Manual Therapy 14 (2009) 611-617



Contents lists available at ScienceDirect

Manual Therapy

journal homepage: www.elsevier.com/math



Original Article

The association between degenerative hip joint pathology and size of the gluteus maximus and tensor fascia lata muscles

Alison Grimaldi^{a,*}, Carolyn Richardson^a, Gail Durbridge^b, William Donnelly^c, Ross Darnell^a, Julie Hides^{a,d}

^a Division of Physiotherapy, School of Health and Rehabilitation Sciences, The University of Queensland, Brisbane 4072, Australia

^b Centre for Magnetic Resonance Imaging, Brisbane, Australia

^c Brisbane Orthopaedic Specialist Services, Brisbane, Australia

^d The UQ/Mater Back Stability Clinic, Mater Health Services, Raymond Terrace, South Brisbane, Queensland 4101, Australia

ARTICLE INFO

Article history: Received 10 August 2007 Received in revised form 28 October 2008 Accepted 8 November 2008

Keywords: Hip osteoarthritis Gluteus maximus Tensor fascia lata Magnetic resonance imaging

ABSTRACT

The aim of this study was to obtain, using Magnetic Resonance Imaging (MRI), muscle volume measurements for the gluteus maximus (upper: UGM and lower: LGM portions) and tensor fascia lata (TFL) muscles in both healthy subjects (n = 12) and those with unilateral osteoarthritis (OA) of the hip (mild: n = 6, and advanced: n = 6). While control group subjects were symmetrical between sides for the muscles measured, subjects with hip joint pathology showed asymmetry in GM muscle volume dependent on stage of pathology. The LGM demonstrated atrophy around the affected hip in subjects with advanced pathology (p < 0.05), however asymmetry of the UGM (p < 0.01) could be attributed largely to hypertrophy on the unaffected side, based on between group comparisons of muscle volume. TFL showed no significant asymmetry, or difference compared to the normal control group. This study highlights the functional separation of UGM and LGM, and the similarities of the UGM and TFL, both superficial abductors appearing to maintain their size around the affected hip. Further research is required to determine the specific changes occurring in the deeper abductor muscles. This information may assist in the development of more targeted and effective exercise programmes in the management of OA of the hip.

© 2008 Elsevier Ltd. All rights reserved.

1. Introduction

Therapeutic exercise has been cited as an important approach used in management of osteoarthritis (OA) of the hip (Hochberg et al., 1995; Altman et al., 2000; Smidt et al., 2005; National Collaborating Centre for Chronic Conditions, 2008; Zhang et al., 2008). There is however, a distinct scarcity of literature investigating the effectiveness of therapeutic exercise of the hip. Programmes have often been quite generalised with small to moderate short term effects and poorer long term effects (van Baar et al., 2001; Tak et al., 2005). Outcomes may be improved through the development of more specific programmes based on a greater understanding of muscle function and dysfunction around the hip joint. One of the most consistent findings in subjects with hip dysfunction is an inability to maintain adequate lateral control of the hip and pelvis in single leg stance (Hardcastle and Nade, 1985). Studies assessing hip abductor muscle strength in subjects with OA of the hip have found deficits of up to 31% (Murray and Sepic, 1968; Jandric, 1997; Arokoski et al., 2002), while others have found no significant losses in abductor strength (Teshima, 1994; Sims et al., 2002). These apparent inconsistencies may be associated with specific changes occurring within muscles of the abductor synergy, and the association of these changes with stage of pathology.

While strength testing provides information on global abductor muscle function, a resultant effect of all synergists, specific changes within the synergy will only become evident by addressing each muscle individually. Muscles of the abductor synergy providing lateral stability of the hip and pelvis could be divided into superficial muscles that provide their effect via insertion into the iliotibial band (ITB), and deeper muscles that act via insertion into the greater trochanter. Muscles of the superficial system include the tensor fascia lata (TFL) muscle and the gluteus maximus (GM) muscle. The deep system would include the gluteus medius (GMED), piriformis (PIRI) and gluteus minimus (GMIN) muscles. This paper will focus on the study of muscles of the superficial system, while the deep muscle system will be addressed in a further publication (Grimaldi et al., unpublished).

In clinical rehabilitation settings, the GM muscle has been targeted for strengthening exercises, due to its reported tendency to

^{*} Correspondence to: Alison Grimaldi, PhysioTec Physiotherapy, 23 Weller Road, Tarragindi, Brisbane, Queensland 4121, Australia. Tel./fax: +61 7 3342 4284.

E-mail address: info@physiotec.com.au (A. Grimaldi).

