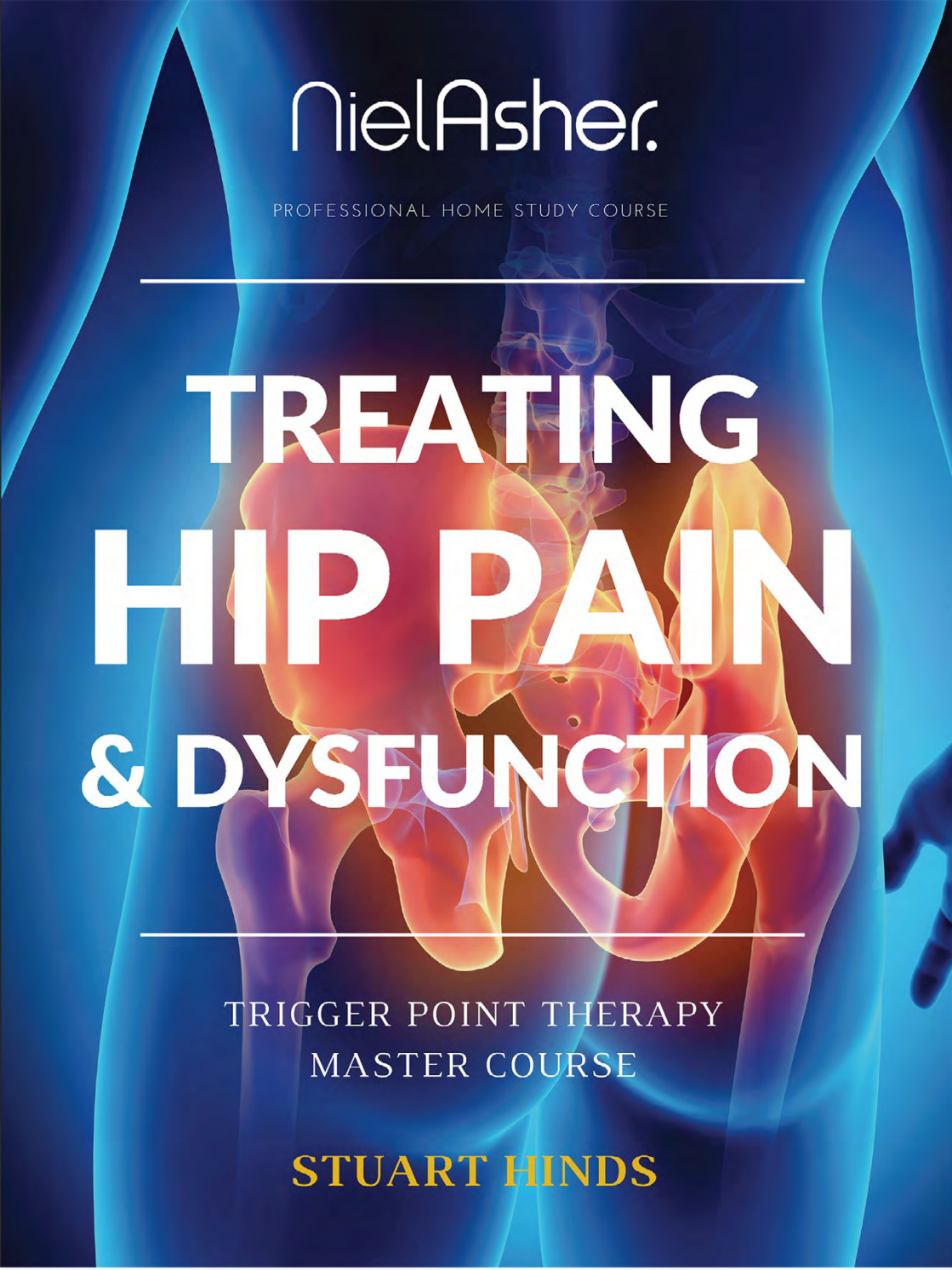
NielAsher.

PROFESSIONAL HOME STUDY COURSE

TREATING HIP PAIN & DYSFUNCTION



TRIGGER POINT THERAPY
MASTER COURSE

STUART HINDS

NAT Pro Series:

Treating Hip Pain & Dysfunction With NAT

Trigger Point Therapy Course

STUART HINDS

WELCOME

I am delighted and excited to share these Hip Treatment techniques with you. This approach was born out of the principles of the Niel-Asher Technique (NAT) as it is applied to shoulder dysfunctions.

Let's not forget that the hip is the back shoulder in a quadruped!

Hip pain is both common and debilitating, especially in athletes or people who demand high performance. Over my 20+ years career I have seen countless patients with hip pain and have often been frustrated by the lack of progress I could make.

Sure, freeing the soft tissues seems to offer temporary relief, but then the client would return again and again without any real progress.

NOW this has all changed. After using the techniques described in this course for a number of years, I and my colleagues have learned how to effect changes that are both profound and long lasting.

These changes don't just improve the hip alone but all of the associated soft tissues and the lumbar spine mechanics.

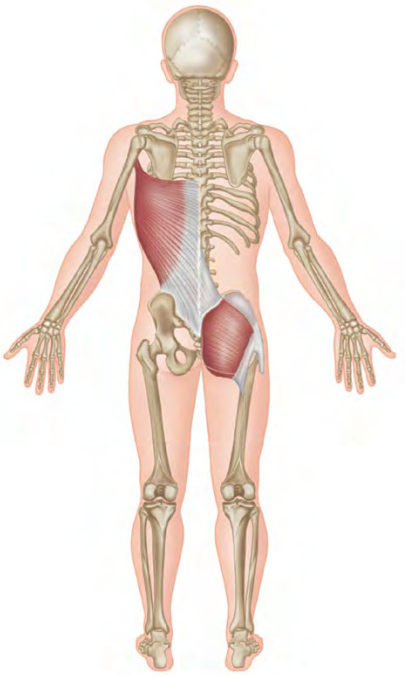
We hope that you will find this useful!

Stuart Hinds

Foreword

The hip and the shoulder share an intimate relationship, especially when we think of locomotion and 'four-legged' dynamics. Our shoulders are a modified forelimb which has been externally rotated (supinated) and our hind limbs (legs) internally rotated (pronated).

There are several fascinating and tantalizing insights connecting hip and shoulder mechanics.



Tom Myers, in his seminal work *Anatomy Trains*, explores some of the myofascial connections for distribution of load and integrated movement through both the 'shoulder and spiral lines'.

Movement and performance guru Paul Chek also has an interesting insight about a posterior oblique sling mechanism which translates rotational forces from the gluteus maximus on one side to the latissimus dorsi on the other, in concert with an anterior oblique sling mechanism translating anterior forces from the adductor complex and psoas fibers to the opposite internal oblique, as well as to the mediastinal fascia and shoulder via the diaphragm.

Chek furthermore suggests that ambulatory walking of less than 0.75km per hour translates hip and shoulder forces to the same side, whilst a gait of >0.75km per hour tends to engage the opposite limb and sling mechanism.

The hip and shoulder, as you can see, are wired to work as a unit. This is just one of the reasons why I have taken such an interest in Stuart's work in this area, and have so much enjoyed co-operating with him to present this NAT Hip course.

Simeon Niel-Asher

Introduction

Having used the NAT shoulder protocol successfully for some time, I was fascinated by its consistency and success.

Over my many years of working with elite athletes, I had often felt frustrated when it came to my ability to treat hip pain and dysfunction in particular.

It was this frustration that led me to start thinking about the integration of some key NAT principles to the treatment of the hip.

I should note that I was first introduced to the NAT shoulder technique in 2007, and have been implementing the technique regularly and with great success ever since.

Outstanding

Presented with a number of hip pain cases within Team Australia at the London Olympics 2012 and under pressure to deliver quickly, I began to put some of my thoughts regarding NAT hip to the test. The results were outstanding.

Since 2012, I have successfully treated literally hundreds of complex hip problems with the techniques and approach that we are presenting in this course.

Not only does NAT hip produce consistently good results, but the ideas behind it and their implications for treating other body regions are extremely exciting.

Holding Patterns

For example, the possibility that hip syndromes might manifest as a generalized neurological holding pattern are very compelling.

The notion that this holding pattern might be driven by nociceptive pain (via trigger points) and altered reciprocal inhibition seems to make a lot of clinical sense.

As does the idea of deliberately using nociceptive pain to change the relationships between the periphery and the cortex.

Algorithm

As with NAT Shoulder, the NAT Hip technique is based on a soft tissue algorithm that works to "unlock" the hip 'holding pattern'. This antalgic response can be seen in a variety of hip joint dysfunctions.

NAT Hip aims to address 3 common types of hip dysfunction patterns. These are:

1) Hip extension - anterior
2) Hip abduction - lateral
3) Hip rotation or complex restrictions

The NAT Hip treatment protocol has been shown to be effective for unlocking all three of these patterns in one smooth soft-tissue algorithm.

CONTENTS:

Common types of hip pain for NAT Hip

1. Arthritis, Post-operative stiffness
2. Labral tears & Femoral Acetabular Impingement
3. Groin Strain
4. Osteitis Pubis
5. Bursitis & Tendonitis
6. Snapping Hip Syndrome

The high performance hip

1. History of how the technique was developed
2. A Lightbulb moment

Common Types of Hip Pain

The Arthritic Hip

Osteoarthritis is the most common joint disorder in the Western World and is the leading cause of disability in the elderly, with nearly 200,000 total hip replacements performed annually in the USA alone. X-Ray evidence of osteoarthritis of the hip is present in about 5% of the population over the age of 65 years. However, not all patients with radiographic evidence of osteoarthritis have consistent symptoms. It is important to note that there often is an inconsistent relationship between radiographic changes and symptoms. It is not uncommon for patients to have severe OA changes on x-ray but very little hip pain (even with severe stiffness).

The main presenting feature of OA hip are pain and stiffness. As the OA advances, spinal pain and fixed flexion of the hip joint can be seen. The level of pain or stiffness patients present with is mainly related to how advanced the arthritic changes are (as well as the sensitivity of the patient). In advanced cases, the patient often presents with a tell-tale limp and loss of lumbar spine mobility, with a positive Thomas Test.

Stages of Hip OA

Osteoarthritis begins with degenerative changes of the articular cartilage in a localized, nonuniform manner. This process is followed by a subsequent thickening of the subchondral bone, new bony outgrowths at joint margins (osteophytes), and mild-to-moderate synovial inflammation (synovitis).

OA hip is categorized as primary (idiopathic) or secondary (systemic or localized) disease. Risk factors for primary osteoarthritis of the hip include old age, high bone mass, a genetic predisposition for the disease, increased BMI, participation in weight-bearing sports (e.g., running at an

elite level), and occupations that require prolonged standing, lifting, or moving of heavy objects.

Secondary causes (systemic) include hemochromatosis, hyperparathyroidism, hypothyroidism, acromegaly, hyperlaxity syndromes, Paget's disease, gout, and chondrocalcinosis.

Localized risk factors include joint injury, developmental deformities (e.g., slipped capital femoral epiphysis), Legg–Calvé–Perthes disease, acetabular dysplasia, osteonecrosis, and rheumatoid or septic arthritis as a result of cartilage damage.

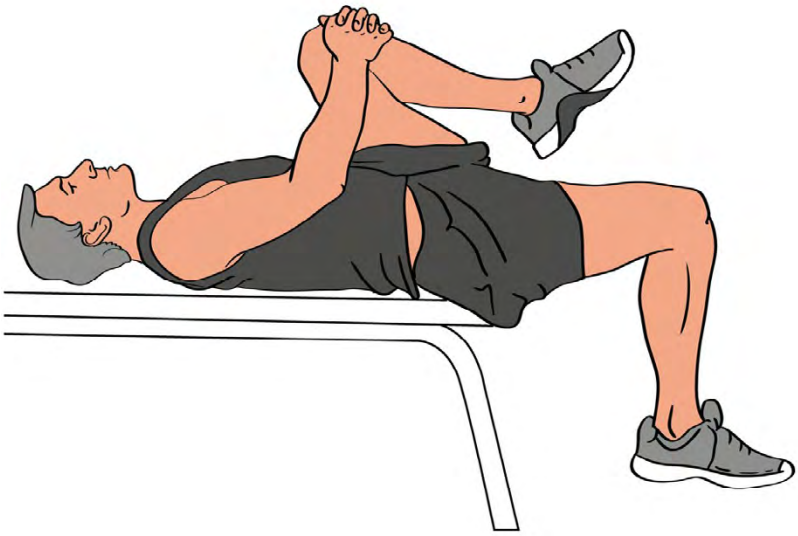
Thomas test

The **Thomas test** is used to rule out hip flexion contracture and psoas syndrome. Often associated with runners, dancers, and gymnasts who complain of hip "stiffness" and reported "snapping" feeling when flexing at the waist.

The patient lies supine on the examination table and holds the uninvolved knee to his or her chest, while allowing the involved extremity to lie flat. Holding the knee to the chest flattens out the lumbar lordosis and stabilizes the pelvis. If the iliopsoas muscle is shortened, or a contracture is present, the lower extremity on the involved side will be unable to fully extend at the hip. This constitutes a positive Thomas test. Sometimes with a very flexible patient, the Thomas test will be normal despite a psoas dysfunction being present. However, in the patient with a normal hip joint, a positive test is a good indicator of psoas hypertonicity.

Other signs from the Thomas test:

- Opposite/contralateral hip flexes without knee extension - tight iliopsoas
- Hip abducts during the test - tight tensor fasciae latae
- Knee extension occurs - tight rectus femoris
- Lateral rotation of tibia - tight biceps femoris



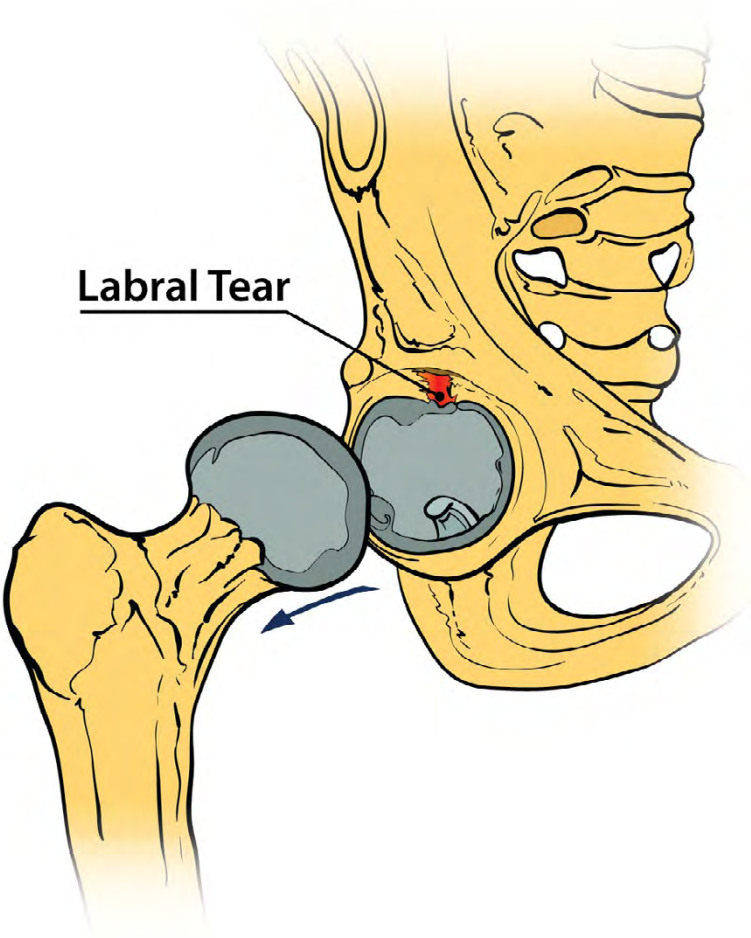
Labral Injury and Femoral Acetabular Impingement (FAI)

The Labrum

The acetabular labrum is a ring of fibrocartilaginous cartilage extending across the majority of the acetabulum, increasing its depth. The labrum acts as a type of suction seal around the femoral head, maintaining the synovial fluid within. This fluid both protects, cushions and nourishes the joint. The labrum is an integral component of hip stabilization. Hence when it is injured it can have a significant biomechanical impact.

Tears

Symptomatic labral tears are most common between the ages of 25 and 40 with equal prevalence in men and women. Labral tears typically result from an underlying problem. They are actually pretty common, with a prominent cadaver study (McCarthy et al.) demonstrating at least one labral tear in 53 or 54 specimens. Byers et al. found the labrum was detached from the articular surface of the acetabulum in 88% of people over the age of 40.


Labral Tear

Causes

1) Bony

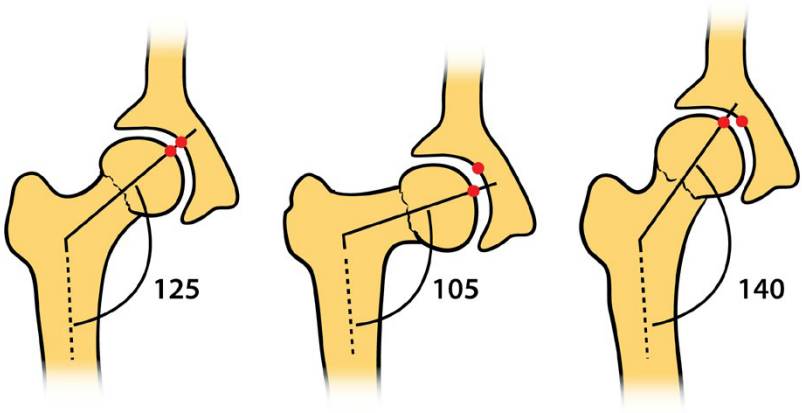
a. **Static Overload** from femoral anteversion, acetabular dysplasia (ant/lat), or valgus of femoral neck positioning

b. **Dynamic impingement** Cam impingement, Pincer impingement, Femoral retroversion

2) **Soft Tissue** Laxity (hypermobility - Ehlers-Danlos Trait), Psoas impingement

3) **Traumatic** Dislocation, Subluxation

Angle of inclination



A Normal **B** Coxa vara **C** Coxa valga

History and Presentation of Labral Tears

Overuse activities are common in labral tears, especially external rotation and hyperabduction. Patients often present with groin pain localized to the anterior hip/inguinal region. Pain can be intermittent and refer into the thigh. Sleep may be disturbed secondary to the pain. The pain is described as sharp and deep which is reproduced with high degrees of flexion and internal rotation. Prolonged sitting or activity can increase pain.

