# Gibbons SGT, Strassl H 2012 Can altered movement pattern and muscle imbalance be related to FAI and other hip disorders? Manuelle Therapie. (German). 16: 119-131

## Introduction

Femoroacetabular impingement (FAI) is a relatively newly recognised clinical presentation (Standaert et al. 2008, Inman and Khanduja 2011). The aetiology is still unclear along with the specific roles of genetic, personal, and environmental factors in the development and progression of the condition (Jaberi and Parvizi 2007, Keogh and Batt 2008, Audenaert et al. 2011, Inman and Khanduja 2011, Balch Samora et al. 2011).

The conceptual model of FAI implies that there is abnormal contact between the femur and acetabular rim at the end range of hip motion, particularly flexion, eventually resulting in the development of various pathologies. The abnormal contact is related to anomalies of the femur, the acetabulum, or both. Two distinct types of FAI have been described, predominantly based on whether the anomalous morphology occurs in the femur (cam impingement) or acetabulum (pincer impingement) (Standaert et al. 2008), although they frequently occur together (Gosvig et al 2008). The anatomical malformations themselves do not cause any symptoms, but the model proposes that instead, that the sequelae of repetitive impingement damages surrounding structures leading to pain (Imam and Khanduja 2011).

The prevalence of FAI has been reported to be 10-15% (Keogh and Batt 2008) and as high as 17% (Gosvig et al. 2008) in the general population. It may be present in up to 24% of athletes (Keogh and Batt 2008). In asymptomatic volunteers cam-type FAI in one hip was present in 14% while 3.5% had bilateral deformities (Hack et al. 2010). In another asymptomatic group, 13.95% of males had a cam-type FAI with 14.88% being borderline based on their measurements. In the female group, 5.56% were pathological and 6.11% were borderline (Jung et al. 2011).

Physiotherapy for FAI has been suggested to be counterproductive (Jaberti and Parvizi 2007). Evidence for conservative management for FAI without other pathology is limited to case studies (Ames and Heike 2010, Wright and Hegedus 2011) and case series (Emara et al. 2011). Non clinical trial evidence supports the use of surgical interventions for FAI for short term outcomes (Larsonet al. 2008, Ng et al. 2010, Matsuda et al 2011), however the quality of literature reporting outcomes of surgical intervention is limited (Bedi et al. 2008). There remains some debate regarding the conservative management, the best surgical procedure and post surgical rehabilitation of FAI.

It is unlikely that one treatment will help all subjects with FAI. Sub-classification is recommended to help better direct patient management. In this process, patients are given treatments that match their own specific requirements based on their individual presentations. Table 1 highlights a detailed sub-classification process that addresses all the known changes that are associated with musculoskeletal pain (Gibbons 2012a). Sub-classification has been shown to produce better outcomes for low back pain (Fersum et al. 2010, Hill et al. 2011, Gibbons and Clarke 2009, Gibbons and Newhook 2012). To more specifically target FAI with conservative

management, a sub-classification approach is recommended that specifically addresses the deficits that clients present with. Movement pattern control (MPC) and translation control are sub-categories within the motor function sub-classification. Muscle imbalance changes and gait disturbances have the potential to place extra stress on the hip region which could contribute to the development and maintenance of local pathology (Dannenburg 1993, Lewis and Sahrmann 2006, Lewis et al 2007, Grimaldi 2009, Lewis et al 2010, Grimaldi 2011). As well, deficits such as reduced balance, proprioception, and local muscle dysfunction occur in regions following pain that are independent of the region (Gibbons 2012a) which could also contribute to the maintenance of the presentation. Core strengthening, sensory motor control, alignment optimization and the elimination or modification of movements that exacerbate the pain have been recommended (Balch Samora et al. 2011), however sub-classification strategies or experimental evidence is lacking. Clinically, it is observed that changes in movement patterns, muscle imbalance and reduced translation control of the femoral head appear to be associated with FAI. The purpose of this paper is to describe the sub-classification of MPC and translation control for the hip as well as some of the relevant background theory.

Table 1: A suggested sub-classification strategy that considers the variety of known differences in presentation between people with chronic pain and those without.

| **Patho-anatomical** | **Movement & Motor Function** | **CNS Coordination** | **Pain Mechanisms** | **Behavioral Factors** |
|---|---|---|---|---|
| • Myofascial<br>• Articular<br>• Neurodynamic<br>• Connective tissue | • Movement pattern control<br>• Translation control<br>• Respiratory control<br>• Motor fitness | • Sensory motor function<br>• Neurocognitive function<br>• Neurological soft signs<br>• Midline awareness | • Nociceptive<br>• Neurogenic<br>• Neuropathic<br>• Central sensitization<br>• Central body image disruption | • Clinical disorders<br>• Personality & developmental disorders<br>• Psychosocial factors |
| **Individual factors** (e.g. Medical & physical conditions; Expectations & beliefs; Cultural, gender & age influences; Motivation & compliance; Health behaviors) | | | | |

**Muscle Function and Classification**

Muscle classification is somewhat artificial at first appearance, however a deeper understanding shows that it can be useful and assist in clinical reasoning with exercise prescription and in various manual therapies. Muscles are required for segmental control, control of postural alignment, the control of movement and force production. All muscles have the ability to contribute to stability, however some muscles are better suited for the above roles because of their anatomical location and structure, biomechanics, muscle spindle capacity (and ability to provide proprioceptive feedback) and neurophysiology. These characteristics, along with how the muscle acts during low and high load requirements, and following an episode of pain can be used to functionally classify muscles (Gibbons 2005).

The most functional model of muscle classification divides muscles into local stabilizers, global stabilizers and global mobilizers (Comerford and Mottram 2001, Gibbons and Comerford 2001).

Contemporary research shows that it is too simplistic to place all muscles in one category. The model should be considered as a model of best function where a muscle may have more than one dominant function and thus may be placed under two or even three categories. An example of this would be gluteus maximus with upper and lower distinctions (Grimaldi et al. 2009), and deep sacral fibres (Gibbons 2007a). On the other hand, a muscle may have multiple functions, but one is minor. Here, the muscle would be categorized under its primary function. An example of this would be the rotation role of transversus abdominis (Urquart et al. 2005). Table 2 summarizes the classification model.

**Muscle Imbalance**

Awareness of global muscle imbalance is necessary since this contributes to uncontrolled movement; muscle shortness in mobilizer muscles which may develop into restrictions to normal movement (and resultant compensations); and greater force on joint structures. Biomechanical models have provided evidence of the potential for greater strain on the hip when there is reduced activity from stabilizer muscles such as gluteus maximus, gluteus medius and iliacus-psoas major (Sims 1999, Simoes et al. 2000, Lewis et al. 2007, Grimaldi et al 2009, Lewis et al. 2010).

Testing strength provides information on high load gross muscle function, however specific changes within muscle synergies will only become evident by addressing each muscle individually (Grimaldi 2009). Both low and high load function can be assessed by taking advantage of kinesiology principles and standardized movements (Gibbons 2012b).