¹³⁵⁶⁻⁶⁸⁹X/\$ – see front matter \odot 2008 Elsevier Ltd. All rights reserved. doi:10.1016/j.math.2008.11.002

weaken and atrophy (Janda, 1983; Sims, 1999; Sahrmann, 2002). In contrast, the TFL muscle has been targeted for lengthening techniques, due to its reported tendency to become excessively active (Janda, 1983; Sims, 1999; Sahrmann, 2002). There has been little attention paid in either research or clinical settings, to the impact of the functional differentiation of the GM muscle on joint mechanics and the prescription of therapeutic exercise. The upper portion of the GM muscle (UGM) arises from the posterior iliac crest, while the lower portion of the GM muscle (LGM) arises from the inferior sacrum and upper lateral coccyx (Williams et al., 1989). Despite a lack of fascial separation in adult humans, studies on morphogenesis of the GM muscle have revealed that it arises from two muscle primordia with a loose connective tissue separation between cranial and caudal portions in the foetus followed by fusion in the prenatal period (Tichy and Grim, 1985). The UGM, acting above the centre of rotation of the hip, has a primary function of hip abduction, and does not have a role in hip extension. While both portions may externally rotate the femur, the lower portion of the GM muscle (LGM), acting below the centre of rotation of the hip, is the primary hip extensor (Stern, 1972; Stern et al., 1980; Lyons et al., 1983; Jaegers et al., 1992) playing an important protective role in absorbing ground reaction forces at heel strike during gait.

The role of the hip abductor synergy in joint protection is less clear. While hip abductor strengthening is generally considered as a priority in patients with hip pain, an *in vivo* study on joint loads during gait revealed that peak joint loads were associated with peaks in hip abductor muscle activity during stance phase rather than solely loads applied from body weight (Krebs et al., 1998). Contrary to common clinical belief, the authors from this study recommended that clinicians aiming to reduce joint load should *reduce* hip abductor activity.

Another important aspect that should be considered in the prescription of therapeutic exercise for patients with OA of the hip is the stage of pathology. While global atrophy of hip muscles may be present in end stage pathology, in the earlier stages of the condition, more specific changes in the muscles of the hip abductor synergy may occur. It has been proposed that these changes can result in alteration of the orientation of the resultant hip joint vector, and ultimately result in joint damage over time (Kummer, 1993; Sims, 1999). Further information pertaining to hip muscle dysfunction at different stages of pathology would be useful as it could be used in the development of more specific and possibly more effective conservative intervention or prevention programmes for those with degenerative hip joint pathology.

Imaging studies provide an excellent opportunity to analyse individual muscles of the hip. Only one study has measured muscle size in subjects with OA of the hip. Arokoski et al. (2002) used magnetic resonance imaging (MRI) to measure hip muscle cross sectional area (CSA) in men with and without hip OA. Two axial slices through the pelvis provided a single CSA for LGM and a combined CSA of all hip abductors, including the UGM. This measure unfortunately failed to provide specific information of individual muscles of the abductor synergy. Furthermore, volume measurements rather than single slice CSA measurements, may be more representative of the complex pelvic musculature. One study has reported muscle volume measurements of the hip muscles for three healthy subjects (Jaegers et al., 1992), but no volume measurements have been reported in subjects with hip OA.

The main aim of this study was to investigate size of the muscles of the superficial lateral stability mechanism of the hip, TFL and GM muscles, in subjects with either mild or advanced degenerative pathology of the hip. Subjects with unilateral pathology were selected in order to provide both within and between subject comparisons. The specific aims were to examine i) if there was significant side *asymmetry* in the superficial muscles across 3 groups (mild degenerative change, advanced degenerative change, matched controls), ii) if there were significant differences in *actual muscle size* among the pathology and control groups, and iii) if the functionally separate portions of the GM muscle, UGM, and LGM, display similar patterns of change in subjects with hip pathology. This study also examined the association of both stage of pathology, and muscle size, with the factors of age, height, weight, pain, function and activity levels.

The hypotheses of the study were that ia) there would be significant asymmetry in size of the UGM, LGM, and TFL in subjects with hip joint pathology, but not in controls, ib) asymmetry would be greater in subjects with advanced pathology, ii) the affected side LGM muscle would be smaller that the comparable side in control subjects, based on clinical expectation (Sims, 1999; Sahrmann, 2002), and iii) changes in the UGM would more closely reflect changes in the TFL muscle based on their close functional relationship.

2. Methods

2.1. Subjects

Twenty-four subjects (12 subjects with hip joint pathology and 12 control subjects) were recruited for this study via community advertisement and via contact with medical practitioners. Control subjects were recruited to match each subject with pathology by sex and age. The age of the control subject was required to be within 5 years of the age of the matched subject with hip pathology. There was an equal distribution of males and females in each group. Subject details are listed in Table 1.

Subjects with hip joint pathology were included in the study if they had both a medical diagnosis and radiographic evidence of unilateral degenerative hip joint pathology. Radiographic evidence included X-Ray or MRI demonstrating OA or atraumatic, degenerative labral pathology. OA of the hip joint was classified by an experienced radiologist using the Kellgren/Lawrence (K/L) global scoring system (Kellgren and Lawrence, 1957; Hirsch et al., 1998). Six subjects with early joint space narrowing and osteophytes (K/L grades 1–2) were recruited for the 'Mild Group' and 6 subjects with moderate to severe joint space narrowing and osteophytes (K/L grades 3–4) were recruited for the 'Advanced Group'. Seven subjects had left sided pathology and five subjects had right sided pathology.

