

Movement Patterns during a Small Knee Bend Test in Academy Footballers with Femoroacetabular Impingement (FAI)

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Abstract

Background: Femoroacetabular impingement (FAI) is common in footballers and causes hip pain, which may arise from abnormal morphologic features involving the proximal femur and/or acetabulum. Early detection and treatment are important to prevent the development of osteoarthritis (OA). Despite extensive publications on FAI, little is known about hip movement patterns associated with FAI, which may indicate mechanisms of dysfunction to inform development of effective interventions.

Design: Observational pilot study

Methods: Nine male academy footballers aged 12–18 years with hip/groin pain, diagnosed with FAI on magnetic resonance imaging, were studied. The hip and pelvis were observed whilst the participant performed a small knee bend test, to see if any abnormal movement patterns were present.

Findings: In all nine cases, abnormal movement patterns were observed clinically. Participants were unable to control hip flexion in one or more aspects, mostly seen as the trunk leaning forwards and the hip moving into increased flexion. Participants also demonstrated poorly controlled hip medial rotation.

Discussion: These preliminary findings suggest impaired movement control exists in academy footballers with symptomatic FAI. Identifying and classifying these movement faults may prove necessary for effective prevention and management of symptoms by controlling movement adaptations. Further studies are warranted to validate these findings against motion analysis technology and muscle activity using electromyography, and to further understand the mechanisms of movement dysfunction. Since FAI is a strong predictor in the development of hip OA, it is vital that strategies are developed to prevent FAI and its progression to OA.

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Conflict of interest statement:

Disclosure: Sarah Mottram and Mark Comerford are Co-Directors of Movement Performance Solutions Ltd, which educates and trains sports, health and fitness professionals to better understand, prevent and manage musculoskeletal injury and pain that can impair movement and compromise performance in their patients, players and clients. None of the other authors has any conflict of interest to declare

Introduction

Femoroacetabular impingement (FAI) is a hip condition involving abnormal morphologic features of the proximal femur and/or the acetabulum (Ganz et al. 2003). Cam and pincer are two types of FAI described. Cam impingement occurs when the femoral head has an abnormally large radius, with a loss of the normal spherical junction between the femoral head and neck, while pincer impingement involves over-coverage of the acetabulum (Ito et al. 2001). In both cam and pincer impingement, bony contact occurs with the combined movement of hip flexion, adduction and internal rotation (Ganz et al. 2003). This abnormal contact between the femur and the acetabular rim at the end of hip

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range of motion (ROM), is an increasingly recognised cause of hip pain in young people, resulting in development of deep chondral injuries, labral detachment and a precursor for osteoarthritis (OA) of the hip (Ganz et al. 2003; Beck et al. 2005; Harris-Hayes & Royer 2011; Agricola et al. 2013).

Football places a high demand on the hip joint, as it involves sprinting, jumping and kicking, which subject the hip to high loads and torsional forces; thereby affecting the joint, surrounding capsule, ligaments and associated muscles (Saw & Villar 2004). Cam-type deformities can be recognisable from the age of 13 years and are more prevalent and pronounced in young football players than in their non-athletic peers (Agricola et al. 2011). Monazzam et al. (2013) found that cam and pincer morphology can occur as early as 10 to 12 years of age in a population with no known orthopaedic hip complaints. Professional football players are likely to start sporting activities at a young age (Kapron et al. 2011), therefore high physical demands placed on their joints during the critical stages of hip development may lead to abnormalities consistent with FAI and could cause later OA (Leunig et al. 2007; Agricola et al. 2014). In addition, continued sports participation could cause FAI to become symptomatic, as the increased loading may exacerbate the labral or articular cartilage damage (Kapron et al. 2011).