Table 2: Function and characteristics of local stabilizer, global stabilizer and global mobilizer muscles in normal function and after the presence of pain (dysfunction) (Adapted: Comerford and Mottram 2001)

| Muscle | Function | Dysfunction |
|---|---|---|
| **Local Stabilizer** | ● Biomechanical influence for translation control<br>● Minimal length change in functional movements<br>● Anticipatory recruitment to movement<br>● Independent of the direction of movement | ● Reduced cross sectional area<br>● Motor recruitment deficit<br>- Altered patterns of recruitment<br>- Altered timing |
| **Global Stabilizer** | ● Generates force to control movement<br>- Low threshold eccentric deceleration of movement<br>- Bias is towards rotation | ● Imbalance in low threshold recruitment between synergists and antagonists<br>● Length associated change affecting muscle efficiency |
| **Global Mobilizer** | ● Generates force to produce range of movement<br>- Concentric acceleration of movement (primarily in the sagittal plane)<br>● Activity is phasic (on –off pattern with repetitive movement)<br>● High load stability | ● Myofascial shortening<br>● Overactive low load or low threshold recruitment<br>● Reacts to pain and pathology with increased activity |

Table 3: The types of muscle imbalances described in the literature (from Gibbons 2012b)

| **Muscle Imbalance** | |
|---|---|
| **Global Muscle Imbalance** | **Traditional Muscle Imbalance** |
| ● Altered order of recruitment between synergists or kinetic chain movement<br>● Altered activation time between synergists<br>● Altered amount of activity between a group of synergists or kinetic chain movement<br>● Reduced inner range holding efficiency of a global stabilizer compared to a standardized comparison group | ● Strength ratio between agonist and antagonistic muscles or muscle groups<br>● Strength of a group of synergists when compared to the opposite limb or standardized comparison group |

Table 4: Possible muscle imbalances around the hip between global stabilizers and global mobilizers.

| Hip Movement | Stabilizer(s) | Mobilizer(s) |
|---|---|---|
| Flexion | Iliacus and anterior psoas major | Rectus femoris, tensor fascia latae and iliotibial band, sartorius |
| Extension | Lower gluteus maximus | Hamstrings |
| Abduction | Posterior gluteus medius, upper gluteus maximus | Tensor fascia latae and iliotibial band |
| Adduction | Pectineus, adductor brevis, short head of adductor magnus | Gracilis, adductor longus, long head of adductor magnus |
| Internal rotation | Gluteus minimus, anterior gluteus medius | Tensor fascia latae and iliotibial band |
| External rotation | Posterior gluteus medius and gluteus maximus | Piriformis |

## Movement Pattern Control

The underlying hypothesis of movement as a link to musculoskeletal symptoms is that the way the central nervous system (CNS) coordinates movement can influence tissue loading. The CNS has numerous motor control options when producing a movement. Neuromotor function is the process whereby the CNS uses the available sensorimotor information and prioritizes the current requirements (e.g. functional requirements, neurocognitive demands, psychological arousal) to coordinate movement. In normal function we need the ability to vary postures and movement patterns, or kinetic chain sequence, in order to avoid tissue overload. It is normal and necessary to use our end range movements, however it is abnormal to continuously use the same movement pattern or end range movement. If the ability to vary the kinetic chain and control movement is lost, tissue load can be exceeded, tissue repair can become compromised and pathology may result. To explain how habitual movement can lead to pain presentations, Sahrmann (2002) proposed the concept of relative stiffness. In this model, a relatively less stiff region will compensate (increase movement in a specific direction) for a muscle system with greater stiffness. It does not require that muscles are tight or strong, just relatively more stiff than their adjacent region. Gibbons (2012c) expanded on this and proposed a sensory motor model. Here, the CNS requires constant sensory motor feedback from the body and there is competition within the CNS for the available resources and processing. There is an overlap in the CNS where neurocognitive, sensory, motor and psychological function as well as information related to body image are processed. If there is a deficit in one, there may be a deficit in processing of other functions. If sensory information is limited, or inadequately processed, the body may move further into end range to gain information (proprioceptive, tactile) from compression or stretch from joint structures and myofascial structures. When global muscle imbalance and dysfunctional movement patterns become familiar to the CNS, they can be maintained and altered movement pattern control (MPC) may result. Neurological movement patterns, or remnants of primitive reflexes, may result to fill the void of normal movement (Gibbons 2009a, 2012c).

## Mechanisms of Altered Movement Pattern Control

To fully understand the sub-classification of MPC, we need to further understand why the motor system would adopt a dysfunctional movement pattern. This has not been specifically considered in other classifications (Sahrmann 2002, 2011, O'Sullivan 2005, Comerford and Mottram 2001, 2012). There are numerous potential mechanisms that may disturb motor control and movement patterns. The most common mechanisms are summarized in table 5 along with suggested rehabilitation options. An understanding of the mechanisms of movement pattern control deficits (MPCD) can allow rehabilitation to be targeted more specifically. It should be appreciated that there are numerous other possible mechanisms that affect movement or can indirectly influence it by affecting CNS competition, however only the most common we see clinically are listed below (Gibbons 2011a, Gibbons 2012c).

Table 5: The most common mechanisms of MPCD along with suggested rehabilitation options

| Mechanism of MPCD | Rehabilitation Option |
|---|---|
| Sensory motor deficits | Sensory rehabilitation (e.g. proprioception, two point discrimination) |
| Fatigue | Endurance training (within the constructs of movement pattern control) |
| Repetitive movements | Endurance training (within the constructs of movement pattern control)<br>Ergonomics (activity modification) or adjustment of training schedules |
| Restrictions to movement: The underlying mechanism of the restriction must be understood | Articular: Manual therapy<br>Neurodynamic: Neural mobilization (this will be influenced by manual therapy and muscle tone)<br>Muscle tone (summary of the mechanism that restrict active and passive movement)<br>● Primitive reflexes: primitive reflex inhibition<br>● Reflex stiffness: muscle imbalance rehabilitation<br>● Intrinsic stiffness: muscle stretching and passive techniques<br>● Spinal reflexes: various neurological techniques |
| Weakness | Strength training (within the constructs of movement pattern control) |
| Prolonged postures at end range | Sensory rehabilitation (e.g. proprioception, two point discrimination)<br>Ergonomics<br>Endurance training (within the constructs of movement pattern control) |
| Dual tasking | Integration of the rehab program into functional activities |

## Movement Pattern Control Testing

MPC testing is the clinical procedure of assessing the relationship between movement and risk for tissue strain. Further to the above theory, the premise is that the inability to consciously control movement is associated with habitual and uncontrolled movement into this direction which places extra stress on the tissues of the region. There is considerable support for this model in the lumbar spine (for summary see Lehtola et al. 2012). The procedure for MPC testing is outlined in table 6. We must also consider the natural functional movement pattern, or kinetic chain sequence. Due to influences such as endurance, dual tasking, ergonomics, or conscious focus, that are not part of the assessment of MPCD, the testing of MPCD undoubtedly lacks some sensitivity (e.g. a person may appear to pass a MPC test when in fact the person does exhibit excessive movement in that region during their natural functional movements such as sport or work). If a person exhibits excessive movement in a region during a kinetic chain sequence or during manual testing, it should be taken into consideration in rehabilitation.

