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The Layer Concept: The Key to Rehabilitation of the Non-Arthritic Hip

Combined Sections Meeting
American Physical Therapy Association
Las Vegas, NV
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February 6, 2014

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Disclosure: I do not have a financial relationship with any commercial interest.

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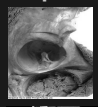
Layered Anatomical Approach to the Hip

Layer 1: Osteochondral Layer
Mechanics of joint

Layer 2: Inert Layer



Layer 3: Dynamic Layer

Layer 4:
Neuromechanical
Layer

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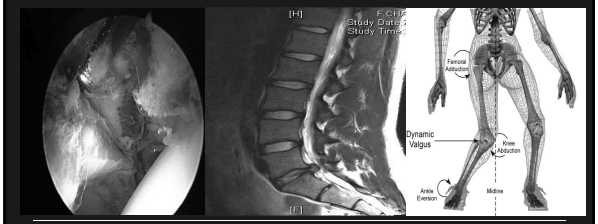
Hip Differential Diagnosis

- **Is the hip the SOURCE of the problem?**
- **Is the hip the SITE of the problem?**
- **Is the hip the SOLUTION of the problem?**



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Layer I: The Osteochondral Layer

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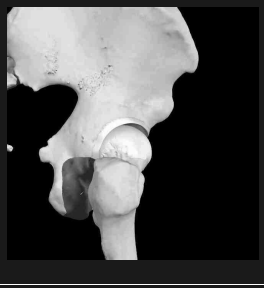
Pathoanatomy

Structures: Femur, Pelvis, Acetabulum
Purpose: Joint congruence and normal osteo / arthro kinematics

- ***Dynamic Impingement***
  - Cam Impingement
  - Rim Impingement
  - Femoral Retroversion
  - Femoral Varus
- ***Static Overload***
  - Acetabular Dysplasia
  - Femoral Anteversion
  - Femoral Valgus



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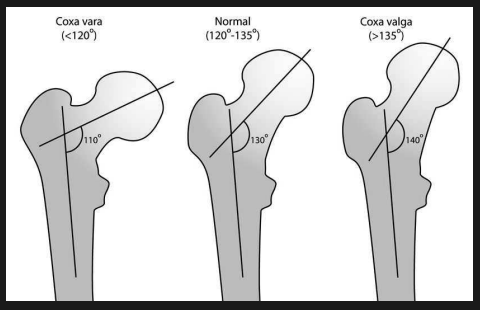
Angle of Inclination



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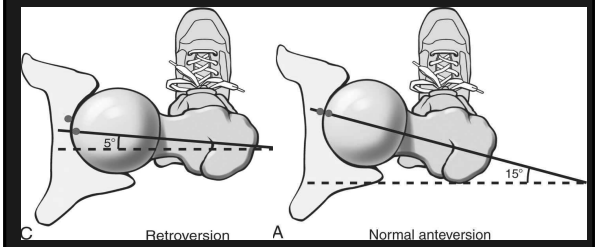
Femoral Torsion



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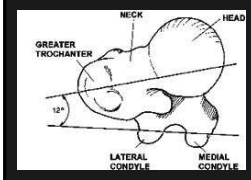
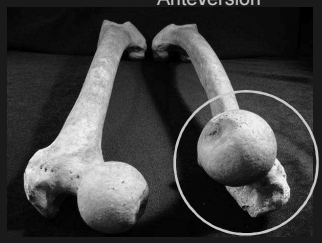
Femoral Version/Torsion

Anteversion





Retroversion < 8° > NORMAL < 15° > Anteversion
Craig's Test: Clinical measurement

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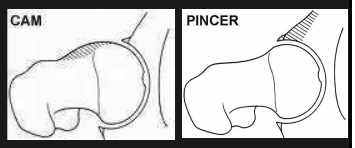
Femoroacetabular Impingement (FAI)

Cam Lesion
- Decreased femoral head-neck offset ($\alpha > 55°$) Neumann M et al, 2008
- Incomplete or late separation of growth plate
- SCFE
- Post-traumatic changes
- Femoral anteversion
- Coxa vara, extreme coxa valga Austin A et al, 2008

Pincer Lesion
- Acetabular retroversion
- Coxa profunda/protrusio
- Abnormal stress across anterior labrum → bony proliferation



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Acetabular and Femoral Retroversion

External Rotation

Internal Rotation

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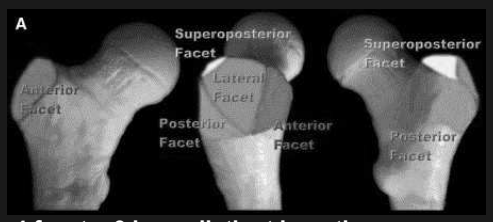
Dwek J. Pfirrmann C. Stanley A. Pathria M. Chung CB. **MR imaging of the hip abductors: normal anatomy and commonly encountered pathology at the greater trochanter.** Magnetic Resonance Imaging Clinics of North America. 13(4):691-704, vii, 2005 Nov



- **4 facets, 3 have distinct insertions**

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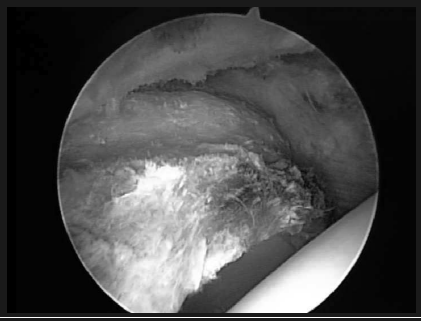
Lateral Rim



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Lateral Rim Impingement

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Should we avoid squatting with hip impingement?

