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Title: Coordination of deep hip muscle activity is altered in symptomatic femoroacetabular impingement

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Abstract

Diagnosis of femoroacetabular impingement (FAI) is increasing, yet the associated physical impairments remain poorly defined. This morphological hip condition can cause joint pain, stiffness, impaired function, and eventually hip osteoarthritis. This exploratory study compared coordination of deep hip muscles between people with and without symptomatic FAI using analysis of muscle synergies (i.e. patterns of activity of groups of muscles activated in synchrony) during gait. Fifteen individuals (11 males) with symptomatic FAI (clinical examination and imaging) and 14 age- and sex-comparable controls without morphological FAI underwent testing. Intramuscular fine-wire and surface electrodes recorded electromyographic activity of selected deep and superficial hip muscles. A non-negative matrix factorization algorithm extracted three synergies which were compared between groups. Information regarding which muscles were activated together in the FAI group (FAI group synergy vector) was used to reconstruct individual electromyography patterns and compare groups. Variance accounted for (VAF) by three synergies was less for the control (94.8 (1.4)%) than FAI (96.0 (1.0)%) group (P=0.03). VAF of obturator internus was significantly higher in the FAI group (P=0.02). VAF of the reconstructed individual electromyography patterns with the FAI or control group vector were significantly higher for the FAI group (P<0.01). Following reconstruction, VAF of quadratus femoris was significantly more reduced in controls (P=0.04), indicating greater between-subject variability. Coordination of deep hip muscles in the synergy related to hip joint control during early swing differed between groups. This phase involves movement towards the impingement position, which has relevance for the interpretation of synergy differences and potential clinical importance.

Key Words: Femoroacetabular impingement, hip joint, muscle coordination, gait, electromyography

INTRODUCTION

The prevalence of symptomatic femoroacetabular impingement (FAI) appears to be increasing, yet the associated physical impairments remain poorly defined (1). FAI is a morphological hip disorder that can cause joint stiffness, pain, muscle weakness, and impaired performance in younger active adults (2-4). An association with structural damage and future development of hip osteoarthritis has been proposed (2, 3). Structural FAI is also frequently identified in individuals with no symptoms (5). Whether hip joint biomechanics are altered during walking in symptomatic FAI is unclear (6-10). Although small differences in range of motion (ROM) have been universally reported, there is little evidence for altered hip kinetics (11). Hip muscle activity during gait in symptomatic FAI has not been studied but may provide further insight into the physical impairments.

Although the role of anatomical factors in FAI is clear, the potential contribution of altered muscle coordination to functional deficits and/or symptoms, or whether it contributes to post-operative physical impairments (e.g. altered lower limb biomechanics during walking and stair climbing) (6, 9), has not been investigated. The deep hip external rotator and extensor muscles may be relevant to consider in aetiology, perpetuation or progression of FAI as their mechanical actions directly oppose impingement (12) and they have a potential role in hip joint stability (13). Although some aspects of the deep hip muscles function can be inferred from anatomy (12) and strength measures, recordings of activity are necessary to assess neuromuscular function during gait in those with and without FAI. Few studies have investigated

deep hip muscle function in any context (14-16) due to the necessary invasive recording methods.

An inherent problem in studies of muscle activity is how to interpret the activity from many muscles involved in a function. This is particularly relevant in the context of FAI as multiple small muscles (including deep hip external rotator muscles) can contribute to hip control, including avoidance of the potentially provocative position of hip joint impingement (internal rotation in deep flexion (2)). Analysis of muscle synergies can simplify interpretation of action of multiple muscles. Muscle synergies involve groups of synchronously activated muscles and have been proposed as a simplified neural control mechanism (17, 18). In gait, muscles with similar biomechanical function tend to activate in synchrony and distinct muscle synergies have been identified related to functional sub-tasks of the gait cycle (e.g. heel strike, swing) (19-21). By extracting muscle synergies, electromyographic (EMG) activity recorded over multiple muscle sites can be reduced to a more interpretable limited set of muscle synergies and their corresponding activation patterns (18). Although robust synergies have been identified during dynamic functions in healthy individuals (18, 19, 21), they can differ secondary to sports training (22-24) and neuropathology (brain and spinal cord injury (25, 26)). In gait, differences in synergies between individuals/groups can be gleaned from the information related to the muscles that are activated together in synchrony and the activity pattern these muscles have across the gait cycle (27).