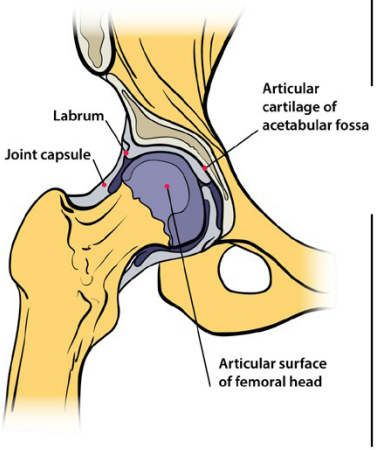
Symptoms of Labral Tear
• 92% of people complain of anterior groin pain
• 33% of people recall a traumatic onset
• 66% are suspected to be degenerative in nature
• 56-71% complain of night pain
• Up to 89% report limping
• 67% report clicking
• >50% describe locking or catching

AAOS Classification of Labral Tears	
Stage 0	Labral contusion with synovitis
Stage 1	Discreet labral tear with normal articular cartilage
Stage 2	Tear with focal articular damage to subjacent femoral head, no acetabular cartilage abnormality
Stage 3A	Tear with focal acetabular cartilage lesion <1cm
Stage 3B	Tear with focal acetabular cartilage lesion >1cm
Stage 4	Extensive acetabular labral tear with associated diffuse osteoarthritis

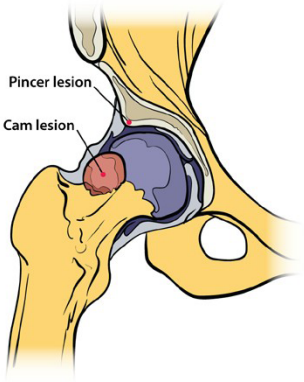
Femoral Acetabular Impingement (FAI)

Impingement occurs when the ball shaped head of the femur rubs abnormally or pinches (impinges) the acetabular socket. Damage to the hip joint can occur to the articular or labral cartilage.

Normal Hip Anatomy



Lesions that occur with Impingement



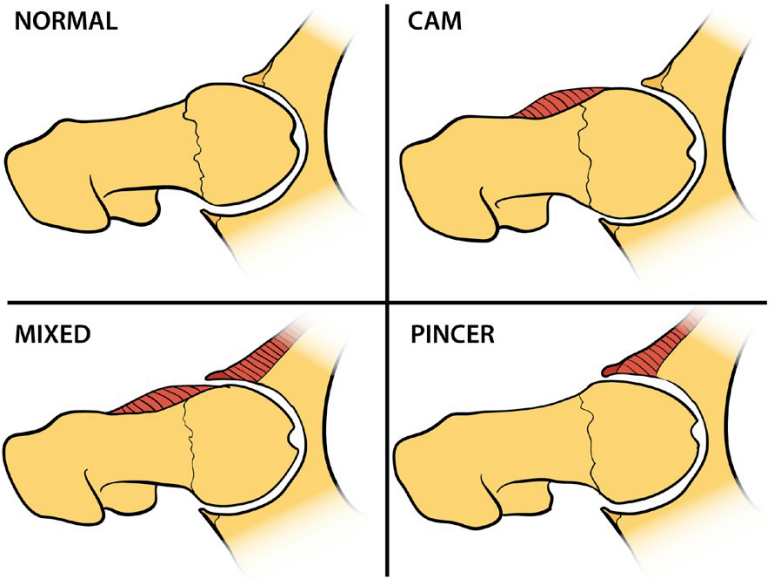
Impingement

People with FAI usually present with pain (or sometimes a dull ache) in the inguinal/groin area and sometimes more toward the outside of the hip. Sharp stabbing pain may occur with turning, twisting, and squatting. FAI occurs because the hip bones do not form normally during childhood in the growing years. The deformity of a cam bone spur, pincer bone spur, or both, leads to joint damage and pain. When the hip bones are shaped abnormally, there is little that can be done to prevent FAI.

There are three types of FAI mechanism.

1) Cam deformity - An excess of bone along the neck of the proximal femur
2) Pincer deformity - Due an excessively deep socket or an abnormal tilt of the acetabular socket
3) A mixture of the preceding two forms (most common scenario)

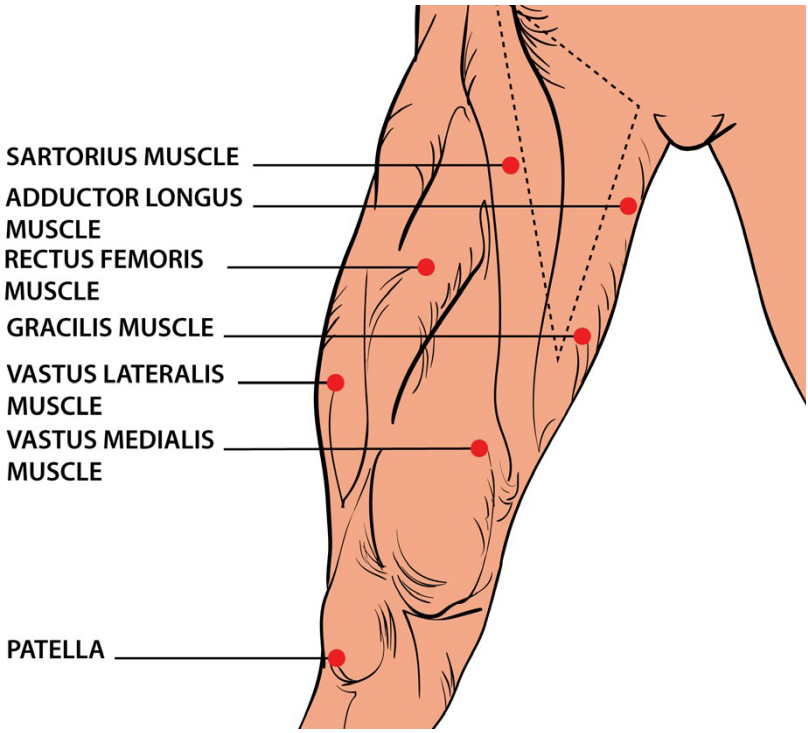
The result of any of these deformities is increased friction/catching between the acetabular cup and femoral head which may result in pain and loss/reduction of hip function.


NORMAL
CAM
MIXED
PINCER

Groin Strain

Groin injuries comprise 2 to 5 percent of all sports injuries. Early diagnosis and proper treatment are important to prevent these injuries from becoming chronic and potentially career-limiting. Adductor strains and osteitis pubis are the most common musculoskeletal causes of groin pain (in athletes). Most common in athletes that play sports like hockey, ice hockey, fencing, handball, cross-country skiing, hurdling & high jump; it may comprise 5-7% of all soccer related injuries (Westlin 1997). The diagnosis is complicated and may remain unclear in 30% of cases (Ross 1997).

Muscles of the femoral triangle



Adductor Strains

The most common cause of groin pain in athletes is probably adductor strain. This is certainly true among soccer players: in this sport, rates as high as 10 to 18 groin injuries per 100 players have been reported, and 62 percent of these have been diagnosed as adductor strains. They are fairly easily diagnosed on physical examination, with pain on palpation of the involved muscle and pain on adduction against resistance. They must, however, be distinguished from osteitis pubis and "sports hernias," which can present with pain in similar locations.

Ultrasound is useful for diagnosing muscle and tendon tears, but not muscle strains. **The most common site of strain is the musculotendinous junction of the adductor longus or gracilis.** Complete avulsions of these tendons also occur, but much less frequently. As well as treating the strain, it is well worth considering biomechanical factors that may predispose to injury; foot and lower leg malalignment, muscular imbalances, leg length discrepancy, gait or sports-specific motion abnormalities can all theoretically place abnormal loads on the adductors.

Tears and Chronicity

This has important therapeutic and prognostic implications. If an acute tear occurs at the musculotendinous junction, a relatively aggressive approach to rehabilitative treatment can be undertaken. When an acute partial tear occurs at the tendinous insertion of the adductors into the pubic bone, a period of rest must be completed before pain-free manual therapy is possible. Chronicity of symptoms is another huge factor in the clinical presentation. Complete tears of the tendinous insertion from the bone, though rare, may do better with surgical repair.

Osteitis Pubis

Osteitis pubis occurs commonly in soccer players and distance runners and is a frequent cause of groin pain. It is characterized by symphysis pain and joint disruption. It may be difficult to distinguish from adductor strains, and the two conditions may occur concomitantly in the same patient. Factors such as limitation of internal rotation of the hips or fixation of the sacroiliac joint also place excessive stresses on the joint. Leg length discrepancy and valgus or varus of the hip or knee may also play a factor, as well as abnormal Q-angles. Clinically, the patient reports exercise-induced pain in the lower abdomen and medial thigh. Symptoms are gradual in onset, slowly increasing in severity if activities are not stopped. The pain may present in differing areas:

Osteitis Pubis Presenting Symptom Area	
Adductor pain	80 percent
Pubic symphysis	40 percent
Lower abdominal pain	30 percent
Hip pain	12 percent
Referred scrotal pain	8 percent

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Hip Bursitis (Bursopathy) and Tendonitis

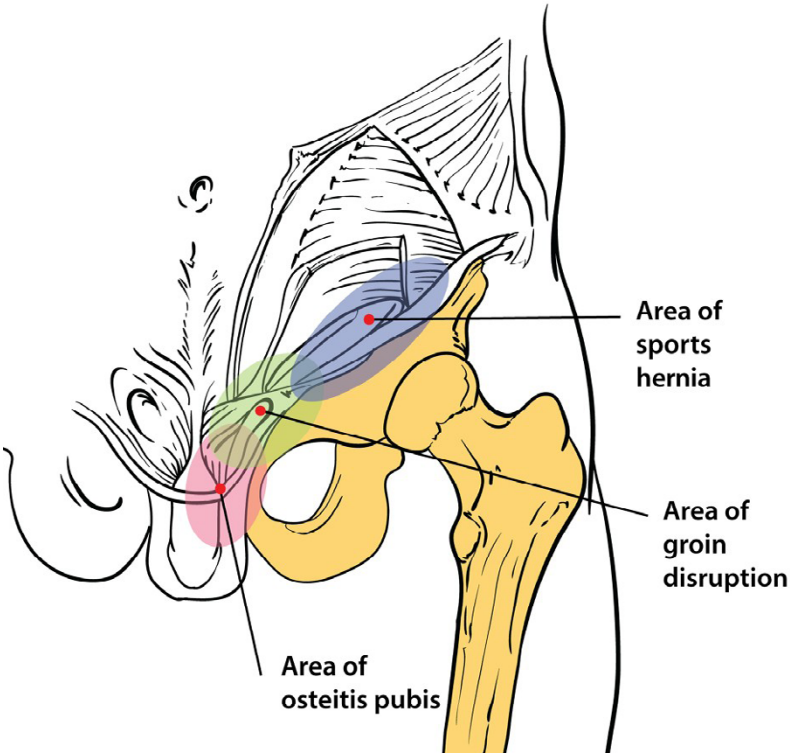
We will focus on the two most commonly presenting types of hip bursitis – Trochanteric and Iliopsoas.

Iliopsoas Bursopathy

The iliopsoas bursa is the largest bursa in the body and communicates with the hip joint in 15 percent of patients.

Bursitis is caused by overuse and friction as the tendon rides over the iliopectineal eminence of the pubis. The pain is difficult for patients to localize and difficult for clinicians to reproduce. In fact, the average time from the onset of symptoms to diagnosis is 31 to 42 months.

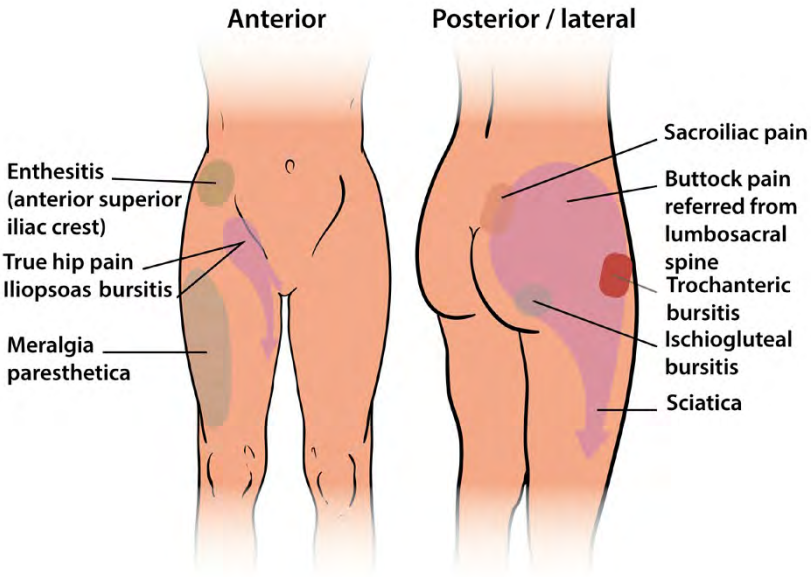
The condition is associated with sports requiring extensive use of the hip flexors (e.g., soccer, ballet, uphill running, hurdling, jumping). Iliopsoas bursitis is characterized by deep groin pain, sometimes radiating to the anterior hip or thigh, often accompanied by a snapping sensation. If severe enough, it may be accompanied by a limp.



Areas of sports hernia, groin disruption, and osteitis pubis.

Trochanteric Bursopathy (TB)

Is one of several conditions that manifest as greater trochanteric pain syndrome (GTPS). Trochanteric bursopathy is a common cause of hip pain and is frequently attributed to trochanteric bursitis and distension of the subgluteal bursae. Patients are suffering from pain radiating to the **posterolateral aspect of the thigh**, paraesthesiae in the legs, and tenderness over the iliotibial tract. The pain can be quite debilitating, with pain on walking and often pain which wakes the patient during sleep. It is also a common complication of total hip replacement. **TB** is considered to be a consequence of inflammation of the bursa between the greater trochanter and the fascia lata (also known as the iliotibial band). This inflammation can be caused acutely by trauma following a fall or during contact sports, or due to repetitive trauma as the fascia lata rubs over the greater trochanter. It can also be the result of overuse (starting a motorcycle for example). Less frequently, it can be related to sleeping on the same side night after night on a poor quality mattress.


Anterior
Posterior / lateral
Enthesitis (anterior superior iliac crest)
True hip pain
Iliopsoas bursitis
Meralgia paresthetica
Sacroiliac pain
Buttock pain referred from lumbosacral spine
Trochanteric bursitis
Ischiogluteal bursitis
Sciatica

Snapping Hip Syndrome

Snapping hip syndrome refers to a snapping sensation felt about the hip with movement. Less than one third of patients experience pain with snapping. Snapping hip may be classified **as external/lateral or as interior/medial/anterior.**

The more common external/lateral syndrome occurs when the iliotibial band, tensor muscle of fascia lata, or gluteus medius tendon rides back and forth across the greater trochanter, where bursitis may also develop.

Internal causes of snapping hip include the iliopsoas tendon passing over the iliopectineal eminence, acetabular labral tears, and subluxation of the hip and loose bodies.

You should attempt to identify the source of the clicking on physical examination. Correcting any contributing biomechanical abnormalities and stretching tightened muscles (e.g., iliopsoas muscle, iliotibial band) are important.

The High Performance Hip

The HP-Hip technique has been a culmination of years treating athletes of all levels, whether it was Olympic, International Elite, National, State, or the Amateur status. This technique developed out of the numerous discussions with athletes, coaches, biomechanists, reading of up-to-date research papers, and also from lecturing classes, where inquisitive minds tease out the questions of 'How and Why'.

It has been a long process of trial and error to formulate an algorithm for releasing the soft tissue restrictions of the hip that can be reproduced simply and consistently by others. I have used this technique for many years with huge success on athletes I have worked with, both in the past and many with whom I am still working.

Having observed the similarities between the shoulder and the hip 'holding patterns', a big question drove me, "Can I develop a technique that can release the soft tissue restrictions of the hip joint but understand and consistently achieve a change?"

The result and answer to that question would result in helping the athlete perform more efficiently; this has been my passion and desire for over two decades.

As many who know me know, the technique did not develop overnight; instead, like all good things it started with a thought, a seed if you like, and I watered it over the months and years with informative inputs from many different gardens, some gardens supplying more growth than others. It was that love and discussion which provided some of the crucial answers.

A LIGHT BULB MOMENT

It was whilst working at the London Olympics in 2012 that I started to formulate my protocol in earnest. I had been working with the NAT soft-tissue protocol for frozen shoulders for many years, and it was whilst working with this protocol I had a light bulb moment!

NAT is an advanced trigger point technique—advanced because it utilizes the neurophysiology behind myofascial trigger points in a novel and deliberate way. The technique uses deep stroking massage and compression/inhibition techniques blended together in a choreographed algorithm. In NAT, trigger points are regarded not as the familiar knots of muscular dysfunction, but as "inputs" to the spinal cord and central nervous system. This is because trigger points are exquisitely painful and, as discussed above, stimulating the pain pathways has many profound effects on the nervous system, including the attenuation of peripheral and central sensitization.

During most hands-on treatments, a haphazard stream of various mechanoreceptors are stimulated. In NAT, the number of inputs is reduced to an absolute and purposeful minimum. Inputs are made via the trigger points in repeatable sequences, which always include the manipulation of STPs (super trigger points) as well as trigger points in agonists and antagonists. Part of the NAT input sequence is performed three times: repeating something three times (either verbally or somatically) seems to help the nervous system "get the point." Stroking massage is performed in "one direction" only, and compression techniques are performed to the point of pain (and are sometimes held for up to 10 minutes).

NAT was originally developed in 1997 by Simeon Niel-Asher to treat frozen shoulder syndrome (adhesive capsulitis). The shoulder is one of the most complex regions in the body, involving four joints and eighteen muscles. A frozen shoulder is considered by many to manifest the worst components of all other shoulder problems put together. It is in many ways an enigma, and like other enigmas, solving the puzzle helps us gain many truths and insights of the inner workings of the nervous system. Simeon observed that specific tender/trigger points seemed to be present in the same distribution in every single patient. To his surprise, by manipulating these points in a sequence, he discovered that patients' long-standing frozen shoulders seemed to melt away in as few as one or two treatments. This rapid "defrosting" could only be explained by a neurological process which led to the current theoretical model.

NAT Theory

Intentionally stimulating mechanoreceptors embedded within and around the trigger points (and joints) generates a novel "neural signature," which affects the spinal cord and the somatic cortices. NAT deliberately utilizes some of the automatic reflexes associated with trigger points, including:

- Kohnstamm Reflex
- Co-coordination
- Reciprocal inhibition
- Post-isometric relaxation
- Post-activation depression
- Pure facilitation
- Co-facilitation
- Autonomic (ANS) responses
- "Pain gate"
- Spinal cord reflex responses
- Neuroplasticity

The nervous system responds to these input sequences by releasing the "holding pattern," normalizing motor unit output, and improving co-coordination. Clinically, after each NAT session, patients describe a sense of joints being "oiled inside" or feeling that "normal" muscular control has been regained. With regard to somatic dysfunction, NAT is readily used to reinvigorate and release protective joint postures (such as with spondylolisthesis) and/or treat protective spasm around joint problems (such as an arthritic hip).