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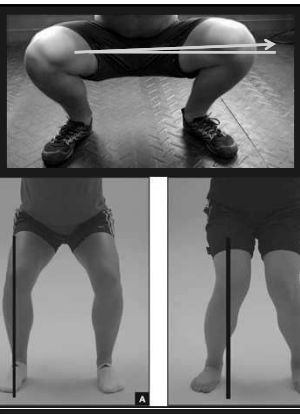

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Structural Factors- Osseous Over-Coverage

Restrictions to Motion

- Acetabular Retroversion
- Acetabular Protrusio/Profunda
- Coxa Vara
- CAM formation on femur
- Femoral Retroversion

↓

Structural Limitation into Hip Flexion and IR

- Toed out foot position
- Pain in groin
- Pain with full squat
- Pain with sitting

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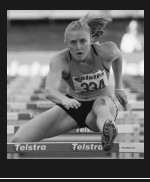
Overcoverage- Provocative Activities

- **Sitting**
- **Squatting**
- **IR, pivoting**
- **Running**
- **Pain is:**
  - Sharp
  - Catching
  - Achy



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Dysplasia (Osseous Under-coverage)

- Improper orientation of the acetabulum to the femoral head
- Decreased contact area
- 1.4 incidence / 1000 births
- Tonnis angle: < 10°
- Center Edge angle: < 25°

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Joint Stress

- Loss of lateral and anterior coverage of the femoral head by the acetabulum increases contact pressure and pressure on the anterolateral edge of the acetabulum
- The smaller the center edge angle → the abductor force and direction of force

Genda E et al., *J Biomech* 2001

- Labrum supports 1-2% of load across a normal hip; 4-11% of load across a dysplastic hip

Henak et al., *J Biomech* 2011

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Structural Factors- Osseous Under-Coverage and Joint Overload

Loss of Articular Stability

– Acetabular Anteversion
– Dysplasia
– Femoral Anteversion
– Coxa Valga
– Coxa Vara

↓

Structural Loss of Stability

– Pain at lateral hip
– Pain with walking or standing

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Undercoverage- Provocative Activities

- **Standing**
- **Walking**
- **Running**
- **Pain is:**
  - Aching
  - Throbbing
  - Heavy

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Layer II: The Inert Layer

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Inert Tissue

- **Capsule**
- **Ligamentum Teres**
- **Labrum**
- **Ligaments**
- **Bursae**

Purpose: Provides static stability to the joint

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Inert Tissue

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Labral Function

Finite element analyses, animal and in vitro models of labrum:

1. **Protects cartilage contact by limiting contacts pressure**
2. **Controls joint stability**
3. **Protects cartilage by Suction Seal effect**
   - **Labrum adds resistance to fluid flow path expressed from cartilage layers**

Ferguson. JOS 2001; J Biomech 2000, 2001, 2003

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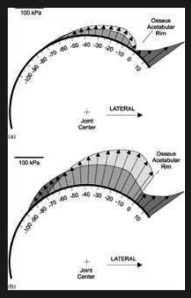
Biomechanics: Cartilage Compression

- Finite Element Analysis
- Contact stresses in acetabular cartilage increase by 92% w/ no labrum
- Cartilage layers deform 40% more w/ no labrum



Ferguson. J Biomech 2000

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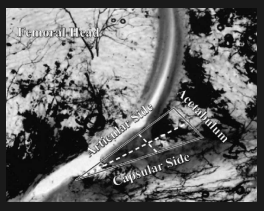
Healing Potential of Labrum

- Relatively avascular tissue
- Better vascularity shown in the capsular side of the labrum when compared to the articular side



Kelly et al. Arthroscopy 2005

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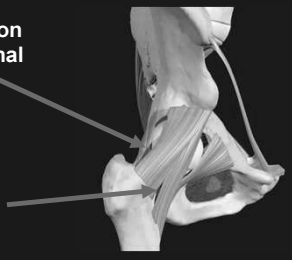
Iliofemoral ligament

- The lateral arm has dual control of external rotation in flexion and both internal and external rotation in extension.
- The medial arm is the greatest inhibitor to external rotation in extension



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Intracapsular Ligament

- Ligamentum Teres
  - Triangular-shaped band arising from the acetabular fossa and transverse acetabular ligament to the fovea and femoral head
  - Though this ligament conducts vessels to the head of the femur in most people; it has a minimal role in vascularity

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"Ligamentum Teres has an abundance of type III mechanoreceptors. When discharged they have a potent inhibitory effect on all rotators but facilitate the gluteal muscles."

…Dee, Annals of the Royal College of Surgeons of England, 1969

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Anterior Instability Test

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Anteverted Hip

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Superficial Back Line

- Plantar Aponeurosis
- Gastrocnemius
- Hamstring
- Sacrotuberous Ligament
- Sacral Fascia
- Iliocostalis
- Semispinalis Capitis and Cervicis
- Epicranial Fascia

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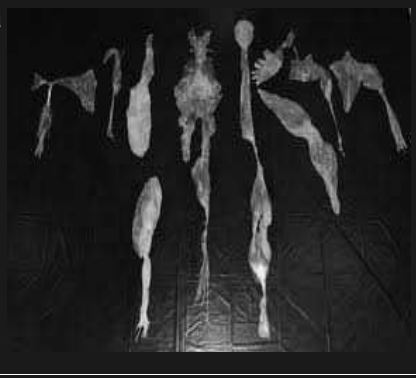
Fascia



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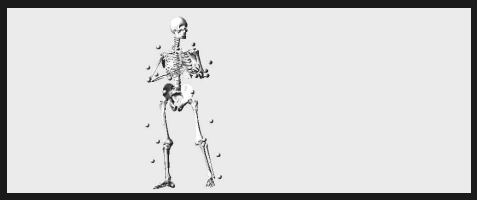
Kinematics and Kinetics of hip injuries in athletes



Hip loaded pelvis usually rotates over fixed femur
creating anterior and medial forces with rotary moments

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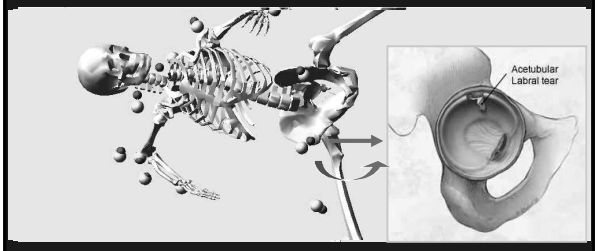
Tae Kwan Do



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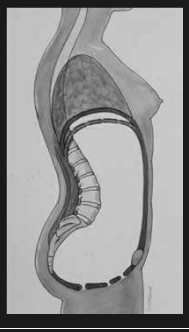
Layer III: The Contractile Layer