The bony anatomy causing FAI is common, particularly in active populations (Tibor & Leunig 2012). Prevalence in the general population is 14% to 35%; more frequent in males (Gosvig et al. 2010; Hack et al. 2010) and is as high as 72% in professional footballers (Gerhardt et al. 2012). A cam deformity has been recognized as major risk factor for the development of hip OA (Agricola et al. 2013) and in youth soccer players there was a significant increase in the prevalence of a cam deformity during skeletal maturation (Agricola et al. 2014). In boys aged 12 and 13 years, the prevalence of a flattened head-neck junction increased significantly during follow-up from 13.6% to 50.0% ($p=0.002$) (Agricola et al. 2014); putting them at greater risk of developing OA in later life. Studies on retired professional footballers have shown an increased risk of developing OA in the hip compared to the general population, with an earlier onset of symptoms. However,

the prognosis and identification of those patients who ultimately develop OA is still unclear (Bardakos & Villar 2009; Clohisy et al. 2011).

Studies of hip kinematics, muscle activation and biomechanics associated with FAI (Austin et al. 2008; Kennedy et al. 2009b; Kennedy et al. 2009a; Lamontagne et al. 2009; Lamontagne et al. 2011; Rylander et al. 2011; Morrissey et al. 2012; Hunt et al. 2013), indicate abnormal hip and pelvic movement. However, movement faults contributing to the impairment of the ability to control hip and pelvic movement associated with FAI has not been studied and may indicate mechanisms of dysfunction and inform development of effective interventions. The efficiency of movement control can be evaluated with movement control tests, in which a person is asked to cognitively control movement at a specific joint (e.g. the hip), whilst challenging the ability to maintain this control with movement at an adjacent joint (Comerford & Mottram 2001; Comerford & Mottram 2012; Roberts 2013; McNeill 2014). Such tests of movement control

are suggested for identifying deficits (Mottram & Comerford 2008) and people with pain often fail these tests and demonstrate impaired movement control (Luomajoki et al. 2008; Worsley et al. 2013). Impaired movement control can imply disturbance or abnormality in the movement system (Sahrmann 2002; O'Sullivan 2005). It is based on the basic principle that loss of precise movement is the result of repetition of movements and positions in specific directions with activities (Sahrmann 2002). The loss of this movement precision is proposed to contribute to repeated stresses to tissues, causing alterations in control strategies. Also, it has been suggested that impaired movement control at the hip and pelvis has the potential to produce compensation and injury at other joints (Reiman et al. 2009; Powers 2010). In particular, there is evidence that movement impairments at the hip and pelvis may trigger injuries such as anterior cruciate ligament tears (Hewett et al. 2005), iliotibial band syndrome (Noehren et al. 2007), and patellofemoral joint pain (PFJP) (Powers 2003; Powers 2010). Therefore, improvement in movement

