nature would have direct clinical implications for the conservative treatment of hip OA.

Thus this study aimed at comparing the hip abductors of a group of subjects with unilateral clinical hip OA with those of a group of healthy older adults. We proposed the hypothesis, in accordance with clinical theory and some research evidence, that the magnitude of GMD activation would be smaller and TFL larger (*a*) in the affected limb than in the unaffected limb in subjects with clinical unilateral hip OA; (*b*) in the affected limb in subjects with clinical unilateral hip OA than in a control limb in a group of healthy older adults; (*c*) in the unaffected limb in subjects with clinical unilateral hip OA than in a control limb in a group of healthy older adults.

Abbreviations: AP, anteroposterior; COP, centre of pressure; EMG, electromyography; GMD, gluteus medius; ML, mediolateral; OA, osteoarthritis; RM-ANOVA, repeated measures analysis of variance; RMS, root mean square; TFL, tensor fascia lata; VAS, visual analogue scale; WOMAC, Western Ontario and McMaster University Osteoarthritis (index)

Table 1 Clinical characteristics of patients with hip OA (HOA)

Patient No	Age	Skill dominant limb	HOA leg	Flexion (HOA)	Internal rotation (HOA)	Duration of morning stiffness (min)	Duration of symptoms (years)	VAS rating (0–10)	WOMAC disability score (0–68)
1	64	R	R	100	10	<45	15	3	22
2	68	R	L	105	10	<30	5	4	2
3	72	R	R	85	5	<30	8	1	17
4	78	R	R	104	10	<15	3	1	24
5	76	R	R	110	10	<45	2	2	8
6	79	R	L	110	14	<15	2	1	15
7	75	R	L	100	14	<30	15	2	18
8	76	R	L	95	10	<15	10	1	4
9	74	R	R	100	12	<30	3	1	12
10	70	R	L	90	13	<30	6	0	34
11	70	R	R	115	20*	<15	2	2	7
12	66	R	L	115	20*	<30	10	1	5
13	61	R	L	120	20*	<45	3	0	7
14	79	R	L	110	25*	<30	5	2	10
15	70	R	L	110	28*	<30	10	4	32
16	64	R	R	115	20*	<45	15	3	31
17	52	R	R	100	20*	<15	5	1	21
18	65	R	R	110	0	<15	6	0	12
19	74	R	R	110	12	<60	10	0	13

*Indicates pain on internal rotation.

SUBJECTS AND METHODS

Subjects

Thirty eight community dwelling older adults (19 healthy people, aged 71.7 years (range 60–88), mean height 162.4 cm (range 151–185), mean weight 64.8 kg (range 54–84.5); and 19 subjects with unilateral clinical hip OA, aged 70.2 years (range 52–79), mean height 165.6 cm (range 151–177), mean weight 72.9 kg (range 43–92.5) volunteered for this study. The healthy older adults (four men, 15 women) had all reported no history of neurological disorders, hip pain, or other major musculoskeletal injuries. The group with clinical hip OA (seven men, 12 women) had experienced hip pain, but most remained active and had not had radiographic investigation. Subjects were recruited through community advertisements and medical practices in Eugene, Oregon.

To determine the presence of symptomatic hip OA a detailed history was obtained and clinical examination performed. Adults were classified as having clinical hip OA by using the criteria determined by the American College of Rheumatology.[23] Thus, all subjects with hip OA had to have hip pain, internal rotation $<15°$ and hip flexion $\leqslant 115°$ or internal rotation $\geqslant 15°$, to have morning stiffness $\leqslant 60$ minutes, to be >50 years of age, and to have pain on internal rotation. All subjects had pain in one or more of the following areas: the lateral hip, posterior hip, groin or anterior thigh, as pain in these areas is commonly found in people with hip OA.[24] No subjects had pain in the lumbar spine or radiating past the knee. Table 1 summarises the clinical characteristics. These same clinical criteria were also used to determine the absence of hip OA in the contralateral limb.

