# Red Flags for Potential Serious Conditions in Patients with Pelvic, Hip, or Thigh Problems

| Red Flags for the Pelvis, Hip, and Thigh Region | | |
|---|---|---|
| **Condition** | **Red Flag<br>Data obtained during Interview/History** | **Red Flag<br>Data obtained during<br>Physical Exam** |
| Colon Cancer[1] | Age > 50 years old<br>Bowel disturbances (e.g., rectal bleeding, black stools)<br>Unexplained weight loss<br>History of colon cancer in immediate family<br>Pain unchanged by positions or movement | Later stages: may have hypo-or hyper active bowel sounds from obstruction<br>Possible tenderness to palpation of abdomen in area of cancer<br>May have ascites<br>First sign may be of metastases to liver, lung, bone, or brain |
| Pathological Fractures of the Femoral Neck[2,3] | Older females (>70 years) with hip, groin, thigh or knee pain<br>History of a fall from a standing position | Severe, constant pain – worse with movement<br>A shortened and externally rotated lower extremity |
| Osteonecrosis of the Femoral Head[4]<br>(aka Avascular Necrosis) | History of long-term corticosteroid use (e.g., in patients with RA, SLE, asthma)<br>History of AVN of the contralateral hip<br>Trauma | Gradual onset of pain; may refer to groin, thigh, or medial knee; worse with weight-bearing<br>Stiff hip joint; restrictions primarily in IR, flexion, adduction |
| Legg-Calve-Perthes Disease[5] | 5-8 year old boys with groin/thigh pain | Antalgic gait<br>Pain complaints aggravated with hip movement, especially hip abduction and internal rotation |
| Slipped Capital Femoral Epiphysis[6] | Overweight Adolescent<br>History of a recent growth spurt or trauma | Groin aching exacerbated with weight-bearing<br>Involved leg held in external rotation<br>ROM limitations of hip internal rotation |
| Septic Hip Arthritis[7] | Child or older adult with vague hip aching who had a recent bacterial infection | Unwillingness to weight bear on or move the involved hip |
| Inguinal hernia[8] | Pain in groin, and/or scrotum in males<br>Consider “sports hernia” (internal disruption of the inguinal canal) in an athlete with unresolving groin pain | Sx’s exacerbated by coughing, sneezing or resisted sit-up<br>Tenderness in area of inguinal canal |
| Appendicitis[9] | RLQ pain, then nausea and vomiting<br>Retrocecal appendix may refer pain to right thigh or testicle | Abdominal rigidity, rebound tenderness<br>Positive McBurney’s point<br>Positive Psoas and Obturator sign |
| Ovarian Cyst[10] | Female of childbearing age<br>Sudden, severe abdominal or pelvic pain<br>Menstrual irregularities and pain | |


References:

1. Suadicani P, et al. Height, weight, and risk of colorectal cancer. An 18-year follow-up in a cohort of 5249 men. *Scandinavian Journal of Gastroenterology*. 1993;28:285-288.
2. Tronzo RG: Femoral neck fractures. In Steinburg ME, ed. *The Hip and Its Disorders*. Philadelphia, Saunders. 1991:247-79.
3. Guss DA: Hip fracture presenting as isolated knee pain. *Ann Emerg Med.* 1997:29:418-420.
4. Stulberg BN, Bauer TW, Belhobek GH, et al. A diagnostic algorithm for osteonecrosis of the femoral head. *Clin Ortho*. 1989;249:176-182.
5. Wenger DR, et al. Current concepts review: Legg-Calve-Perthes disease. *J Bone Joint Surg Am*. 1991;73:778-788.Busch MT, Morrissy RT. Slipped capital femoral epiphysis. *Orthopedic Clinics of North America*. 18;637-647, 1987Kocher MS, Zurakowski D, and Kasser JR. Differentiating between septic arthritis and transient synovitis of the hip in children: an evidence-based clinincal prediction algorithm. *J Bone Joint Surg Am*. 1999;81:1662-1670.
8. Kesek P, Ekberg O. Herniographic findings in athletes with unclear groin pain. *Acta Radiologica* 2002;43;603-608.
9. Graff L, Russell J, Seashore J, et al. False-negative and false-positive errors in abdominal pain evaluation: failure to diagnose acute appendicitis and unnecessary surgery. *Academic Emergency Medicine*. 2000;7:1244-1255.
10. Kumar, S. Right-sided low inguinal pain in young women. *R Coll Surg Edinb.* 1996;41:93-94.