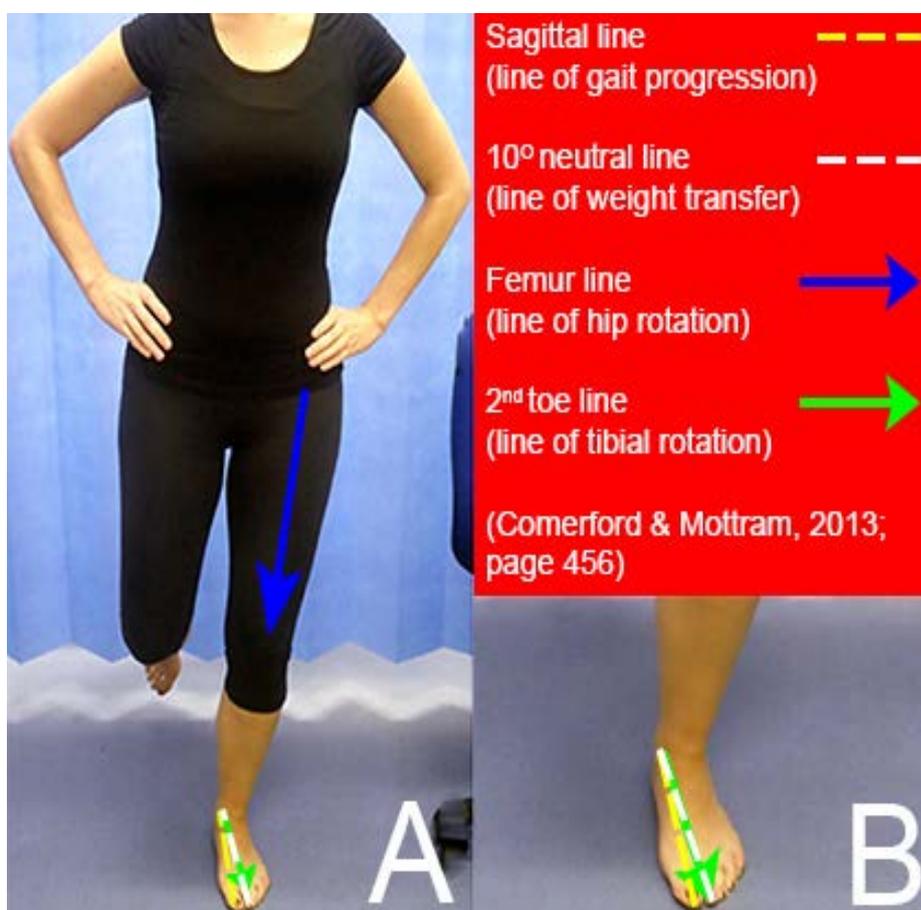


Figure 1. (a) Ideal alignment during Small Knee bend test (b) Foot lines



Figure 2. Small Knee Bend motor control test

control at the hip and/or pelvis may also contribute to the prevention of joint injuries more distally in the kinetic chain. There is a need to explore the association of FAI with pelvic and hip movement patterns. Furthermore, it is unknown whether there are impaired movement control patterns in the unaffected limb of subjects with FAI, similar to the injury-associated muscle imbalance in the unaffected limb of those with chronic groin pain (Morrissey et al. 2012) and hip OA (Sims et al. 2002). Information of this nature could have direct clinical implications for the conservative treatment of FAI. Also, understanding the characteristics and impaired movement patterns of the hip may provide ways to detect and treat FAI early. The present pilot study describes the movement impairments and abnormal movement control patterns observed during a small knee bend (SKB) test, in young footballers with symptomatic FAI.

Methods

A pilot study was carried out to document the movement patterns observed during a unilateral SKB motor control test in young footballers with symptomatic FAI. This test resembles situations of daily life,

making it functional and easily administered by clinicians. The reliability and validity of similar tests used to observe medio-lateral knee motion have been termed the unilateral squat (Chmielewski et al. 2007) and single-limb mini squat (Ageberg et al. 2010).

Participants

Nine footballers aged 12-18 years with unilateral hip or groin pain, diagnosed with FAI on magnetic resonance (MR) imaging were recruited using convenience sampling from an English Premiership football club academy. Exclusion criteria were: hip or groin pain secondary to muscular, lumbar and/or sacro-iliac joint problems; any musculoskeletal, neurological or systemic diseases, or a history of lower limb, pelvic or spinal fractures. Ethical approval was obtained from the Faculty of Health Sciences Ethics Committee, University of Southampton. The purpose of the study and data collection procedure were fully explained to all participants (ages 12-18) and their guardian or parent if they were aged 12-17. Participants over 17 years gave their informed consent, while the parents/guardian signed consent for those participants below 17 years to take part in the study.

Procedure

Each participant was given an introductory and practice trial of the SKB movement control test by the same investigator (NB). The participant stood on one leg, which was placed in a position with the 2nd metatarsal aligned along the 10° neutral line of weight transfer, while the 1st metatarsal and medial border of the foot was in the sagittal plane; ensuring a correct foot position (Figure 1. a and b).

The participant was instructed to flex the knee and dorsiflex the ankle while keeping the heel on the floor. The movement control benchmark was to keep the body weight through the heel, keeping the knee over the 2nd metatarsal, the trunk vertical, without any forward leaning of the trunk or posterior shift of the hips and pelvis i.e. no increase in hip flexion (Figure 2) from a forward lean. The same investigator observed performance of the task for impaired movement control patterns and movement faults were recorded as listed in Table 1.