Exclusion criteria for both groups included a history of congenital or adolescent hip disease, hip trauma, inflammatory joint disease, and clinical signs of bilateral hip disease or lumbar spine disease. In addition, because pain may influence muscle activation, it was monitored immediately before testing with a visual analogue scale (VAS) (on a scale from 0 to 10). Subjects with a VAS>5 or in discomfort at the time of testing were excluded from the study. The level of clinical disability was quantified by using the Western Ontario and McMaster University Osteoarthritis (WOMAC) index.[25] Disability was assessed using the physical function section of WOMAC, which contains 17 questions relating to functional disability, scored from 0 to 4 by the subject. Subjects with high levels of clinical disability (>34 on the WOMAC scale) were excluded as it was unlikely that they could perform the experimental protocol consistently. All the subjects with hip OA and 17 of the healthy older adults were right leg skill dominant. Informed consent was obtained from all subjects and the study received approval from the human subjects research committee at the University of Oregon, as the study was undertaken there.

Experimental protocol

Before laboratory testing the lumbopelvic complex of all subjects was assessed by an experienced physiotherapist, during which the hip range of motion was measured with a standard goniometer. Muscle strength of hip abduction was recorded (average of three repetitions of 100% effort) with a hand held dynamometer. Subjects lay down with the leg to be tested abducted to a neutral position, the dynamometer was placed perpendicular to the thigh above the lateral femoral condyle, and the examiner met the resistance of a five second maximal isometric hip abduction contraction. Repeatability of these measures (ICC>0.7) was established in a subset of eight subjects (four healthy, four with hip OA).

The laboratory task consisted of a reaction time stepping task which had previously been shown to challenge the hip abductors in controlling balance in the ML direction.[18] This task was chosen as it was felt that it would provide a more consistent response from the hip abductors than gait, which can be highly variable[26] and is more technically difficult to record. Subjects stood on a dual force platform system (University of Oregon, Institute of Neuroscience technical group) with feet placed parallel, half a foot's length apart. On a light cue they placed either the right or left leg as quickly as possible onto a 15 cm high step placed 15 cm in front of the foot so that the entire foot made contact in a central 20 cm area of the step. One light signalled as a warning to get ready to step, a second signalled a step with the right foot, and a third signalled a step with the left foot. The attainment of equal weightbearing between the limbs at the start and the end of the step was monitored online. Subjects performed 4–5 practice trials, and then after resting 20 stepping trials were recorded (10 with each leg), with the order random across the randomised trials. Trials that did not meet any of the criteria (for example, foot placed too laterally) were repeated.

Instrumentation and data analysis

EMG activity was recorded from the GMD and TFL bilaterally. Bipolar surface electrodes (1 mm×10 mm, DE-02, Delsys Inc, Massachusetts) were placed on the skin over the muscles, parallel to the muscle fibres. The data were band pass filtered