27 Muscles Crossing the Hip Joint

- **Muscles supporting and stabilizing the lumbo-pelvic complex**
  - Core "canister": Transversus abdominis, multifidi, pelvic floor, diaphragm
  - All other muscles with direct or fascial attachments to the pelvis



Primary Abductors

- Gluteus medius
- Gluteus minimus
- Tensor fascia lata

Secondary Abductors:
- Piriformis
- Sartorius

Primary Hip Flexors

- Iliopsoas
- Sartorius
- Tensor fascia lata
- Rectus femoris
- Adductor longus
- Pectineus

Secondary:
- Adductor brevis
- Gracilis
- Anterior fibers of gluteus minimus

Primary Hip Extensors

- Gluteus maximus
- Hamstrings
- Posterior head of Adductor magnus

Secondary:
- Posterior fibers of gluteus medius
- Anterior fibers of adductor magnus

Primary Hip Adductors

- Pectineus
- Adductor longus
- Gracilis
- Adductor brevis
- Adductor magnus

Secondary:
- Biceps femoris (long head)
- Gluteus maximus
- Quadratus femoris

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Primary Rotators

External Rotators:
- Gluteus maximus
- Short rotators
  - Piriformis, obturator internus, gemellus superior, gemellus inferior, qudratus femoris, obturator externus

Internal Rotators:

Anatomical Position: NONE

Secondary (torque increases closer to 90° HF)
- Anterior fibers gluteus minimus, medius
- Tensor fascia lata
- Adductor longus and brevis
- Pectineus

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The Iliocapsularis muscle: an important stabilizer in the dysplastic hip

Babst D

- Compared Dysplastic vs over-coverage hips
- Increase in width, circumference, cross-section and volume and with less fatty infiltration in the dysplastic hips

- Conclusion: Iliocapsularis muscle is a dynamic stabilizer of the femoral head in a deficient acetabulum.

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Hypotheses of Altered Neuromuscular Control

- **Global Stabilization System vs Local Stabilization System (Bergmark)**
  - larger muscles of the trunk vs local segmental muscles
- **Reflex Inhibition**
  - Caused by effusion, pain, ligamentous stretch, capsular compression
- **Central Excitation (Facilitated Segment)**

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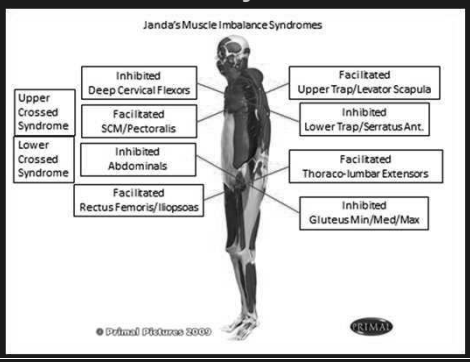
Janda's Crossed Syndrome



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Spinal and Pelvic Causes of Altered Hip Mechanics

Sacral Torsion

Posterior lateral disc protrusion

Segmental Instability

Congenital scoliosis

Thoracic Spine

Cascade of spinal segmental and neuromuscular changes lead to altered mechanics at the hip and pelvis

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Gluteal Timing

- Palpate over multifidi, gluteals and hamstrings
- Assess timing: (1) Multifidi (2) Gluteals (3) Hamstrings
- Dysfunction: Variations of hamstring firing prior to Multifidi and/or gluteal firing

RESULT of Improper Timing: Anterior translation / fulcruming of femoral head in anterior direction

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Psoas Function with Hip Pathology

Thought to be a dynamic anterior stabilizer of the hip in presence of changes in the hip anterior capsule, stress to anterior structures or mirco-instability

DO NOT CONFUSE OVER USE WITH "TIGHTNESS"

DO NOT STRETCH IF IT IS NOT SHORT

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Tightness vs. Tone

Tightness
- Adaptive shortening
- Resistance at the end of range

Tone
- Protective tone
- Myotomal
- Resistance through the range

- Must differentiate
- Muscles are fighting to stabilize
- Over stretching feeds into the vicious cycle
- Re-educate instead

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Layer IV: Slings, Rings and Other Things
The Effect of the Kinematic Chain

Neuromechanical Layer

- **Kinematic Chain**
  - Other levels of functional rotation
    - Upper cervical spine
    - Thoracic spine
    - Subtalar joint
- **Neurological Components**
  - Efferents
  - Afferents
  - Proprioceptors
- **Vascular Components**

Questions to be Answered

- **When is the pinch not osseous impingement?**
- **What factors play a role in dynamic impingement and hip pain?**
- **When is hip pain secondary to other factors in the kinematic chain?**
- **What is creating anterior groin pain in the dysplastic hip?**

Hip Joint Translation

Model-Based Tracking of the Hip: Implications of Novel Based Analyses of Hip Pathology

- Martin DE, et al. *J Arthrop 26(1); 2011*
- **Model-based tracking vs. Metal implant bead based tracking**
- **Walking and Chair Ascent**
- **Translation average: 3mm**
- **Rotation average: 8°**

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STRUCTURE AND FUNCTION OF HIP JOINT INNERVATION

Arnott Demonstration delivered at the Royal College of Surgeons of England
on
4th February 1969
by

Roger Dee, M.A., F.R.C.S.
Neurological Laboratory, Royal College of Surgeons of England, and
Department of Orthopaedic Surgery, Middlesex Hospital, London

- **"No nerve endings of any description are present in the synovial tissue of the hip, as is the case with all other synovial joints" (Wyke, 1967)**

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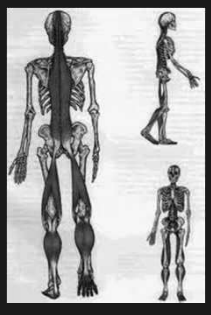
The Fascial Connection



Anatomy Trains – Tom Myers

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Core Stability Training Principles