Exclusion criteria for all subjects included any systemic disease affecting the muscular or nervous system, history of congenital or adolescent hip disease, hip trauma or previous surgery, inflammatory joint disease, presence of tumour, any lower limb injuries in the previous 2 years, participation in unilateral sports, use of a walking aid, and factors that would preclude them from MRI

Table	-1
Tanie	
IUNIC	

Subject characteristics for each group.

Group	No	Sex M:F	Age Mean(SD)	Weight(kg) Mean(SD)	Height(cm) Mean(SD)	AMI Mean(SD)	MHHS(P) Mean(SD)	MHHS(F) Mean(SD)	MHHS(Total) Mean(SD)
Mild	6	3:3	46.5(9.5)	80.4 (15.1)	171.3 (9.7)	63 667 (23 884)	25 (10.5)	41.5 (3.0)	73.2 *(11.3)
Adv	6	3:3	57.7 (6.7)	78.3 (8.5)	172.0 (7.4)	82890 (75410)	16.7 (5.2)	36.2 (5.5)	58.1 *(58.7)
Con	12	6:6	51.8 (9.7)	73.5 (13.3)	168.2 (10.2)	123 175 (68 766)	-	-	-

No = Number. BMI = Body Mass Index. AMI = Activity Metabolic Index. MHHS = Modified Harris Hip Score. P = Pain. F = Function. M:F = Male:Female. SD = Standard deviation. Adv = Advanced Pathology. Con = Control. *Significant difference between pathology groups (p < 0.05).

scanning procedures (eg. pacemaker, metal implants, pregnancy, claustrophobia). Subjects in both groups were also excluded if they had experienced any lower back pain in the previous 2 years or if there had been any significant lifetime history of lower back pain that resulted in a period of immobility, or required further investigation or treatment. Subjects in the control group were excluded if they had any history of hip pain.

Information on the study was sent to the subjects prior to admission to the study. Ethical approval was obtained from the institutional review boards and informed consent was obtained from all subjects.

2.2. Procedure

2.2.1. Self-report questionnaires

Information on subject activity levels was gathered using a 12 month Leisure Time Physical Activity questionnaire providing an activity metabolic index (AMI) (Taylor et al., 1978; Arokoski et al., 2002). Activities were coded using the intensity code provided (Taylor et al., 1978).The AMI for each activity the subject participated in was calculated with the formula: AMI = Intensity code (mean metabolic units) × average number of times per month - × the number of months per year (frequency) × the time the activity was performed per occasion (duration). Total AMI reflects the addition of AMI for all activities (Taylor et al., 1978) and provides a measure of metabolic units used per year.

The Modified Harris Hip Score (MHHS) was used to assess pain and function in the subjects with OA of the hip (Byrd and Jones, 2000). The pain section consisted of 44 points, where a score of 44 represents a pain-free state. The function section consisted of 47 points, where a score of 47 points represents full, normal function. The multiplier 1.1 was used to achieve a total score out of a possible 100 (pain-free normal function).



Fig. 1. Diagramatic representation of the portions of the GM muscle. UGM _____; LGM _____; ITB _____.

2.2.2. Testing of leg dominance

Subjects were also tested for leg dominance. Kicking was used as the test function (Herneth et al., 2004). The weight-bearing leg was recorded as "stance dominant" and the kicking leg as the "skill dominant" leg.

2.2.3. MRI assessment

Subjects were first screened for contraindications to MRI by a medical practitioner. Subjects were positioned in supine lying with legs extended to a neutral position. Leg rotation was controlled with the use of sand bags. A 1.5 Tesla Siemens Sonata MR system was used. A T2 True Fast Imaging with Steady State Progression (FISP) sequence using 2 series of 28×6 mm contiguous slices from the iliac crest to the most distal extent of the GM muscle was employed (Time to Repetition (TR): 3.78 ms/Echo Time (TE): 1.89 ms/Field of View (FOV): 390 mm).

2.2.4. Measurement procedure

An MRI measurement software package (Osiris) was used to measure CSA (cm²) of UGM, LGM and TFL muscles on each image in which the muscle appeared. Muscle volume (cm³) was calculated by multiplying CSA by slice width and then adding the volumes from each slice to determine the total muscle volume (Fukunaga et al., 1992; Alkner and Tesch, 2004) (Fig. 2). The two functionally separate parts of GM were measured (UGM and LGM). The UGM includes that part of the muscle acting above the centre of rotation of the femoral head. These fibres insert almost exclusively into the ITB via a thick laminar tendon (Lieberman et al., 2006). The LGM inserts below the centre of rotation, superficial fibres into the ITB. deep fibres into the gluteal ridge of the femur (Lieberman et al., 2006). This anatomy is depicted in Fig. 1. In this study the largest CSA of the femoral head was used as an anatomical landmark to functionally separate the UGM from the LGM muscle, to approximate the centre of rotation of the femoral head (Stern, 1972).