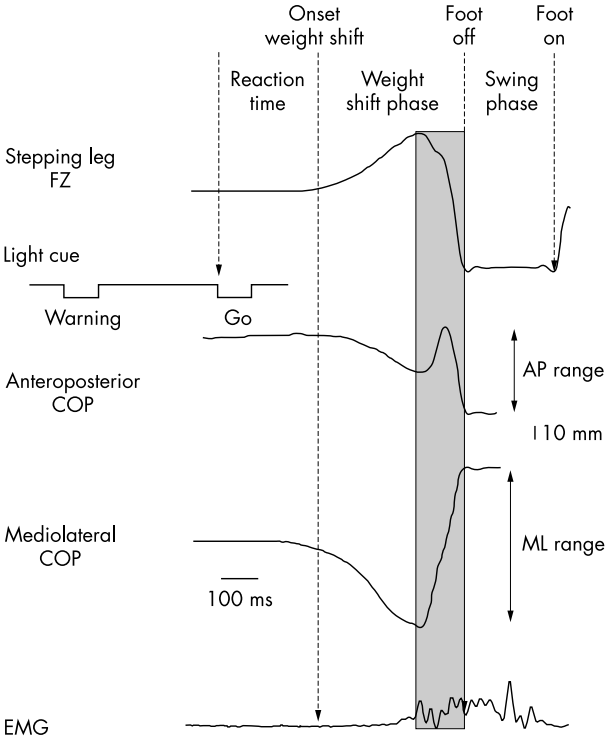

Figure 1 Data from a representative subject showing vertical force (FZ) under the stepping leg, the light cues, centre of pressure (COP) traces, and EMG signal. Temporal markers include the go signal, the onset of weight shift, foot off, and foot on. Timing phases include reaction time, weight shift time, and swing phase. The anteroposterior (AP) and ML COP ranges are displayed and a representative rectified GMD EMG trace is included. The shaded area is the time period over which the RMS of the EMG signal was calculated.

between 20 and 500 Hz and sampled at 1000 Hz with an AMLAB data acquisition system (AMLAB Int). Further signal processing and analysis was performed in MATLAB (Mathsworks Inc).

To capture the amplitude of the EMG signal, it was rectified, low pass filtered (50 Hz 6th order Butterworth filter), and the root mean square (RMS) of the EMG signal was calculated from the onset of the ML weight shift towards the stance leg, until foot off (fig 1). This time period was determined from the vertical force under the stepping leg and was chosen to capture the magnitude of muscle activity occurring during the component of the step in which the stance limb hip abductors control sideward weight shift. To permit comparison across subjects, the RMS activity was normalised to the middle five seconds of EMG activity recorded during a standard submaximal contraction (hip abduction against gravity for eight seconds in side lying). A submaximal contraction was preferred as a more reliable measure than a maximal contraction.[27] A maximal contraction has also been regarded as an inaccurate means of normalisation,[28] particularly in subjects who may be limited by pain.[29] The examiner ensured that the hip was maintained in a neutral position of flexion and extension, internal and external rotation.

Force platform data were collected to monitor how subjects performed the stepping task. Temporal parameters of the stepping movement were calculated from the vertical ground reaction forces and are outlined in fig 1. The onset of weight shift was the first increase in vertical force (>3SD above pre-movement level) under the stepping leg.[30] Foot off (start of single leg stance) and foot on (end of step) were also calculated from the vertical forces under the stepping leg. The weight shift (preparatory) phase was the period from the onset of weight shift to foot off. The swing phase started at foot off and ended at foot on. COP motion was calculated from the force platforms. The peak range of COP motion in the AP and ML directions during weight shift was determined, and the velocity of the COP in the AP and ML directions during the weight shift phase was calculated by dividing the distance of COP motion by the weight shift time.

Statistics

The means of the 10 stepping trials were used for analysis. To determine whether the magnitude of hip muscle activity differed according to group or side as suggested, separate repeated measures analyses of variance (RM-ANOVA) were performed for the TFL and GMD using SPSS 10. Both models investigated a group effect (hip OA *v* healthy older adult), side effect (affected *v* unaffected), and group×side interactions. In the 19 subjects with hip OA the affected limb was the right in 10 subjects and the left in nine. This meant that in the group with hip OA there was a roughly equal proportion of left and right steps in which the affected limb was the stance limb. To prevent laterality being a confounding variable, 10 healthy older adult subjects were assigned a right affected limb and nine a left affected limb.

The same RM-ANOVA model was also performed to compare the strength of the hip abductors between groups and sides. To investigate the temporal parameters of the stepping movements an RM-ANOVA was performed investigating group, side, and measure (weight shift *v* swing phase time) effects and associated interactions. A similar model was used to determine whether the range and velocity of COP motion during the weight shift phase differed between groups or sides but investigated the following measures: MLvelocity, ML-range, APvelocity, and APrange.