Furthermore, NAT sequences seem to tone or reactivate the muscles around the joint; as an automatic response, there is also an increase in strength and power.

Hip Holding Patterns

The most common patterns is the **lateral hip pain pattern** – this is associated with: non-specific bilateral lower back pain/stiffness and insentient hamstring tightness. On palpation excessive tension and sensitivity to the gluteus medius/minimus can be felt; this is also consistent with chronic overloading.

In the past, I found that treatment to lower back tissues helped relieve the symptoms but these improvements were short-lived. When these cases presented they often became chronic, with an ongoing cycle of continued overload and restriction.

Once I starting assessing passive hip abduction, extension, internal rotation range-of-motion, I figured out the 'holding patterns'.

- **Hip abduction** restriction was indicating **adductor magnus** hypertonicity.
- **Hip extension** restriction was indicating **iliopsoas hypertonicity** just superior to the lesser trochanter attachment.
- **Hip internal rotation** restriction was individually present in 4 common regions: **adductors, tensor fascia latae, hip flexor** or **deep external rotators.**

I thought to myself, "surely these restrictions must be neurologically mediated" – similar to that observed by Simeon in his frozen shoulder holding patterns.

Unlocking the hip

The first major key was unlocked when I realized that the tonicity housed in the **adductor muscles** was the key to the **gluteus overload** - once this was cleared with trigger point manipulation, the gluteus overload was resolved.

Similarly, with the **hip extension restriction** - once the **hip flexor attachment** trigger point in the **iliopsoas attachment** and the **rectus femoris** was deactivated, the hip was free to move into extension without restriction at all.

The final icing was the hip **internal rotation restrictions** cleared up. These varied from athlete to athlete, sometimes presenting as a simple restriction, other times it was multiple and slightly more complex.

The consistent changes were very evident with both patient feedback and the clear changes in tissue tone. At the end of using my algorithm, there was an objective release in all the hip restrictions as well as the soreness in the hip, lower back, and hamstrings.

The results of using my NAT algorithm were **transformative** for the athletes. Not only did it mean we were no longer dealing with their complex hip restrictions but they no longer presented with overload, pain, and fatigue. It was clear to see that the **NAT hip protocol improved their performance and their stamina.**

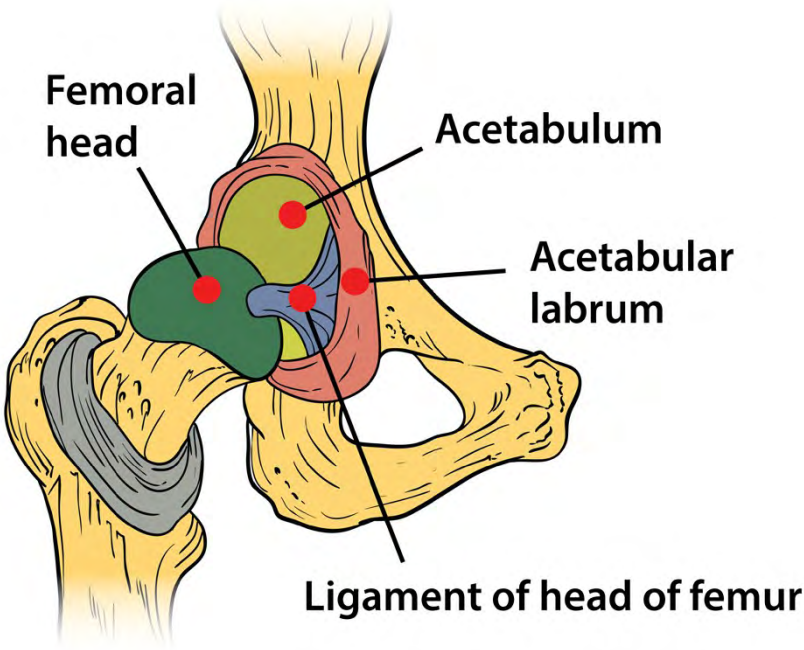
Simple!

HIP JOINT ANATOMY AND PHYSIOLOGY

The hip joint consists of an articulation between the **head of femur** and **acetabulum** of the pelvis.

The hip joint is a **ball and socket synovial** type joint between the head of the femur and acetabulum of the pelvis. It joins the lower limb to the pelvic girdle. The hip joint is designed to be a stable weight-bearing joint. To achieve this, a large range of movement is sacrificed for stability.

Structures of the Hip Joint
Articulating Surfaces



The femoral head and acetabulum of the hip joint.

The acetabulum is a cup-like depression in the lateral side of the pelvis (much like the glenoid fossa of the scapula). The head of femur is hemispherical and fits completely into the concavity of the acetabulum.

Both the acetabulum and head of femur are covered in **articular** cartilage, which is thicker at the places of weight bearing.

Ligaments

The ligaments of the hip joint act to increase stability. They can be divided into two groups – intracapsular and extracapsular.

Intracapsular

The only intracapsular ligament is the **ligament of head of femur**. It is a relatively small ligament that runs from the acetabular fossa to the fovea of the femur. It encloses a branch of the obturator artery, which comprises a small proportion of the hip joint blood supply.

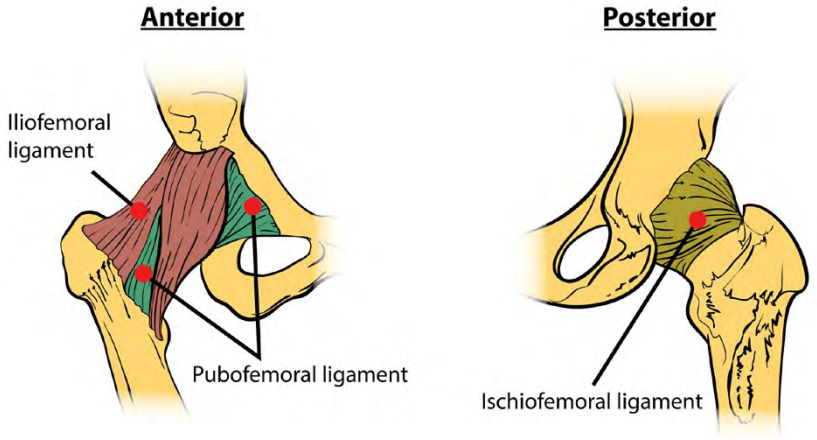
Extracapsular

There are three extracapsular ligaments. They are continuous with the outer surface of the hip joint capsule.

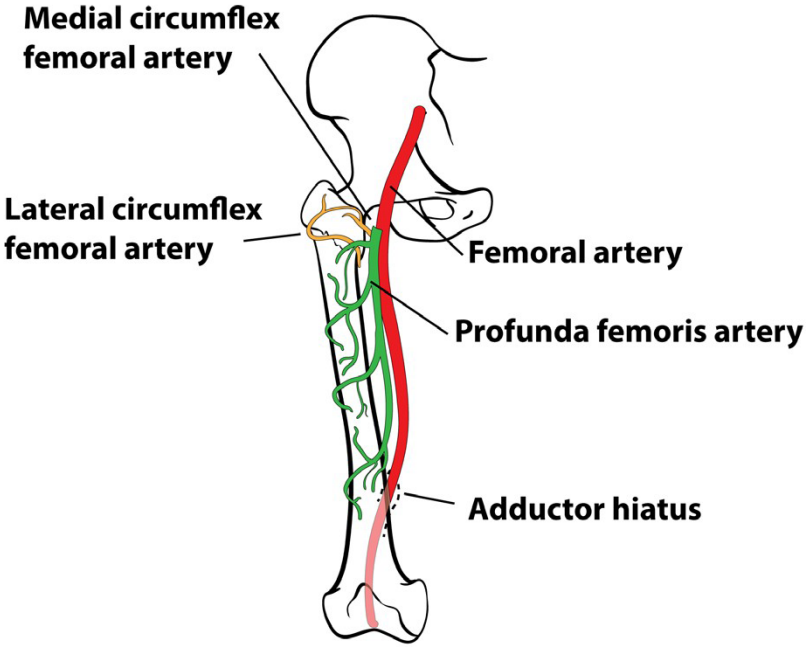
- **Iliofemoral**: Located anteriorly. It originates from the ilium, immediately inferior to the anterior inferior iliac spine. The ligament attaches to the intertrochanteric line in two places, giving the ligament a Y-shaped appearance. It prevents hyperextension of the hip joint.
- **Pubofemoral**: Located anteriorly and inferiorly. It attaches at the pelvis to the iliopubic eminance and obturator membrane, and then

blends with the articular capsule. It prevents excessive abduction and extension.

- **Ischiofemoral**: Located posteriorly. It originates from the ischium of the pelvis and attaches to the greater trochanter of the femur. It prevents excessive extension of the femur at the hip joint.



The extracapsular ligaments of the hip joint.



The medial and lateral circumflex arteries.

Vascular supply to the hip joint is achieved via the medial and lateral **circumflex femoral arteries**, and the artery to the head of the femur. The circumflex arteries are branches of the profunda femoris artery. They anastomose at the base of the femoral neck to form a ring, from which smaller arteries arise to the supply the joint itself.

The **medial** circumflex femoral artery is responsible for the majority of the arterial supply (the lateral circumflex femoral artery has to penetrate through the thick iliofemoral ligament to reach the hip joint).

Damage to the medial circumflex femoral artery can result in **avascular necrosis** (AVN) of the femoral head.

The hip joint is innervated by the femoral nerve, obturator nerve, superior gluteal nerve, and nerveto quadratus femoris.

Stablising Factors

The primary function of the hip joint is to **bear weight**. There are various structures present that increase its stability.

The first structure is the **acetabulum**. It is deep, and encompasses nearly all of the head of the femur. This decreases the probability of the head slipping out of the acetabulum, and causing a dislocation. There is a fibrocartilaginous collar around the acetabulum which increases its depth. It is known as the **acetabular labrum**. The increase is depth provides a large articular surface, thus improving the stability of the joint.

The iliofemoral, pubofemoral, and ischiofemoral ligaments are very strong, and along with the thickened joint capsule, they stabilise the joint greatly. These ligaments have a unique **spiral orientation**; this causes them to become tighter when the joint is extended, which adds stability to the joint, and also means less energy is needed to maintain a standing position.

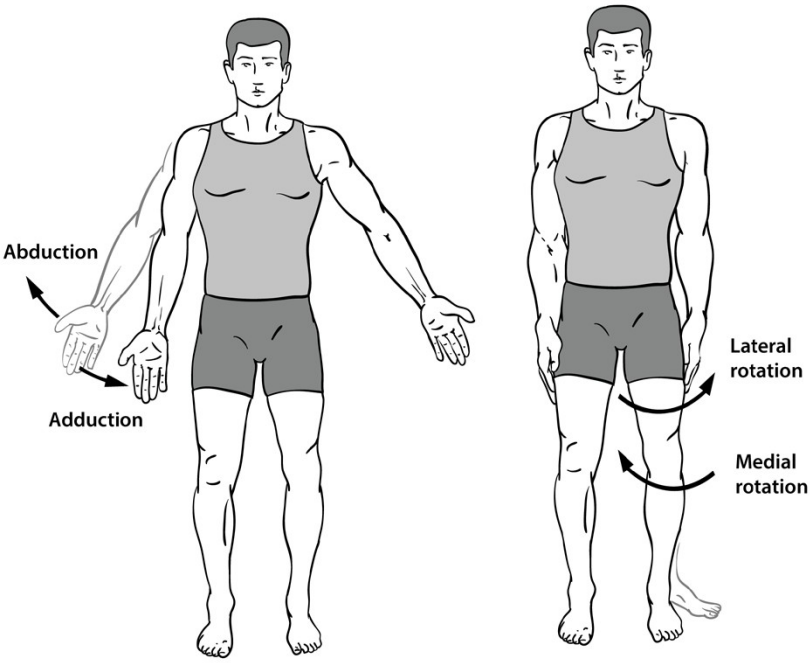
Muscles and ligaments work in a reciprocal fashion at the hip joint:

- **Anteriorly**, where the ligaments are strongest, the medial flexors (located anteriorly) are fewer and weaker.
- **Posteriorly**, where the ligaments are weakest, the medial rotators are greater in number and stronger – they effectively 'pull' the head of the femur into the acetabulum.

Movements and Muscles

The movements that can be carried out at the hip joint are flexion, extension, abduction, adduction, and medial/lateral rotation.

The degree to which flexion at the hip can occur depends on whether the knee is flexed, which relaxes the hamstrings and increases the range of flexion.


Abduction
Adduction
Lateral rotation
Medial rotation

Adduction, abduction, and rotation.

Extension at the hip joint is limited by the joint capsule, and in particular, the iliofemoral ligament. These structures become taut during extension to limit further movement.

Listed below are the movements of the hip joint, and the principle muscles responsible for those movements:

- **Flexion**: Iliopsoas, rectus femoris, sartorius
- **Extension**: Gluteus maximus, semimembranosus, semitendinosus, and biceps femoris
- **Abduction**: Gluteus medius, gluteus minimus, and the deep gluteals (piriformis, gemelli, etc.)
- **Adduction**: Adductors longus, brevis and magnus, pectineus, and gracilis
- **Lateral rotation**: Biceps femoris, gluteus maximus, and the deep gluteals (piriformis, gemelli etc)
- **Medial rotation**: Gluteus medius and minimus, semitendinosus, and semimembranosus

DIFFERENTIAL DIAGNOSIS - What Else Could It Be?

Differential diagnosis is an essential tool to assess the risk of any problems when they come to the office for treatment. Early detection and treatment can facilitate effective recovery — plus, we don't want to hurt someone who comes to us in pain!

We highly recommend using the pathological sieve CDFIMNRT to sift through the potential alternatives to a hip issue, of course; it is possible to have two or more conditions happening at the same time. This sieve is a quick and easy format that you can apply to all complaints. Don't forget to add '**emotional**' to this list.

PATHOLOGICAL SIEVE	
HIP & GROIN PAIN	
CONGENITAL	Slipped Upper Femoral Epiphysis (SUFE), Congenital Hip Dislocation (CDH), Legg-Calve-Perthes disease
DEGENERATIVE	Hip arthritis, bursitis, labral tears, osteoporosis, acetabular dysplasia, osteonecrosis, referred lumbar disc disease (L4 NRI), stress fractures
FUNCTIONAL	Impingement, quadriceps tendonitis, bursitis, synovitis, FAI, osteitis pubis, postpartum
INFECTIVE	Tuberculosis, osteomalacia, osteomyelitis, abscess, Staph infections, septic arthritis, intra-abdominal disorders (e.g., aneurysm, appendicitis, diverticulosis, inflammatory bowel disease), genitourinary abnormalities (e.g., urinary tract infection, lymphadenitis, prostatitis, scrotal and testicular abnormalities, gynecologic abnormalities, nephrolithiasis)

METABOLIC	Hormonal: hypo- or hyperparathyroidism, pituitary disease related, inflammatory arthritis (HLA-B27 - <0.3%), psoriatic arthropathy, inflammatory bowel disease, polymyalgia rheumatica, hemochromatosis, acromegaly, hyperlaxity syndromes, gout, chondrocalcinosis
NEOPLASTIC	Primary soft tissue (rhabdomyosarcoma) or secondary metastases
RETICULO-ENDOTHELIAL	Systemic lupus erythematosis (SLE), multiple myeloma, Brown Tumor, Paget's disease, rheumatoid arthritis
TRAUMATIC	Fractures of hip, impingement, femoral or cutaneous nerveimpingement – meralgia paraesthetica

Differential Signs and Symptoms of Hip Pain

Condition	History	Features of Physical Examination	Laboratory or Radiographic Features
Trochanteric bursitis	Lateral hip pain aggravated by direct pressure; intolerance of sleeping on affected side	Point tenderness over trochanteric bursa, about 2.5 cm posterior and superior to the greater trochanter	None
Meralgia paresthetica (lateral femoral cutaneous nerve entrapment)	Localized area of pain on lateral aspect of hip that can range from numbness and tingling to burning pain	Not affected by direct pressure, hip movement, or lower back movement	None
Lumbar radiculopathy	Lateral or posterior hip pain that can radiate down the leg and into the foot with or without associated foot numbness (especially at the L4-L5 level); commonly with low back pain; possible numbness and weakness in the lower leg	Straight leg raise eliciting pain in the leg, buttock, or back at 60 degrees or less of leg elevation; if test is positive, suggestive of L5 or S1 nerve-root irritation; possibly with reduced strength and sensation and impaired reflexes in the lower leg†	Narrowing of intervertebral disk spaces, disk herniation, and nerve-root swelling or impingement, as seen on MRI of lower lumbar spine
Lumbar spinal stenosis	Lateral and posterior hip pain that may radiate	Usually normal; reflexes of ankle or knee may be	Narrowing of the intraspinal canal, as seen

	to the lower leg or groin, exacerbated by walking or standing and relieved by sitting and leaning forward or lying down; back pain characteristic; possibly with numbness and weakness in the lower leg, especially after ambulation	impaired; muscle weakness in the lower leg, especially after ambulation	on MRI or CT of lumbar spine
Chronic inflammatory arthritis (including rheumatoid arthritis and spondyloarthropathies)	Prominent morning stiffness, possibly with pain in other joints and other systemic manifestations of disease	Swelling and tenderness in other joints	Increased erythrocyte sedimentation rate or level of C-reactive protein; inflamatory synovial fluid (>2000 white cells per cubic millimeter)
Osteonecrosis	Anterior groin pain with joint use that can lead to thigh and buttock pain; frequent pain on walking and at rest; often with a history of corticosteroid use	Tipically with pain on range of motion	Normal radiographic findings in early stages of disease, possibly with visible areas of femoral necrosis on MRI before changes in femoral head are visible on radiography

Iliotibial band syndrome	Lateral hip pain (aching or burning) that radiates down the side of the leg and is exacerbated by running or walking	Pain and tenderness along the iliotibial band with the patient lying with the involved hip up and the involved leg lowered and then moved forward and downward	None
Metastatic cancer of the femur	Lateral hip pain aggravated by direct pressure or weight bearing, possibly with nocturnal or continuous pain; often associated with a known primary cancer	Possible tenderness on direct palpation	Metastatic bone involvement on radiography of hip
Gout or pseudogout	Episodes of acute pain with history of similar episodes in other joints	Tenderness or swelling in other joints	Crystals in inflammatory synovial fluid; possible evidence of crystal deposition within the joint on radiography

Anderson BC. Evaluation of the adult with hip pain Online no. 15.7. UpToDate, 2007.

Symptoms that may indicate an underlying pathology:

- Pain following major trauma such as a road accident or fall from a height
- Pain for no apparent reason

Red Flags

Some signs and symptoms need a more careful investigation; if you are concerned, we recommend not treating the patient but sending them back to their family doctor with a note of your concerns.