Reliability of the assessor's measurement technique was tested by retracing all slices of one subject (44 slices) with an interim period of 6 weeks. Intra-tester reliability was tested for each separate measurement on each slice using a two sided bootstrapped interval of intraclass correlation coefficient (ICC_{2,1}). Intrarater reliability was found to be good, with correlation coefficients ranging from 0.87 to 0.99. Standard error of measurement (SEM) was calculated using the formula SEM = pooled SD × (1-ICC)^{1/2} (Wallwork et al., 2007). Standard deviation of the difference (SDD) was also calculated as the standard deviation of the differences between measurement 1 and 2. SEM for the GM muscle was 0.495 cm² and the SDD was 3.87 cm², while for the TFL muscle the SEM was 0.536 cm² and the SDD was 2.44 cm². These values represent good measurement stability with low error.

2.3. Statistical analysis

The comparison of muscle volumes among groups and between sides was performed using a mixed linear model describing muscle volume with group as a between-subject factor, and side as a within-subject factor (Dependent variable = muscle volume, Independent variables = sides and groups). Each muscle was analysed separately. Contrasts of means were performed to compare sides within groups. Muscle volumes around the affected and unaffected hips of the subjects with hip joint pathology were compared with muscle volumes of the corresponding sides of their matched control subjects. That is, if the pathological side was left, the left side muscle volume of the matched control subject was used for comparison, and the right compared with the unaffected side value of the pathology group counterpart. Percent differences were calculated using the formula: % Difference = [(larger value – smaller value)/larger value] × 100 (Hides et al., 1996).



Fig. 2. Axial MRIs through the pelvis: images A,C & E through ilia showing UGM in the proximal pelvis; Images B, D & F showing LGM and TFL just below the hip joint. A & B: control subject; C & D: subject with mild left hip OA (right side as viewed in image); E & F: subject with advanced left hip OA. GM _____ TFL _____.

Analyses were also conducted to assess participant characteristics in relation to a) the similarity of the groups and b) the extent of association with muscle size. One way analysis of variance was used to assess group equivalence across each of the dependent measures of age, height, weight, pain, function, and metabolic activity. The association between these patient characteristics and UGM, LGM, or TFL muscle size was assessed using analysis of covariance.

3. Results

3.1. Side to side differences in muscle volumes within groups

There were no significant side to side differences in the control or mild pathology groups. While LGM size was smaller on the affected side in all but one subject in the group with mild joint changes, the asymmetry was not great enough to be statistically significant. In the group with advanced pathology there were significant between side differences in the GM but not the TFL muscle. The asymmetry was greater in the UGM muscle (mean difference 21%, p < 0.01) than the LGM muscle (mean difference 19.7%, p < 0.05). Means, standard deviations, and percentage difference in muscle volumes are reported for each group in Table 2. Examples of side to side differences are illustrated for each group in Fig. 2.

3.2. Differences in muscle volumes between groups

No significant differences in muscle volumes were found between the mild and advanced pathology groups. The UGM muscles were significantly larger on the unaffected side (Mean difference 30.5%) of the subjects in the advanced pathology group when compared with matched controls (p < 0.05, Table 3). No other

 Table 2

 Side to side differences in muscle volume (cm³), and percentage differences within groups for UGM, LGM, and TFL muscles.

GROUP	SIDE	UGM Mean (SD)	LGM Mean (SD)	TFL Mean (SD)
Mild	Affected	405 (70)	508 (118)	82.5 (20)
	Unaffected	421 (60)	539 (120)	/3.8 (19)
	% Difference	3.8%	5.8%	10.5%
Advanced	Affected	378 (96)	457 (158)	86.2 (38)
	Unaffected	479 (118)	569 (144)	89.5 (27)
	% Difference	21.0%	19.7%*	3.8%
Control	Left	352 (106)	453 (130)	74.3 (24)
	Right	359 (125)	495 (158)	80.6 (29)
	% Difference	2.0%	8.6%	7.8%

SD = Standard Deviation. UGM = Upper gluteus maximus muscle. LGM = Lower gluteus maximus muscle. TFL = Tensor fascia lata muscle. p < 0.01 * p < 0.05.

comparisons reached statistical significance although LGM volumes were on average 15.2% larger (p = 0.12) on the *unaffected* side in the group with advanced pathology, compared with controls, statistical analysis did not reveal a significant difference in this relatively small sample size. Means, standard deviations, and percentage difference in muscle volumes around matched hips of the pathology and control groups are reported in Table 3.