- **Inner Unit –** ***Hodges et al.***
  - Pelvic Floor
  - Multifidus
- **Outer Unit -** ***Vleeming***
  - **Anterior Oblique** – Oblique Abdominal/Contralateral Adductor
  - **Posterior Oblique** – Gluteus Maximus/Contralateral Latissimus/Thoracodorsal Fascia
  - **Deep Longitudinal** – Hamstrings/Erector Spinae/Sacrotuberous Ligament
  - **Lateral System** – Gluteus Medius-Minimus/Ipsilateral Adductor

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Integrated Systems Model

Lee and Lee, 2007, 2011

- **View the body system as a series of rings**
- **Identifying the "Primary Driver" for a dysfunction in a "meaningful task".**
  - Cervical Spine
  - Thoracic Rings (LJ Lee)
  - Pelvic Ring/SI Joint
  - Hip
  - Foot/Ankle

- **Dysfunctions which lead to "Failed Load Transfer" of a "meaningful task"**

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One Leg Standing - Hip

Lee & Lee, 2004

- **Unilateral Stance**
  - Femoral head should remain centered
  - No translation or rotation
- **Improper load transfer and muscle imbalance indicated by anterior translation**

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Squat Test

- Palpate proximal femur during squat motion
- Should feel femoral head settle back into acetabulum during squat
- Imbalance: anterior translation
- Assess ability to load equally through bilateral LE's

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Poor Dynamic Stability of Lumbo-pelvic girdle in a Squat Task

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Treatment Algorithm and Approach

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Etiology of Injury

Traumatic **Atraumatic**

Intrinsic Factors

Extrinsic Factors: training errors, surface, shoes, equipment, inadequate nutrition

Structural:
- Bony
- Inert Tissue
- Contractile Tissue

Neuromuscular: Timing, control, fatigue, weakness, hypertonicity

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CAN IT BE THIS BASIC?

- **Can Over-coverage be considered a kinematic problem and Under-coverage be considered a kinetic problem?**
- **Static Diagnostic Tests should never be used solely as an outcome predictor for a truly dynamic problem, especially with undercoverage**
- **Can an FAI problem be resolved with an AFC solution?**

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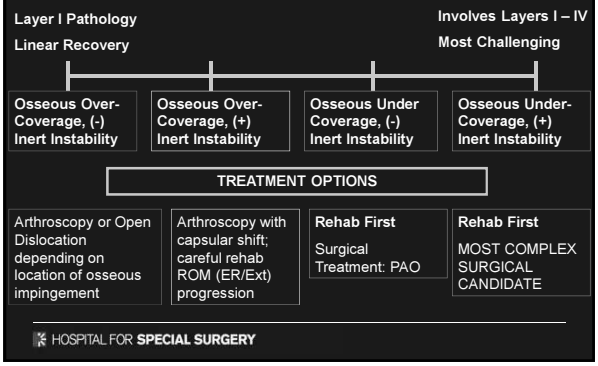
Acute Soft Tissue Issues

Hip Pathology Treatment Algorithm



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Differential Diagnosis of Motion Limiting Structures

- Bony Structure (Layer I) vs. Inert or Soft Tissue (Layer II/III)
- Capsular (Layer II) vs. Soft Tissue (Layer III)
- Soft Tissue: Tightness vs. Tone

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The Dysplastic versus the Overcoverage Hip

Dysplastic	Overcoverage
• Less osseous stability	• More osseous stability
• Dynamic overload: lateral / abductor fatigue and pain	• Impingement pain: anterior groin or posterior gluteals
• Relies more on inert tissue and neuromuscular control for stability	

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Treatment Tips

- **Dysplastic Hips: Primary Driver is often somewhere in kinematic chain (thoracic, lumbar, foot/ankle)**
- **Stabilize the base**
  - TA, pelvic floor, multifidus firing
- **Co-contraction and stabilization of lumbo-pelvic girdle/hip muscles**
- **Supine → Prone → Quadruped → Bilateral Stance → Unilateral Stance**
- **Proprioception is key**

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Joint Compression Supine

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Joint Compression Seated

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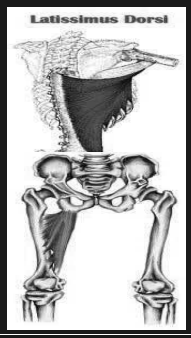
Relationships



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Imbalance Paradigms

- Biomechanical – Repeated or sustained postures can lead to changes in muscle length, strength and stiffness leading to movement impairments
- Global vs Local – Classification of back muscles where global is superficial/FT and tend to shorten and local deep stabilizers prone to weakness
- Neurological – Lack of movement or repetitive movement disorders. Imbalance because of role in motor dysfunction. Neural control unit may alter muscle recruitment strategy to stabilize joints
- HARDWARE OR SOFTWARE PROBLEM

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Potential Breakdown Regions

- Robb et al – Baseball Overhead Athlete
- Ellera Gomes et al – Soccer Non-contact ACL
- Vad et al – Golf LBP
- Bedi et al – Football Spondy/Met Fx/ACL

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CONSIDERATIONS

- THINK PROXIMAL
- THINK CORE
- THINK STRUCTURE
- THINK LINK

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TAKE HOME CLINICAL REHAB PEARLS

- If you load it, check it
- Don't forget about a functional muscular adjustment period following FAI surgery
- Know whether you are dealing with over coverage, under coverage, a neuromuscular issue or a structurally intact hip
- Is it protective tone or true muscle tightness
- Is the hip the source, site or solution
- Dx from inside out & treat from outside in
- Transition and Threshold are critical inflammatory elements
- Form and fatigue dictate exercise volume and intensity
- Neuromuscular control of the pelvis is essential
- Avoid the PERFECT STORM
- HIP REHAB IS NOT LINEAR

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PERFECT STORM

- Bad Bone – Layer 1
- Inert Insufficiency – Layer 2
- Neuromuscular Amnesia – Layer 3
- Kinetic Collapse – Layer 4
- Cognitive Uncertainty- Layer 5

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REHABILITATION

Thank You

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Curr Rev Musculoskelet Med (2012) 5:1–8
DOI 10.1007/s12178-011-9105-8

The layer concept: utilization in determining the pain generators, pathology and how structure determines treatment

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Published online: 28 February 2012
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Abstract The level of understanding of pain in the non-arthritic hip has made significant strides in the last couple of decades beginning with the discoveries of Reinhold Ganz, MD. However, even with the detection of subtle bony abnormalities, including femoroacetabular impingement, a clinician's ability to differentiate pain generators in the hip has been ambiguous. Deciphering the etiology of the pathology versus the pain generator is essential in prescribing the proper treatment. The Layer Concept developed by Dr. Bryan Kelly, is a systematic means of determining which structures about the hip are the source of the pathology, which are the pain generators and how to then best implement treatment. Four layers will be discussed in this article. Layer I, the osseous layer, Layer II, the inert tissue layer, Layer III, the contractile layer and Layer IV, the neuromechanical layer.