- Age of appearance <20 or >50
- Severe Trauma
- Constant, nonmechanical unrelenting pain
- Prior history of:
    - Cancer
    - Steroid use
    - HIV or Drugs
    - Generally unwell (fever, weight loss, etc.)
- Structural defect
- Blood test/ESR > 25 (other inflammatory markers)
- Widespread neurological dysfunction
- Pain that develops gradually, and slowly gets worse and worse over days or weeks
- Pain, if the patient has been on steroid tablets for more than 3 months

Beliefs, Attitudes, and Emotions

Beliefs and attitudes about pain have a role to play in all types of pain. Sometimes even if the pain improves, the patient becomes 'hyper' symptom focused and is unable to recover fully. Pain behavior is complex; it is influenced by many factors as diverse as anger, depression, and hopelessness to childhood and family pain behavior. Other factors such as family problems, work problems, litigation issues, or even diagnostic and treatment issues may also have a role to play. We need to keep all of these factors in mind as we approach treatment.

Which Muscles Are Implicated In Hip Pain?

Primary

- Quadriceps Femoris
- Psoas
- Pectineus
- Gracilis
- Adductor Magnus
- Adductor Brevis
- Adductor Longus

Secondary

- Lumbar Erector Spinae
- Gluteus Medius
- Gluteus Minimus
- Small Hip Muscles
- Tensor Fascia Lata

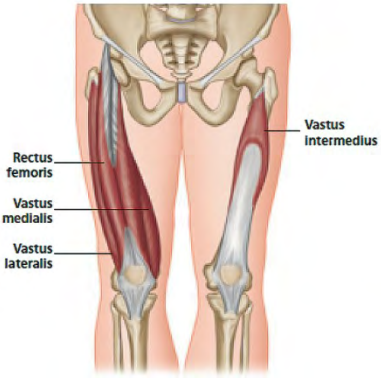
Overview

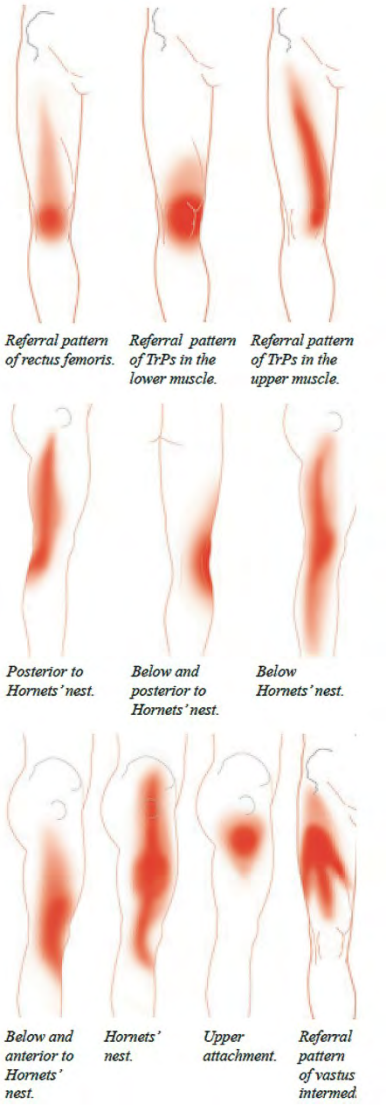
Trigger points in any of these muscles will reduce their efficiency, so it is worth checking them individually for taut bands. Remember that posture, aging, and other health issues may also influence trigger point formation and location. Also remember to look at the trigger point pain maps and the anatomy before you start; sometimes the referred pain is in a distal

location to the muscle. If you can find a taut band in one of these muscles that reproduces the patient's symptoms, then 'bingo' – go for it.

However, often there's more to it; the body tends to shut down around pain to avoid further noxious stimuli in a 'holding pattern' (pain inhibition). Part of the way it does it is by using trigger points. Depending on how long the symptoms have been there, we see certain 'classic' active trigger points in the hip holding patterns. This pattern includes the hip flexors (especially iliopsoas insertion and rectus femoris), abductors (specifically gluteus medius), adductors, and lower back muscles (erector spinae and multifidus). We will address this holding pattern with the NAT algorithm later in the course.

Rectus Femoris Anatomy and Trigger Point Map





Latin, quadriceps, four-headed.

Origin

Rectus femoris: Anterior inferior iliac spine (AIIS) and groove above the rim of the acetabulum.

Vastus medialis: Anterior intertrochanteric line, medial lip of the linea aspera, and proximal aspect of the medial supracondylar line.It is interesting to note the attachment into the tendons of the adductor longus and magnus and into the medial intermuscular septum.

Vastus lateralis: Intertrochanteric line and greater trochanter, gluteal tuberosity and lateral lip of the linea aspera, and lateral intermuscular septum.

Vastus intermedius: Anterior lateral surface of the proximal two-thirds of the femur, distal half of the linea aspera, and lateral intermuscular septum.

Articularis genu: Two slips from the anterior femur below the vastus intermedius (pulls the capsule superiorly).

Insertion

All of the quadriceps muscles wrap up the patella (sesamoid bone), with each having a unique and specific line of pull or directional force acting on the patella. They share a common tendon (patellar tendon or ligament) and attach to the tibial tuberosity.

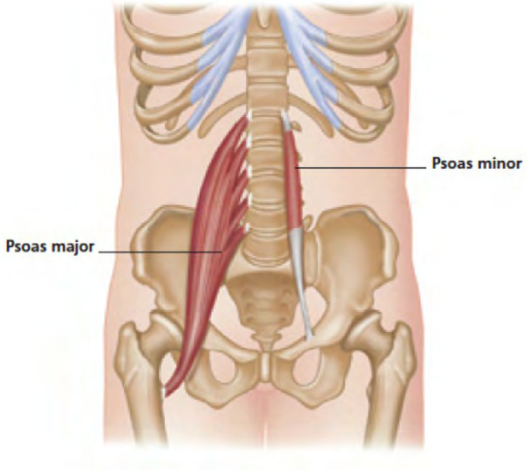
Action

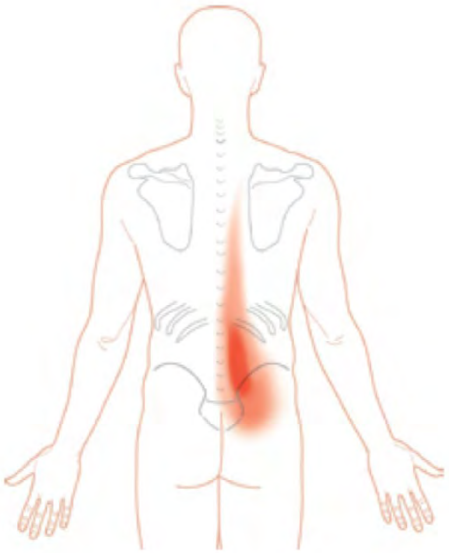
Extend the knee joint. Rectus femoris additionally flexes the hip joint.

Nerve

Femoral nerve L2- L4.

Psoas Anatomy and Trigger Point Map





Greek, *psoa*, muscle of the loin. **Latin**, *major*, larger.

Origin

Transverse processes of L1- LS, bodies of T12- LS, and intervertebral discs below bodies of T12-L4.

Insertion

Middle surface of the lesser trochanter of the femur.

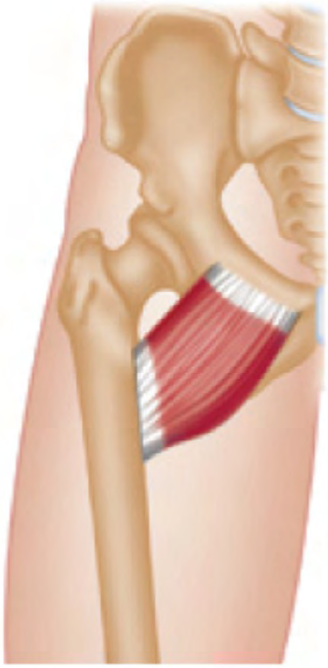
Action

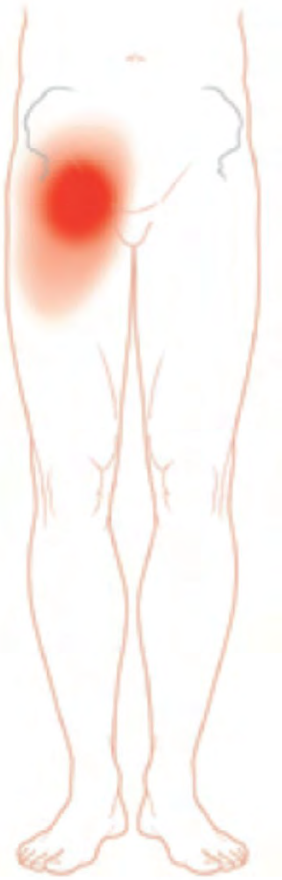
Flexes and medially rotates the hip.

Nerve

Ventral rami of lumbar nerves L1-L4.

Pectineus and Trigger Point Map





Latin, *pecten*, comb; *pectinatus*, comb-shaped.

Origin

Pectineal line of the pubis, between the iliopubic eminence and the pubic tubercle.

Insertion

Pectineal line of the femur, from the lesser trochanter to the linea aspera.

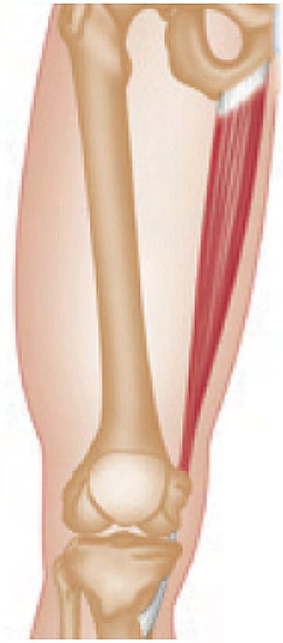
Action

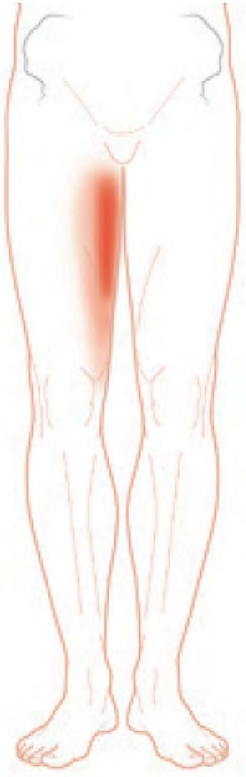
Adducts the hip joint. Flexes the hip joint.

Nerve

Femoral and obturator nerves L2-L4.

Gracilis Anatomy and Trigger Point Map





Latin, *gracilis*, slender, delicate.

Origin

Anterior lower half of the symphysis pubis, and medial margin of the inferior ramus of the pubis.

Insertion

Front and medial surface of the shaft of the tibia, just below the condyle.

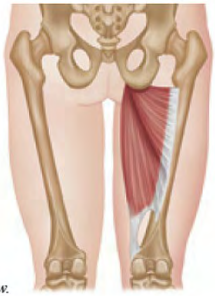
Action

Adducts the hip joint. Flexes the knee joint. Medially rotates the knee joint when flexed.

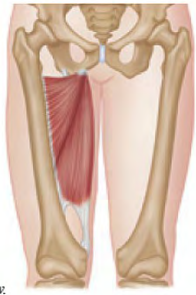
Nerve

Anterior division of obturator nerve L2- L4.

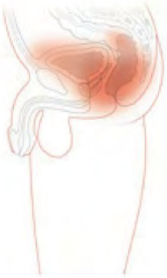
Adductor Magnus Anatomy and Trigger Point Map



Posterior view.



Anterior view.



Latin, *adducere*, to lead to; *magnus*, large.

Origin

Anterior fibers: Inferior or anterior ramus of the pubis, in the angle between the crest and the symphysis. Posterior fibers: Ischial tuberosity.

Insertion

Entire length of the femur, extending from the gluteal tuberosity along the linea aspera, medial supracondylar line, and adductor tubercle on the medial condyle of the femur.

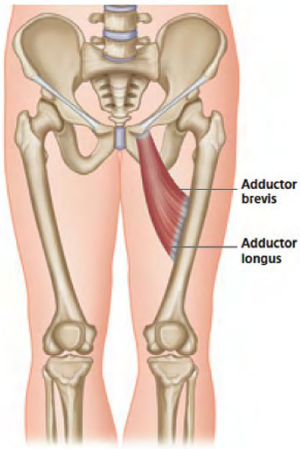
Action

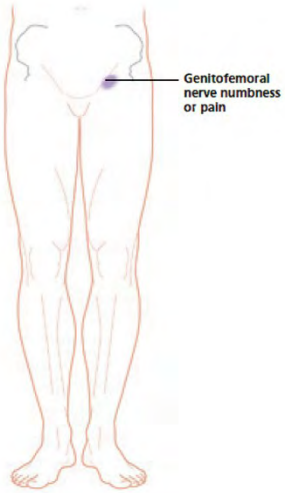
The upper fibers of the adductor magnus adduct and laterally rotate the hip joint. The vertical fibers from the ischium may assist in weak extension of the hip joint.

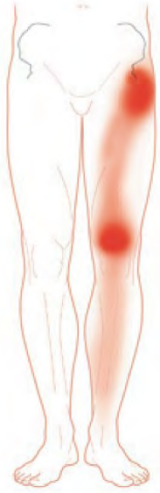
Nerve

Tibial portion of sciatic nerve L4, L5, S1. Posterior division of obturator nerve L2-L4.

Adductor Longus Anatomy and Trigger Point Map







Latin, *adducere*, to lead to; *longus*, long.

Origin

Anterior of the pubis in an angle between the crest and the symphysis.

Insertion

Middle third of the medial lip of the linea aspera.

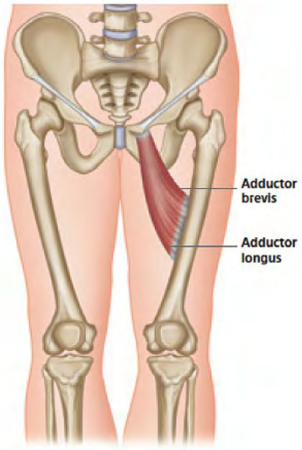
Action

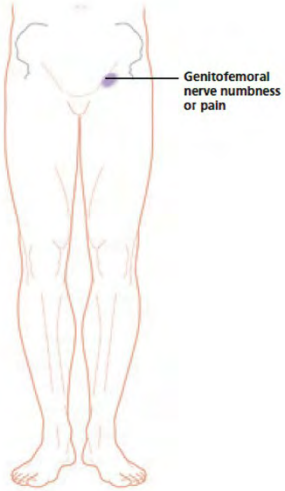
Adducts the hip joint. Flexes the extended femur at the hip joint. Extends the flexed femur at the hip joint. Assists in lateral rotation of the hip joint.

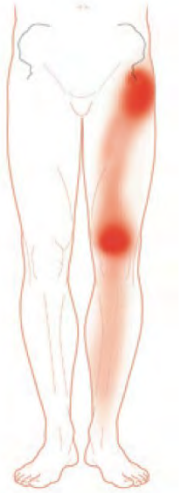
Nerve

Anterior division of obturator nerve L2- L4.

Adductor Brevis Anatomy and Trigger Point Map







Latin, *adducere*, to lead to; brevis, short.

Origin

Outer surface of the inferior ramus of the pubis.

Insertion

On a line extending from the lesser trochanter to the upper part of the linea aspera.

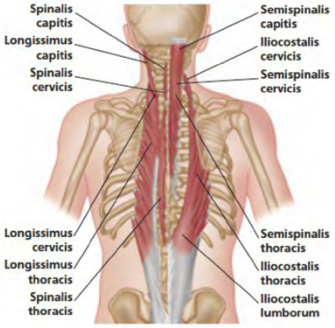
Action

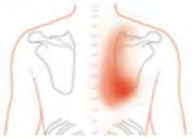
Adducts the hip joint. Flexes the extended femur at the hip joint. Extends the flexed femur at the hip joint. Assists in lateral rotation of the hip joint.

Nerve

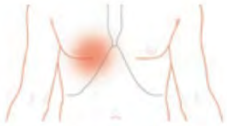
Anterior division of obturator nerve L2- L4.

Adductor Brevis Anatomy and Trigger Point Map

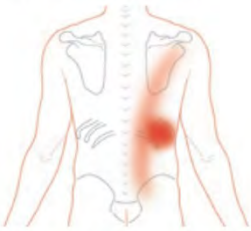




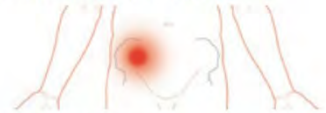
Upper iliocostalis posterior referral pattern.



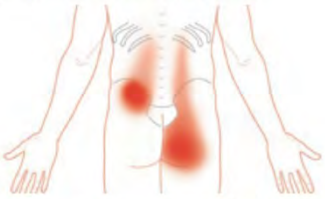
Upper iliocostalis anterior referral pattern.



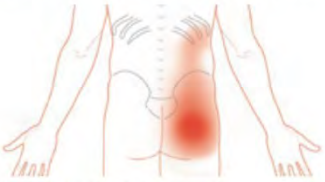
Posterior iliocostalis thoracis referral pattern.



Anterior iliocostalis thoracis referral pattern.



Lower thoracic longissimus thoracis referral pattern.



Iliocostalis lumborum referral pattern.

Latin, *erigere*, to erect; *spinae*, of the spine; *sacrum*, sacred; *spinalis*, relating to the spine.

Origin

All of the many erector spinae muscles conjoin with the thoracolumbar fascia that attaches at several different angles to the crest of the ilium and sacrum, and to the spinous processes of the lumbar and eleventh and twelfth thoracic vertebrae.

Insertion

Many different attachments to the posterior costal bones, the spinous and transverse processes of the thoracic and cervical vertebrae, and the mastoid process of the temporal bone.

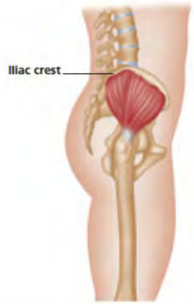
Action

Extends the vertebral column while the deep rotators and multifidi erectors rotate the spinal column to the opposite side. The semispinalis extends the vertebral column and the head.

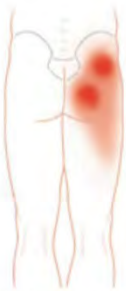
Nerve

Dorsal rami of cervical, thoracic, and lumbar spinal nerves.

Gluteus Medius Anatomy and Trigger Point Map

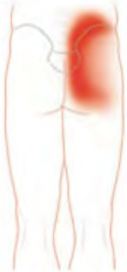


TrPs in the gluteus medius often occur in a line along and below the iliac crest.



Referral pattern of TrPs usually found under the middle portion of the muscle along and under the iliac crest.