RESULTS

The main aim of this study was to determine whether subjects with unilateral clinical hip OA exhibited changes in EMG activation of the hip abductors when compared with the unaffected side and with a group of healthy older adults.

Electromyography

The normalised magnitude of GMD activation during the weight shift phase was analysed with an RM-ANOVA and showed a group effect ($F_{1,34}$=4.69, p=0.037) but no side effect ($F_{1,34}$=0.12, p=0.733). The magnitude of GMD activation was higher in the group with hip OA on both sides compared with the healthy older adults. The normalised magnitude of TFL activation during the weight shift phase showed neither a group ($F_{1,34}$=0.13, p=0.72) nor a side ($F_{1,34}$=2.20, p=0.147) effect (figs 2 and 3). There were no group×side interactions for either muscle.

Dynamometry

The strength of the hip abductors was measured with a hand held dynamometer and analysed with an RM-ANOVA which showed neither a group effect ($F_{1,30}$=0.002, p=0.969), nor a side effect ($F_{1,30}$=0.169, p=0.684), nor a group×side interaction. These results indicate that the hip abductor strength was similar between the two groups and between one limb and another.

Centre of pressure

To provide an overview of performance, two temporal parameters of the stepping movement were compared between groups and between sides (table 2). The RM-ANOVA did not demonstrate a group ($F_{1,36}$=0.446, p=0.508) or a side ($F_{1,36}$=0.945, p=0.338) effect in the weight shift or swing phase time. This indicates that both groups performed the task at a similar speed. The COP motion was used to monitor the performance of the stepping task. The RM-ANOVA

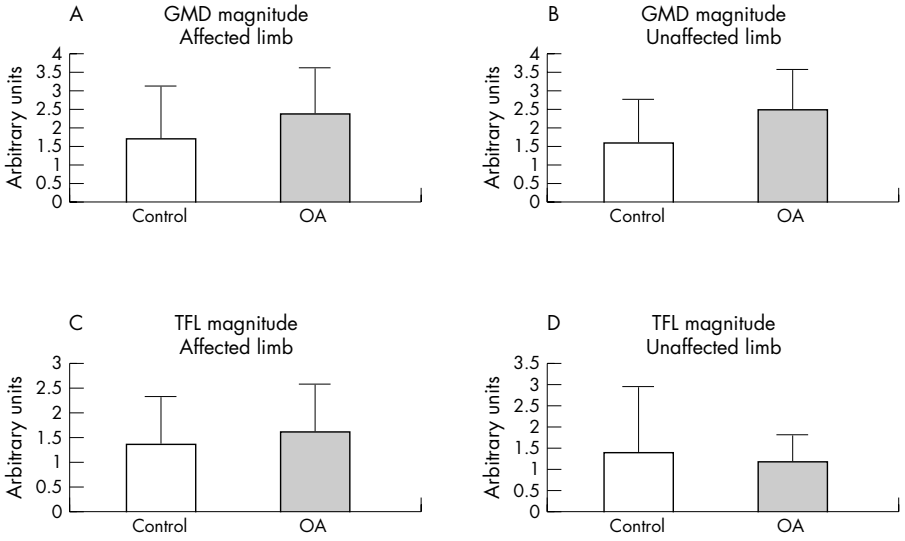

Figure 2 The mean magnitude of EMG activity (normalised to the submaximal contraction) of (A) the GMD of the affected limb, (B) the GMD of the unaffected limb, (C) the TFL of the affected limb, (D) the TFL of the unaffected limb. A group difference was found for GMD (A and B combined).