This exploratory study aimed to analyse muscle synergies during gait to compare the coordination of deep hip muscles between individuals with and without symptomatic FAI. We hypothesised that symptomatic FAI would involve an altered motor coordination strategy to

control the hip joint, and this would be most apparent when moving in the direction of impingement.

METHODS

Participants

Fifteen volunteers aged 18-33 years diagnosed with cam-type or combined FAI were recruited to participate in this exploratory cross-sectional study (level 3 evidence). A convenience sample of participants was used due to the invasive nature of the measurement procedures and the fact that there are no relevant published data in this population upon which an a priori sample size could be calculated. All participants were scheduled for arthroscopic surgery and identified by screening the surgical records of the study orthopaedic surgeon (JO). Participants had definitive signs of FAI on imaging ((X-ray and/or magnetic resonance imaging (MRI)); alpha angle > 55° (cam FAI), and lateral centre edge angle > 39° and/or positive crossover sign (combined FAI)) (28, 29) and tested positive for a clinical impingement test (2). The indication for surgery was persisting symptoms, activity limitation for at least three months, with no significant improvement following conservative treatment. Participants with bilateral FAI were tested on the more symptomatic side. Potential participants were excluded if they had only pincer-type FAI. The less common pincer-type, characterized by excessive acetabular coverage (2), typically presents in older individuals (30-40 years) (5), and likely presents with unique neuromuscular strategies in the absence of a bony deformity of the femoral head. Other exclusion criteria included: (i) any history of hip surgery; (ii) moderate/severe radiographic osteoarthritis, as determined by the treating physician; (iii) lower limb injury or pain limiting function in the past

month; or (iv) other forms of arthritis, diabetes, cardiac circulatory conditions that limit everyday activities.

Fifteen healthy control participants with no history of hip/groin pain, or hip joint surgery were recruited from the community. Where possible, control participants were comparable to the FAI group with respect to age, sex, activity level, and leg dominance. Control participants underwent a standard hip MRI with a 3-Tesla MR scanner (Siemens Magnetom Triotim syngo MR B17) and a 16-channel body coil (coupled with a Siemens Spine array) to ensure they did not have morphological FAI. The alpha angle was measured on the oblique sagittal plane MRI (30, 31); the coronal plane was used to measure the lateral centre edge angle (CEA); where necessary, the localizer sequence was used to correct for pelvic obliquity (30). Angles were measured using OsiriX imaging software (Pixmeo SARL, Switzerland) and the Orthopaedic Studio v1.2 Plugin (Spectronic AB, Helsingborg, Sweden). Participants with alpha angles $< 50°$ and centre edge angles $< 40°$, commonly considered to represent "normal" (28, 29), were eligible to participate.

Participants provided written informed consent. The institutional ethics committee approved the study. One control participant was excluded after they were unable to have the necessary screening MRI. Participants were also involved in another study (10).

Procedures

Participants completed six gait trials (three in each direction) across a 10-meter walkway at a preferred, self-selected walking speed wearing standardised footwear (Dunlop Volley, Pacific Brands, Australia). Motion analysis data were collected using a Vicon MX 12-camera motion analysis system (Oxford, UK) at 120 frames/s. Reflective markers were applied to lower

limbs bilaterally according to Besier et al. (32). Two embedded OR6-6 force platforms (AMTI Inc., Watertown, USA) collected ground reaction force data at 3000 samples/s. Gait cycle times were determined from force plate contacts and marker trajectory data.