3.3. Self-report questionnaires

Results of the AMI for all subjects and the MHHS for subjects with OA of the hip are shown in Table 1. Pain and function scores were lower for the group with more advanced radiological changes, reflecting higher pain levels and more functional disability, as measured by the MHHS. These scores considered alone were not significantly different statistically, however when the total score was calculated there was a significant difference between scores in the mild and advanced pathology groups (p < 0.05). There was no statistically significant difference between groups for the AMI.

3.4. Leg dominance

All subjects were left stance dominant/right skill dominant.

3.5. Effect of subject characteristics on muscle size

Results of the analyses indicated the groups were comparable in terms of age, height, weight, and metabolic activity (all p > 0.05). In addition there was no significant relationship between these patient characteristics, or pain and function, and UGM, LGM or TFL muscle volume (p > 0.05).

Fable 3	
Between group differences in muscle volume (cm ³) for UGM, LGM, and TFL muscle	es.

SIDE	GROUP	UGM Mean (SD)	LGM Mean (SD)	TFL
Affected	Mild	405 (70)	508 (118)	82.5 (20)
	Advanced	378 (96)	457 (158)	86.2 (38)
	Control ^a	354 (103)	460 (128)	74.9 (24)
Unaffected	Mild	421 (60)	539 (120)	73.8 (19)
	Advanced	479 (118)*	569 (144)	89.5 (27)
	Control ^a	361 (119)	489 (150)	75.4 (26)

UGM = Upper gluteus maximus muscle. LGM = Lower gluteus maximus muscle. TFL = Tensor fascia lata muscle. SD = Standard Deviation. Side refers to the named side in the pathology group, and for the control group side is aligned by matched pair dependent on side of pathology; * p < 0.05.

^a Reference group for significance values.

4. Discussion

This study investigated the influence of degenerative hip joint pathology on size of the GM and TFL muscles.

4.1. Side to side differences in muscle volumes within groups

The results of this study showed that subjects with demonstrated unilateral hip joint pathology exhibited marked side to side differences in the size of the GM muscle, specific to stage of pathology. While asymmetry in LGM size in subjects with mild joint pathology was not great enough to be statistically significant, in those with advanced joint changes the mean volume of the LGM muscle was on average 19.7% smaller on the affected side (p < 0.05). The only previous study to investigate muscle size in those with OA of the hip/s reported that the mean CSA of the LGM muscle was 9% smaller on the side of the worse hip in those with either unilateral or bilateral OA (Arokoski et al., 2002). The most likely explanations for the smaller percent difference are the inclusion of subjects with bilateral pathology in the latter study which would be expected to reduce the degree of side to side difference demonstrated, and the inclusion of subjects with both mild and advanced joint pathology in the analysis. Some explanation may also be provided by the different measurement techniques. A single CSA measurement may not provide a true reflection of change in total muscle volume.

The UGM muscle similarly showed no significant side to side difference in those with mild joint pathology. In the presence of advanced pathology, the UGM was on average 21% smaller on the affected side, representing a significant side to side difference in muscle size (p < 0.01).

The TFL muscle was not significantly different between sides in either pathology group, although the mild group was on average 10.5% larger on the affected side. In contrast Arokoski et al. (2002) reported that the CSA of the TFL muscle was 13% smaller on the more affected side in men with OA. This difference is again most likely due to differences in subject selection and/or measurement technique.

Another important consideration when interpreting side to side differences in muscle size is that in the absence of longitudinal data, the determination of side to side differences as atrophy or hypertrophy around weight-bearing joints must be approached with caution. Side to side differences could reflect either atrophy or hypertrophy. Decreases in muscle size on the affected side could occur in response to pain (Lund et al., 1991) or reflex inhibition (Stokes and Young, 1984). However, as pain causes an instinctive shift in weight-bearing towards the unaffected side, side to side volume differences may occur due to disuse atrophy around the affected hip and/or overuse hypertrophy of the unaffected side. For this reason, a control group was included for comparison of actual muscle volumes between groups, thereby assisting in the interpretation of side to side differences.

4.2. Differences in muscle volumes between groups

As with Arokoski et al. (2002) study, the current study was unable to demonstrate any between group difference in LGM size. This may be simply due to the inherent variability within the population and the relatively small sample size. Another consideration is the fact that the measurement of muscle size by tracing around the perimeter of a muscle in the subjects with pathology of the hip joint may underestimate the loss of contractile muscle tissue. Replacement of normal viable muscle tissue with intramuscular fatty or connective tissue has been reported as 'fatty atrophy' at the hip in the GMED muscle (Pfirrmann et al., 2005). Differences in tissue quality of the LGM muscle are observable as increased black markings within the muscle on the side of the affected hip in Fig. 1D and F. This assists in the support of the assumption that side to side differences in the LGM muscle in those with hip pathology are at least in part due to atrophy around the affected hip. It is most likely however that together with atrophy around the affected side LGM secondary to patterns of antalgic weight shift. The finding that advanced group subjects LGM volumes were 15.2% larger on the *unaffected* side than matched control subjects (p = 0.12), provides some support for this effect although not reaching statistical significance.