Even though the movement pattern control tests are non functional, we must keep in mind that the 'natural' movement pattern is no longer normal. The strategy is therefore to test and rehabilitate the non functional pattern along with the associated muscle imbalance and integrate this into function with the correction of the kinetic chain sequence. It may be permissible to start with correcting the kinetic chain sequence, however our clinical observation is that these individuals normally do not have the sensory motor skills to start at this level without having first learned MPC. In acute situations or when the kinetic chain sequence still aggravates tissues (provokes symptoms) MPC rehabilitation is favoured since the region of strain is not moved and less strain is placed upon it.

**Movement Pattern Control of the Hip**

During functional movements (with and without pathology), the hip commonly moves into excessive flexion, extension, internal rotation and external rotation (and combinations of these).

Tables 7-11 describe some basic MPC screening tests for the hip.

Figures 1&2 accompany table 7 and display the assessment of the kinetic chain sequence of trunk and hip flexion in various functional movements as well as the standing hip flexion control test.

Figures 3-5 accompany table 8 and display the assessment of the kinetic chain sequence of trunk and hip extension in various functional movements as well as the assessment of hip extension range (using the modified Thomas Test) and the standing hip extension control test.

Figures 6-8 accompany table 9 and display the assessment of the kinetic chain sequence of the squat as well as the assessment of hip rotation range (in prone) and the squat hip rotation control test.

Figures 9 & 10 accompany table 11 and display the assessment of the kinetic chain sequence of lunge as well as the short lunge hip rotation control test.

Table 6: Clinical procedure for assessing movement pattern control

| Movement Pattern Control Assessment Procedure | |
|---|---|
| Observe normal active pattern through full range | Test the kinetic chain sequence that is related to the MPC test |
| Observe movement pattern during a functional demonstration of an aggravating movement | Observe the kinetic chain sequence within the functional movement |
| Assess available range of movement (passive or auto-assisted) | Teach the client how to place the test region in neutral and perform the test movement. The therapist may need to manually assist. The test direction movement usually involves moving above or below the test region or controlling a movement of the test region (e.g. rotation). The region of movement could be remote if that region naturally challenges movement at the test site. It involves conscious control to keep the region being tested in neutral and independently move another region. |
| Assess possible restrictions (if above range is limited) | The therapist should assess the myofascial, neurodynamic and articular structures that may limit normal movement and the test movement |
| Teach ideal movement pattern control | The therapist should use (1) visualization and mental imagery (2) sensorimotor feedback (3) motor facilitation strategies to teach the MPC exercise (Gibbons 2011b) |
| Test the ability of the client to consciously control movement without assistance (sensory motor feedback or motor facilitation) | Make a clinical judgement if a deficit exists or not. For example:<br>Do they understand the MPC test?<br>Can they perform the test correctly?<br>Are they confident of their ability to perform the exercise?<br>Do they require sensory motor feedback?<br>Do they experience any fatigue?<br>Do they have a high sensation of effort?<br>Can they integrate this movement into a functional task? |
| If a MPCD exists: Assess mechanism of the MPCD | Are there restrictions to movement?<br>Is sensory motor feedback required?<br>Does the history suggest a lack of endurance, end range postures, repetitive movements or dual tasking?<br>Is there a strength deficit?<br>Are primitive reflexes present that involve the MPCD? |

Table 7: Standing hip flexion control

| Standing Hip Flexion Control | |
|---|---|
| Test region | Hip |
| Direction of movement | Flexion |
| Starting position | Standing with calcaneus under hips with lumbar spine and hips in neutral |
| Move | Move trunk into flexion |
| Control | Hip stays in neutral (or stationary) |
| Test description | Maintain the hip in a neutral (or stationary) position and flex the trunk |
| Normal movement pattern control | Trunk flexes 30° without movement of the hips |
| Therapist monitoring strategy | ASIS for hip movement and angle between ASIS and thoracolumbar junction for gross trunk movement |
| An example of a client monitoring strategy (if required) | Hands on ASIS and greater trochanter (if required). There should be no movement of the ASIS and the two points should not approximate each other. |

Figure 1: Assessment of kinetic chain sequence of trunk and hip flexion. in: a. standing. Note excessive hip flexion (> 70°), b. sitting and c. four point kneeling. Note excessive hip flexion in b. & c. (> 120°)

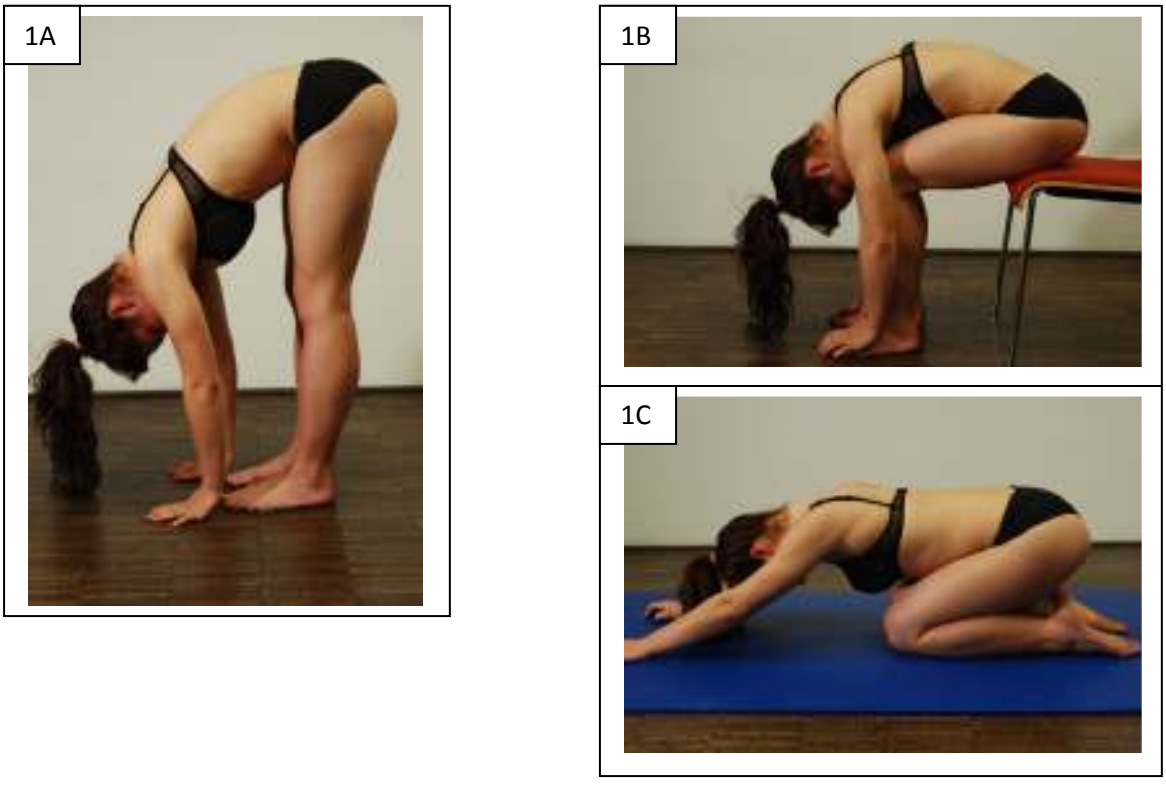


Figure 2: Standing hip flexion control. Note that the hip stays in neutral while the spine moves into flexion.