Keywords Femoroacetabular impingement · Hip arthroscopy · Bony pathology · Capsular laxity · Neuromuscular control

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Introduction

The history of hip impingement can be traced as far back as 1920 when Sir Robert Jones reported at the British Orthopedic Association meeting on hip osteoarthritis (OA) that he had relieved the pain of a house painter by performing a cheilectomy on the head of his femur [1]. In 1936 Smith-Petersen reported performing an acetabuloplasty for a case involving a slipped upper femoral epiphysis [2] and in 1949 Heyman reported performing the same procedure for the same pathology in 42 cases [3]. In 1965 Murray [4] reported the anterior-posterior (AP) pelvis "Tilt Deformity" describing the same findings of Stulberg and Harris in 1974 [5], which they coined the term "Pistol Grip" from their interpretation on an AP pelvis x-ray. For more than 50 years, observations have been made suggesting structural deformity and its relationship to early onset of OA in the hip joint. Although this conclusion was mainly drawn from childhood hip pathologies ranging from slipped capital femoral epiphysis (SCFE) and Legg Calves Perthes (LCP) to undercoverage issues such as degenerative disease of the hip (DDH), it was becoming ever so clear that an irregular shaped ball housed in an irregular shaped socket would create irregular mechanical forces across the joint. Early theories on mechanical malalignment as part of the etiology of hip OA in the 1970's [6] have certainly been substantiated with the recent work of Ganz et al. [7•, 8]. Ganz reported that subtle, often unrecognized bony deformities and motion of the joint can cause acetabular rim damage by femoroacetabular impingement (FAI) [8]. This work provides clinicians and researchers the opportunity to finally distinguish the etiological differences existing between those who develop early hip OA from structural overcoverage and those who develop hip OA as a result of structural undercoverage. Recognizing and attempting to understand these osseous,

inert, contractile, and neuromechanical relationships and differences, as they relate to normal osseous structure, osseous overcoverage and osseous undercoverage, is what led to the development of the Layer System. (Table 1)

Diagnostic testing for identifying osseous, inert and soft tissue hip pathology has included x-ray, magnetic resonance imaging (MRI), computed tomography (CT) Scan, delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) studies, diagnostic injection and clinical special tests[9, 10]. Computer navigation surgical planning software, such as A2, can be used to confirm and model osseous impingements. X-ray views of AP lateral and Dunn view can be used along with 3 dimensional CT scans to identify osseous hip pathologies [11]. These studies provide structural blueprints for determining alpha angles, beta angles and McKibbon indices [12]. dGEMRIC studies can be used, when indicated, to determine the health of the cartilage[9]. Intra-articular injections have proven extremely reliable for differentiating between intra and extra articular hip pathology [13]. MRI has been diagnostically sensitive to Layer II inert tissue (labrum, capsule, ligament and ligamentum teres) pathology, as well as Layer III contractile tissue direct involvement and indirect enthesiopathies.

During the diagnostic process it may be helpful to categorize the hip as structurally normal, structurally overcovered or structurally undercovered. A structurally normal hip will have values that fall within a normal range for center

Table 1 The layer concept

Layer	Name	Structure	Purpose	Pathology	
I	Osteochondral	Femur	Joint congruence	Developmental	Dynamic
		Acetabulum	Arthrokinematic movement	Dysplasia	Cam Impingement
		Innominate		Femoral Version	Rim Impingement
				Acetabular Version	Trochanteric Impingement
					Delamination
				Femoral Inclination	Sub-spine impingement
				Acetabular Profunda/Protrusio	
II	Inert	Capsule	Static Stability	Labral Tear	
		Labrum		Capsular Instability	
		Ligamentous Complex		Ligamentum teres tear	
		Ligamentum Teres		Adhesive capsulitis	
III	Contractile	Musculature crossing hip	Dynamic Stability	Hemi-pelvic Pubalgia:	
		Lumbosacral muscles		Anterior Enthesiopathy	
		Pelvic floor		Hip flexor strain	
				Psoas impingement	
				Rectus femoris impingement	
				Medial Enthesiopathy	
				Adductor tendinopathy	
				Rectus abdominus tendinopathy	
				Posterior Enthesiopathy	
				Proximal hamstring strain	
				Lateral Enthesiopathy	
				Peri-trochanteric space	
				Gluteus medius tear	
III	Neuromechanical	Thoroco-lumbar mechanics	Communication, timing and sequencing of the kinematic chain	Neural	Mechanical
		Lower extremity mechanics		Nerve entrapment	Foot structure and mechanics
		Neuro-vascular structures referring to and regional to the hip		Referred Spinal Pathology	Scoliosis
		Regional mechanoreceptors		Neuromuscular Dysfunction	Pelvic posture over femur
				Pain syndromes	Osteitis Pubis
					Pubic symphasis pathology
					SI dysfunction

edge angle, hip valgus, and hip version values [14]. A structurally undercovered hip will diagnostically present with anteversion, hip valgus or dysplastic characteristics [14]. Comparatively, overcoverage will diagnostically present as cam lesion at head neck junction, rim lesion, often associated with acetabular retroversion, acetabular profunda or acetabular protrusio [14].

Many activities take place when the feet are on the ground. When this occurs the pelvis moves over a fixed femur as opposed to the femur moving under the pelvis. Therefore, it fair to imply that dynamic impingement may occur under these conditions, when there is a lack of neuromuscular pelvic control. This type of impingement occurring as the pelvis moves over a fixed femur may be referred to as acetabular-femoral impingement. Instruct a person who experiences anterior hip pain to perform a single leg squat, both with and without trunk stability cueing, and see how their lower quarter responds.