Referral pattern of TrPs usually found under the front portion of the muscle along and under the iliac crest.



Referral pattern of TrPs usually found under the back portion of the muscle close to the sacrum, along and under the iliac crest."

Greek, *gloutos*, buttock. **Latin**, *medius*, middle.

Origin

Outer surface of the ilium, between the posterior and middle gluteal lines.

Insertion

Posterolateral surface of the greater trochanter of the femur.

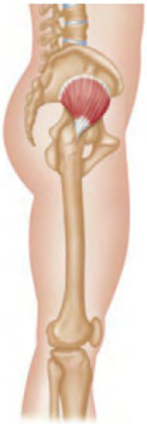
Action

Abducts and both externally/internally rotates the hip. Tilts the pelvis when walking.

Nerve

Superior gluteal nerve L4, L5, S1.

Gluteus Minimus Anatomy and Trigger Point Map



Anterior portion.

Posterior portion.

Greek, *gloutos*, buttock. **Latin**, *minimus*, smallest.

Origin

Outer surface of the ilium, between the middle and inferior gluteal lines.

Insertion

Anterior surface of the greater trochanter of the femur.

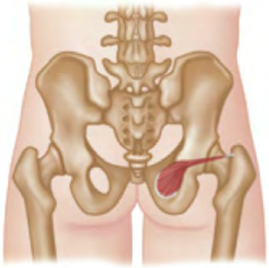
Action

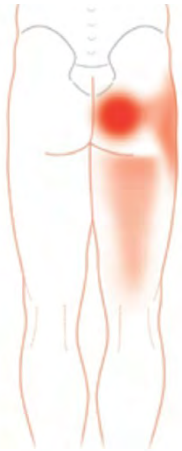
Abducts and medially rotates the hip. Assists in tilting the pelvis when walking.

Nerve

Superior gluteal nerve L4, L5, S1.

Small Hip Muscles – Obturator Internus Anatomy and Trigger Point Map





Latin, *obturare*, to obstruct; *internus*, internal.

Origin

Pelvic surface of the obturator membrane, and bony margin of the obturator foramen.

Insertion

Anterior part of the medial surface of the greater trochanter of the femur.

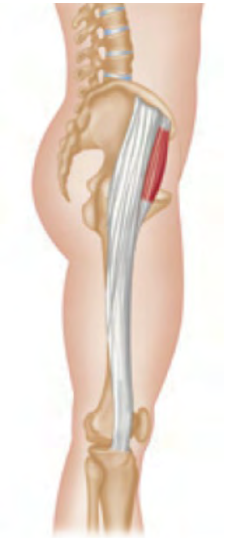
Action

Laterally rotates the extended thigh at the hip, stabilizes the hip, and produces horizontal extension. Abducts the flexed thigh.

Nerve

Branch of ventral rami of lumbar nerve L5, and sacral nerves S1, S2.

Tensor Fascia Lata Anatomy and Trigger Point Map



Latin, *tendere*, to stretch, pull; *fasciae*, of the band; *latae*, of the broad.

Origin

Anterior superior iliac spine, outer lip of the anterior iliac crest, and fascia lata.

Insertion

Iliotibial band.

Action

Assists in stabilizing and steadying the hip and knee joints by putting tension on the iliotibial band of fascia.

Nerve

Superior gluteal nerve L4, L5, S1.

Special Testing for the Hip

Special Testing for the Hip	
FABER TEST	Hip flexion, abduction, & external rotation
THOMAS TEST	Flex hips and lower the affected side slowly
IMPINGEMENT TEST	Flexion, abduction and internal rotation
OBER'S TEST	Knee/Hip extension, hip abduction
LATERAL RIM IMPINGEMENT	Flexion>Extension whilst in abduction
CRAIG TEST	Rotate limb until greater trochanter is parallel to the floor
ELY TEST	Flex knee and draw lower leg to thigh

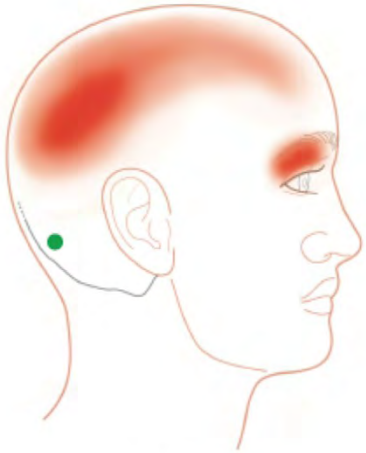
Trigger Points 101

Coined in 1942 by Dr. Janet Travell to describe painful lumps or nodules felt within tight bands of muscle. Trigger points all seem to have the following characteristics:

- Pain, often exquisite, is present at a discrete point.
- A nodule is embedded within a taut band in the muscle.
- Pressure reproduces the pain symptoms, with radiations in a specific and reproducible distribution (map).
- Pain cannot be explained by findings from a neurological examination.

About Trigger Points

- Trigger points develop in the muscle belly; so multipennate (several heads) muscles such as the Deltoid or Serratus Anterior may have several trigger points at once.
- They are the result of overstimulation of the muscle spindle, which becomes sticky and permanently 'switched-on'; this is the lump that we feel.
- They are often embedded in the muscles remotely from where the pain is felt.
- They make the host muscle shorter and fatter and reduce its efficiency; this can lead to pressure on nerves and blood vessels.
- Reduced efficiency = increased risk of injury



Occipitalis

How do trigger points develop?

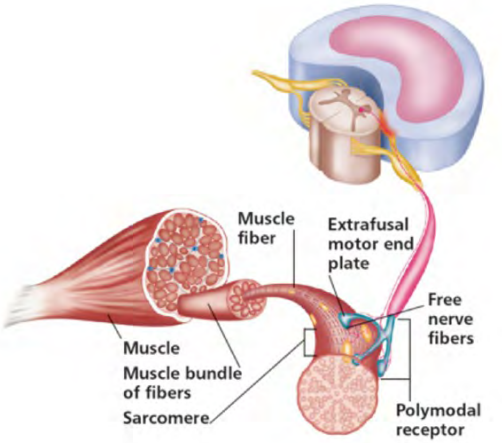
There are several possible trigger point mechanisms[1]:

- Low-level muscle contractions
- Uneven intramuscular pressure distribution
- Direct trauma
- Unaccustomed eccentric contractions
- Eccentric contractions in unconditioned muscle
- Maximal or submaximal concentric contractions

PATHOPHYSIOLOGY OF TRIGGER POINTS

The Integrated Trigger Point Hypothesis is the current theory/working hypothesis: it explains most of the trigger point phenomena, and is based on the best electrodiagnostic and histopathological evidence to date. First introduced by Travell and Simons in 1981 as the "energy crisis theory"[2], the ITPH has been expanded over the years by many others in the field.

Trigger points manifest in the region where sarcomeres and extrafusal motor endplates become overactive. Microscopy has demonstrated that actin and myosin myofilaments (sitting within a taut band) stop sliding over one another and get stuck[3]. Others have reported "pathological alterations" in mitochondria within these myofilaments, as well as an increase in the width of A bands and a decrease in the width of I bands. The affected sarcomere(s) becomes permanently "switched on," leading to a contraction and "wind-up." The swollen, contracted actin and myosin filaments may actually get stuck in the Z band because of the gel-like titin molecules ratcheting the fibers in place and preventing detachment (Dommerholt et al. 2006).



Recent electrophysiological investigations have revealed that the electrical activity of "active trigger points" arises from dysfunctional extrafusal motor endplate zones rather than from (as previously thought) muscle spindles. Electrical discharge frequencies of 10–1000 times normal have been demonstrated in the "endplate zone" in horses, rabbits, and humans (Simons et al. 2002, Dommerholt et al. 2006).

Histological investigation indicates abnormal calcium and ACh levels, and a shortage of ATP in the vicinity of the trigger point. It is worth noting that Grinnel et al. (2003)[4] demonstrated that stretching and/or hypertonicity of muscles causes a pulling of integrin protein peptides at the motor nerve terminal, triggering excessive ACh release without the need for calcium. Other abnormal chemicals present in the milieu of "active" trigger points include[5]:

- Prostaglandins
- Substance P
- Cytokines

- Bradykinin (BK)
- Hydrogen (H+)
- Calcitonin gene-related peptide (CGRP)
- Tumor necrosis factor (TNF- α)
- Interleukins IL-1 beta, IL-6, and IL-8
- Serotonin
- Norepinephrine

These chemicals have many interactions and are part of various feedback loops. For instance, bradykinin is known to activate and sensitize muscle pain fibers (nociceptors). This may help to explain some of the inflammatory hyperalgesia, tenderness, pain, and lowered pain thresholds seen in patients with chronic trigger points.

Vicious Cycle of Energy Crisis

Sustained dysfunction and sarcomere contraction leads to local intracellular and extracellular chemical changes including:

- Localized ischemia/hypoxia
- Increased metabolic needs
- Increased energy (required to sustain contraction)
- Failed reuptake of calcium ions into the sarcoplasmic reticulum
- Localized inflammation (to facilitate repair)
- Compression or watershed effect on local vessels
- Energy crisis
- Production of inflammatory agents (which sensitize local autonomic and nociceptive - pain - fibers)

If this situation is allowed to continue over a significant period of time, the above changes lead to a vicious cycle. Calcium is unable to be taken into the actin and myosin myofilaments, leading to sarcomere “failure.”

Bengtsson et al. (1986)[6], Hong (1996)[7], and Simons et al. (1998) have all proposed variations of the energy crisis theory. This theory suggests that the body attempts to resolve sarcomere and endplate failure (outlined above) by changing the blood supply to the sarcomere (vasodilation). One further result of this anomalous situation is the migration of localized acute and chronic inflammatory cells. Inflammation is a cascade: this cascade mechanism starts to occur around the dysfunctional sarcomere. Inflammation brings with it sensitizing substances, such as bradykinin and substance P, a peptide present in nerve cells, which not only increases the contractions of gastrointestinal smooth muscle, but also causes vasodilation. This has the effect of stimulating both local (small) pain fibers and local autonomic fibers, which in turn leads to increased ACh production and hence a vicious cycle.

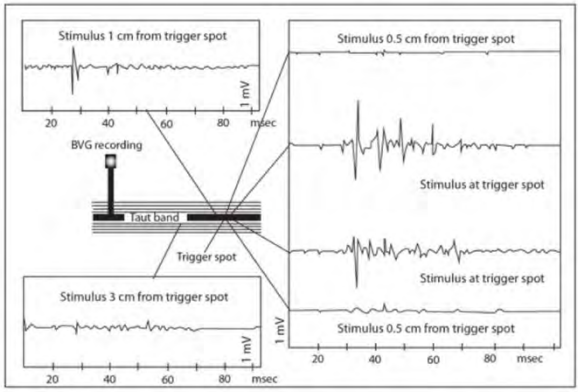
Eventually, the brain sends a signal to the muscle in which the trigger point manifests to cause it to rest. This leads to hypertonia, weakness, shortening, and fibrosis (muscle stiffness) of the muscle, along with reflex inhibition of other muscle groups. Under microscopy, these fibers have been described as "ragged red." Treatment is thus aimed at interfering with and attenuating this vicious cycle.

Other theories include radiculopathic theory[8, 9] and polymodal theory[10].

Evidence for trigger points

Studies over the past decade have imaged trigger points[11, 12] shown that their activation results in CNS activation through fMRI scanning[13], demonstrated electrophysiological activity at the trigger point[14], and have shown biochemical changes in the trigger point zone[15]. Further studies have shown that manipulation of the trigger point modulates muscle function[16], and induces local and referred pain[17].

Here's a picture of the twitch response, stimulated in a rabbit gastrocnemius muscle. Notice the increase (spike) in electrical activity as the trigger point is stimulated.



Local twitch response (LTR) in a rabbit tender spot. LTRs are elicited only when the needle is placed accurately within the trigger spot. (Adapted from: Hong 1996.)

For more information the book 'Muscle Pain' by Mense[18]

Trigger Point Classification

Trigger points are described according to location, tenderness, and chronicity as: central (or primary), satellite (or secondary), attachment, diffuse, inactive (or latent), and active.

Central (or Primary) Trigger Points

These are the most well-established and "florid" points when they are active, and are usually what people refer to when they talk about trigger points. Central trigger points always exist in the center of the muscle belly, where the motor endplate enters the muscle.

Note: Muscle shape and fiber arrangement is of importance in this regard. For example, in multipennate muscles (such as the deltoid), there may be several central points. Also, if muscle fibers run diagonally, this may lead to variations in trigger point location.

Satellite (or Secondary) Trigger Points

Trigger points may be "created" as a response to the central trigger point in neighboring muscles that lie within the referred pain zone. In such cases, the primary trigger point is still the key to therapeutic intervention: the satellite trigger points often resolve once the primary point has been effectively rendered inactive. As a corollary it is also true that satellite points may prove resilient to treatment until the primary central focus is weakened; such is often the case in the paraspinal and/or abdominal muscles.

Attachment Trigger Points

Myofascia is a continuum. It has been noted that the area where the tendon inserts into the bone (tendo-osseous junction) is often "exquisitely" tender (Simons et al. 1998; Davies 2004). This may well be

the result of the existing forces travelling across these regions. It has also been suggested by the same authors that this may result from an associated chronic, active myofascial trigger point. This is because the tenderness has been demonstrated to reduce once the primary central trigger point has been treated; in such cases, the point is described as an attachment trigger point. Furthermore, it has been suggested that if a chronic situation occurs where the primary and attachment trigger points remain untreated, "degenerative changes" within the joint may be precipitated and accelerated (Simons et al. 1998).

Diffuse Trigger Points

Trigger points can sometimes occur where multiple satellite trigger points exist secondary to multiple central trigger points. This is often the case when there is a severe postural deformity, such as a scoliosis, and an entire quadrant of the body is involved. In this scenario, the secondary points are said to be diffuse. These diffuse trigger points often develop along lines of altered stress and/or strain patterns.

Inactive (or Latent) Trigger Points

This applies to lumps and nodules that feel like trigger points. These can develop any where in the body and are often secondary. However, these trigger points are not painful, and do not elicit a referred pain pathway. The presence of inactive trigger points within muscles may lead to increased muscular stiffness. It has been suggested that these points are more common in those who live a sedentary lifestyle (Starlanyl & Copeland 2001). It is worth noting that these points may reactivate if the central or primary trigger point is (re)stimulated; reactivation may also occur following trauma and injury. Latent trigger points may have associated autonomic symptoms with pain, and their presence results in a limited range of motion, muscle fatigability, and muscle weakness as in the active[19, 20] presentation.

Active Trigger Points

This can apply to central and satellite trigger points. A variety of stimulants, such as forcing muscular activity through pain, can activate an inactive trigger point. This situation is common when activity is increased after a road traffic accident (RTA), where multiple and diffuse trigger points may have developed. The term denotes that the trigger point is both tender to palpation and elicits a referred pain pattern.

Ligamentous Trigger Points

There is some evidence that ligaments may develop trigger points[21] but the relationships are not clear. The sacrotuberous and sacrospinous ligaments can refer pain down to the heel, and the iliolumbar ligament can refer pain down to the groin and even into the testicles or vagina (Hacket 1958). Trigger points in the sacrotuberous ligaments can have profound effects on low back, lumbar Erector Spinae, and pelvic pain[22], and may also be associated with backache, neck pain, and even vocal dystonia[23]. As well as stabilizing structures, ligaments have strong proprioceptive functions (Varga et al. 2008). Working on ligament trigger points therapeutically can be clinically useful as part of the neuroplastic model (explored later). Trigger points may manifest in the Anterior Longitudinal Ligament (ALL) of the spine (e.g. after whiplash) which may result in neck instability[24]. The Fibular Collateral Ligament has a similar referred pain pattern to the Vastus Lateralis and trigger points in the ligamentum patellae are profoundly useful for treating knee pain syndromes.

NAT THEORY AND THE HIP

BEYOND THE TRIGGER POINT

Identifying and treating myofascial trigger points can be uniquely effective therapeutically; trigger points, however, rarely develop in isolation and may return if the underlying cause is not identified and addressed. Long-standing trigger points may lead to secondary (and even tertiary) changes in the nervous system (sensitization) and to trigger point formation elsewhere remote from the original problem. While trigger points may develop as a result of trauma, injury, or overuse, there may be other mechanisms at play.

The fact that trigger points are so ubiquitous in the population as a whole (from babies to the elderly) needs to be explored. So far, models have focused on the "where" and "how" but not the "why." Our mechanical systems are imbued with self-awareness, self-healing, and self-regulation, so what is the body trying to achieve and why? I believe it will help us to stand back and think about the why by exploring some other relevant models.

Protection

We are born with a number of protective mechanisms prewired into our nervous system. When we touch something hot, we quickly withdraw our hand; when we smell something unpleasant, we turn or move away. As a rule, the body reacts to noxious stimuli by "switching off" or pulling away from the stressor. Mechanical pain is relayed back to the brain via a number of mechanoreceptors; the brain then responds by initiating movements for maximal efficiency. Muscle groups are then arranged hierarchically into functional units of agonist, antagonist, fixator, and synergist.

When a structure is damaged or strained in the low back, "switch-off" mechanisms kick in to avoid further noxious stimuli. We are forced to recruit synergists, fixators, and agonists, often in a less efficient manner, to perform our daily tasks. This is fine in the short term, but over time it can lead to neuroplastic changes in the spinal cord and brain (sensitization). These mechanisms often include reflexes maintained locally in the spinal cord, and centrally in the brain.

Muscular conflict can be palpated around a region of pain as a result of these protective mechanisms. It is worth noting that, as humans, we often "push through" these barriers to carry on with our complex lives.

This "switching-off" mechanism is universal throughout the body. On a cellular level the "switch-off" phenomenon has been observed in a diverse range of diseases and conditions. In cancer, for example, some of the latest ideas center around the fields of the "immune-neural cortex" and "immune oncology." In these fields, the cancer cells have been observed to suppress or "switch off" our immune surveillance mechanisms by creating an immunosuppressive microenvironment around them: they fool our "immune checkpoints" and self-tolerance systems. Chronic viral infections, such as hepatitis, have a similar effect on the immune system. The latest HIV research, for example, suggests that the virus acts as a chronic noxious stimulus; this not only fools the immune surveillance mechanisms into "switching off" but over time, also makes T-cells both hyperactive and unresponsive (or silent) at the same time. The immune and nervous systems operate as a continuum. In the musculoskeletal system we are able to observe both the "switching-off" and the hyperactivation in the peripheral (spinal cord) and the somatosensory S1 and motor M1 cortices.