# PELVIC/HIP PAIN MEDICAL SCREENING QUESTIONNAIRE

NAME: _____ DATE: _____
Medical Record #: _____

| | Yes | No |
|---|---|---|
| **1. Have you recently had a trauma, such as a fall?** | ☐ | ☐ |
| **2. Have you ever had a medical practitioner tell you that you have osteoporosis?** | ☐ | ☐ |
| **3. Have you ever had a medical practitioner tell you that you have a problem with the blood circulation in your hips?** | ☐ | ☐ |
| **4. Are you currently taking steroids or have you been on prolonged steroid therapy?** | ☐ | ☐ |
| **5. Does your pain ease when you rest in a comfortable position?** | ☐ | ☐ |
| **6. Do you have a history of cancer?** | ☐ | ☐ |
| **7. Has a member of your immediate family (i.e., parents or siblings) been diagnosed with cancer?** | ☐ | ☐ |
| **8. Have you recently lost weight even though you have *not* been attempting to eat less or exercise more?** | ☐ | ☐ |
| **9. Have you had a recent change in your bowel functioning such as black stools or blood in your rectum?** | ☐ | ☐ |
| **10. Have you had diarrhea or constipation that has lasted for more than a few days?** | ☐ | ☐ |
| **11. Do you have groin or thigh pain that increases when you cough or sneeze?** | ☐ | ☐ |
| **12. Do you feel sick to your stomach to the point where you feel like vomiting?** | ☐ | ☐ |
| **13. If female, have you recently experienced increased pain during your menstrual period?** | ☐ | ☐ |

# Hip Mobility Deficits

*ICD-9-CM codes*: 843.1 Sprain of the ischiocapsular ligament

*ICF codes*: Activities and Participation Domain codes:

**d4500** Walking short distances (Walking for less than a kilometer, such as walking around rooms or hall ways, within a building or for short distances outside.)

**d4500** Walking long distances (Walking for more than a kilometer, such as across a village or town, between villages or across open areas.)

**d4154** Maintaining a standing position (Staying in a standing position for some time as required, such as when standing in a queue.)

Body Structure code: **s75001** Hip joint

Body Functions code: **b7100** Mobility of a single joint

*Common Historical Findings*

Ache in groin/medial thigh after activity – especially after prolonged weight bearing

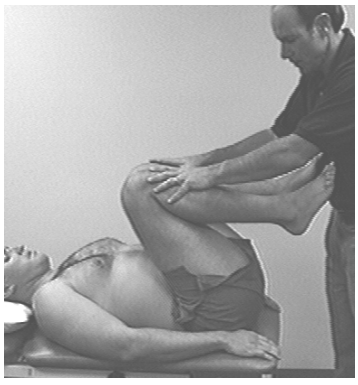
Progresses to stiffness

*Common Impairment Findings* - Related to the Reported Activity Limitation or Participation Restrictions:

| | | Norms |
|---|---|---|
| Motion limitations of | extension: | 20° |
| | internal rotation: | 40° |
| | abduction: | 40° |

Pain at end range - one motion is worse than others and reproduces symptoms

*Physical Examination Procedures*:



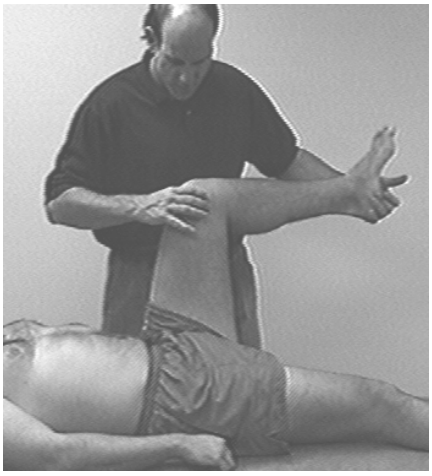
Hip Extension ROM
Starting Point

Hip Extension ROM
Measuring Point

Performance Cues:

Flex both hips and posteriorly rotate pelvis to the point where lumbar spine is flush against the table

Lower the involved femur - keeping it in 0° abduction - determine hip extension ROM



Hip Internal Rotation ROM

Performance Cues:

Flex to 90° - maintain

Maintain 0° abduction/adduction

Internally rotate femur to barrier - measure along line of tibia

Hip Abduction ROM

Performance Cues:

Abduct femur until pelvis begins to move

Measure angle of both ASIS's and abducted femur

# Hip Mobility Deficits: Description, Etiology, Stages, and Intervention Strategies

The below description is consistent with descriptions of clinical patterns associated with the vernacular term "**Hip Capsulitis.**" There is not an ICD-9 code for a category representing a clinical pattern suggesting "hip capsulitis" or "hip adhesive capsulits." The ICD-9 category is most consistent with this pattern is probably 843.1 "sprain of the ischiocapsular ligament." However, the hip mobility deficits are likely the sequelle of inflammation induced capsular fibrosis following a hip capsular or "ischiocapsular" sprain.

*Description:* Hip capsulitis, also known as degenerative joint disease (DJD), osteoarthritis (OA), or "wear and tear" arthritis, is the most common type of arthritis and a leading cause of disability worldwide. The incidence of OA increases with age and OA Hip, along with OA of the knee, affects the ability to walk and climb stairs more than any other disease. It is a slow degeneration of joint cartilage, with possible inflammation, pain, muscle weakness, or even loosening of the ligaments of the hip. The normal hip joint has articular cartilage that provides a smooth and cushioned surface between bones. With DJD, the smooth surface becomes rough and pitted. In advanced stages, the smooth surface may wear away completely and the synovium of the joint may be inflamed.