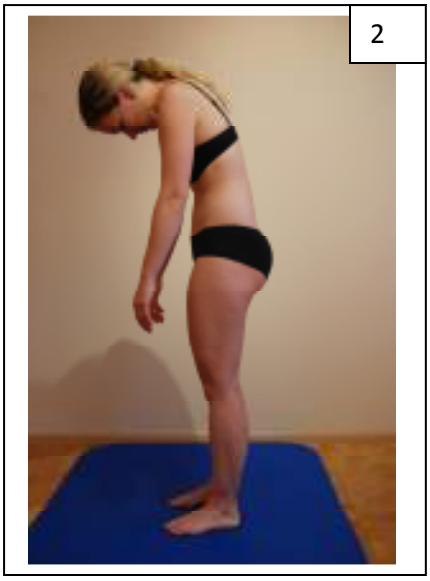
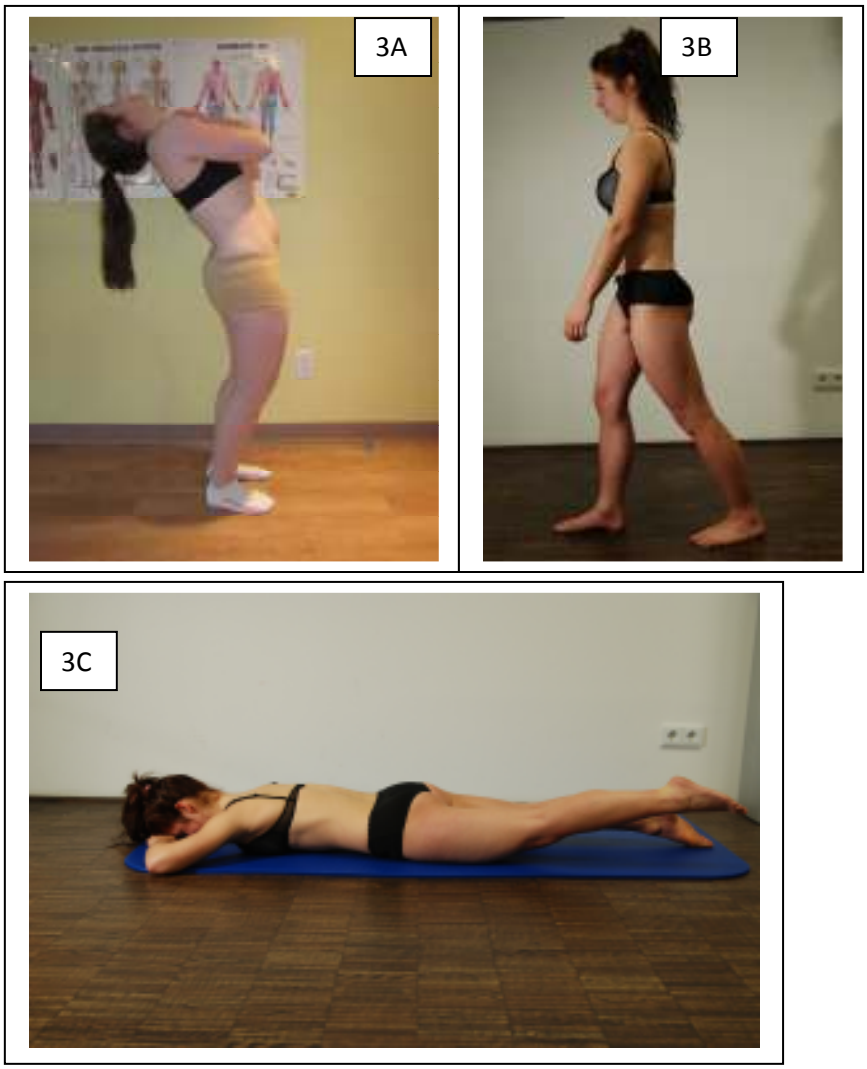



Table 8: Standing hip extension control

| **Standing Hip Extension Control** | |
|---|---|
| Test region | Hip |
| Direction of movement | Extension |
| Starting position | Standing with cancaneus under hips with lumbar spine and hips in neutral |
| Move | Move trunk into extension |
| Control | Hip stays in neutral (or stationary) |
| Test description | Maintain the hip in a neutral (or stationary) position and extend the trunk |
| Normal movement pattern control | Trunk extends 20° without movement of the hips |
| Therapist monitoring strategy | ASIS for hip movement and angle between PSIS and thoracolumbar junction for gross trunk movement |
| An example of a client monitoring strategy (if required) | Hands on ASIS and greater trochanter (if required). There should be no movement of the ASIS and the two points should not move away from each other |

Figure 3: Assessment of the kinetic chain sequence of trunk and hip extension in: a. standing. Note the restricted hip extension with compensatory knee flexion and excessive low lumbar extension, b. walking. Note excessive hip extension of > 10-15° and c. prone. Note neutral lumbar spine while hip extension reaches benchmark of 10°.




Fgure 4: Assessment of hip extension range using Thomas Test, a. startposition, b. end position. Note excessive range of 15° hip extension.

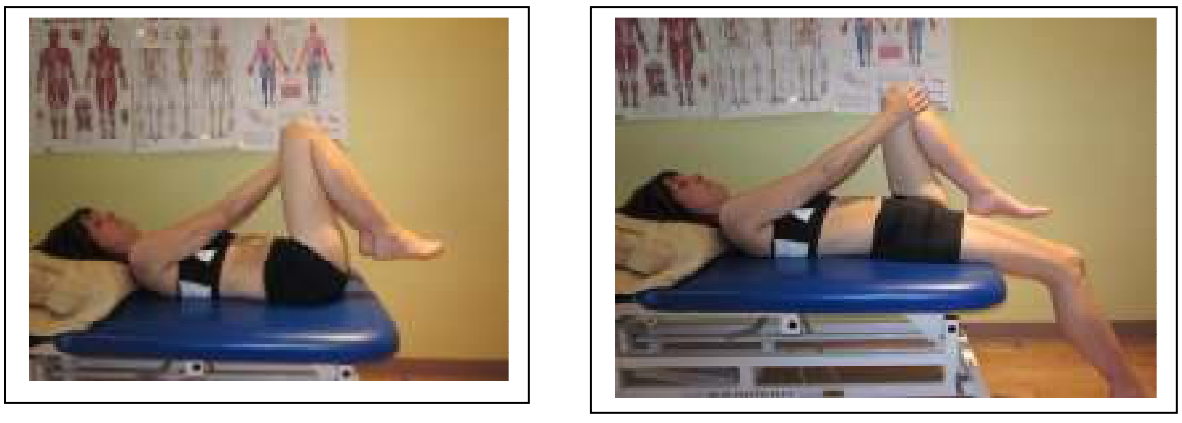


Figure 5: Standing hip extension control. Note that the hip stays in neutral while the spine moves into extension.