Between group differences for the UGM muscle showed that the mean muscle volume of the UGM muscle on the *unaffected* side in those with advanced pathology was significantly (Mean difference 30.5%) larger than the corresponding muscles in the control group subjects. This finding suggests that the significant asymmetry (Mean difference 21%) observed in subjects with advanced joint pathology may be largely attributable to hypertrophy on the unaffected side. Some degree of atrophy on the affected side however cannot be discounted although fatty atrophy was not commonly observed in the UGM muscle. Around the affected hip neither the UGM muscle, nor the other superficial hip abductor, the TFL muscle, were significantly different in size to a normal population.

The other information that was assessed with regard to the subjects of this study was gathered through self-report questionnaires and leg dominance testing. While pain, function and leg dominance had no significant effect on GM or TFL muscle size, the information collected provided 2 important pieces of information.

4.3. Pain, function and radiological change

The first of these relate to the association between pain, function, and radiological change.

It has been previously noted that there is often no clear relationship between severity of radiological change in an osteoarthritic joint and severity of pain or degree of disability (Hurley, 1999). In studies of subjects with OA of the knee, advanced radiological change may in some people be accompanied by very little pain, while others with only mild degenerative change may experience severe disabling pain (Claessens et al., 1990; McAlindon et al., 1993). Arokoski et al. (2002) in their study of men with OA of the hip were unable to demonstrate a correlation between grade of severity of OA and pain measured on a visual analogue scale. There was however significantly more pain within individuals on the side with the highest radiographic OA score. Similarly the findings of the current study reflect the difficulty in linking a pain score alone to degree of radiographic change. By combining measures of pain and function, the MHHS was able to demonstrate significant differences between subjects with early radiographic change and those with advanced radiographic change. This may suggest that this particular combination of questions may be more sensitive to degree of radiographic change than those available for OA of the knee.

4.4. The influence of leg dominance

The second finding of importance relates to the potentially confounding variable of leg dominance. Although there is evidence that dominance has an effect on muscle strength (Balogen and Onigbinde, 1992), particularly in upper limb strength in those involved in unilateral sports (Ducher et al., 2005; Ellenbecker et al., 2006), there is a much weaker link between *leg* dominance and muscle strength (Hunter et al., 2000; Zakas, 2006), and little evidence to link leg dominance to asymmetry in muscle size. Greater muscle strength of the dominant limb may be associated with improved neuromuscular functioning, rather than muscle size alone. In the current study the exclusion of all subjects involved in unilateral sports sought to avoid the effect of this potentially

confounding variable on muscle symmetry. The results of this study were able to demonstrate that for the normal control subjects tested there was no significant asymmetry in muscle size for the muscles measured. All subjects were left stance dominant which, if this factor were imparting an effect, would favour a larger muscle volume on the left side particularly for the weight-bearing LGM muscle. This was not the case, allowing greater clarity in interpretation of results for the pathology groups.

4.5. Possible clinical implications

The balance of muscle activity around a joint may either protect a joint from injury or accelerate destructive joint forces. Both the UGM and LGM muscles are known to be active at heel strike in gait to help absorb ground reaction forces causing lateral pelvic drop and flexion moments at the hip and knee (Stern et al., 1980; Lyons et al., 1983). While reduced activation of the GM muscle may fail to absorb these ground reaction forces, excessive activation in the abductor muscles, may lead to an increase in joint loading (Krebs et al., 1998). So both atrophy of the LGM muscle around the affected hip, and hypertrophy of the UGM muscle around the unaffected hip may have negative effects on their respective underlying joints. Hurley (1999) has suggested that the presence of bilateral muscle dysfunction may help to explain why unilateral OA years later often becomes bilateral OA. The findings of this study imply that the LGM and UGM muscles should be assessed individually, and on both sides, with clinical management directed towards restoring normal symmetrical weight-bearing patterns and muscle bulk.

Further, the finding that *neither* of the superficial hip abductor muscles appear to be affected on the side of pathology, and recommendations to reduce recruitment of the hip abductor muscles in order to reduce peak acetabular pressures during gait (Krebs et al., 1998), the current clinical rationale for generalised hip abductor muscle strengthening could be questioned. While some authors have reported hip abductor muscle strength deficits of up to 31% (Murray and Sepic, 1968; Jandric, 1997; Arokoski et al., 2002), others have reported no significant difference (Teshima, 1994; Sims et al., 2002). These variable findings may be a reflection of the relative degrees of atrophy of individual muscles of the abductor synergy. If both superficial abductor muscles are not significantly affected by pathology, strength changes may possibly reflect weakness in the deeper abductor muscles. Together with the information provided by this study, further information on the response of the deep muscle system to degenerative change of the hip may provide further insight into specific changes within the abductor synergy. Greater specificity in exercise prescription around the hip may allow development of interventions that achieve more significant and longer lasting changes in pain and function scores in patients with OA of the hip.