Electromyography was recorded with a Noraxon DTS 2400 wireless telemetry system (Noraxon, AZ, USA) from eight muscles of the study limb (symptomatic leg for participants with FAI; leg with imaged hip for controls). Intramuscular electrodes were used to record activity from deep hip muscles that have an external rotator function (amongst others): posterior gluteus medius (GM), piriformis (PI), obturator internus (OI), and quadratus femoris (QF). Bipolar fine-wire EMG electrodes were fabricated from 75µm Teflon-coated stainless steel wire using established methodology (33). Wires were threaded into a hypodermic needle (Terumo, Somerset, USA, 0.65 x 70 mm); 1 mm of insulation was removed to form the recording surface; exposed tips were bent back at 1.5 and 4 mm to form hooks. Fine wire electrodes were inserted with ultrasound guidance. Standard sterile procedures were used.

Surface electrodes were placed over the muscle bellies of semimembranosus (SM), upper gluteus maximus (UG), sartorius (SA), and tensor fascia latae (TF). The skin was cleaned with alcohol and shaved to reduce impedance prior to attaching Ag/Ag-Cl electrodes (10 mm diameter; Covidien, Kendall foam electrodes, Dublin, Ireland). Electrodes were placed in a bipolar configuration (inter-electrode distance: 20 mm) along the orientation of the muscle fibres.

EMG signals were pre-amplified 500 times, low pass filtered (common mode rejection ratio >100 dB; input impedance of >100 Mohm), wireless-transmitted then output via Noraxon DTS Analog Module, and digitized with 16-bit resolution at 3000 sample/s using the Vicon

analog-to-digital converter. A high sampling rate was used because of the higher frequency of intramuscular recordings (because of proximity to the motor unit and lesser filtering by soft tissue) compared to surface recordings. A fixed 312ms wireless transmission delay was set and corrected in analysis.

A modified Tegner Activity Scale was administered to grade level of physical activity in all participants (34). Physical function of participants with FAI was assessed with the international Hip Outcome Tool (iHOT-33) (35) and the Copenhagen Hip and Groin Outcome Score (HAGOS) (36). Hip pain experienced during gait testing was assessed in the FAI cohort immediately following walking trials using an 11-point self-reported Numerical Rating Scale (range 0-10; 0 = no pain, 10 = worst pain possible).

Data Analysis

Data analysis was undertaken using custom programs written in Matlab, version 2013a (Mathworks, USA). A vertical contact force threshold of 20 N was used to detect time of heel strike and toe-off. EMG data were high-pass filtered at 50 Hz (fine-wire EMG) or 20 Hz (surface EMG) with a dual-pass, zero-lag fourth order Butterworth filter. EMG signals for all completed gait trials were visually inspected for artefacts by three researchers. Knowledge of EMG waveform characteristics was used to identify trials without an acceptable signal-to-noise ratio. Three gait cycles (from heel strike to subsequent heel strike) were selected for analysis. As recordings from all muscles are required to undertake synergy analysis, it was decided to exclude data for UG, SA, and TF from the model. Movement artefact, an inherent issue with EMG data (37), and low signal-to-noise ratios led to rejection of data from at least one of these muscles for many participants. For three control participants, three good cycles were not available for all five

included muscle sites, so they were excluded from further analysis. EMG data were then rectified and smoothed using a 6 Hz dual-pass, zero-lag fourth order, low-pass Butterworth filter given our interest in the slower changing characteristics of the muscle recordings that emerge from summation of many individual motor unit firings during gait. These lower frequency EMG components relate primarily to muscle force and movement (38). Data were subsequently amplitude-normalized to the average of the peak values across the three cycles, as done previously in muscle synergy analysis (19).

A non-negative matrix factorization was applied using the Lee and Seung algorithm (39) to extract the muscle synergies for five muscles, as previously described (24, 40) (Figure 1). EMG was selected over one gait cycle and then spline interpolated to 101 time points. This was done for three cycles, and concatenated to make up 303 points per muscle per participant. The resulting EMG matrix (*E*) was 5 rows and 303 columns. Non-negative matrix factorization results in *muscle synergy vectors*, which correspond to the relative weighting of each muscle within each synergy and *synergy activation coefficients*, which represent the activity pattern across the gait cycle of the muscle synergy (24). The mean total variance accounted for (VAF) was calculated (40, 41) for each participant by varying the number of extracted synergies between 1 and 5. The lowest number of synergies that accounted for more than 90% of the variance for all participants was selected (42). The same number of synergies (i.e. three) was extracted for each participant to enable comparison between groups. The order of output of individual synergies for each participant may differ following the application of the non-negative matrix factorization algorithm. For this reason, it was necessary to re-order the synergies by visual inspection based on comparison of the synergy activation coefficient and/or the muscle synergy vectors to a representative participant.