Table 9: Standing squat hip rotation control

| **Standing Squat Hip Rotation Control** | |
|---|---|
| Test region | Hip |
| Direction of movement | Rotation |
| Starting position | Standing with cancaneus under hips with lumbar spine and hips in neutral |
| Move | Initiate a squatting movement (so the hips and knees flex and the ankles dorsiflex) |
| Control | Hip stays in neutral rotation (or stationary) |
| Test description | Maintain the hip in a neutral (or stationary) position and flex the hips and knees, and allow ankle dorsiflexion |
| Normal movement pattern control | The hips maintain neutral rotation during 30° hip and knee flexion. Neutral rotation is considered with the femur in line with the second metatarsal. This would need to be modified to accommodate any lower limb structural restrictions (e.g. anteversion). The trunk should also be neutral (this test may also be used to test for lumbar flexion control during squatting). |
| Therapist monitoring strategy | Line of femur over second metatarsal (other MPCD may occur at the lumbar spine, knee or foot). |
| An example of a client monitoring strategy (if required) | Hands on ASIS and greater trochanter (if required). There should be no movement of the ASIS and the greater trochanter should not rotate. This can also be monitored visually. |

Figure 6: Kinetic chain sequence of squat. Note how hip goes into excessive flexion while spine stays extended almost through full range.

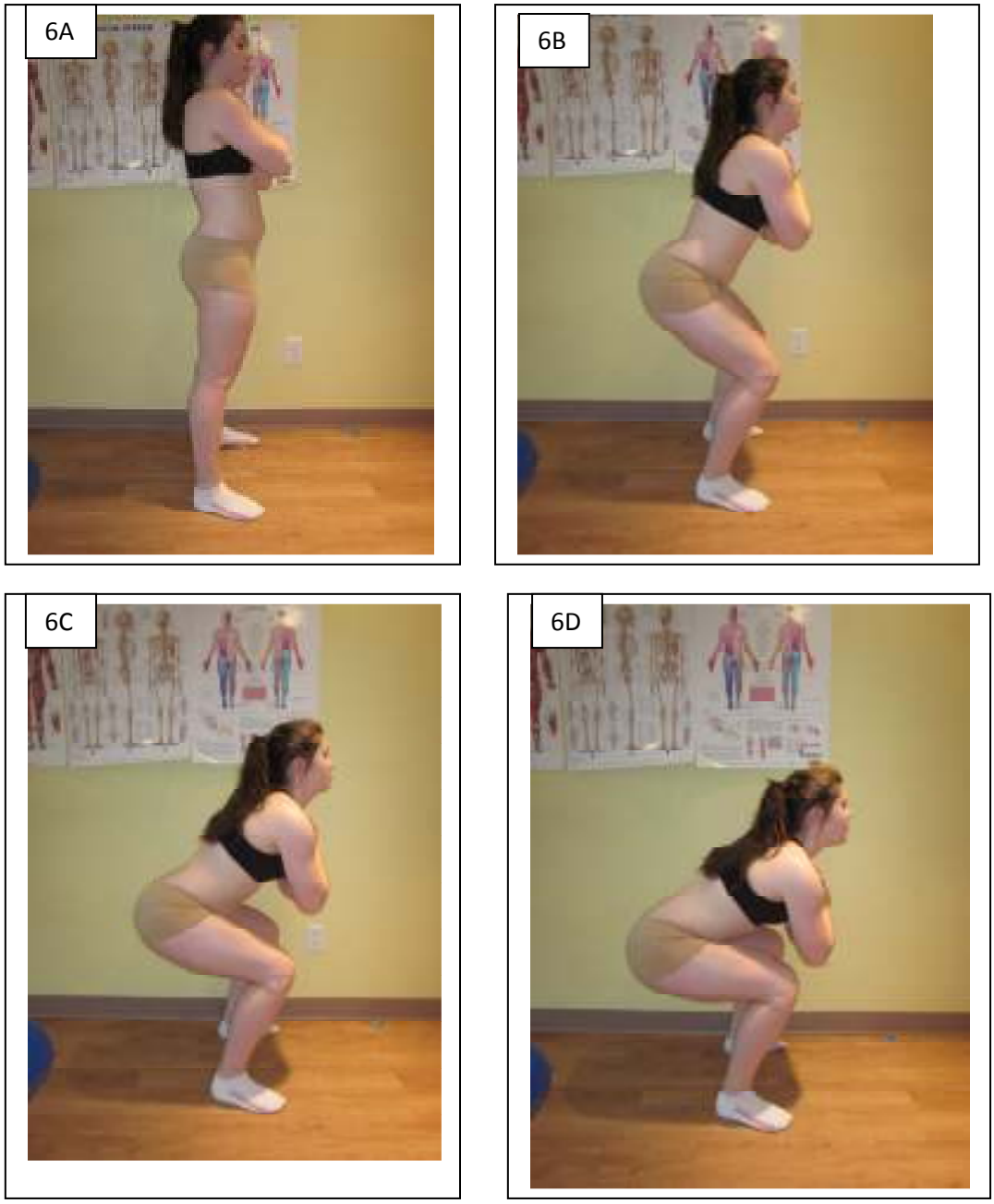


Figure 7: Assessment of hip rotation range, a. start position, b. external rotation. Note restriction < 35°. c. internal rotation. Note excessive movement of > 35°.

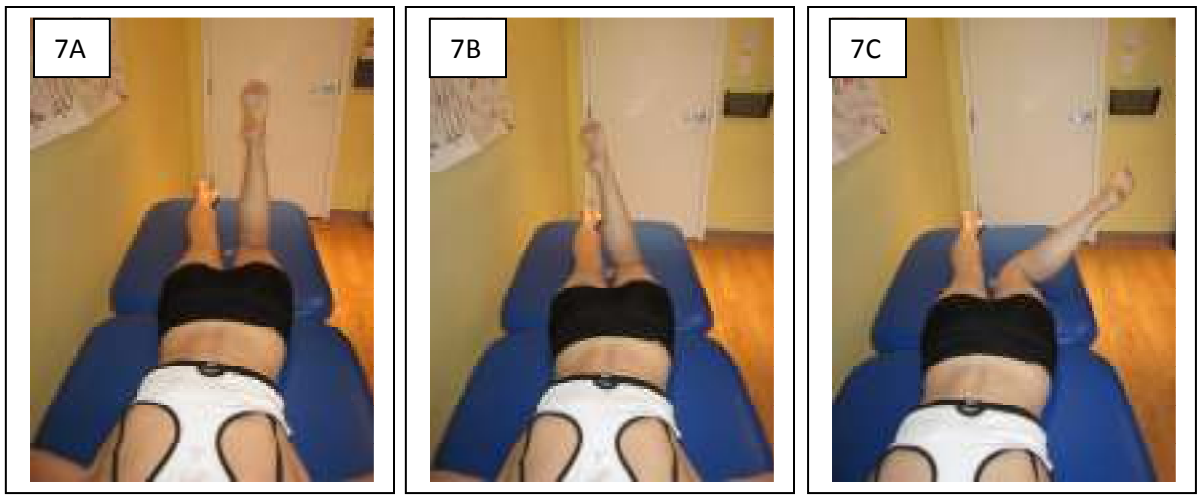



Figure 8: Squat hip rotation control. a. Lack of rotation control. Note that the femures go medial of the first metatarsal b. Control of rotation. Note the femures over the second metatarsals.