4.6. Limitations and future directions

The main limitation of this study was low subject numbers. Valuable additional information may be gained by subsequent studies with larger subject numbers and the inclusion of a method to measure quality of muscle tissue. Furthermore, this study assessed only two of many hip muscles which may be associated with hip pathology. Further investigation of other muscles, such as the deeper abductor muscles, is required to provide a more complete picture of muscle dysfunction.

5. Conclusion

This study has demonstrated that the GM muscle should be considered as 2 functionally separate entities, the UGM a hip abductor and the LGM, a hip extensor, these muscles having differing responses to the presence of joint pathology. The UGM muscle like its functional counterpart, the TFL, appears unaffected on the side of joint pathology, while the LGM muscle demonstrates local atrophy. The lack of affect on the superficial hip abductors suggests that muscle weakness demonstrated in subjects with OA of the hip may be related to changes in the deeper hip abductors (GMED, GMIN and PIRI) and require more specific therapeutic exercise intervention.

References

- Alkner BA, Tesch PA. Knee extensor and plantar flexor muscle size and function following 90 days of bed rest with or without resistance exercise. European Journal of Applied Physiology 2004;93:294–305.
- Altman RD, Hochberg MC, Moskowitz RW, Schnitzer TJ. Recommendations for the medical management of osteoarthritis of the hip and knee:2000 update. Arthritis and Rheumatism 2000;43:1905–15.
- Arokoski MH, Arokoski JPA, Haara M, Kankaanpaa M, Vesterinen M, Niemitukia LH, et al. Hip muscle strength and muscle cross sectional area in men with and without hip osteoarthritis. The Journal of Rheumatology 2002;29:2185–95.
- Balogen JA, Onigbinde AT. Hand and leg dominance: do they really affect limb muscle strength? Physiotherapy Theory and Practice 1992;8(2):89–96.
- Byrd JWT, Jones KS. Prospective analysis of hip arthroscopy with 2-year follow up. Arthroscopy 2000;16(6):578-87.
- Claessens AA, Schouten JS, van den Ouweland FA, Valkenburg HA. Do clinical findings associate with radiographic osteoarthritis of the knee? Annals of the Rheumatic Diseases 1990;49:771–4.
- Ducher G, Courteix D, Mémé S, Magni C, Viala J, Benhamou C. Bone geometry in response to long-term tennis playing and its relationship with muscle volume: A quantitative magnetic resonance imaging study in tennis players. Bone 2005;37(4):457–66.
- Ellenbecker TS, Roetert EP, Riewald S. Isokinetic profile of wrist and forearm strength in elite female junior tennis players. British Journal of Sports Medicine 2006;40:411–4.
- Fukunaga T, Roy RR, Shellock FG, Day MK, Lee PL, Kwong-Fu H, et al. Physiological cross-sectional area of human leg muscles based on magnetic resonance imaging. Journal of Orthopedic Research 1992;10(6):926–34.
- Grimaldi A, Richardson C, Stanton W, Durbridge G, Donnelly W, Hides J. The association between degenerative hip joint pathology and size of the gluteus medius, gluteus minimus, and piriformis muscles, unpublished.
- Hardcastle P, Nade S. The significance of the trendelenberg test. The Journal of Bone and Joint Surgery British 1985;67B:741-6.
- Herneth A, Philip M, Pretterklieber M, Balassy C, Winkelbauer F, Beaulieu C. Asymmetric closure of ischiopubic synchondrosis in pediatric patients: correlation with foot dominance. American Journal of Radiology 2004;182(2):361–5.
- Hochberg MC, Altman RD, Brandt KD, Clark BM, Dieppe PA, Griffin MR, et al. Guidelines for the medical management of osteoarthritis. Part 1. Osteoarthritis of the hip. Arthritis and Rheumatism 1995;38:1535–40.
- Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 1996;21(23):2763–9.
- Hirsch R, Fernandes RJ, Pillemer SR, Hochberg MC, Lane NE, Altman RD, et al. Hip osteoarthritis prevalence estimates by three radiographic scoring systems. Arthritis and Rheumatism 1998;41(2):361–8.
- Hunter SK, Thompson MW, Adams RD. Relationships among age-associated strength changes and physical activity level, limb dominance, and muscle group in women. Journal of Gerentology 2000;55A(6):B246–72.
- Hurley MV. The role of muscle weakness in the pathogenesis of osteoarthritis. Rheumatic Disease Clinics of North America 1999;25(2):283–98.
- Jaegers S, Dantuma R, deJongh H. Three dimensional reconstruction of the hip on the basis of magnetic resonance images. Surgical Radiologic Anatomy 1992;14:241–9.
- Jandric S. Muscle parameters in coxarthrosis. Medicinski Pregled 1997;50(7-8): 301-4.
- Janda V. Muscle function testing. London, Boston: Butterworths; 1983.
- Kellgren J, Lawrence J. Radiological assessment of osteoarthritis. Annals of the Rheumatic Diseases 1957;16:494–502.