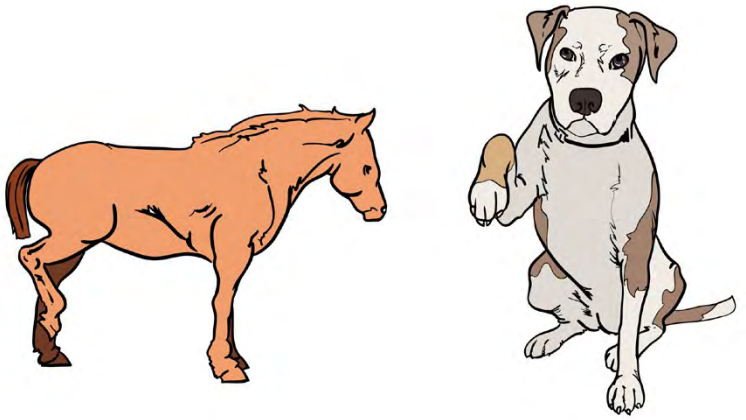
Holding patterns

A few years ago, we were stuck in an airplane for almost an hour, circling around Heathrow Airport, waiting for a "landing window." The Captain informed us that we were in a holding pattern and should be landing shortly.

We have thought a lot about this phrase ever since. For us, it neatly encapsulates the way we see a patient when they present in the therapeutic setting. Patients may come with acute or chronic symptoms, but, whatever the origin, the body's myofascial framework adapts and changes in a protective "holding pattern." Over time the "normal" muscle functioning fails, often resulting in multiple trigger point formation. The longer a problem persists, the more rigid these patterns may become. Chains of sarcomeres fail and chronic recalcitrant trigger points form. Peripheral and central sensitization play a role in maintaining this holding pattern, but so does the adapted myofascial infrastructure.

Hip holding

Janda (1989)[25] proposed that in response to pain or injury the hip and associated muscles stiffen up. This is similar to the decerebrate (default) posture seen in stroke patients, manifesting as tightness of the tonic flexor muscles and weakness of the phasic extensor muscles. The posture of someone with a severe hip issue mirrors that of a stroke victim, this suggests the pattern is a centrally mediated response to injury, i.e. a holding pattern. These patterns are recognizable and consistent from one patient to the next. This is an ancient pattern pre-wired into our nervous system; it can be seen in other animals too.



Pain Is a Big Stimulus

With regard to myofascial trigger points, the stressor is acute or chronic pain, either in a joint or in the myofascial matrix. In both cases, the body "switches off" around the stimulus; this switching is maintained both locally and centrally. The phenomenon is observable in muscles around a fracture site, a slipped disc, or, for example, a frozen shoulder. Painful stimuli are often mediated by inflammation and its noxious exudates, which is part of a well-demonstrated cascade. When our feedback mechanisms are altered, the brain is forced to adapt and compensate. Pain is a highly motivating symptom for the nervous system: it is our alarm bell that something is wrong.

Research into central sensitization has introduced the concept of polymodal receptors. Kawakita et al. (2002)[26] suggested that these "sensitized neural structures," may be proto-trigger points, or "trigger points in situ." In this scenario the brain switches on "trigger points on demand" where needed as part of the myofascial protective mechanism.

Trigger points on demand—TODs

Ever heard of "Video on Demand"? Because trigger points make the host muscles weak, they are a useful mechanism for rapidly switching off muscle power around an injury (sometimes called pain inhibition). This is essential if, for example, there is a fracture: it is an important part of our defense, protect, and repair mechanisms. The nervous system uses myofascial trigger points as part of its feedback vocabulary to accomplish this. This may also help to explain the local and rapid neurogenic responses in the muscles to acute injury or fracture.

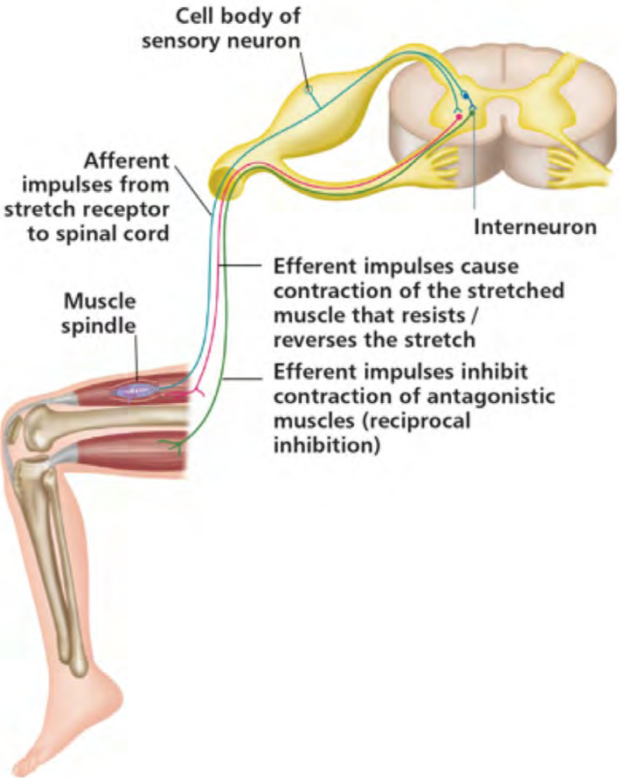
Reciprocal Inhibition

Reciprocal inhibition is an important reflex within our nervous system and has a major role in the control of voluntary movement. It describes the "automatic" process that occurs when muscles on one side of a joint relax to accommodate contraction on the other side of that joint. Joints are controlled primarily by opposing sets of muscles, extensors, and flexors, which must work in synchrony for smooth movement.

When a muscle spindle is stretched and the stretch reflex is activated, the opposing muscle group must be inhibited to prevent it from working against the resulting contraction of the homonymous muscle. This inhibition is accomplished by the actions of an inhibitory interneuron in the spinal cord.

The primary (Ia) afferent fiber of the muscle spindle bifurcates in the spinal cord. One branch innervates the alpha motor neuron that causes the homonymous muscle to contract, producing the reflex. The other branch innervates the inhibitory interneuron, which in turn innervates the alpha motor neuron that synapses onto the opposing muscle. Because the interneuron is inhibitory, it prevents the opposing alpha motor neuron from firing, thereby reducing the contraction of the opposing muscle. This is a part of our protective mechanism; without this reciprocal inhibition,

both groups of muscles might contract simultaneously and work against each other.



The implications for this are clear: not only do trigger points interfere with host muscle efficiency but they also have a reciprocal effect on antagonist muscles. This effect is increased with the chronicity of the condition and needs to be recognized and addressed during treatment. This reflex also offers the therapist the opportunity to treat acute myofascial trigger points via their antagonist.

PERIPHERAL AND CENTRAL SENSITIZATION

Pain is a complex area of medicine, and current research has thrown up a number of discoveries relevant to trigger point manifestation and perpetuation. Pain systems need to be sensitive enough to detect potentially harmful stimuli. But in the case of trigger points, these systems eventually become too sensitive, causing us pain with no benefit. Hypersensitivity arises because our pain pathways actually increase in sensitivity when they relay pain messages, and, with regard to myofascial trigger points, the mechanisms of this sensitization are now coming to light.

Peripheral Sensitization

Within 48 hours of developing, and if untreated, MTPs cause inflammation, chronic facilitation, and changes in feedback from the host muscle. Physiologically, there is a drop in the excitation threshold of polymodal nociceptors (discussed above) so that even normally innocuous, light stimuli activate them. After sensitization of "pain fibers," stimuli that as a rule are non-painful can cause pain (Schaible 2006[27]); in addition, mechano-insensitive nerve fibers can become mechano-sensitive. "This recruitment of silent nociceptors adds significantly to the nociceptive input to the spinal cord. Resting discharges may be induced or increased in nociceptors" (Schaible 2006). This occurs because of chronic active trigger points providing a continuous afferent barrage into the spinal cord.

The suspected mechanism is:

- Substance P, released from nociceptor terminals, carries nociceptive signals for central processing, and alters local microcirculation and vessel permeability, leading to local edema, activating both

mechanoreceptors and nociceptors, with subsequent increased tenderness and pain.

- Persistent activation with these pain-inducing (algogenic) substances leads to changes in nociceptor responsiveness both peripherally and centrally.

It has been shown that up to 50% of muscle nerves may be made up of nociceptors, and that nociceptors also innervate the connective tissue surrounding muscle. This could account for the severity of pain and exquisite tenderness found in muscles on palpation. Persistent activation of nociceptors leads to peripheral sensitization whereby primary afferent nociceptors exhibit an enhanced responsiveness to natural stimuli.

Central Sensitization (Spinal Hyperexcitability)

In the course of time the peripheral changes move deeper into the nervous system and the pattern becomes established centrally. The superficial, the deep, and the ventral spinal cord show pronounced changes in their response properties (Schaible 2006). This is a form of neuroplasticity: after sensitization, an increased percentage of neurons in a segment respond to stimulation of an inflamed tissue. The sensitivity of the spinal cord neurons becomes enhanced, so that an input that was previously subthreshold may now be sufficient to activate the neurons. This effect is magnified up and down the spinal cord over several segmental levels both caudally and cephalically, which may lead to lowered activation thresholds for other MTPs.

The implications of this are profound: it may well be that a chronic trigger point in one area may sensitize levels of the spinal cord above and below the input level. Over time, this may lead to a type of neuroplastic change in the CNS. This will decrease the pain threshold in other regions remote from the original source and possibly lower the threshold potential for other trigger points within the pain map. Central sensitization can persist

for weeks, months, and even years, depending on the chronicity of the stimulus.

The suspected mechanism is:

- Repetitive stimulation of primary afferent nociceptors leading to a progressive increase in action potential discharge—a phenomenon called wind-up, which may lead to a 20-fold increase in neuronal sensitivity.
- The result is an increase in intensity of pain and sensitization of neurones in the dorsal horn of the spinal cord because of the activation of N-methyl-D-aspartate (NMDA) receptors— central sensitization.
- Sensory neurones from the dorsal root ganglia become sensitized to mechanical stimuli, so that only mildly painful stimuli become more painful —mechanical hyperalgesia.
- Sustained nociceptive input from active trigger points may not only sensitize dorsal horn neurons, leading to hyperalgesia and allodynia, but also generate expanded referred pain regions.

Potential mechanisms for this phenomenon are the activation of previously redundant synapses at the dorsal horn, and the sprouting of new spinal terminals that broaden synaptic contacts at the dorsal horn, which may explain the referred pain seen with active trigger points.

Both peripheral and central sensitization can have serious unwanted effects. The advice, therefore, is to interfere with this process as soon as possible. The good news is that myofascial trigger point release (and dry needling techniques) has been reliably demonstrated to reduce these effects (Mense S, Ger win RD. Muscle Pain: Understanding the Mechanisms. Springer, Heidelberg, 2010).

Hip holding patterns, Movement Patterns and Muscular Imbalances

Movements of the Hip Joint involve a particular pattern of sequential muscle activation. The specific patterns of muscular movement that we will look at are hip extension, hip abduction, as well as hip rotational movements.

All of these patterns are made possible by the coordination of specific muscle groups. Some of the main muscles involved in these movements are the **Gluteus Maximus** and **Gluteus Medius**, the **Iliopsoas**, **Quadratus Lumborum**, **Erector Spinae**, **Piriformis**, **Tensor Fasciae Latae**, the **Biceps Femoris**, the **Adductors** of the hip, as well as the **Rectus Femoris**.

These muscles could also be grouped into a hierarchy of influence during a certain movement. There are thus the **primary movers** or the **agonists**, the **antagonists**, as well as the **fixators** and **synergists**.

A primary mover or an agonist will be vital in the initiation of a specific joint movement, and it will be aided by the synergist in this particular movement. On the other hand, the antagonist will act in an opposing manner to the agonist and as such it will lead to movement of the joint in the opposite direction.

Extension of the hip joint involves the contraction of three muscles acting in a coordinated sequence. The **Gluteus Maximus** is the primary mover in this movement while the **Biceps Femoris** and the **Erector Spinae** act as the synergists. A deviation in the activation of a specific muscle will be picked out as an 'altered movement pattern'. This will lead to hip extension dysfunction, muscle imbalance, or pain; over time, this will cause a change in posture of an individual.

The normal sequence of muscle contractions will be initiated with the contractions of the **Gluteus Maximus** as well as the **Biceps Femoris** of

the **ipsilateral** side. This will be promptly followed by the contractions of the **Erector Spinae** of the **contralateral** side and then by the contraction of the Erector Spinae muscle of the ipsilateral side.

1. The primary mover during hip extension is the **Gluteus Maximus**. During this movement the **Biceps Femoris** will assist in flexing the knee. The contractions of the contralateral and the ipsilateral **Erector Spinae** muscles are meant to stabilize the lumbar spine as well as the pelvis during hip extension. A change in this sequence of muscle contractions will be considered as an 'altered movement pattern.' The most commonly seen change is that of the contraction of the ipsilateral **Erector Spinae** before the **Gluteus Maximus** contracts in the initiation of the movement of the hip movement. You can best pick out this when the patient is lying in the prone position while performing the hip extension movement. This erroneously makes the **Erector Spinae** to be the primary mover of the hip joint in hip extension instead of the **Gluteus Maximus**. This weakness of the **Gluteus Maximus** makes it unable to act as the primary mover of the hip joint in hip extension.

Causes

Causes of an altered hip extension movement:

The **Gluteus Maximus** can be weakened or inhibited due to an injury to it, or the muscle may become deconditioned as a result of an injury or an illness which limits activities such as walking. It could also be caused by the overuse of the muscle occasioned by excessive repetitive motion or the over-activation of an antagonist muscle. The usual primary mover, the **Gluteus Maximus** will display signs such as a weakened contraction, a flattened shape, and trigger points will also be found in the muscle.

The **Iliopsoas**, which is a hip flexor and an antagonist muscle to the **Gluteus Maximus**, could also be the likely culprit in the case of an altered

hip extension movement. Tightness or hypertonicity of this muscle, which may be occasioned by prolonged sitting in a flexed position, could also lead to mechanical restriction of the motion of hip extension.

Having a tight Psoas muscle will restrict one's hip extension range of motion, the normal being about 20 degrees, and as a result one may end up with a decreased stride. Postural changes in the lumbar spine may also be occasioned by tightness of the Psoas and this will lead to a decrease in the lumbar lordosis. It is worth noting that the **Rectus Femoris** muscle is also antagonistic to the Gluteus Maximus, as it also assists in hip flexion.

As such, the **Rectus Femoris** and the **Iliopsoas** could both mechanically lead to inhibition of the full range of motion of the hip in extension. Inhibition of the **Gluteus Maximus** will lead to the recruitment of a synergistic muscle.

As such, the **Erector Spinae** muscle will initiate hip extension. The **Erector Spinae** will then assume the function of the primary mover in hip extension and this will lead to an altered movement pattern.

The **Erector Spinae** muscle will contract on the ipsilateral side of hip extension before the **Gluteus Maximus** does in the initiation of hip extension as well as the stabilization of the pelvis. With the **Erector Spinae** acting as the primary mover in hip extension, there will be increased strain on the lumbar spine.

Over time, the muscle fibres will become hypertonic and this will lead to:

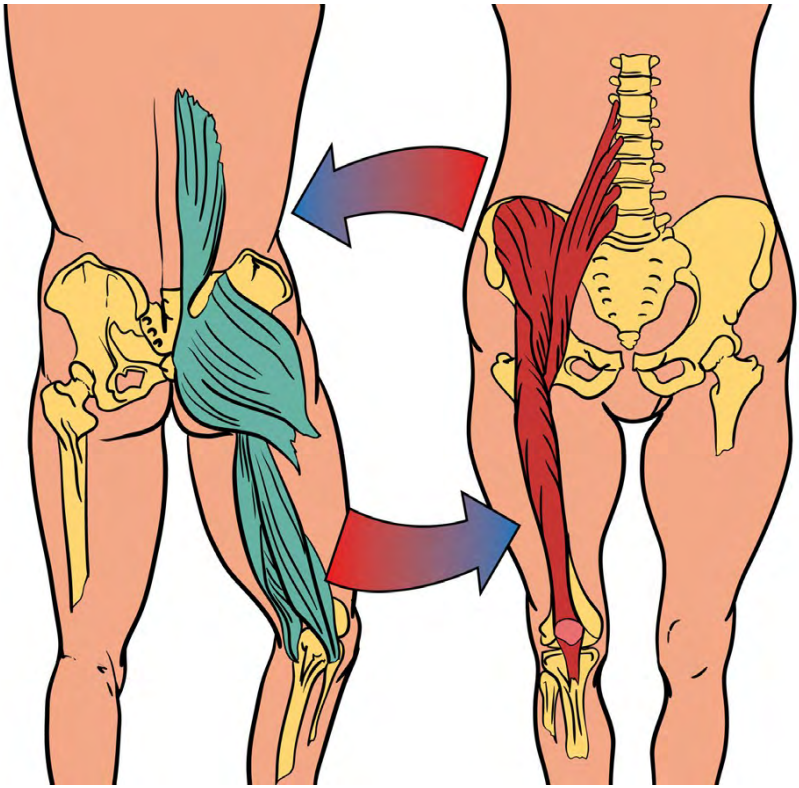
- An increase in the stress/load on the lumbar facets.
- Increasing pain in the lumbar spine while walking – this may also be accompanied by a decrease in the passive and active ranges of motion during extension of the hip.

• Tightness of the **Iliopsoas** and the **Rectus Femoris**

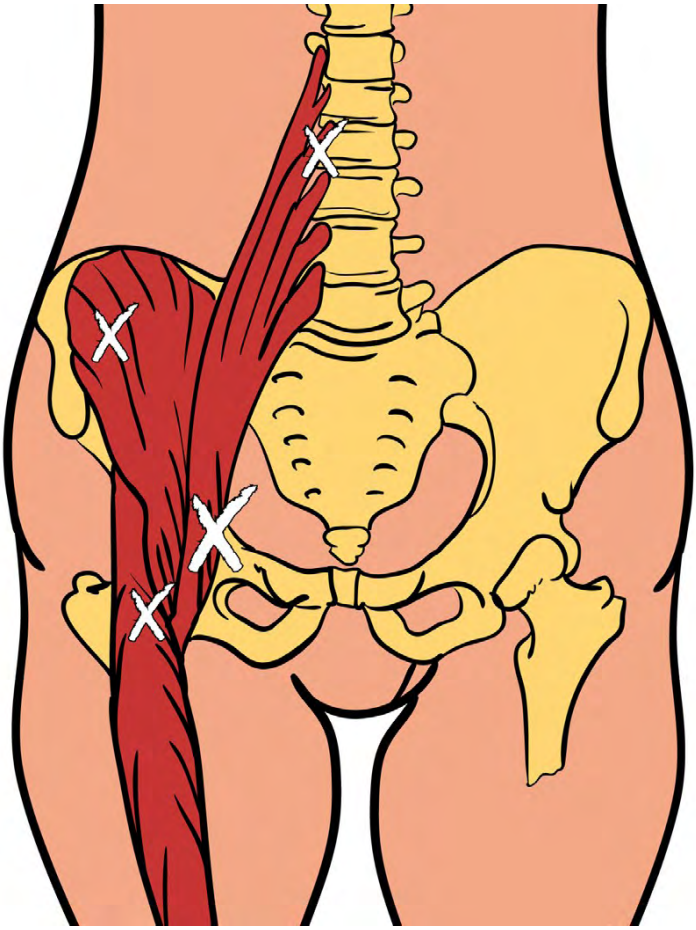
- Altered hip extension - either unilateral or bilateral.