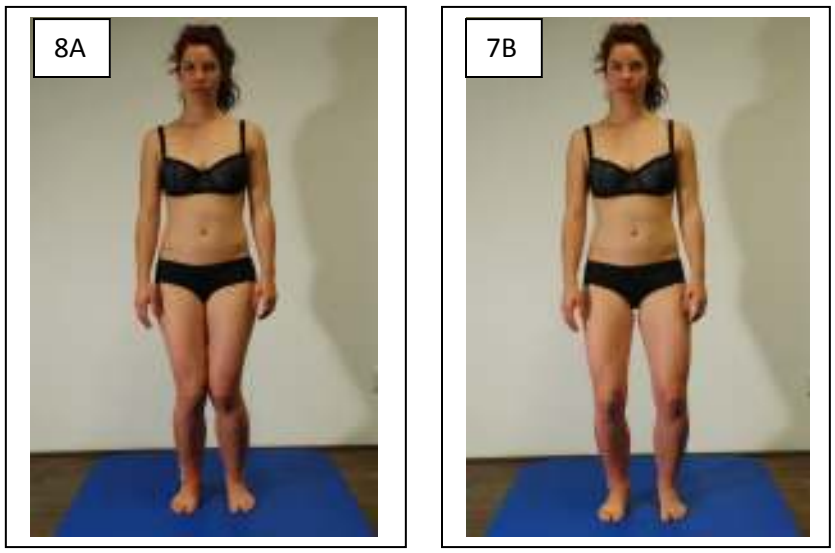


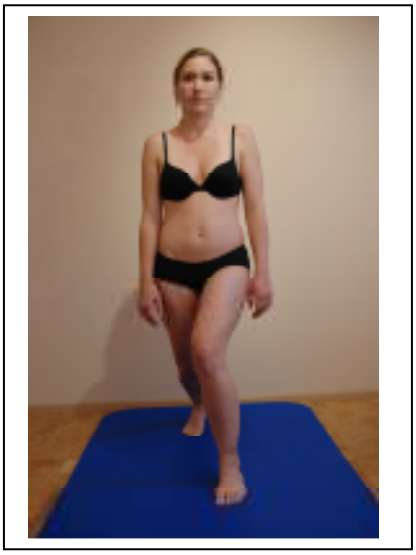
Table 10: Standing one leg squat hip rotation control

| Standing One Leg Squat Hip Rotation Control | |
|---|---|
| Test region | Hip |
| Direction of movement | Rotation |
| Starting position | Standing with cancaneus under hips with lumbar spine and hips in neutral |
| Move | Transfer weight onto one leg. Stand on that leg and squat while balancing (flex the hips and knees, and dorsiflex the ankle. |
| Control | Hip stays in neutral rotation (or stationary) |
| Test description | Maintain the hip in a neutral (or stationary) position, stand on one leg and squat (flex the hips and knees, and dorsiflex the ankle) |
| Normal movement pattern control | The hips maintain neutral rotation during 30° hip and knee flexion. Neutral rotation is considered the line of the femur is in line with the second metatarsal. This would need to be modified to accommodate any lower limb structural restrictions (e.g. anteversion). The trunk should also be neutral (this test may also be used to test for lumbar flexion control during squatting). |
| Therapist monitoring strategy | Line of femur over second metatarsal (other MPCD may occur at the lumbar spine, knee or foot). |
| An example of a client monitoring strategy (if required) | Visual monitoring with or without a mirror. In general, a line dropped from the middle of the patella to the second metatarsal (or sometimes first) can be used by the client. |

Table 11: Standing short lunge hip rotation control

| Standing Short Lunge Hip Rotation Control | |
|---|---|
| Test region | Hip |
| Direction of movement | Rotation |
| Starting position | Standing with cancaneus under hips with lumbar spine and hips in neutral |
| Move | Take a step forward (two – thirds of maximum) and allow the knee to follow through over the toes. |
| Control | Hip stays in neutral rotation (or stationary) |
| Test description | Maintain the hip in a neutral (or stationary) position and flex the hips and knees, and allow ankle dorsiflexion |
| Normal movement pattern control | The hips maintain neutral rotation during 30° hip and knee flexion. Neutral rotation is considered the line of the femur is in line with the second metatarsal. This would need to be modified to accommodate any structural restrictions (e.g. anteversion). The trunk should also be neutral (this test may also be used to test for lumbar flexion control during squatting). |
| Therapist monitoring strategy | Line of femur over second metatarsal (other MPCD may occur at the lumbar spine, knee or foot). |
| An example of a client monitoring strategy (if required) | Visual monitoring with or without a mirror. In general, a line dropped from the middle of the patella to the second metatarsal (or sometimes first) can be used by the client. |

| Figure 9: Kinetic chain sequence of lunge. Note that the femur goes medial of the first metatarsal. | Figure 10: Short lunge hip rotation control. Note good control with the femur over the second metatarsal. |
|---|---|



**Multi-Regional Deficits**

The lumbar spine, the sacro-iliac joint and the hip are artificially separated into distinct anatomical locations, however they are intimately linked in function and dysfunction. Muscles such as psoas major and gluteus maximus have a stability role in all three regions (table 12). For this reason, MPCD and translation control deficits (see below) may be seen in all three areas at the same time. When this multi-region breakdown in motor control occurs, the hip may develop strain with atypical patterns of uncontrolled movement since the stability of the whole lumbo-pelvic-hip complex may be compromised. This presentation generally requires more rehabilitation to correct.

Table 12: Examples of stabilizer muscles that are involved in the hip and other joint regions

| Muscle | Regions |
|---|---|
| Psoas major, iliacus | Lumbar spine<br>Sacro-ilaic joint<br>Hip |
| Gluteus maximus, gluteus medius | Lumbar spine<br>Sacro-ilaic joint<br>Hip<br>Lower limb alignment |
| Oblique abdominals; transversus abdominis | Lumbar spine<br>Sacro-iliac joint |

**Translation control**

Translation of the femoral head is a normal part of the physiological motion of the hip and is highly variable between individuals (Harding et al 2003). The clinical literature suggests that it does seem plausible for scenarios to exist in which the normal translation of the femoral head would be increased (Martin et al. 2006, Lewis et al 2007, Standaert et al. 2008, Groh et al. 2009, Smith and Sekiya 2010, Boykin et al. 2011, Shu and Safran 2011). There are a number of terms used in the literature which imply an increased motion of the femoral head. These are listed in table 13.

Table 13: Terms used in the literature which imply an increased motion of the femoral head

| |
|---|
| Instability (Boykin et al. 2011) |
| Dislocation (Boykin et al 2011b) |
| Subluxation (Boykin et al. 2011a) |
| Subtle joint subluxation (Standaert et al 2008) |
| Capsular laxity (Boykin et al. 2011) |
| Capsular redundancy (Smith and Sekiya 2010) |
| Focal capsular redundancy and laxity (Shu and Safran 2011) |
| Ligamentous laxity (Shindle et al. 2006, Boykin et al. 2011) |
| Microinstability (Shindle et al. 2006, Boykin et al. 2011) |
| Subtle or gross instability (Smith and Sekiya 2010) |
| Subtle instability (Sahrmann 2002) |
| Subclinical instability (Bowman et al 2010) |
| Increased anterior gliding (Lewis et al. 2007) |
| Hypermobility (Groh et al. 2009) |

The causes of increased femoral head motion may include: underlying systemic disease (Boykin et al. 2011); congenital body or soft tissue abnormalities (Martin et al. 2006, Smith and Sekiya 2010, Boykin et al. 2011, Shu and Safran 2011); mild osseous hip dysplasia not meeting radiographic diagnosis (Shu and Safran 2011); hormonal influences (Groh et al. 2009) and acquired abnormalities. The literature appears to be in a consensus that capsular laxity may be caused by repetitive rotation with axial loading (Martin et al. 2006, Smith and Sekiya 2010, Boykin et al. 2011, Shu and Safran 2011). Another proposed mechanism is when hip extension occurs with inefficient activity of gluteus maximus and iliopsoas (Lewis et al. 2007).