- Krebs DE, Robbins CE, Lavine L, Mann RW. Hip biomechanics during gait. Journal of Orthopedic and Sports Physical Therapy 1998;28(1):51–9.
- Kummer B. Is the Pauwels theory of hip biomechanics still valid? a critical analysis, based on modern methods. Annals of Anatomy 1993;175:203–10.
- Lieberman D, Raichlen D, Pontzer H, Bramble D, Cutright-Smith E. The human gluteus maximus and its role in running. The Journal of Experimental Biology 2006;209(11):2143–55.
- Lund JP, Donga R, Widmer CG, Stohler CS. The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Canadian Journal of Physiology and Pharmacology 1991;69(5): 683–94.
- Lyons K, Perry J, Gronley JK, Barnes L, Antonelli D. Timing and relative intensity of hip extensor and abductor muscle action during level and stair ambulation. Physical Therapy 1983;63(10):1597–605.
- McAlindon TE, Cooper C, Kirwan JR, Dieppe PA. Determinants of disability in osteoarthritis of the knee. Annals of the Rheumatic Diseases 1993;52: 258–62.
- Murray MP, Sepic SB. Maximum isometric torque of hip abductor and adductor muscle. Physical Therapy 1968;48:1327–35.
- National Collaborating Centre for Chronic Conditions. Osteoarthritis: national clinical guideline for care and management in adults. London: Royal College of Physicians; 2008.
- Pfirrmann CWA, Notzli HP, Dora C, Hodler J, Zanetti. Abductor tendons and muscle assessed at MR imaging after total hip arthroplasty in asymptomatic and symptomatic patients. Radiology 2005;235:969–76.
- Sahrmann S. Diagnosis and treatment of movement impairment syndromes. St. Louis: Mosby; 2002.
- Sims K, Richardson CA, Brauer SG. Investigation of hip abductor activation in subjects with clinical unilateral osteoarthritis. Annals of the Rheumatic Diseases 2002;61:687–92.
- Sims K. The development of hip osteoarthritis: implications for conservative management. Manual Therapy 1999;4:127–35.
- Smidt N, de Vet HCW, Bouter LM, Dekker J, for the Exercise Therapy Group. Effectiveness of exercise therapy: a best evidence summary of systematic reviews. Australian Journal of Physiotherapy 2005;51:71–83.
- Stern JT. Anatomical and functional specializations of the human gluteus maximus. American Journal of Physical Anthropology 1972;36:315–40.
- Stern JT, Pare EB, Schwartz JM. New perspectives on muscle during locomotion. Electromyographic studies of rapid and complex behaviours. Journal of the American Orthopedic Association 1980;80(4):287–91.
- Stokes M, Young A. The contribution of reflex inhibition to arthrogenous muscle weakness. Clinical Science 1984;67(1):7–14.
- Tak E, Staats P, van Hespen A, Hopman-Roc M. The effects of an exercise programme for older adults with osteoarthritis of the hip. The Journal of Rheumatology 2005;32:1106–13.
- Taylor HL, Jacobs DR, Schucker B, Knudsen J, Leon AS, Debacker G. A questionnaire for the assessment of leisure time activities. Journal of Chronic Diseases 1978;31:741–55.
- Teshima K. Hip abduction force in osteoarthritis of the hip. Acta Medica Nagasakiensia 1994;39(3):21–30.
- Tichy M, Grim M. Morphogenesis of the human gluteus maximus muscle arising from two muscle primordia. Anatomy and Embrology 1985;173(2): 275–7.
- van Baar M, Dekker J, Oostendorp R, Bijl D, Voorn T, Bijlsma J. Effectiveness of exercise in patients with osteoarthritis of hip or knee: nine months' follow up. Annals of the Rheumatic Diseases 2001;60:1123–30.
- Wallwork TL, Hides JA, Stanton WR. Intrarater and interrater reliability of assessment of lumbar multifidus muscle thickness using rehabilitative ultrasound imaging. Journal of Orthopedic and Sports Physical Therapy 2007;37(10): 608–12.
- Williams P, Warwick R, Dyson M, Bannister L. Grays anatomy. 37th ed. Edinburgh: Churchill Livingstone; 1989.
- Zakas A. Bilateral isokinetic peak torque of quadriceps and hamstring muscles in professional soccer players with dominance on one or both two sides. Journal of Sports Medicine and Physical Fitness 2006;46:28–35.
- Zhang W, Moskowitz RW, Nuki G, Abramson S, Altman RD, Arden N, et al. OARSI recommendations for the management of hip and knee osteoarthritis, Part II: OARSI evidence-based, expert consensus guidelines. Osteoarthritis and Cartilage 2008;16:137–62.