Table 1. Small Knee Bend motor control test observed faults (Faults 1 to 5 associated with altered hip flexion; Faults 6 and 7 associated with altered hip medial rotation; Faults 5, 8 and 9 may be associated with restrictions of knee and ankle flexion contributing to the altered hip flexion)

Observed Movement Faults	
1	Trunk leans forward
2	Increased hip flexion
3	Anterior pelvic tilt
4	Hips sway back
5	Shift body weight forefoot
6	Functional femoral line falls medial
7	Hip hitching
8	Knees not move past 2nd toe
9	Knee alignment <2cm past toes

Other measures included passive hip internal rotation (IR) range of motion (ROM) tested in prone and recorded using a plurimeter. Ideally there should be 35° hip IR ROM when measured in prone lying (Hoppenfeld 1976 pg 158; Kenyon & Kenyon 2009 pg 61). Also, age,

height, weight, symptom duration and body mass index (BMI) were measured.

Data Analysis

Descriptive statistics were used to describe and summarise the data. The sums of the faults were calculated for the symptomatic and asymptomatic side, dominant and non-dominant side and if the participant performed the test with no observed faults then the test was considered controlled with no impaired movement control.

Results

Sample Characteristics

Participant demographic and clinical data are reported in Tables 2 and 3. Participants were aged between 12 and 18 years with a mean (SD) age of 14.6 (1.9) years, with a mean (SD) height of 167.8 (13.7) centimetres, weight of 60.6 (14.9) kilograms and BMI of 21.2 (2.5). Symptom duration.

Results in Table 3 suggest the passive range of hip IR in prone is reduced on both the symptomatic and asymptomatic sides with a mean (SD) of 31.0° (6.6) and 31.3° (6.3) respectively. Furthermore, five of the nine participants had a positive hip impingement test.

Table 3. Descriptive statistics hip internal rotation range of motion of the symptomatic and asymptomatic side

Participant	Age (years)	Symptomatic Side IR ROM (degrees)	Asymptomatic Side IR ROM (degrees)
1	18	37.0	28.0
2	17	29.0	35.0
3	15	30.0	28.0
4	14	25.0	30.0
5	14	19.0	20.0
6	14	30.0	30.0
7	14	40.0	40.0
8	13	31.0	31.0
9	12	38.0	40.0
Mean	14.6	31.0	31.3
Std. Dev	1.9	6.6	6.3
(min-max)	12-18	19.0-40.0	20.0-40.0

Small Knee Bend Test Descriptive Measurements

Impaired movement control patterns were observed in all nine cases, as detailed in Table 4, which shows that participants were unable to control hip flexion in one or more aspects of hip control. The observed movement faults indicating impaired control of hip flexion were increased hip flexion (7/9 participants on the symptomatic side and 6/9 on the asymptomatic side), trunk leaning forwards

(6/9 participants on both sides), hips swaying back (4/9 participants on both sides), shifting the body weight to the forefoot (5/9 participants on the asymptomatic side and 4/9 on the symptomatic side) and anterior pelvic tilt (1/9 participants on both sides). These faults are all variations of ways individuals exhibit their predisposition for poor control of hip flexion compared to the standard benchmark.

Table 2. Demographic data for participants

Participant	Age (years)	Height (cm)	Weight (kg)	BMI	Period Pain (months)	Symptomatic leg	Dominant leg
1	18	180.0	72.5	22.4	12	Left	Right
2	17	180.5	83.5	25.6	24	Left	Left
3	15	177.0	64.0	20.4	4	Right	Right
4	14	170.5	69.0	23.7	4	Right	Right
5	14	171.8	62.0	21.0	3	Right	Right
6	14	178.0	64.0	20.2	6	Left	Right
7	14	144.8	36.0	17.2	12	Right	Right
8	13	159.0	52.0	20.6	8	Left	Left
9	12	148.5	42.5	19.3	1	Left	Right
Mean	14.6	167.8	60.6	21.2	8.2		
Std. Dev	1.9	13.7	14.9	2.5	7.0		
(min-max)	12-18	144.8-180.5	36.0-83.5	17.2-25.6	1-24		

Table 4. Faults observed for each participant on the symptomatic and asymptomatic side