In order to make comparisons between groups, a cross validation procedure was used (19, 22, 24, 42). Individual EMG patterns for all FAI participants were concatenated to represent the FAI group EMG in a 5 x 15 x 303 = 5 x 4545 matrix. From this FAI group EMG matrix, the group synergy vector was extracted. The FAI group synergy vector was then used to reconstruct the individual EMG patterns for each participant in both the control and FAI groups. In doing so, the FAI group synergy vector (*W*) was held fixed in the non-negative matrix factorization algorithm, and the synergy activation coefficients (*C*) were free to vary. C matrix was initiated with random values, and then iteratively updated until convergence.

$$E = WC + e$$

From the EMG reconstruction, the total VAF and the VAF of each individual muscle reconstruction were calculated. The VAF reduction was calculated as the difference in VAF resulting from the original factorization and VAF resulting from reconstruction with the FAI group vector. Greater reduction in VAF equates to a poorer reconstruction/less similarity with the FAI group synergy vector. The same cross validation procedure was performed where the mean control group synergy vector was used to reconstruct the individual EMG patterns for each participant in both the control and FAI groups.

Statistical Analysis

Statistical analyses were performed using Statistical Package for the Social Sciences (SPSS), version 22 (IBM, New York, USA). The Shapiro-Wilk test was used to explore all data for normality. Between-group comparisons for VAF values were made using independent t-tests and Mann-Whitney U tests where required. Spatiotemporal and demographic variables were

examined for between-group differences using independent t-tests and Pearson's chi-square. Significance was set at $P<0.05$.

FIGURE 1 HERE

RESULTS

FAI and control groups were comparable for age, body mass index, sex, and dominant leg (Table 1). Sporting activity level (Tegner activity scale) was significantly higher in the control group at the time of testing ($P = 0.03$). Activity level was reported in the "physical fitness, moderate to strenuous work" category for both groups. Participants with FAI had symptoms for 5-48 months (mean 28) prior to testing. The iHOT-33 and HAGOS subcategory scores are reported in Table 1. Participants with FAI reported a mean pain of 1 (range 0-7) on the NRS after completing the walking trials. Eight participants with FAI (53%) reported bilateral symptoms with concurrent radiographic findings. There were no significant between-group differences for walking speed or other spatiotemporal gait variables (Table 2).

Individual Synergy Analysis

Individual EMG patterns are shown in Figure 2. Three muscle synergies were extracted for all participants, which resulted in a lower VAF for the control (94.8 ± 1.4%) than FAI (96.0 ± 1.0%) groups ($P = 0.03$). The cumulative percentage of VAF for each group is depicted in Figure 3. The VAF for individual muscles ranged from 92.7 ± 5.3% (QF - control) to 97.5 ± 1.8% (OI -

FAI) (Table 3). The VAF for OI was significantly higher in the FAI than control group (P = 0.02).

TABLE 1 HERE

TABLE 2 HERE

FIGURE 2 HERE

FIGURE 3 HERE

TABLE 3 HERE

Synergy activation coefficients for all participants and the average difference between control and FAI participants are depicted in Figure 4. The synergy activation coefficients (Figure 4, rows 1 and 2) and the synergy vectors determine the overall muscle activity pattern. When examined together, they can be related to biomechanical sub-tasks of the gait cycle. FAI and control group mean synergy coefficients and vectors are depicted in Figure 5. *Synergies 1* and *2*. include a major contribution from SM and GM, which have been included in other studies (17-19). *Synergy 1* primarily involved SM with contribution from GM, and was active during late swing/early stance phase to decelerate the swing leg and prepare for weight acceptance. *Synergy 2*, which mainly involved GM and piriformis, was active during the weight acceptance phase of the gait cycle. *Synergy 3* was active during swing, primarily involved the deep hip muscles (OI,

QF), and is likely to assist in hip joint control through the early swing phase. The control group demonstrated substantial inter-subject variability with respect to *Synergy 3* (Figure 4: row 1 column 3), which contrasts the more homogeneous pattern observed for the FAI group (Figure 4: row 2 column 3). The difference between group synergy coefficients is particularly evident during early swing (Figure 4: row3 column 3). Mean synergy vectors in Figure 5 illustrate that OI is a primary contributor to this synergy; VAF of OI was increased in the FAI group ($P = 0.02$).