The diagnosis of the clinical entities in table 14 are not well defined compared with other pathologies and thus present a clinical challenge (Smith et al. 2010). There is also considerable overlap in the clinical presentation of a number of hip conditions (FAI, acetabular tears and atraumatic instability) with regards to symptoms and aggravating activities (Shindle et al. 2006). A better understanding of the diagnosis and rehabilitation of these clinical presentations is relevant since they may be involved in the aetiology of FAI (Smith and Sekiya 2010, Boykin et al. 2011).

We propose a more general and simplified concept to aid in sub-classification and rehabilitation - translation control deficits. A translation control deficit (TCD) would be "increased translational movement, or displacement of the femoral head, beyond normal physiological parameters for a specific individual". It may also be possible that reduced translational movement of the femoral

head could occur when it is held in a displaced axis of rotation by increased muscular activity (Martin et al 2006, Smith and Sekiya 2010). Within this general sub-classification, both increased and decreased femoral head movement would constitute a TCD. As noted above in table 1, a diagnosis of specific tissue pathology should also be made concurrently with a TCD (if possible).

A TCD is useful sub-classification since it provides a rehabilitation directive for the presentation irrespective of whether a specific patho-anatomical diagnosis can be made or not. Hence, in any scenario when a TCD is present, exercises for translation control can be prescribed in rehabilitation. This presentation would also involve the rehabilitation of MPCD and muscle imbalances, however clinical observations allow us to hypothesize that the reverse is not necessarily the case in that you may have a MPCD without a TCD. The concept of a more general term such as TCD may also appropriately address the spectrum of this clinical presentation from subtle increased movement of the femoral head (e.g. micro-instability) to gross movement (e.g. instability) and thus aid in greater reliability of physical assessment. A possible weakness of this more general approach to sub-classification is that people with true instability may not achieve the same outcomes as those with subtle or micro instability, however there are a number of factors that could influence this.

From a rehabilitation perspective, the approach to address a TCD is to specifically target the muscles that control translation, and integrate with the other exercise approaches (as well as into function). Core stability has been recommended for conservative management of FAI (Smith and Sekiya 2010, Boykin et al. 2011). Core stability has not been well defined and involves a spectrum of exercises that aim to control translation; improve posture and alignment; normalize movement patterns, and improve the capacity of the body (strength and endurance) (Gibbons 2007b). Information is available elsewhere relating to specific translation control exercises for the hip (Gibbons et al 2002, Gibbons 2007c), muscle imbalance (Page et al. 2010, Grimaldi 2011) and movement pattern control (Sahrmann 2002, Lewis and Sahrmann 2006, Sahrmann and Associates 2011). Some common clinical tests for translation control are described in table 14. The tests described do not rely on pain reproduction or range of motion, but rather the therapist's monitoring of the femoral head. Clicking or clunking are not considered a positive sign unless the femoral head movement occurs with the click or clunk. Other tests such as the dial test or standard impingement test should still be performed to gain information about translation control and joint structures.

Table 14: Translation control tests of increased femoral head motion

| Test | Position and Test Movement | Monitoring and Interpretation |
|---|---|---|
| One-leg standing test – hip[1] | For right hip: Standing with most of their weight on the left leg. Weight shift onto the right leg and lift the left hip into end range hip flexion | On the weight bearing leg, palpate the greater trochanter and the femoral head. There should be no movement of the femoral head or the greater trochanter**. A positive test is if any motion if felt. |
| Active straight leg raise: concentric*[2] | For the right hip: supine with hands by their side. The right hip is flexed to end range. If painful, this test can be modified to a heel slide and hip flexion. | Palpate the greater trochanter and the femoral head (anterior). There should be no anterior displacement of the femoral head or the greater trochanter. A positive test is if any motion if felt. Any motion is best palpated when the lever is the greatest (as soon as the hip flexes). |
| Active straight leg raise: eccentric[3] | For the right hip: supine with hands by their side. The right hip is flexed to end range and held passively by the therapist. The patient then eccentrically lowers the hip from flexion. | Palpate the greater trochanter and the femoral head (posterior). There should be no anterior displacement of the femoral head or the greater trochanter. A positive test is if any motion if felt. |
| Eccentric lowering from hip flexion[4] | For the right hip: supine with hands by their side. The right hip is flexed to end range with the knee flexed relaxed and held passively by the therapist. The patient then eccentrically lowers the hip from flexion while actively flexing the knee. | Palpate the greater trochanter and the femoral head (posterior). There should be no anterior displacement of the femoral head or the greater trochanter. A positive test is if any motion if felt. |
| Prone hip extension from flexion[5] | For the right hip: lie prone over a bed so that the hips are flexed to at least 45° and the pelvis is on the bed. The right hip is actively extended to horizontal with the knee extended. | Palpate the greater trochanter and the femoral head (anterior). There should be no anterior displacement of the femoral head or the greater trochanter. A positive test is if any motion if felt. |

*The active straight leg raise for the hip has not been researched as it has been for the sacro-iliac joint, therefore compression of the pelvis or hip region is difficult to interpret
** Medial or lateral movement of only the greater trochanter is a sign of a MPCD into rotation and not a TCD

Acknowledgements: [1]This test was interpreted and modified from Lee (2010). [2]This test was interpreted from Shirley Sahrmann. [3]This test was independently extrapolated from test 4. [4]This test was interpreted from Mark Comerford. [5]This test was independently developed.

## Gait

The human hip joint withstands high contact forces during normal walking and is therefore susceptible to injury and structural deterioration over time (Correa et al 2010). Changes in gait pattern can put additional stress along the whole kinetic chain, including the hip joint. For forward propulsion in normal gait we need to have adequate mobility – e.g.: heel roll, ankle dorsiflexion, toe extension, mid foot collapse and resupination, contralateral hip extension and trunk rotation. With any of the above being restricted, the body has to compensate to keep functioning.

Functional hallux limitus is the condition in which the first metatarsal is restricted into extension (Dannenburg 1993). During the single support phase of gait, 30° - 40° of pivotal function is required between the foot and the supporting ground surface for sagittal motion. There are numerous compensations that may occur for this restriction. These include: midfoot rotation, rearfoot eversion, tibial lateral rotation, knee hyperextension, femoral rotation, femoral anterior translation, pelvic rotation, and trunk flexion. This presentation, as well as limited dorsiflexion may occur due to the presence of primitive reflexes in the foot such as the plantar grasp reflex, heel grasp reflex or foot tendon guard reflex. If these reflexes are abnormally present they can increase muscle tone in the calf and long toe flexors and limit motion (Gibbons 2012d). Lewis and Sarhmann (2006) recommend to assess for a lack of appropriate knee flexion at heel-strike and early stance phase, prolonged foot flat during stance, and knee hyperextension that causes hip hyperextension. As well, look for walking with the hip in lateral rotation as an improper correction of femoral anteversion. These observations make it clear that a gait assessment is a vital part of the assessment of movement patterns of the hip.