Both diagnostic testing and clinical special tests have identified that impingement can occur in more locations than the literature recognizes. It has been confirmed via CT Scan with dynamic simulation software. (Table 2)

Layer I: osseous layer

Layer I consists of the femur, pelvis and acetabulum. The purpose of this layer is to offer joint congruence and structurally guide normal osteo and arthro kinematics. It is in this layer that structural pathologies exist and can be classified as either developmental or dynamic. Developmental pathologies include dysplasia, femoral version, acetabular version, femoral inclination and acetabular profunda/protrusio. Dynamic related pathologies include cam/pincer impingement, trochanteric impingement, sub-spine impingement and delamination. (Table 1) Loss of femoral head sphericity and joint congruity, due to cam impingement can lead to edge loading [15], labral tears or delamination of acetabulum at contact site of the articular cartilage [14]. The hyaline cartilage is divided into 3 layers and resembles cartilage from other joints within the body. The main difference is the varied thickness on both the acetabulum and the femoral head. The most superficial layer comprises 10–20% of the total cartilage thickness, the middle layer comprises 40–60% of the articular volume whereas the deepest layer is 30% of the overall thickness [16]. The work of Ferguson et al. [17] demonstrated the relationship and interaction which exist between Layers I and II. Forces to distract the head of the femur by 3 mm after venting the capsule and creating a labral tear decreased by 43% and 60% respectively. Loss of maintaining a suction seal would decrease intra-articular hydrostatic pressure and potential nutritional loss to the joint.

Layer II: inert layer

Layer II consists of the labrum, capsule, ligamentous complex and ligamentum teres. The primary purpose of this layer is to provide static stability to the joint. The most commonly affected structure at this layer is the labrum.

Table 2 Special test for the layer system

Layer I	Layer 2	Layer 3	Layer IV
Anterior Superior Acetabular Impingement- Flexion Adduction Internal Rotation (FADIR)	Anterior Instability Test- extension and external rotation over the end of the table	Thomas Test	Gait Observation- loss of hip extension, hip drop, decreased stride length, foot mechanics
Sub-Spine Impingement- Pure Flexion	Log Roll	Manual Muscle Testing	Functional Squat- loss of hip flexion
Superior-Lateral Acetabular Impingement- Flexion, abduction, External Rotation (FABER)	Distraction Test	Supine to Prone ROM comparison	Single Leg Stance- monitor hip drop or palpation of femoral head to translate or spin with weight bearing
Lateral Rim Impingement- Pure Abduction	Ligamentum Teres	Supine Hamstring ROM	Single Leg Squat- hip drop, genu valgum
Ischiofemoral Impingement- External Rotation and Extension	Straight Leg Raise Test		Forward Step Up- hip drop, genu valgum
Posterior Impingement- Side-lying Extension	Quadruped Posterior Rock		Forward Step Down- hip drop, genu valgum
Trochanter Sub-spine Impingement- 30 deg flex, 30 abd, IR (ant facet of gr troch impinge with sub-spine)			Foot Mechanics- Full kinematic chain rotation over the foot; assess supination and pronation
Craig's Test			Spine Screening Exam
			Gluteal timing- prone hip extension; monitor ability of gluteals to fire prior to hamstrings

However, at this layer it is not uncommon to experience labral insult, ligamentum teres tear, capsular instability, ligament tears and adhesive capsulitis. The loss of labral suction seal has been shown to have increased femoral head displacement in cadaveric hips [17–19]. Translation of hip joint center may be as much as 2–5 mm for that loose hip further stressing inert and contractile tissue [20, 21]. Akiyama et al. [22] studied the center edge displacement for both the normal and dysplastic female hips. They concluded the amount of excursion in the normal and dysplastic hips as 1.12 mm and 1.97 mm respectively. The excursion was measured while moving from a neutral joint position to the Patrick position [22]. The strongest ligament of the hip is the iliofemoral ligament, also called the Y ligament of Bigelow [23]. Henak et al. [24] demonstrated in vivo that acetabular labrum supported between 4 and 11% of the force across the joint compared to 1–2% in the normal structured hip. Safran et al. [25] reported the greatest strain change of an intact labrum occurred in the posterior labrum while most tears present clinically anterior and anterolateral. Philippon [26] has arthroscopically shown the anterior superior capsule is thick and taut while the posterior inferior portion is thinner. The acetabular labrum served as a secondary stabilizer. Smith et al. [27] were able to show continued resistance to femoral head translation, in spite of chondral-labral separation, when joint compression was applied to neutrally position femoral head. A limitation of that study is that it cannot be correlated to functional or athletic activity where the hips are in various, non-static positions. Field et al. [28] reported increased biomechanical for reconstructing the labrum, when possible.

Studies have also examined the properties of the ligamentous structures of the hip point. Myers et al. [29] found the iliofemoral ligament served as a primary stabilizer for limiting external rotation and preventing anterior translation of the femoral head Martin et al. [30] conducted a retrospective study of 350 surgical patients that 20 patients identified with complete ligamentum teres rupture. A string model was used to determine the ligament excursion of these subjects. It was concluded that ligamentum teres laxity may be most exposed in a hip with inferior acetabular insufficiency placed in the position of flexion/external rotation or extension/internal rotation. Byrd et al. [31] reported ligamentum teres rupture as the third most common arthroscopic finding.