EMG reconstruction with FAI group synergy vector

The reconstructed individual EMG patterns using the FAI group synergy vector for all participants resulted in a VAF that was less for the control (90.2 ± 2.4%) than FAI (93.3 ± 2.3%) groups ($P < 0.01$). Although, values above 90% demonstrate that the synergies are robust across groups, the VAF was reduced by a greater amount for the control (4.6 ± 2.3%) than FAI (2.7 ± 1.7%) group ($P = 0.02$). Both the significantly lower VAF (mean difference 3.1 [95% CI 1.1 to 5.0] %) and greater reduction of VAF for the control group than the FAI group (when data were reconstructed using the FAI group synergy vector) indicate that synergies differed between groups. Synergies were more similar among FAI patients than among individuals in the control group. This could be related to either: (i) more variation between individual in the control group; or (ii) different synergistic patterns between groups. Reduced VAF per muscle (Table 4), resulting from reconstruction with the FAI group vector, reveal a significantly greater reduction in VAF for QF in the control than FAI group ($P = 0.04$).

EMG reconstruction with control group synergy vector

The reconstructed individual EMG patterns using the control group synergy vector for all participants resulted in a VAF that was significantly lower (mean difference 2.5 [95% CI 0.3 to 4.6] %) for the control (90.4 ± 2.3%) than FAI (92.9 ± 2.8%) groups (P = 0.03). Synergies were again more similar among individuals in the FAI group than among individuals in the control group, even when using the control group synergy vector for reconstruction. Consequently, the difference in synergies between groups must be best explained by greater variation within the control group. Reduced VAF per muscle resulting from reconstruction with the control group vector reproduced the finding of significantly greater reduction in VAF for QF in the control than FAI group (P = 0.04) (Table 4). This suggests greater between-subject variability in the control than FAI group for this muscle.

FIGURE 4 HERE

FIGURE 5 HERE

TABLE 4 HERE

DISCUSSION

The aim of this exploratory cross-sectional study was to determine whether coordination of the group of hip muscles that have the anatomy to oppose the impingement position in FAI is altered in people with symptomatic FAI. This was achieved through examination of muscle synergies involving these muscles during gait. Between participants with FAI and an

asymptomatic control group with no evidence of morphological FAI, findings demonstrate differences in several features of muscle synergies that may have relevance for people with FAI. Differences were most apparent for the contribution of the deep hip rotator muscles involved in a synergy related to control of the hip during the early swing phase of gait (*Synergy 3*). A novel observation was that the control group demonstrated greater inter-subject variability with respect to this synergy than the relatively homogeneous pattern of the participants with FAI. This was most apparent for activation of OI and QF, which are important hip external rotator muscles. An interpretation of this observation is that whereas individuals without FAI have the flexibility to vary muscle activation of these particular deep muscles, those with symptomatic FAI have tightly constrained activation of these muscles as the hip initiates movement towards the provocative position.

During gait in both control and FAI groups, three muscle synergies accounted for the majority of variability in the EMG signals of five lower limb muscles relevant for hip control. This is the first time that deep hip muscle synergies have been reported. Although the synergies are robust and consistent across groups, the variance accounted for by three synergies was significantly higher in the FAI group. The control group demonstrated substantial inter-subject variability with respect to *Synergy 3* (lower VAF of OI, a primary contributor to this synergy). Reconstructions with both group synergy vectors also highlight that synergies are less variable in the FAI group. Both reconstructions reduced the VAF for QF by a greater amount for controls than FAI participants. Synergies were more similar among individuals in the FAI group than among individuals in the control group, regardless of which group mean was used to reconstruct the patterns. This confirms that the synergies differed between groups, and importantly, that this difference can be attributed to greater variability in the controls. Individuals without FAI appear

less constrained to use these muscles in a specific manner during gait which suggests muscles are activated flexibility to adapt to stride-by-stride changes in task constraint.