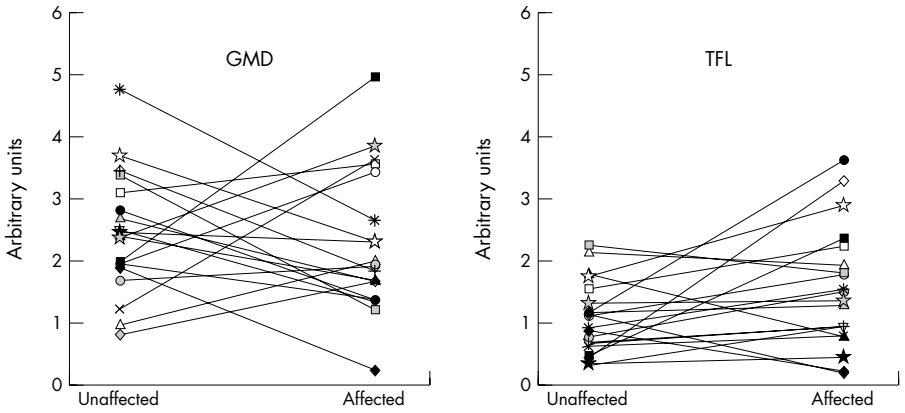

Figure 3 Individual data of all subjects with hip OA showing no between-sides differences in the GMD and TFL magnitude.

Table 2 Force platform data

	Controls		Hip OA	
Parameter	Affected	Unaffected	Affected	Unaffected
ML velocity (m/s)	0.22(0.07)	0.22(0.09)	0.26(0.07)	0.27(0.07)
ML range (m)	0.18(0.05)	0.17(0.06)	0.2 (0.04)	0.2 (0.04)
AP velocity (m/s)	–0.07(0.05)	–0.07(0.05)	–0.07(0.05)	–0.06(0.05)
AP range (m)	0.05(0.02)	0.05(0.02)	0.05(0.02)	0.05(0.02)

ML, mediolateral; AP, anteroposterior.

demonstrated neither a group ($F_{1,36}$=3.29, p=0.078) nor a side ($F_{1,36}$=0.158, p=0.694) effect in any of the four variables analysed. These results show that subjects performed the task in a similar manner regardless of group or side. Thus any changes in the normalised EMG values are unlikely to be due to differences in the speed or the way in which the movement was performed.

DISCUSSION

This study found that the group with hip OA had greater GMD activation than the healthy older adults. No difference in the magnitude of GMD activation between the affected and unaffected limbs was found in the group with hip OA. The magnitude of activation of the TFL did not differ between groups or sides. The increase in GMD activation and lack of change in TFL were contrary to the original hypothesis that their activation would reduce and increase, respectively. This hypothesis was based on clinical observation[22] and research evidence demonstrating GMD inhibition and TFL overactivity in some subjects with hip OA.[19] To explain these seemingly contradictory findings possible reasons for increased GMD activation in subjects with hip OA must first be explored.

There may be several explanations for the increase in GMD activation. Firstly, subjects with hip OA might have performed the stepping task in a different way or at a faster speed than the healthy older adults. This explanation can be partly refuted, as the force platform data showed no differences between the groups, suggesting the task was performed similarly. However, it

is possible that subtle changes in motion of the pelvis and trunk in the frontal plane might have been present. The experimental paradigm presented a specific demand for control of frontal plane motion and it has been reported that the hip abductors have an important role in controlling trunk position in the frontal plane.[31] The increased GMD activation may therefore be associated with a greater need to control trunk or pelvic motion. This issue could be resolved using kinematic analysis of subjects with hip OA performing the stepping task.

The current findings are also consistent with other studies showing an increase in the normal level of EMG activation in certain muscle groups in the presence of OA. Abnormal increases in quadriceps activation have been demonstrated in subjects with knee OA during the stance phase of gait[32] and during isometric quadriceps contractions.[14] Higher levels of co-contraction of the quadriceps and hamstrings during gait were found after total knee replacement, which persisted up to two years after the operation.[33] Previous studies on the hip have shown continuous activity in the GMD[20] and the TFL[19] in some subjects with hip OA. Higher levels of EMG activation relative to maximum voluntary contraction have also been demonstrated in the GMD and TFL of subjects with hip OA during gait after osteotomy.[21] An increase in EMG activation may reflect higher muscular forces or simply a compensatory increase in neural drive.