Layer III: contractile layer

Layer III consists of all contractile tissues that support, control and create movement about the hip joint. This layer also includes trunk stabilizers and pelvic floor musculature. The purpose of this layer is to provide dynamic stability to the hip, pelvis and trunk. A multitude of extra-articular pathologies of the muscular tissue may be directly related to the underlying structural pathology of the hip joint. Hip internal snapping of the psoas may occur over the femoral head or iliopectineal eminence. The psoas is displaced laterally with flexion and medially with hip extension [32]. Babst et al. [33••] reported increased cross sectional area of iliocapsularis muscle in dysplastic hips, representative of dynamic stabilization to combat loss of inert tissue integrity. Beck et al. [34] reports that poor dissection around gluteus minimus during open procedures will result in loss of both anatomic and structural stability. Gluteus medius and minimus tears were found in approximately 20% of patients undergoing femoral neck fractures or total hip arthroplasties [35]. Tears in this muscle may clinically result in a positive Trendelenberg sign and weakness ascending stairs. Additional affected sites of pathology include the proximal hamstring and the medial complex consisting of rectus abdominus, conjoin tendon and adductor longus [36]. The mechanism may be acute, traumatic, overuse tendinosis or developmental avulsions [37]. Casartelli et al. [38] report that patients with FAI presented with decreased maximal voluntary contraction levels for the hip adduction (28%), flexion (26%), external rotation (18%) and abduction (11%) when compared with the control group, demonstrating the contractile dysfunction occurring as a result of structural pathology and pain. The tensor fascia lata (TFL) also demonstrated decreased activation during hip flexion in the hip diagnosed with FAI. These are all compensatory responses to layer I and II pathology and may be classified as seen in Table 1. [36]

Green et al. [39] found the adductor longus acts as a hip flexor and adductor magnus acts as a hip extensor from the immediate onset of the respective motion. The adductor longus also may act as a frontal plane stabilizer when the abductor mechanism is weak. Therefore clinically, the adductor group is consistently found to be overactive and at times develop into a tendinosis in this group. Abdominal wall musculature architecture is often affected because of its attachment to the pelvis and there has been clinical correlation demonstrated between the existence of FAI and the development of sports hernias [40]. In addition to the pathologies and pain generators of the respective layers, Janda's work with the Lower Crossed Syndrome theory has further added postural adaptive changes that must be considered when examining Layer III. The Lower Crossed Syndrome is characterized by inhibited abdominals and gluteal muscle groups and facilitated rectus femoris, iliopsoas and thoracolumbar extensors [41]. The effect of spinal pathology and the mytomal response to regional muscle groups cannot be ignored when examining muscle dysfunction about the hip and pelvis. Robb et al. [42•] showed differences between the hips in a baseball pitcher can affect pelvic

and trunk biomechanics in throwing. The end result could be loss in throwing velocity or predisposition to injury. Ellera Gomes et al. [43] showed in a series of 50 soccer players who suffered a non-contact anterior cruciate ligament (ACL) injury, more than 50% presented with radiological hip abnormalities. Both of these examples provide clinical significance for recognizing the importance of clearing the hip when examining kinetic linking activities.

Layer IV: neuro-mechanical layer

The neuro-mechanical layer is a theoretical layer compiled of anatomical structure, physiological events and kinematic changes throughout the chain which drive proprioception and pain within the hip. Locally at the site of the hip, this layer refers to the neuro-vascular structures, mechanoreceptors and nociceptors. On a global level, this layer refers to posture and the position of the pelvis over the femur. This may be affected by the result of lumbar pathology on the hip resulting in sacral torsion, rotation of the innominate or myotomal changes; or changes in foot and ankle mechanics and the response of the lower extremity up to the hip. It also involves looking at functional movement patterns and examining how motor learning affects dynamic movement of the pelvis over the femur or the femur under the pelvis.

The medial circumflex femoral and the lateral circumflex femoral arteries supply blood to the hip joint. Both of these arteries usually arise from the profunda femoris artery (deep artery of the thigh), however at times they are found to arise directly from the femoral artery. A small branch of the posterior division of the obturator artery runs through the ligamentum teres and also contributes to the femoral head. The gluteal and trochanteric anastomosis of the hip are comprised of femoral artery or the deep artery of the thigh and the gluteal arteries. [44, 45] The labrum receives its blood supply from a combination of the obturator, inferior and superior gluteal arteries; however, overall is not a well vascularized structure. One study does indicate significantly more vascularization of the capsular side of the labrum versus the articular side of the labrum [46].

Based on prior studies, it is understood that there exists four categories of nerve endings. Ruffini endings, Pacinian corpuscles, Golgi-tendon organs which are categorized as mechanoreceptors; and free nerve endings which are intra-articular nociceptive receptors [47]. Joint mechanoreceptors may further be categorized as Type I (Ruffini endings) found in the joint capsule, periosteum, ligaments and tendons and are responsible for proprioception. Types II (Pacinian corpuscles and Meissner corpuscles) are found in the deep joint capsule and fat pads; and are responsible for kinesthesia. Type III (Golgi endings) found in intrinsic and extrinsic ligaments; are responsible for proprioception. Lastly, Type IV (free nerve endings) is found in the joint capsule, ligaments, tendons, blood vessels and fat pads; are responsible for pain [47–49]. Type II mechanoreceptors have been described as rapidly adapting receptors with a low threshold. These receptors are inactive at rest, but respond reflexively to movement and point position [50]. Type I mechanoreceptors have a higher threshold and are slow adapting. Richard Dee in 1969, found the Type I receptors in the inferior joint capsule of the hip, but they were much less prominent in other peripheral joints [50]. The purpose of the Type I receptors here being to respond to stress and stretch in the hip joint capsule. The Type II receptors however, were non-existent in the hip joint, but were present in other peripheral joints of the body. This concept has been supported by additional literature examining the hip joint capsule and ligamentum capitus femoris (LCF) of normal hips and dysplastic infant hips [51]. In this study, no mechanoreceptors were found in either the capsule or the LCF of any of the subjects. This is contrary to a study done with adult hips by Leunig [52], which did demonstrate free nerve ending in the LCF. Dee [50] supported this finding clinically by examining a patient who had hip pain for two weeks. It was noted that she was able to stand in unilateral stance without too much trouble, however when she closed her eyes and her visual sense was removed, she demonstrated a significant hip drop when standing on the affected side.

The significance of this finding was to suggest that at the time of injury, the lack of proprioceptive mechanoreceptors found in the hip, will leave the joint more susceptible to neuromuscular inhibition and dysfunction as compared to other peripheral joints. Other peripheral joints may have an abundance of mechanoreceptors to provide a local reflexive response to an injury.