Test	Faults Observed Symptomatic Side	Faults Observed Asymptomatic Side
Small Knee Bend motor control test	<u>Participant 1</u> Functional femoral line falls medial to 10 degree neutral line. Increase hip flexion Trunk leans forward	<u>Participant 1</u> Increase hip flexion Trunk leans forward Shift body weight forefoot
	<u>Participant 2:</u> Functional femoral line falls medial to 10 degree neutral line. Knees not move past 2 nd toe Increase hip flexion Shift body weight forefoot	<u>Participant 2:</u> Functional femoral line falls medial to 10 degree neutral line. Trunk leans forward Hips sway back Shift body weight forefoot
	<u>Participant 3</u> Knees not move past 2 nd toe Increase hip flexion Hips sway back	<u>Participant 3</u> Knees not move past 2 nd toe Increase hip flexion Hips sway back
	<u>Participant 4</u> Increase hip flexion	<u>Participant 4</u> Increase hip flexion Hips sway back
	<u>Participant 5</u> Increase hip flexion Trunk leans forward Hips sway back Shift body weight forefoot	<u>Participant 5</u> Trunk leans forward Hips sway back Shift body weight forefoot
	<u>Participant 6</u> Functional femoral line falls medial to 10 degree neutral line. Increase hip flexion Hips sway back Shift body weight forefoot	<u>Participant 6</u> Increase hip flexion Trunk leans forward
	<u>Participant 7</u> Trunk leans forward Anterior pelvic tilt	<u>Participant 7</u> Trunk leans forward Hips sway back Hip hitching Anterior pelvic tilt
	<u>Participant 8</u> Functional femoral line falls medial to 10 degree neutral line. Increase hip flexion Trunk leans forward Shift body weight forefoot	<u>Participant 8</u> Functional femoral line falls medial to 10 degree neutral line. Trunk leans forward Shift body weight forefoot
	<u>Participant 9</u> Functional femoral line falls medial to 10 degree neutral line. Increase hip flexion Trunk leans forward	<u>Participant 9</u> Increase hip flexion Trunk leans forward Shift body weight forefoot

Figure 3 illustrates that the SKB motor control test revealed that participants presented with impaired movement control of medial rotation in addition to hip flexion. Impaired control of hip rotation was indicated by the femoral line falling medially (knee moving medially to the 2nd metatarsal) and hip hitching, with more participants (5/9) demonstrating poor control on the symptomatic side compared to the asymptomatic side (2/9) (Figure 3). Six of the nine participants' dominant leg was also their symptomatic side. When comparing the dominant and non-dominant side, similar patterns of impaired movement control were observed (Figure 4).

Discussion

The present findings indicated impaired movement control in all nine participants. Current research in lumbopelvic, neck and shoulder pain clearly demonstrates that individuals with pain can present with impaired movement control patterns (Ludewig & Cook 2000; Falla et al. 2004; Dankaerts et al. 2006; Luomajoki et al. 2008; Worsley et al. 2013). In recent years, objective measures of physical function have been increasingly implemented in patients with FAI and research has shown FAI related kinematic alterations of the symptomatic lower limb during dynamic weight-bearing activities (Austin et al. 2008; Kennedy et al. 2009b; Lamontagne et al. 2009; Lamontagne et al. 2011; Rylander et al. 2011; Morrissey et al. 2012; Hunt et al. 2013). However, limited research still exists on the characteristics and hip movement control patterns in FAI. Impaired movement control patterns is a feature of many musculoskeletal pain presentations (Luomajoki et al. 2008; Worsley et al. 2013), therefore identifying these abnormal movement patterns and impaired movement control in footballers with FAI may be needed to effectively prevent damage and manage symptoms by controlling movement adaptations. Abnormal control of femoral translation and femoral rotation has been linked to anterior hip pain, pathologies of the labrum and associated hip capsule and anterior muscles (Sahrmann 2002; Lewis et al. 2007).