Synergies have been associated with the functional sub-tasks of the gait cycle in healthy individuals (19-21). Our new data add to the limited previous investigation of deep hip muscle function (14, 15) and previous synergy findings for other muscle groups (17-19). Similarities are evident in synergies related to late swing leg deceleration/early stance loading control (*Synergy 1*) and weight acceptance (*Synergy 2*), explained by the inclusion of SM and GM, respectively (19). The role of GM during these synergies may relate to frontal plane control of the pelvis relative to the femur; it's contribution to external rotation is highly dependent hip flexion (43).

In the present cohort, the synergy related to hip joint control during early swing (*Synergy 3*) added a VAF of >3% in both groups. Although synergistic activity of these muscles has not been examined previously, a synergy is typically considered a relevant contributor to coordination if its VAF is greater than 3-5% (17, 44). This synergy was most active during early swing, with primary contributions from the OI and QF muscles, which are considered to contribute to external rotation and joint stabilization, with QF also contributing to adduction (12). It is plausible that individuals with FAI activate these deep muscles consistently during early swing in the extended position as it transitions towards the provocative flexion direction for joint stability. Previous studies in healthy individuals, although limited, indicate that OI activation is greatest during static hip extension (15); its activity in gait has not been studied. QF is typically active in stance with minimal activation during swing (16). Given that QF is not in an optimal functional position during early swing (45), the altered coordination of the FAI group, which includes a large contribution from QF, may indicate an effort to increase stability of the pathological joint. Given that FAI patients reported minimal pain during the gait assessment, it is

unlikely that these coordination strategies are a direct consequence of nociceptive input. However, contemporary theories of motor adaptation to pain indicate motor control is often modified for protection of a painful or injured region (46). FAI may invoke altered coordination of the deep hip muscles to avoid provocation as the hip moves to the symptomatic position.

Although highly constrained activation of the deep hip muscles may have protective benefit in the short term, there may be long term consequences with respect to joint loading. It is unlikely that the deep hip muscles are capable of generating substantial joint forces compared to the larger primary muscles (47). However, less variation equates to less load sharing between muscles and potentially less variation in the distribution of load within the joint. This could lead to areas that are consistently under- and over-loaded, with implications given that individuals with symptomatic FAI are at risk for structural damage and early osteoarthritis (2, 3). Further investigation is required to elucidate whether the adopted motor coordination strategy exhibited by individuals with symptomatic FAI is a protective mechanisms imparted to alleviate symptoms, and further, whether hip structure and function are adversely affected.

FAI is now commonly treated in clinical practice with surgical and conservative interventions, but the evidence of efficacy remains limited (1). The results of this exploratory study add to the emerging body of evidence that is identifying differences in hip function between people with and without FAI. Although hip strength deficits (48) and hip rotator imbalances (49) have been identified in individuals with symptomatic FAI, this is the first study to assess neuromuscular function of the hip muscles during gait in this population. Recent lower limb surface EMG data from a squat task in individuals with FAI identified increased levels of co-contraction between the hamstrings and TFL (50). Interpretation of this finding could also point towards increased hip joint stability/protection.

Strengths of this study include the use of an asymptomatic comparison group without morphological FAI, and use of a proven EMG analysis technique (18). This is the first study to assess neuromuscular function of deep hip muscles in individuals with symptomatic FAI. Published data documenting activation of the deep hip muscles is minimal (15, 16), likely due to the necessary invasive recording methods (14). Although our sample size may be considered small, it must be considered within the context of the measurement procedures and the fact that there are no relevant published data in this patient population upon which an a priori sample size could be calculated. Nevertheless, the small sample size may mean that we failed to detect further significant differences that are actually present, and the cross-sectional design makes it impossible to determine whether hip muscle coordination is altered prior to or following pathology development. There are also some methodological considerations. The potential for cross talk between EMG channels was minimized with the use of intramuscular electrodes inserted with ultrasound guidance. Data from three surface muscles were excluded to ensure small between-participant variations and interpretable synergy coefficients. Therefore, the analysis was restricted to recordings from five muscles. EMG data were normalized to the mean of the peak activity for each muscle across the three gait cycles. Although consistent with previous synergy analysis, this normalisation limits interpretation to relative muscle activity and not the absolute level of EMG activity (i.e. relative to maximum voluntary contraction). For this reason interpretation of joint loading is limited and an association with hip muscle weakness cannot be inferred. Further, the three cycles used in this analysis was low relative to some other studies (18, 19) and more trials may have improved validity of synergies. The reported synergies are a representation of the five hip muscles included in the analysis. The data does not support interpretation beyond this, nor is it meant as a generalization of all lower limb muscle synergies