**Influences of Posture and Alignment**

Postural mal-alignment with its associated muscle imbalances can be a factor affecting hip function (Sahrmann 2002). A mal-alignment commonly seen is the sway posture. It is characterised by a forward sway of the pelvis, a consequent short lumbar lordosis and a long kyphosis extending from the mid upper lumbar spine to the upper thoracic spine (Kendall et al. 2005). In addition to the forward sway of the pelvis, the pelvis is frequently found in posterior tilt. This positions the hip in relative extension and decreases that natural anterior stability of the femoral head since there is less anterior coverage of the femoral head by the acetabulum. This is compounded by the normal anteriorly directed forces on the hip during the last 20% - 30% of the stance phase of gait and the anterior orientation of the femoral head (see Lewis and Sahrmann 2006 for overview).

The sway posture is characterized by short and strong hamstrings, and long and weak gluteal muscles and iliacus – psoas major. This is the imbalance that biomechanical modeling has shown places greater anterior stress on the femoral head (Lewis et al 2007) and may be related to instability (Boykin et al. 2011). The tensor fascia latae is also short and strong in the sway posture. This may create more vertical and anterior directed forces on the acetabulum (see Sims 1999 for review).

**Clinical Reasoning**

The principles of clinical reasoning related to movement pattern control and translation control are the same as with other aspects of treatment (Jones and Rivett 2003, Edwards et al. 2004). The key aspect of clinical reasoning is to relate the tissue pathology to the functional movements that may be involved in the presentation. During the subjective history, the clinician should ask questions regarding the aggravating – easing factors and relate them to movement and the relative position of the body. Some examples of these aspects of clinical reasoning are provided in tables 15 and 16. Once this understood, it will lead directly into the clinical tests that can be performed to assess for a MPCD that is related to the client's symptoms. When a diagnosis of a MPCD is made, rehabilitation can be planned. Knowledge of muscle imbalance patterns can be

used to understand which global stabilizer muscles to rehabilitate as a starting point or as a progression and which global mobilizer muscles to target for passive techniques to influence length and tone (e.g. myofascial trigger point release).

An understanding of the main MPCD(s) may lead to providing useful functional advice to help reduce the tissue load and pain provocation. This can also help in deciding what other techniques may be helpful for symptom management (e.g. taping or bracing). MPC can also assist in understanding where the source of mechanical pain is. For example, if flexion related symptoms provoked pain around the hip, but the hip region did not have any MPCD into flexion, it would be wise for the clinician to consider the lumbar spine for a source of referred pain to the hip.

Table 15: Examples of how to relate aspects of the subjective history to movement pattern control tests

| Functional Task | Relative Position of the Hip | MPC Test (examples) |
|---|---|---|
| Sitting, squatting or bending | Flexion | Standing hip flexion control |
| Walking or standing | Extension | Standing hip extension control<br>One leg standing |
| Golf swing | Rotation and extension | Squat hip rotation control<br>One leg standing |

Table 16: Clinical reasoning concepts following the assessment of a movement pattern control test

| Clinical Reasoning | |
|---|---|
| Does the movement pattern control need to be rehabilitated? | Does a movement pattern control deficit exist?<br>Is this movement functionally related to the client's symptoms and aggravating factors? |
| Which are the stabilizing muscles that need to be retrained to control this movement | With excessive movement, the stabilizer muscles often become long and will need to be retrained into their inner range.<br>If a myofascial restriction is present that prevents the ideal movement pattern control from occurring, the stabilizer synergist of this muscle may need to be retrained. |
| What are the possible mechanisms of uncontrolled movement. How would you intervene to rehabilitate these? | Listed above in tables 5 and 6 |
| What functional advice can you give? (see Lewis and Sahrmann 2006 for more detail) | If you understand the MPCD, you should be able to limit exposure to aggravating factor(s) & to modify movement to reduce strain mechanism (e.g. hip extension MPCD: take smaller steps during walking; Hip flexion MPCD: sit with hips higher than knees) |

Specific motor control exercise requires greater sensory motor awareness and neurocognitive function than general exercise (Gibbons 2012e). The Motor Control Abilities Questionnaire is a self report questionnaire designed to predict if people can learn specific motor control exercise (Gibbons 2009b). Success of the rehabilitation program depends largely on how well the precision of the exercises can be performed. Our observations for the hip are the same as the research finding of the lumbar spine in that patients with low self reported sensory motor and neurocognitive deficits and low self reported psychosocial factors do very well with a targeted specific motor control exercise approach (Gibbons 2007d, 2010). Successful conservative

management is less likely if the client cannot learn specific motor control exercise, however other rehabilitation options are available to change motor control (Gibbons 2009c).

**Issues related to implementation into clinical practice**

A number of professional issues are relevant that impact the clinical application of the information presented here. This type of rehabilitation is currently not always taught in physiotherapy undergraduate education. This means that it would have to be learned on continuing education courses. Continuing education is not mandatory in all countries and if so, it does not mean people will take courses related to this or even implement it into their clinical practice. Very few countries have guidelines on how many people can be seen per hour in clinical practice. The clinical trials that use specific motor control rehabilitation generally allow thirty minutes or more per client (Gibbons and Clarke 2009, Gibbons and Newhook 2012) which could impact the appropriate use in clinical practice if less time is spent with clients. Further evidence of related research may influence clinical trends in rehabilitation in this area.

**Summary**

This paper presented some assessment strategies and background theory concerning how altered movement patterns may be related to FAI as well as other hip disorders. It is our opinion that a specifically targeted rehabilitation program based upon appropriate sub-classification can be successful in the conservative treatment of FAI. There are many interpretations and applications of core stability and muscle imbalance in the literature. It is our experience that many of these approaches are not specific enough to change the clinical presentation so the reader should be cautioned regarding the interpretation of interventions when critically appraising any related studies. Certainly much research needs to be done in this field to address the reliability of the assessment, diagnostic accuracy and clinical effectiveness of treatment. The research base for the lumbar spine is growing (Lehtola et al. 2012) and there is preliminary data that motor control patterns can be modified with proximal control using abdominal hollowing (Cynn et al. 2006, Oh et al. 2007, Chance-Larsen et al. 2010, Park et al. 2011, Shirey et al. 2012). From an orthopaedic perspective, the underlying cause of FAI appears to be related to the morphological changes in the femur (Standaert et al. 2008), however the aetiology of FAI is not clear. Keough and Batt (2008) hypothesized that FAI could be induced by repetitive activities. This creates questions related to habitual movement patterns (not just activities) and the development of specific morphological changes related to hip structure, and hence FAI. Whether habitual movement patterns contribute to the development of the structural changes seen in FAI or not, from a rehabilitation perspective, movement pattern control, translation control and muscle imbalance are all important aspects of successful conservative management of FAI.