The ideal observed movement control pattern was to keep the knee over the 2nd metatarsal, the trunk vertical, without any forward leaning of the trunk or posterior shift of the hips and pelvis as

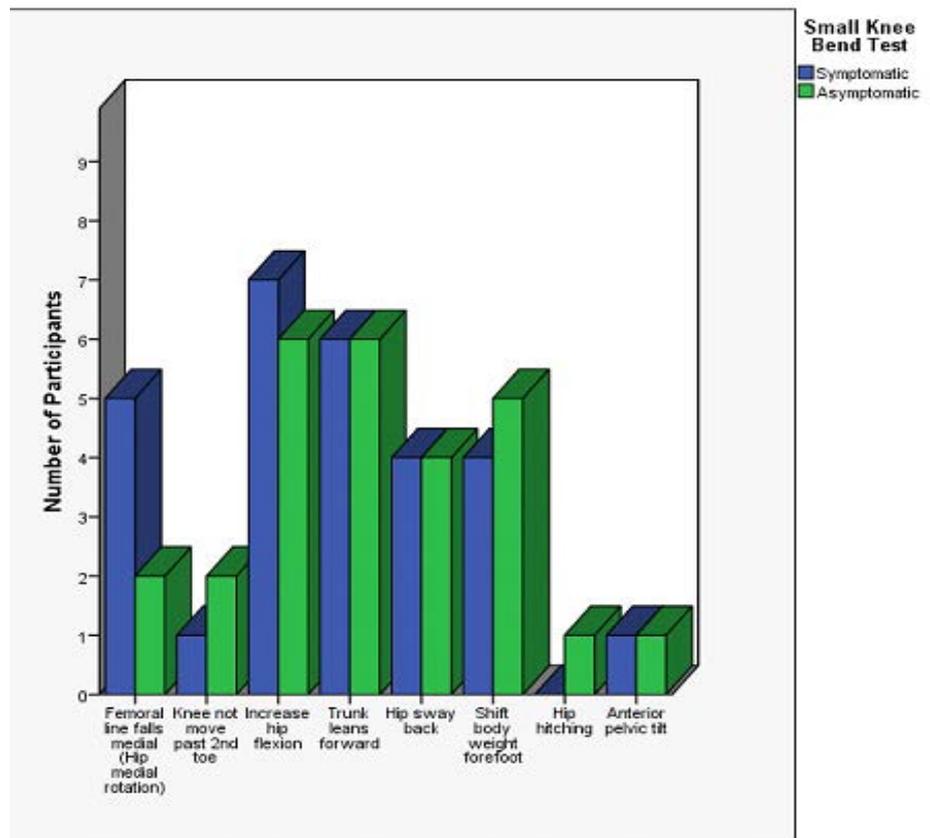


Figure 3. Small knee Bend motor control test cluster bar chart of the total number of faults observed on the symptomatic and asymptomatic side