during gait in individuals with symptomatic FAI. Although our findings suggest that further investigation of the deep hip muscles is warranted, future work could be strengthened by larger patient samples and the inclusion of recordings from additional hip-spanning muscles (i.e. larger primary movers (e.g. gluteus maximus, adductor magnus)).

CONCLUSION

Individuals with symptomatic FAI demonstrate altered coordination of deep hip muscles during gait compared to asymptomatic controls without FAI. Changes were particularly evident in the synergy related to control of the hip joint during early swing. Although the implications of these findings for symptoms and function are not yet clear, they could plausibly relate to enhanced protection for the hip, but with possible long term consequences. Future studies should examine patients prospectively and post-operatively to establish whether treatments targeted at these features would be beneficial.

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Figure Captions

Figure 1. Extraction of muscle synergies from a representative FAI participant. Three gait cycles with good electromyographic (EMG) quality were selected. (a) EMG recordings from semimembranosus (SM), posterior gluteus medius (GM), piriformis (PI), obturator internus (OI) and quadratus femoris (QF) were high-pass filtered at 50 Hz (fine-wire EMG) or 20 Hz (surface EMG). (b) The high pass filtered EMG was rectified and smoothed with a low-pass filter (see methods). (c) Data were amplitude-normalized to the mean of the peaks that occurred within each gait cycle. Values across cycles were interpolated to 101 points per cycle (mean of three cycles shown in c). (d) Non-negative matrix factorization was applied. In this iterative optimization process, (e) the data is approximated by three *muscle synergy vectors (H)*, which correspond to the relative weighting of each muscle within the synergy and three *synergy activation coefficients (W),* which represent the activity pattern across the gait cycle of the synergy. (f) EMG data were then reconstructed using the linear combination of the synergy coefficients and vectors (W x H). (g) The quality of the reconstruction was assessed by calculating the variance accounted for ($VAF = 1 - (\sum error^2 \div \sum EMG^2)$). a.u., arbitrary units.

Figure 2. Electromyographic (EMG) patterns from five muscles for A control (n=11) and B femoroacetabular impingement (FAI) (n=15) participants during a gait cycle. Ensemble-averages are displayed as a thick line. Each profile represents an EMG pattern averaged across three gait

trials. EMG data are normalized to the mean of the peak for each muscle across the three trials. Note the relative similarity in pattern of activity between participants of some muscles for the control (e.g. SM, GM) and FAI (e.g. SM, OI, QF) groups, whereas other muscles were more variable, particularly in the control group (e.g. OI)

Figure 3. Mean percentage of variance accounted for (VAF) with respect to the number of extracted synergies. Three synergies were extracted for all participants in the control (CON) group (95.3 ± 1.9%) and of some muscles were similar (FAI) group (96.0 ± 1.0%).

Figure 4. Synergy activation coefficients for control (n=11, top row) and FAI (n=15, middle row) participants during gait and the average difference between control and FAI participants (mean of FAI - mean of control, bottom row). a.u., arbitrary units.

Figure 5. Extracted muscle synergies: synergy activation coefficients and muscle synergy vectors for control (red) and FAI (blue) groups during gait; a.u., arbitrary units.

Tables

Table 1. Demographic and clinical characteristics of the femoroacetabular impingement (FAI) and control groups.