  This causes changes in the mechanical functions of the **Gluteus Maximus** which lead to its neurological inhibition - normally occasioned by a change in the sequence of muscle activation.

This new neurological sequence for initiating hip extension will be stored in both the cerebellum and neocortex; it will also play a big role in inhibiting the activation of the primary mover, the **Gluteus Maximus**, in hip extension.



Reciprocal Inhibition: hip extensors vs flexors

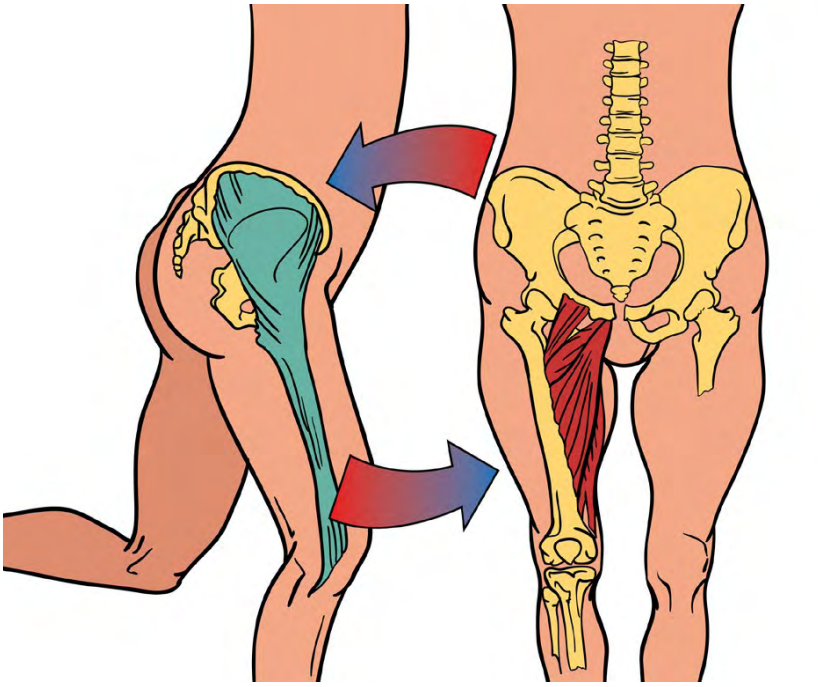


HIP ABDUCTION:

Altered movement pattern can also be manifested in hip abduction.

The primary mover in this case will be the **Gluteus Mediu**s, while the synergistic muscles will be the **Iliopsoas**, **Piriformis**, **Tensor Fasciae Latae**, **Quadratus Lumborum**, as well as the **Rectus Femoris**.

Normally in hip abduction, there will be contraction of the **Gluteus Medius** which will lead to a smooth lateral abduction of the lower extremity away from the body. Any change in the sequence of this movement is referred to as an altered hip abduction movement. These changes are normally occasioned by an inability of the **Gluteus Mediu**s to initiate this movement on its own. The **Gluteus Medius** can be inhibited by some conditions such as an injury or deconditioning of the muscle as a result of an illness or injury which limits the activity of walking. It could also be as a result of overuse of the muscle due to repetitive motion or overactivity of an antagonist. Most of these causes are quite similar to those causing hip extension dysfunction.



Reciprocal Inhibition: hip stabilisers vs adductors

Hip hiking, a sign of altered hip abduction; hip hiking refers to the raising of the pelvis on the side of the body during gait movement due to premature contraction of the **Quadratus Lumborum** (QL) on the side of hip abduction before the contraction of the **Gluteus Medius** on the ipsilateral side.

The sequence of hip abduction is initiated by the contraction of the **Quadratus Lumborum**.

The use of this muscle occurs in cases when the Gluteus Medius is either not strong enough or where it is inhibited in its bid to initiate hip abduction. You could check for hip hiking when a patient is walking, but you could also assess for hip hiking when the patient is lying on their side as they try to perform hip abduction. An alternative sign of altered hip abduction movement may occur if the hip drifts into flexion while one is abducting the hip. Hip hiking occurs due to overactivity of all or one these muscles; the **Iliopsoas**, **Tensor Fasciae Latae** and the **Rectus Femoris**.

The above named muscles will cause the hip to move into flexion in abduction and this could be due to their hypertonicity, mostly in case of weakness of the **Gluteus Medius**, the primary mover in this motion. Another sign of altered hip abduction is the external rotation of the lower extremities; this occurs in hip abduction due to an overactive Piriformis muscle.

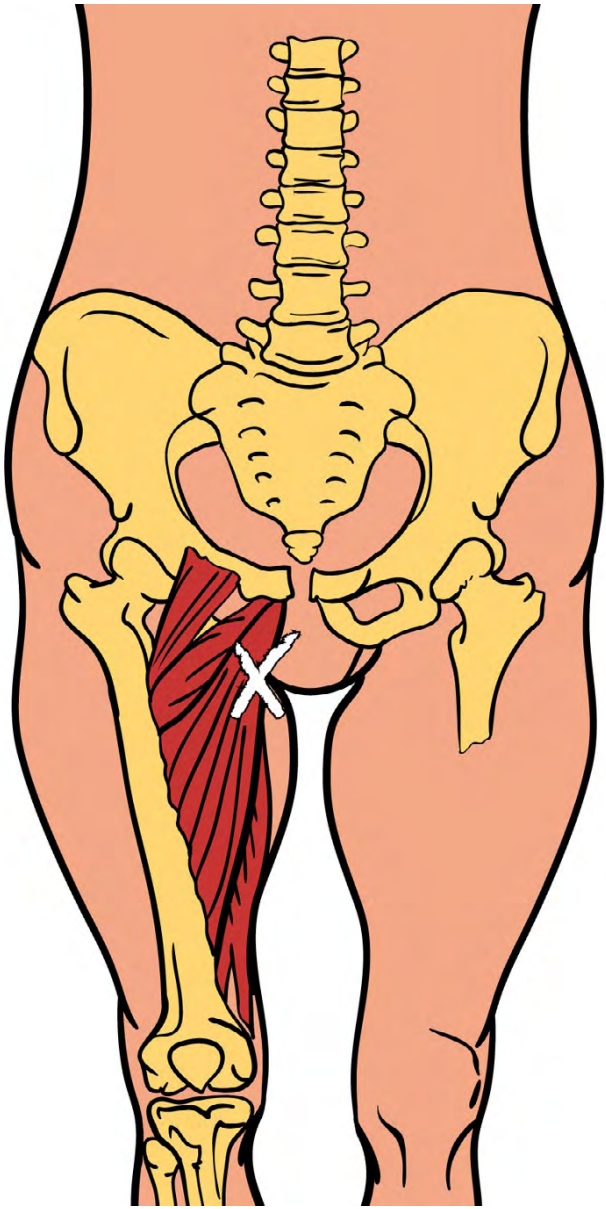
The last two dysfunctions of hip flexion and external rotation could also be assessed with the patient lying on their side while abducting the hip towards the ceiling. In case of **INHIBITATION** of the **Gluteus Medius**, one's gait may be diminished and this may be accompanied by a slight drop in the pelvis along on the swing-leg side.

I feel that tightness or hypertonicity of the adductor muscles leads to inhibition of the gluteus muscles. These adductors are antagonists to

the **Gluteus Medius** during abduction and they limit the normal range of motion of about 45 degrees in abduction. Inhibitation of the **Gluteus Medius** will also be noted by one exhibiting difficulty in standing on one foot. There will be an adaption of shifting the body weight to the stronger limb as the affected patient stands on both feet.

SUPER TRIGGER POINT (STP):

SITE OF TRIGGER POINT AT THE INFERIOR ATTACHMENT OF THE ADDUCTOR MAGNUS AT THE ISCHIAL TUBEROSITY



OTHER INDICATORS

Indications of inhibition of the **Gluteus Medius** could either be an inability to abduct the hip when lying on one side or inability to isometrically hold the hip in abduction for at least 30 seconds.

Another sign of hip muscular imbalances due to hypertonicity of the **Quadratus Lumborum** and the **Adductors** will be leg length deficiency on the ipsilateral side.

Hypertonicity of the **Tensor Fasciae Latae** could lead to lateral knee pain, which may create a lateral shift of the patella which is associated with a groove noted in the lateral thigh. **Sacroiliac joint dysfunction** because of **Gluteus Medius** inhibition will occur and trigger points in the Gluteus Medius will also be present.

Altered hip abduction normally occurs unilaterally but some cases could be bilateral. Just as alluded to earlier in hip extension, primary muscles involved in hip abduction can become inhibited both mechanically and neurologically with time. This is part of the altered movement pattern syndrome.

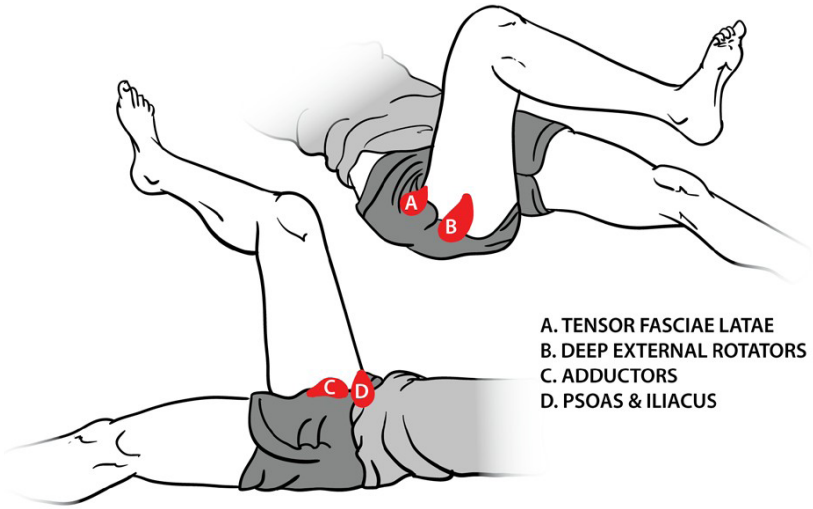
INTERNAL ROTATION

The final phase in the treatment protocol is the treatment to the internal rotation restrictions.

Taking the hip into passive internal rotation with the hip in 90 degrees of flexion and the knee in 90 degrees of flexion, the objective is to highlight where the restriction may be felt at the end of range.

The potential for restriction is highly individualized. As you can see in the illustration below, there are many sites of restrictions; locating these and then treating the sites of restrictions allows the final phase of restriction to be treated.

This process may take several treatments to several sites.



Sites of impingement during internal rotation of the hip

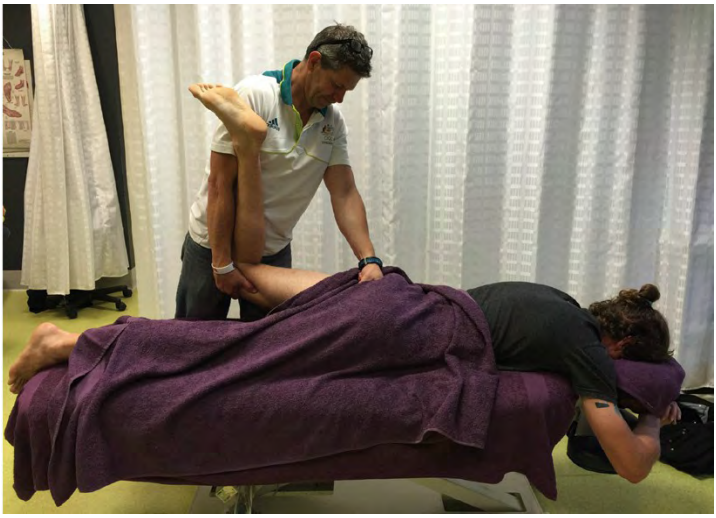
HIP ASSESSMENT:

HIP EX TENSION: STEP 1

- Place client in prone position.
- Stabilise the hip with hand over the superior margins of the Sacroiliac joint.
- With the other hand place underneath the anterior superior knee.
- Knee to be in 90 degrees of flexion.
- Slowly extend hip and assess for passive restriction.

Restriction sites:

- Anterior hip capsule
- Anterior thigh
- Thoracolumbar junction
- Sacroiliac/iliolumbar region

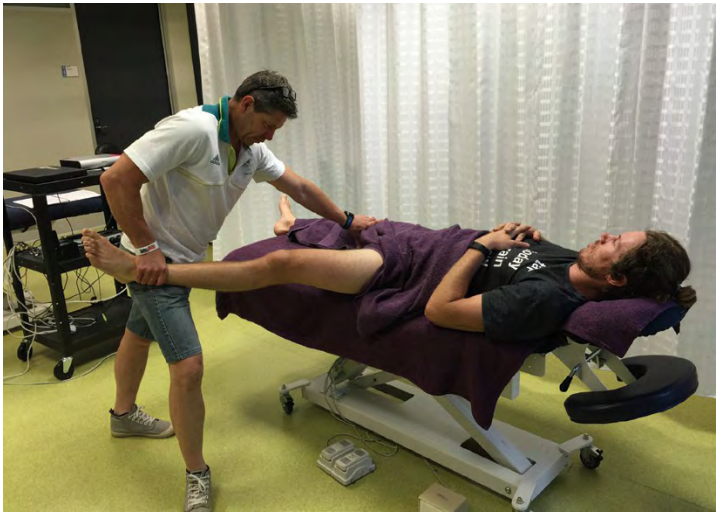


HIP ABDUCTION: STEP 2

- Place client in supine position.
- Stabilise opposite leg to be tested.
- Pick up test leg at ankle and walk out into abduction and feel for passive restriction.

Restriction sites:

- Short Adductors
- Long Adductors
- Lateral Hip



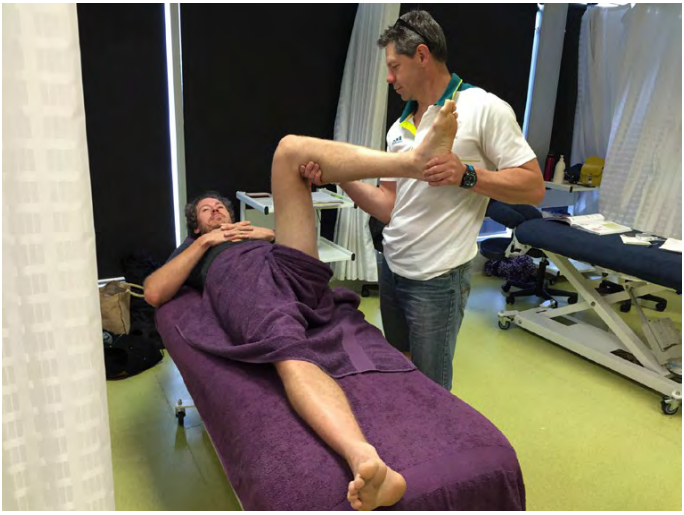
INTERNAL ROTATION:

STEP 3

- Place client in supine position.
- Hip in 90 degrees flexion.
- Knee in 90 degrees flexion.
- Hand placed at the popliteal surface.
- Opposing hand at the medial ankle surface.
- Take hip into internal rotation.
- Check for passive restriction.

RESTRICTIONS SITES:

- Tensor Fasciae Latae
- Iliopsoas trigger point attachment lesser trochanter
- Iliacus trigger point
- Adductor longus/magnus
- Deep external rotators



NAT HIP PROTOCOL:

The treatment protocol success hinges around applying three unique hands-on techniques one after the other.

1) **Trigger point therapy (TPT--NAT approach):** the key here is to identify the super trigger point that creates the greatest restriction. Apply a sustained continued ischemic compression until the discomfort/sensitivity melts away; this process is responsible for breaking the holding patterns that develop at the hip.
2) **Passive release technique (PRT):** is technique used to explore the new range of movement created by the TPT; we engage any lingering fascial restrictions using a sustained tension whilst taking the hip through passive range of movement.
3) **Active Isolated Stretching:**

The Active Isolated Stretching (AIS) method of muscle lengthening and fascial release is a type of athletic stretching technique that provides effective, dynamic, facilitated stretching of major muscle groups, but more importantly, AIS provides functional and physiological restoration of superficial and deep fascial planes.

Over the past few decades, many experts have advocated that stretching should last up to 60 seconds. For years, this prolonged static stretching technique was the gold standard. However, prolonged static stretching actually decreases the blood flow within the tissue, creating localized ischemia and lactic acid buildup. This can potentially cause irritation or injury of local muscular, tendinous, lymphatic, as well as neural tissues, similar to the effects and consequences of trauma and overuse syndromes.

Performing an Active Isolated Stretch of no longer than two seconds allows the target muscles to optimally lengthen without triggering the protective stretch reflex and subsequent reciprocal antagonistic muscle

contraction as the isolated muscle achieves a state of relaxation. These stretches provide maximum benefit and can be accomplished without opposing tension or resulting trauma.

STEP 1 - HIP EX TENSION:

1) ILIOPSOAS (Insertion) TRIGGER POINT

TO BREAK HIP EX TENSION RESTRICTION

This is the most prominent trigger point in the hip extension restriction.

Position:

Hip is placed in slight hip abduction and knee in 90 degrees flexion (leg off table).

Before we apply TPT, a gentle traction to the hip joint by pulling the thigh down in an inferior direction to open the anterior capsule region.

Locating the Trigger Point at the Iliopsoas:

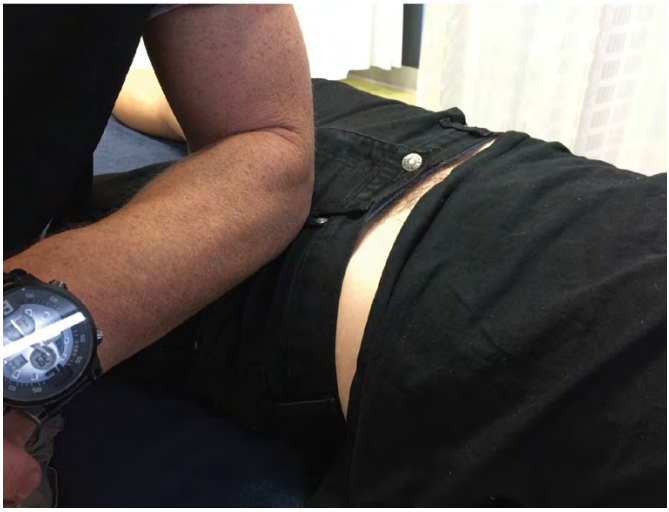
The first sequence is to find the inferior ridge of the greater trochanter. Run an imaginary line across the thigh to the medial thigh. Sink in deep to locate the superior ridge of the lesser trochanter.