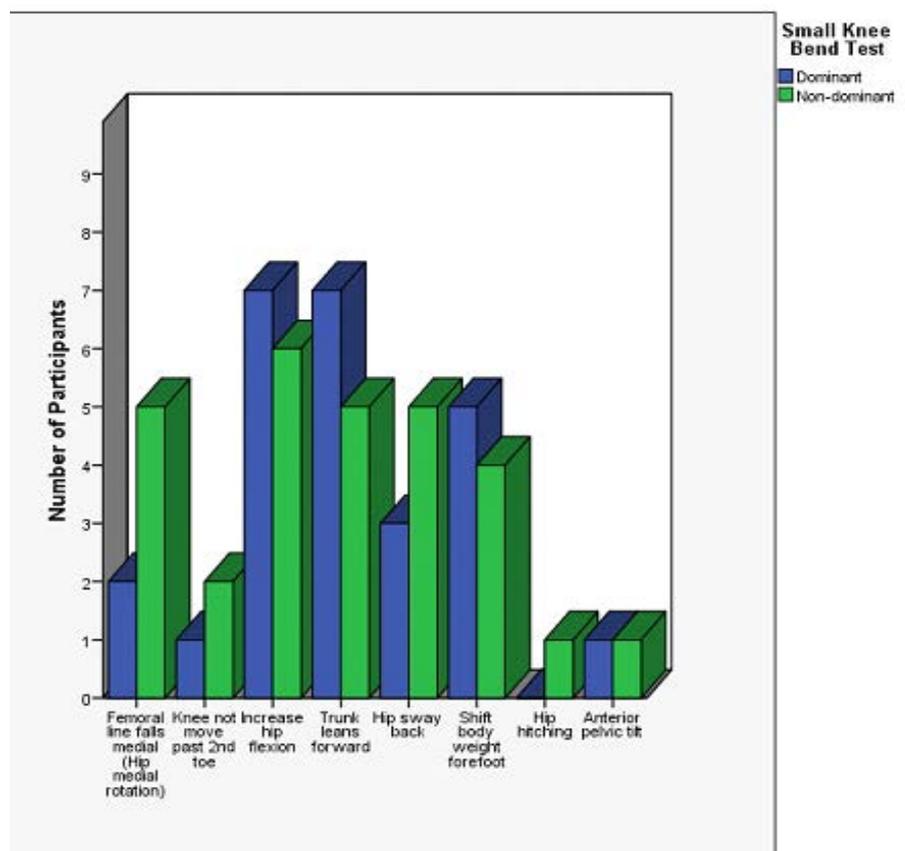


Figure 4. Small knee Bend motor control test cluster bar chart of the total number of faults observed on the dominant and non-dominant side

illustrated in Figure 2 and described in the methods section. The results of the present study demonstrated hip medial rotation impaired movement control with the observed fault of the knee moving medially to the 2nd metatarsal during the single leg SKB motor control test (Ageberg et al. 2010). The observed movement fault of hip hitching may also suggest impaired hip medial rotation control. It is proposed that hip and pelvic coronal plane asymmetry is usually associated with some component of axial or rotation control problem but research is required to support this. If the functional femoral line falls medial to the 2nd metatarsal it may be due to inappropriate foot placement with the 2nd metatarsal not on the 10° neutral line or the line of the femur rotating medially. Either way the possible consequences may be increased valgus stress at the knee, increased mid-foot pronation and/or increased medial rotation at the hip. In a single case study of acetabular hip pathology, Austin et al. (2008) also reported uncontrolled hip medial rotation, while, Levinger et al. (2007) revealed a similar finding in PFJP. It has been suggested that increased hip medial rotation can cause abnormal loading of the anterior hip structures; leading to hip pain and possibly contribute to FAI pathology (Sahrmann 2002; Austin et al. 2008). Altering the frontal and transverse plane hip kinematics decreases hip pain and possibly off loads the anterior hip structures (Austin et al. 2008). Casartelli et al. (2011) suggested that FAI-related hip muscle weakness might result in lower limb kinematic alteration which could cause functional disability. These alterations in movement patterns could exacerbate symptoms, probably due to the increased antero-medial contact stress in the femoroacetabular joint (Yazbek et al. 2011), where bony contact and joint damage can occur. Lewis et al. (2007) reported that the hip demonstrates increased medial rotation if the ilio-psoas force decreases and the tensor fascia latae (TFL) force increases, which causes an imbalance and produces excessive anterior hip loading. Conversely, (Casartelli et al. 2011) reported lower electromyographic (EMG) activity of the TFL in symptomatic FAI compared to controls. This lower EMG activity in TFL may possibly be a protective or guarding response. Since TFL has a combined action of hip flexion and medial rotation, which when combined may provoke hip symptoms. Therefore, preventing this action

may simply be a way of decreasing the provocative loading which may lead to the reduction in TFL activity.