Characteristic	**FAI group n=15**	**Control group n=11**
Age (years)	24.7 (4.9)	26.6 (4.9)
Males, n (%)	11 (73%)	8 (73%)
Height (cm)	176 (9)	177 (8)
Body mass (kg)	76.0 (11.8)	72.9 (11.6)
Body mass index (kg/m^2)	24.4 (2.5)	23.2 (2.1)
FAI type (cam:combined)	11:4	
Test hip (right:left)	9:6	8:3
Dominant side tested, n (%)	10 (67%)	7 (64%)
Bilateral FAI (yes:no)	8:7	
Symptom duration (months)	27.9 (15.0)	
Physical activity level (Modified Tegner Scale)¶	5.2 (2.1)	**6.8 (0.9)**
The international Hip Outcome Tool (iHOT-33)†	51.9 (23.0)	
Copenhagen Hip and Groin Outcome Score (HAGOS)†		
Symptoms	53.6 (21.0)	
Pain	66.5 (18.2)	
ADL	70.3 (22.8)	

	Sport	49.6 (19.5)
	Participation	33.3 (26.2)
QOL		41.7 (20.1)
Pain during gait testing (Numerical Rating Scale) [δ]		0.9 (1.8)

Values are mean (standard deviation) unless otherwise stated; **bold indicates significant difference between groups** $P < 0.05$; [¶]Tegner scale - 0 = disability and 10 = competitive sport at the professional level; [†]iHOT-33 and HAGOS scales – 0 = extreme hip and/or groin problems and 100 = no hip and/or groin problems; [δ]Numerical Rating Scale – 0 = no pain and 10 = worst pain possible; ADL - activities of daily living; QOL - quality of life.

Table 2. Gait variables for femoroacetabular impingement (FAI) and control groups.

Spatiotemporal Variable	FAI (n=15)	Control (n=11)	P - value
Walking speed (m/s)[*]	1.3 (0.2)	1.4 (0.3)	0.51
Stride length (m)	1.5 (0.1)	1.5 (0.2)	0.19
Step length (m)	0.73 (0.07)	0.78 (0.10)	0.18
Cadence (strides/min)	55.4 (3.3)	55.9 (5.3)	0.77

Values are mean (standard deviation); [*]Mann-Whitney U test

Table 3. Variance accounted for (VAF) for each muscle for three extracted synergies.

Muscle	FAI VAF (%) (n=15)	Control VAF (%) (n=11)	P - value
Semimembranosus (SM)	96.8 ± 3.2	96.9 ± 3.6	0.80[*]
Gluteus medius (GM)	93.7 ± 5.6	92.8 ± 3.8	0.26[*]
Piriformis (PI)	95.9 ± 4.7	95.2 ± 2.6	0.10[*]
Obturator internus (OI)	97.5 ± 1.8	93.8 ± 5.4	**0.02**
Quadratus femoris (QF)	93.6 ± 4.1	92.7 ± 5.3	0.61

Values are mean (standard deviation); [*]Mann-Whitney U tests; **bold indicates significance** $P < 0.05$; FAI, femoroacetabular impingement.

Table 4. Change in variance accounted for (VAF) for each muscle when patterns are reconstructed with femoroacetabular impingement (FAI) group (n=15) vector and control group (n=11) vector.

Muscle	FAI group vector			Cont	
	FAI VAF change (%)	Control VAF change (%)	P - value	FAI VAF change (%)	VA

Semimembranosus (SM)	-2.9 ± 3.2	-2.8 ± 3.5	0.61*	-1.5 ± 3.0
Gluteus medius (GM)	3.7 ± 6.3	6.0 ± 9.5	0.61*	4.1 ± 5.9
Piriformis (PI)	3.0 ± 4.0	3.3 ± 3.4	0.68*	2.7 ± 4.6
Obturator internus (OI)	2.9 ± 3.1	2.6 ± 4.4	0.88	2.5 ± 2.7
Quadratus femoris (QF)	5.5 ± 3.4	16.6 ± 16.5	**0.04***	6.4 ± 4.7

Positive values indicate a reduction in VAF. Values are mean (standard deviation); *Mann-Whitney U tests; **bold indicates significance** $P < 0.05$.