The impact of the kinematic chain cannot be ignored in this system of arthro-kinetic reflexes. Whether the hip joint is the cause of dysfunction or the victim, there has been evidence since 1939 that there exists a chain of adaptive reactions changing movement patterns throughout the system. This can occur from the foot up to the pelvis and from the trunk down to the foot [53–59]. Bullock-Saxton [60] and Janda [40] demonstrated this further in examining muscle activation patterns in male athletes who had chronic ankle sprains (> four months). The results demonstrated significantly delayed activation of the gluteus maximus during prone hip extension in the experimental group versus the non-injured control group. These results certainly reveal that neuromuscular and reflexive relationships do exist. The study concluded there was a change in muscle firing patterns at the hip as a result of an injury to the ankle. One could argue that the reverse may also be true. In this case, an existing low back or hip pathology affecting muscle function at the pelvis may change the mechanics from the trunk down and affect how the foot is impacting the ground. Low

back pain in athletes is not uncommon and often involves injury at the L5-S1 level [61, 62]. The local response to such an injury is inhibition of segmental stabilizers, multifidus and transverse abdominus [63]. The myotomal distribution of the L5 and S1 nerve roots will affect the hip abductors, hip external rotators, hip extensors, knee flexors, peroneals, dorsiflexors and plantar flexors [44, 64]. Understanding the neuromuscular relationship of the spine and lower quarter is imperative to successful examination and treatment of hip patients.

Treatment by layer

Clinical treatment of hip pain is closely guided by the examination. Due to the loads and forces placed through the hip joint during both open and closed chain activities, it may be quite difficult to come to an immediate conclusion regarding the etiology of the hip pain. Understanding compensatory movement patterns, as well as femoral and pelvic open and closed chain mechanics should help to isolate the causes and effects. This is why it is not unusual to take a few visits to decide upon and implement a definitive treatment plan. Therefore, following a functional movement exam and a spine screening (Layer IV), the clinical exam of the hip begins from Layer I and moves out toward Layer III. A series of special tests may be used in examining the layers. (Table 2)

Treatment however, begins from Layer IV and progresses in toward Layer I. The kinematic chain must be addressed if a dysfunction is identified. In examining the spine, the ability to recognize a restriction, hypermobility or pelvic obliquity or sacral torsion is of utmost importance. The effect on muscle imbalance or myotome dysfunction will significantly affect the arthrokinematics and muscle timing and performance around the hip. Therefore, addressing these pathologies is imperative. Spinal restrictions and/or pelvic obliquities have been addressed using manual therapy techniques prior to attempting to re-educate muscle function. Equally important is addressing how the foot hits the ground and the effect on the hip and pelvis.

Layer III, the soft tissue layer of fascia and muscle, needs to be assessed for restriction, tightness and tone. Neuromuscular re-education is not affective if these factors are not first addressed. Discrimination of muscle tightness (adaptive shortening) versus tone (protective or myotomally driven) is a vital factor to decipher and the treatment will need to correspond appropriately. Muscle tightness may be differentiated from tone by noting if there is a palpable restriction at the end range of tissue length or is there resistance to movement of the tissue throughout its length. Muscle tone may be driven by a spinal pathology, therefore this clinical finding should match the clinical findings from examining Layer IV. The pattern for protective tone may be categorized into the Lower Cross Syndrome as described by Janda [44] or adductor tone in response to hip pain and inhibition of primary hip stabilizers which is clinically consistent in this group of patients. As described previously, Green demonstrated the extensive function of the adductor group [38]. Therefore, it is understandable that in the presence of pain, the adductor group would respond to assist in protection and stabilization. Soft tissue mobilization and static stretching may be most effective for a tight or shortened muscle, however, soft tissue mobilization alone will not be effective for a muscle with tone. The muscle needs to be neuromuscular inhibited using principles of reciprocal inhibition [65] and then re-educated in proper timing and functional sequence patterns.

Re-education of these core and hip stabilizers has most successfully been achieved in an unloaded position, progressing to an upright loaded position. Dynamic movement from the upper extremity is emphasized in the stabilizing movements to minimize the risk of irritating the hip with repetitive open chain movements. Once core, pelvis and lower extremity stability is demonstrated, then movement patterns may move out of closed chain exercises into more dynamic and functional movement patterns.

Layer II, the inert tissue layer may be addressed through joint mobilization only after all soft tissue restrictions have been addressed. Joint capsules treated with mobilization without definitive clinical reasoning may be detrimental to the recovery process. A hip with normal capsular mobility, but rather a fascial restriction which was over looked, may become an unstable hip, therefore escalating pain and pathology. Once the hip joint is mobilized and new motion is gained, the muscles must then be educated to function through the new range. If the joint capsule is found to be hypermobile or the ligamentum teres has been disrupted, it is still necessary to work through all soft tissue restrictions and then slowly develop a balanced dynamic muscular stabilizing structure around the core and hip.

Layer I, the osseous layer, presents as the most challenging in the rehabilitation setting. Particularly for high level athletes, a restricted range of movement is not an option. However, there are cases in which patient education to avoid positions which create bony impingement (deep flexion, adjust the car seat or desk chair and modification to workouts), may alleviate the patient's pain. Education to drop the opposite leg in the stance while on the field to avoid impingement, may be the instruction required to assist a high level athlete through the end of their season. It is not uncommon to consider an intra-articular injection to help diminish the inflammatory process in this population.

Conclusion

Hip rehabilitation is not linear. Although empirical in nature, overcoverage may be thought of as a dysfunction of Layer I joint kinematics and undercoverage as Layer III kinetic response to Layer I osseous incongruities and Layer II inert insufficiencies. This population of patients may be some of the most challenging to treat. Forces placed on the hip, the number of muscles crossing this joint and the number of anatomical layers under load, require advanced clinical reasoning skills. When the proper treatment recommendation is made, the outcomes are remarkable. However, when improper treatment is prescribed the result is devastating to the patient's quality of life, particularly in cases where surgery was performed, but not appropriately indicated. The purpose of this paper was to introduce the concept of layers so that clinicians may be more systematic regarding the examination and have an algorithm for decisions made regarding treatment.

Disclosure P. Draovitch: none; J. Edelstein: none; B. Kelly: consultant for Pivot medical, Smith and Nephew, and A2 Surgical; stock/stock options with Pivot Medical and A2 Surgical.

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