If the hip abductors in the OA group were weaker, then the central nervous system could compensate by increasing the level of neural drive to achieve a force similar to that of the control group. Several studies have reported a reduction in strength[7 8 34–36] and voluntary muscle activation relative to maximum effort[7 35 37 38] in subjects with knee OA. However, this explanation is not supported by our dynamometry data, which showed no differences in maximum voluntary strength between the groups. This may be because our subjects were not severely affected by pain or disability. In addition, voluntary muscle activation near maximum effort is a different measure from the automatic submaximal activation during a functional task recorded with EMG in this study. Possibly, therefore, the increased level of GMD EMG activation may reflect a failure of the central nervous system to grade the degree of muscular force required in the performance of the stepping task in subjects with hip OA. Investigation of muscular torques is required to confirm this possibility.

An increased level of hip abductor activity is of clinical relevance because it has the potential to cause large compressive forces on the hip. Recent research has shown substantial acetabular loading before heel strike, and peak acetabular pressures in mid-stance before peak ground reaction force, suggesting the influence of muscle contraction on the internal joint forces.[39] During single leg stance, the ipsilateral hip abductor mechanism produces a joint compressive force three to four times the body weight.[40] Barrie, after reviewing 637 femoral heads with OA, proposed that abnormal muscular forces cause asymmetries in wear of the cartilage of the femoral head.[41] Interestingly, tenotomies of the hip abductors, flexors, and adductors in conjunction with other surgical procedures are sometimes used to reduce the load transmitted across the joint.[42]

It is unclear why the level of TFL activation did not vary between groups. There was a non-significant trend towards higher activation in the affected limb of the subjects with hip OA, but not a general increase as was demonstrated in the GMD. One possible explanation is that the step up task challenges the GMD more specifically than the TFL. A previous study using this same methodology in younger adults suggested that GMD activation was more responsive to the ML stress than TFL.[18] Previous studies demonstrating increases in TFL activation were gait studies where TFL is likely to be more active than in a simple step.

The lack of difference in GMD activation between the affected and unaffected side in the group with hip OA was not expected. This might be explained by the presence of a subclinical OA in the contralateral limb, which our clinical criteria failed to detect. Although there appears to be a good correlation between radiographic change and movement loss,[43] structural change may occur in the joint many years before it is radiographically detectable.[44] It is unknown whether clinical or radiological examination is more accurate in the detection of early stage OA. This issue could only be resolved by the use of other diagnostic procedures such as magnetic resonance imaging. Despite these concerns our findings remain consistent with other research which has demonstrated sensorimotor changes in the contralateral limb after injury on one side.[45] Another study showed no difference in proprioceptive acuity between sides in a group of subjects with unilateral knee OA.[11] Although proprioceptive measures were not included in this study, it is possible that a disruption in the normal neurosensory system reduces the precision of the control of the level of muscle activation in both the affected and unaffected limb. Interestingly, it has been suggested that the presence of bilateral muscle dysfunction may explain why unilateral OA often progresses to bilateral OA over several years.[1] Further research is needed to investigate this possible connection.

As far as we know, this is the first study comparing the muscle activation state and hip muscle strength in affected and unaffected hips. It provides evidence of an increased EMG activation of the GMD in subjects with clinical hip OA. It is not possible to say whether the change in muscle activation was a primary or secondary event in the development of hip disease. However, higher levels of EMG activation may represent greater compressive forces acting on the joint, thus contributing to the progressive nature of the disorder. The results of this study suggest that at least part of the muscle dysfunction associated with hip OA may be a loss of precision rather than strength. Rehabilitation programmes for patients in whom pain and disability are not severe should therefore include an emphasis on the fine control of pelvic position during single leg stance. This would require improving the ability of the GMD to operate in its inner range rather than general strengthening. Appropriate rehabilitation is crucial both as a preventive measure and as a critical part of pre- and postoperative care of hip OA.

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