Move 1-2 cm superior, and using a broad forearm, sink in and explore the region for its most sensitive point. Once you've located it, apply as much pressure as the client can accept - this sometimes means just resting there with no weight. Have faith and purpose and wait for the melting. I have at times rested up to 4 minutes in real chronic restrictions.

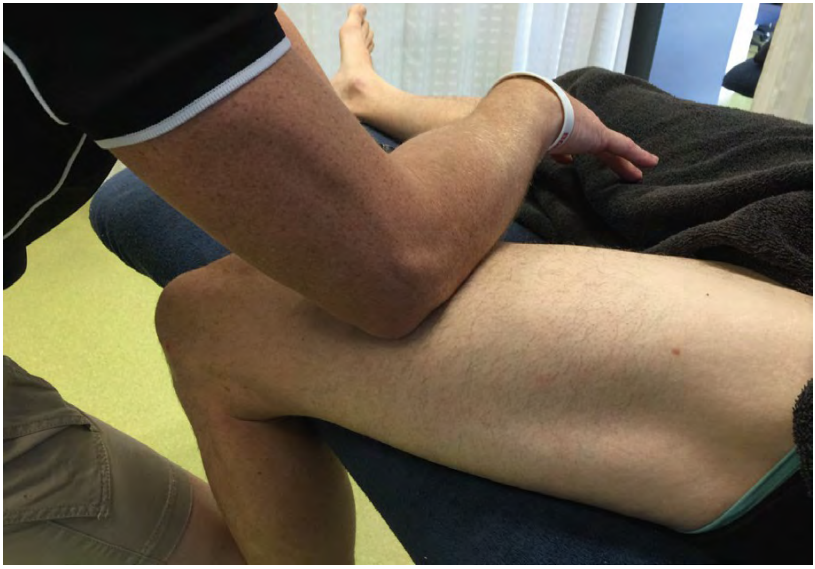
As this melts, explore more of the region either superiorly, inferiorly, medially, or laterally and treat accordingly.

When correctly executed, you should feel your elbow sinking into the hip site and the anterior hip almost feeling like it opens up.

ILIOPSOAS TRIGGER POINT



RECTUS FEMORIS TRIGGER POINT AND RELEASE:



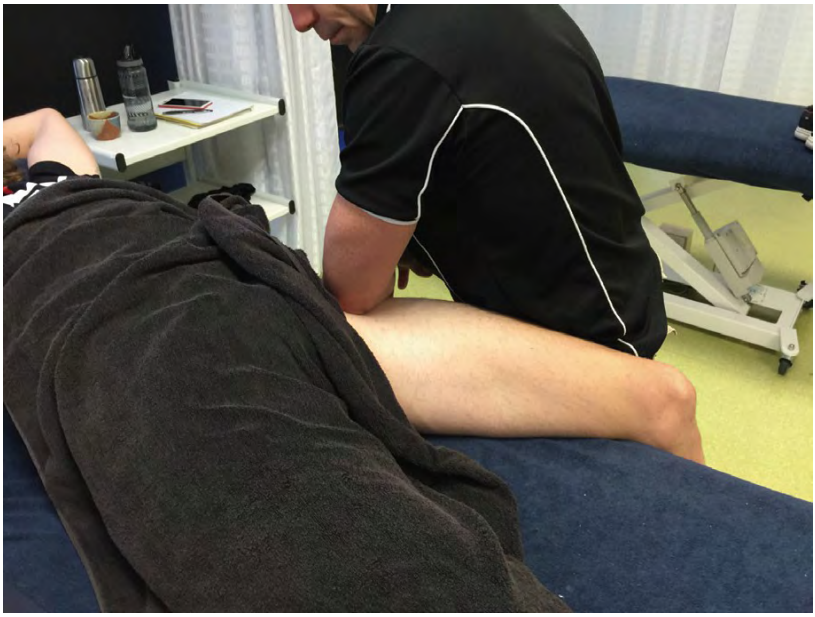
2) RECTUS FEMORIS TRIGGER POINT TECHNIQUE & PASSIVE RELEASE

The second sequence to the hip extension restriction is to locate the Rectus Femoris trigger point.

This trigger point is located just distal to the attachment on the AIIS; the exact same principles and technique procedure applies to this trigger point as described in treating the Iliopsoas trigger point.

The third sequence to breaking this anterior hip restriction is a passive release technique applied to the Rectus Femoris.

First, engage the tissue and work the Rectus Femoris along its length, starting from the superior patella and applying the technique towards the hip. Work the tissue using the analogy of squeezing toothpaste out of a toothpaste tube as you flex the knee, as this is a lengthening technique.

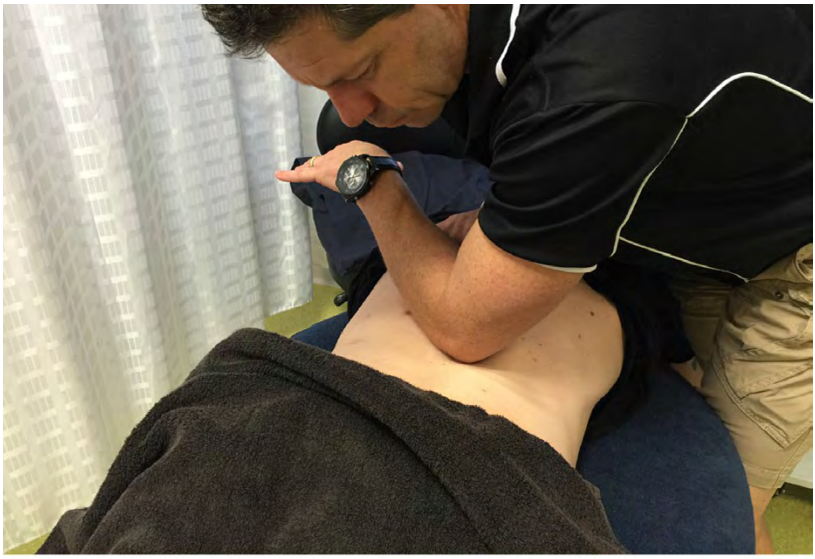


STEP 3 - Thoracolumbar Trigger points:

Be aware that every now and then you may find a client that presents with a restriction at the thoracolumbar junction when performing hip extension assessment. This may have a lot to do with the attachment of the Iliopsoas in this region.

To locate the thoracolumbar trigger points, locate the Erector Spinae muscles from the T10 to L2. Here lies a cluster of trigger points. It is imperative to work superior to inferior in this region. Take your time to treat each trigger point - these trigger points routinely don't take long to dissipate.

THORACOLUMBAR TRIGGER POINTS:



HIP ABDUCTION:

STEP 4

ADDUCTOR MAGNUS/LONGUS:

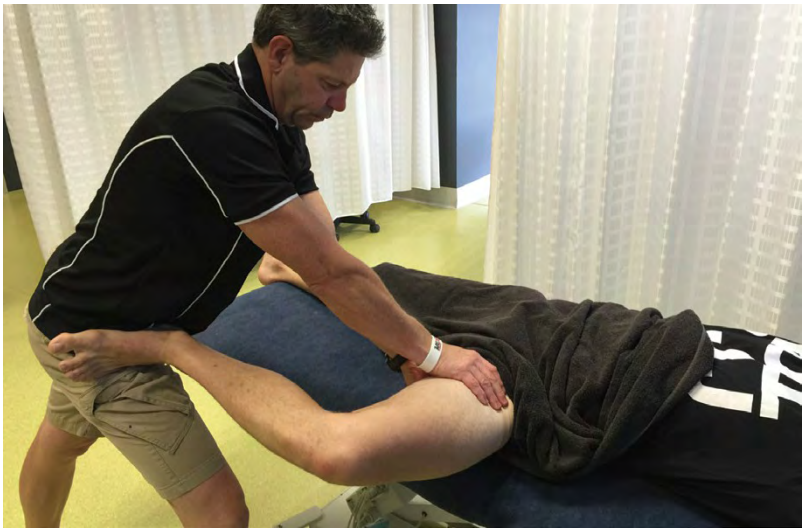
To treat the hip abduction restriction and break the inhibition of the Gluteus Medius, it is important to position the thigh so that you can challenge the range of movement of hip flexion and abduction.

Unlike the previous technique in the hip extension where you apply the ischemic compression to the trigger points, this part of the treatment is predominantly a passive release technique.

With the client's foot resting on your TFL, hip in flexion and adduction, support the knee with your hand. Make sure there is no pain and that it is comfortable - be sure to drape appropriately and consider the modesty of client.

Your tool of choice is a loose fist. 90% of the time you are applying this technique to the Adductor Magnus. Engage the tissue just inferior to the medial knee and slowly work the length of the muscle as you move the hip into flexion and abduction, concentrating on the proximal region of the muscle where the trigger points are located near the attachment. Challenge the tissue at this region, looking for increases in the flexion and abduction.

Usually 4-5 sweeps should suffice.



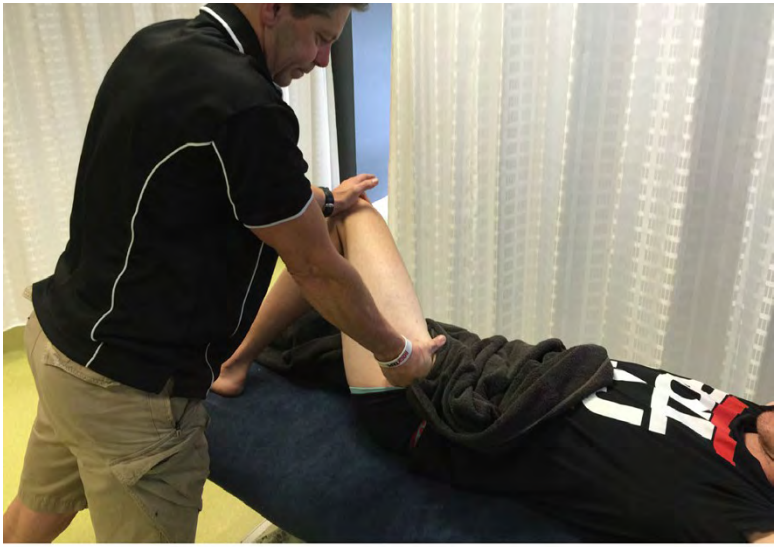
STEP 5 - INTERNAL ROTATION RELEASE

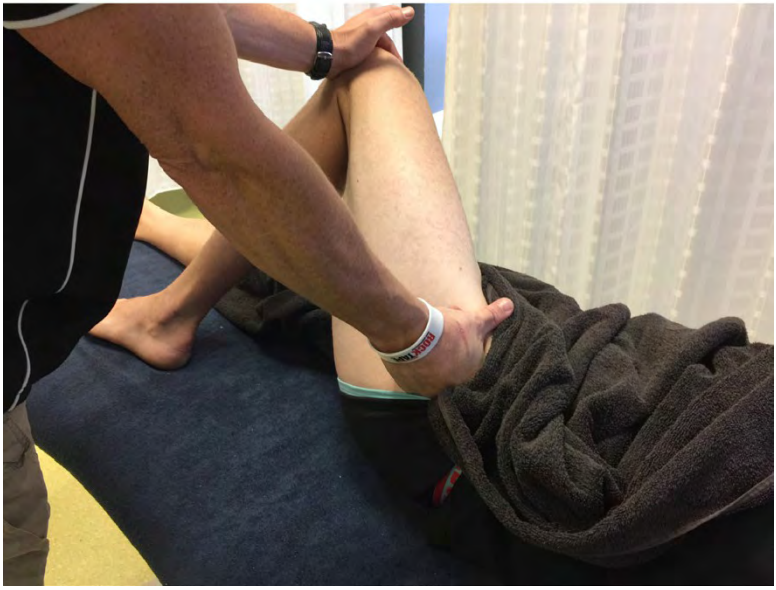
This is the last step in our hip sequence, and by this stage we should already see significant change in passive range of movement of the hip internal rotation from the previous steps. What we are looking for here is any remaining restrictions, which may or may not be present.

To assess if there are any last restrictors to the hip we need to take the hip through passive internal rotation. With hip and knee flexed at 90 degrees, identify any restrictions through feedback from your client who may feel tightness, soreness, and pain around the hip region. I have found that 90% of the time it is the Tensor Fasciae Latae. However it may also be at other sites such as Psoas, Iliacus, Deep External Rotators, or even the Adductors.

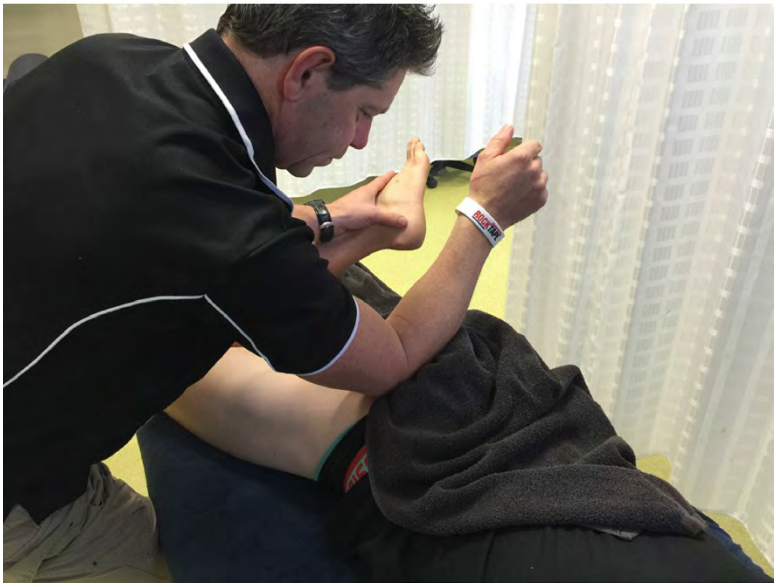
Apply the passive release technique, challenging the tissue and range of movement as described.

TENSOR FASCIAE LATAE RELEASE





EXTERNAL ROTATORS RELEASE:



CASE STUDY

- Had been referred by Physiotherapist.
- Had seen a Sports Doc; Xray/CT of hip: negative.
- Seen numerous Therapists over a 5-month period without change to symptoms.

HISTORY – SUBJECTIVE ASSESSMENT

Female recreational runner – 32 years.

- R hip pain (lateral in origin) then progressed to anterior hip as well, then a feeling of excessive thigh tightness and mild R lumbar spine pain.
- History of running over 15 years with 2 half marathons, 3 times a week running at 10 km.
- She had a break from running to take up CrossFit training.
- Over a period 3 months training CrossFit 3 times a week, she developed R hip/lateral knee pain of a mild nature.
- Decided to move back to running, wasn't long (3 weeks) until the lateral knee pain developed at 5km mark, pain increased until she could no longer run past 6 km.
- Pain at lateral/anterior hip region with increasing knee pain, and "giving away" sensations at hip and knee.

PHYSICAL ASSESSMENT- OBJECTIVE

- Pelvic assessment showed a marked Trendelenburg on the R.
- L upslip ilium and marked tenderness at the L5 spinous process.
- Restricted L lateral flexion lumbar Spine – R QL hypertonicity.
- Positive R prone bend, hip flexion for restricted ROM, confirmed with Mod. Thomas Test.

- L Side lying, hip abduction: 15-second hold reproduces lateral hip pain, weakness, and fatigue. Gluteus medius has trigger points on palpation.
- L Internal Rotation PROM, pain/restriction at pectineus/adductor longus attachments.

TREATMENT –

Referred to Gonstead Chiropractor, adjust S2.

Soft Tissue: - Clear Lumbar spine restrictions- Quadratus Lumborum, thoracolumbar trigger Points

- Hip extension restriction: target hip flexor attachment trigger point, Gluteus maximus trigger points, MTT to rectus femoris.
- Hip Abduction pain: Target gluteus medius/minimus trigger point, Adductor longus/pectineus Passive release technique.
- Hip internal rotation: Chase the restriction- treatment is governed and applied wherever client feels it.
- PRT- to adductor Magnus, TFL: Until free of tension, restoration of ROM.
- 4 treatments in total, running pain free in 5 weeks.
- Return to run plan: walk/run.

CONCLUSIONS

THE ART OF THE TECHNIQUE

I've often found when using a new technique that I can feel clumsy and that I'm not hitting the targets. Don't become frustrated if you feel the same; it's perfectly normally to feel this way. "REPETITION is the mother of all skill".

Take your time to seek and find those areas of tenderness in our 'key restriction sites'. Once an area of tenderness has subsided, make sure to move your pressure slightly around to pick up more areas of tenderness in the 'pain zone'. We are looking to press at that pain/pleasure boundary. Holding and maintaining the pain until it melts away starts the cascade effect we are looking to achieve.

Explore the pain zones and take your time; wait for the melt feeling. Pressing too hard, you may invite resistance; this is the opposite to the therapeutic change we are after.

Work within your clients' tolerance and general health and wellbeing. The first few times you apply this technique you should find a way to allow yourself to simply rest on the tissue as opposed to sinking in. Listen, feel, and slowly work with the tissues with intention to achieve that therapeutic melt.

When these techniques are performed correctly, your client should have a feeling of the joint being oiled from the inside out; there should be a dramatic decrease in any restrictions of the hip joint in both passive and active range of motion. It is at this stage that full range of movement is encouraged via Active Isolated Stretching in all ranges of movement; now the hip holding patterns have been removed and the body is ready for this last stage in the treatment sequence.

As each sequence builds on the last, we are creating a new neurological algorithm. Please don't rush the process — follow the algorithm with your full knowledge and skill; this is imperative to engender long-lasting change.

HOW MANY TREATMENTS

As long as there is no underlying capsular/joint pathology and the technique is applied correctly, 4-5 weekly treatments followed by another in 2 weeks, then another in 1 month. After this, the frequency will depend on the client. Clearly an athlete who performs high performance repetitive activity will need a different approach to a little old lady with an OA hip. You can certainly tailor-make a hip release program to add to your sporting clients, so you can individualize their maintenance.

It is not unusual that there will be secondary tissue issues that will arise in either in lumbar, thoracic, and lower limb regions; these may need addressing briefly as the holding patterns change at the hip. This treatment algorithm can also be used as a tool to reduce holding patterns that develop or occur around any ongoing hip joint pathologies including Osteoarthritis (if not severe). This technique, however, really comes into its own when working with the athletic hip, the high performance arena where this technique was born. It started with high performance and later developed as a comprehensive technique to use in range of hip conditions.

We all look for ward to hearing your feedback on using and developing this highly effective treatment into your box of skills.

STUART HINDS

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