In the present study, impaired movement control patterns were observed on both the symptomatic and asymptomatic sides during the SKB motor control test. The fact that impaired hip flexion movement control was not more prevalent on the symptomatic side, may indicate that the SKB motor control test is more a general measure of altered hip control rather than directly related to loading the impingement biomechanics. However, this was a similar finding to Morrissey et al. (2012) who found the injury-associated muscle imbalance ratio of Gluteus Medius : Adductor Longus (GM:AL) during a standing hip flexion to 90° test in subjects with chronic groin pain was also present in the uninjured limb. This possibly reflects a predisposition to injury, or a bilateral effect of injury, and may have a significant consequence for rehabilitation planning and injury prevention (Morrissey et al. 2012). Also, sensorimotor changes have been demonstrated in the contralateral limb after injury on one side (Denko & Petricevic 1978), while Sharma et al. (1997) showed no differences in proprioceptive acuity between sides in a group of subjects with unilateral knee OA. 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 symptomatic and asymptomatic sides. Morrissey et al. (2012) also suggested that the altered GM activation they observed during hip movement may be indicative of many factors, such as relative abductor muscle inhibition, altered movement patterns or muscle atrophy. Determining GM activation and muscle atrophy was beyond the scope of the present study.

In a kinematic study of level gait, Kennedy et al. (2009b) found that patients with symptomatic FAI had decreased frontal and sagittal hip ROM and frontal pelvic mobility. During a maximum depth squat, Lamontagne et al. (2009) reported differences in sagittal plane pelvic kinematics and overall movement performance between those with and without FAI during a maximum depth squat. As argued by Kennedy et al. (2009b) and Casartelli et al. (2011), these alterations in movement could be the result of strategy adopted by patients to compensate for a hip muscle function deficiency. In the present

study, the observed movement faults of increased hip flexion, trunk leaning forward, hip swaying back and anterior pelvic tilt are all direct observations of different strategies of increasing hip flexion. While, the observed movement faults of the knees not moving past the second toe, knee alignment < 2 cm past the toes and the shift of body weight forward may all indicate reduced ankle and knee flexion, which may have an indirect consequence of increasing the risk of hip flexion as compensation during functional activities. In a second study, Lamontagne et al. (2011) found no significant differences between preoperative and postoperative pelvic motion, or with the pelvic and hip angles at peak squat depth. However, the squat performance improved postoperatively with an increased pelvic posterior pitch during the descent phase of squat. The authors suggested that the increased squat depth and improved pelvic posterior pitch may be due to the corrective surgery having eliminated the mechanical restriction and reduced joint pain, by debridement of the unstable labrum. The possible mechanical restriction and pain within the population studied may provide an explanation for the altered movement patterns observed in the present study. However, wide variability in postoperative test times (8-32 months) could have affected the detection of significant differences between preoperative and postoperative kinematic values. The authors have not mentioned whether the participants had active rehabilitation or movement retraining which may explain some of the positive response postoperatively. Also, it has been reported that hip abductors have an important role in controlling trunk position in the frontal plane (MacKinnon & Winter 1993), which possibly also relates to impaired hip medial rotation control. Limited research exists around motor control issues in hip pathologies and no literature was found specifically investigating hip flexion impaired movement control. Therefore, the cause of the impaired movement control patterns observed cannot be determined by the present study.

The findings of this pilot study add to the limited evidence surrounding impaired movement control in young footballers with FAI. However, the small sample size and the absence of a control group limit our ability to conclusively answer the many questions that exist. Some bias may have been introduced dur-

ing the data collection process of the study. Throughout the study the same researcher conducted the screening of participants and performed the tests. Therefore, the researcher was aware of the subjects' medical history and background information regarding their hip pathology. This can possibly affect the investigator's test interpretation and introduce bias. Future studies should consider the researcher collecting the data to be blinded by having a different researcher conduct the recruitment and screening of the participants. Further research is needed to validate this clinical test using motion analysis and provide kinematic and kinetic data. This will allow for more detailed investigation of altered movement patterns in patients with symptomatic FAI so that the movement impairment can be better understood and then effective interventions developed to prevent and manage FAI.

Conclusions

The present findings demonstrate altered movement patterns during the SKB motor control test. The impaired movement control patterns of hip flexion and medial rotation of the femur may increase loading of the joint, possibly leading to abnormal joint stress overtime. The SKB motor control test is a simple, rapid test that may help identify impaired movement control in the clinical environment to help improve the quality of movements, potentially reducing abnormal loading on joints. Further tests need to be explored and validated to help us understand the mechanisms of movement impairments during functional tasks, to inform strategies for improving movement quality.

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