*Etiology:* Etiology: It is believed that OA results from a combination of genetic abnormalities and joint injuries. Joint injuries may be associated with repetitive manual labor or with repetitive sports, such as ballet. DJD of the hip usually begins gradually, because it normally takes years for the cartilage to become noticeably damaged. Initially, pain is felt at the end of the day. As the disease progresses, pain is commonly felt during the day, especially after exercise or weight bearing activities. Pain commonly subsides with rest, but can later progress to waking the patient at night. Pain is generally felt at the anterior hip, groin, and trochanteric area. The symptoms often progress to significantly deteriorate the quality of life, which may lead to the decision to have a total hip replacement.
Until the 1980s, OA was considered to be primarily a degenerative disorder and a natural occurrence of "wear and tear" on joints as a result of aging. Research evidence is now changing this view, with the most crucial and fundamental change being the shift from thinking of OA as a passive, degenerative disorder for which little can be done to the realization that OA is driven by an active disease process of the joint that can be modified by both mechanical and biochemical (drug) interventions.

<u>Acute Stage / Severe Condition</u>: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b7100.3** SEVERE impairments of mobility of a single joint

- Loss of mobility at community level due to pain with amblulation
- May exhibit a Trendelenburg or gluteus medius compensated gait and may use a cane to decrease force on hip
- Difficulty with taking off shoes, socks, and pants
- Considerable hip flexion, abduction, extension, and internal rotation ROM limitations. Restriction in internal rotation has been shown to be the most predictive sign of radiographic OA, and, when present, can factor into decisions regarding the need for radiography
- Weak and painful gluteus medius and tensor fascia lata

Sub Acute / Moderate Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b7100.2** MODERATE impairments of mobility of a single joint

As above with the following differences:

- Antalgic gait with shortened stance time of the involved extremity
- Muscle atrophy secondary to disuse associated with pain
- Moderate hip flexion, abduction, and internal rotation ROM limitations
- Gait deviations (e.g., compensated gluteus medius gait) associated with the pain and motor control deficits

Settled Stage / Mild Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b7100.1** MILD impairments of mobility of a single joint

As above, with the following differences:

- Morning stiffness or stiffness after inactivity
- Crepitus with active motion
- Pain in the joint after unaccustomed use
- Minimal interference in activities of daily living secondary to joint pain

## Intervention Approaches / Strategies

Acute Stage / Severe Condition

Goals: Maximize tissue and joint health – optimize circulation to the hip's articular cartilage
Decrease pain and inflammation

- Manual Therapy
  - Soft tissue mobilization, joint mobilization and manual stretching to improve hip capsule mobility
  - Remember that it is the tight capsule in a patient with OA at the hip is a primary source of pain and that in the patient with significant pain, the hip joint is more likely to be more "irritable." Thus, progression of the mobilization should proceed slowly and response to treatment monitored closely. Mobilizations with movement – emphasizing painfree active and passive mobility are manual therapy procedures to consider in this stage

- Therapeutic Exercises
  - Stretching exercises to maintain/improve on gains made with manual procedures, i.e., stretching to increase internal rotation, extension and flexion
  - Strengthening exercises to hip muscles – especially for the hip abductors and hip extensors

- Physical Agents
  Ice for 20-30 minutes when joint inflamed; heating pad when not
  Ultrasound for deep heating effect when not inflamed

- Pain Management
  TENS
  Corticosteroid (steroid) injections might be necessary. A total hip replacement might be required if conservative (physical therapy) management is not sufficient to control pain and maintain functional independence.

- Re-injury Prevention Instruction
  Single point cane on side of unaffected limb to lessen load on hip by 60 percent and reduced pain with ambulation.
  Avoid low chairs, sleeping on the affected side, and bending forward at the hip
  The patient may benefit from special adaptations in shoe fasteners and devices to help put on and take off shoes and socks

<u>Sub-Acute Stage / Moderate Condition</u>

Goals: Improve hip mobility and stability
Prevent re-injury to the joint cartilage or capsule
Restore strength of the muscles around hip

- Manual Therapy
  Same as above.
  Proprioceptive neuromuscular facilitation (PNF) techniques – facilitating painfree mobility

- Therapeutic Exercises
  Progress home exercise program (HEP), using concentric and eccentric exercises for strengthening as tolerated
  Stretching exercises and general conditioning exercises, such as Yoga and Tai Chi, that focus on balance and flexibility
  General conditioning exercises, such as cycling, walking, swimming and exercising in the water, are highly recommended. (Jogging and long distance walking be abandoned in favor of non-weight-bearing exercises if they are painful or produce post-exercise pain or stiffness)
  General conditioning and diet changes will allow for slower deterioration of cartilage – especially if weight loss is achieved

- Physical Agents/ Pain Management
  Same as above.
  Medications include acetaminophen, NSAIDs, or COX–2 inhibitors to reduce joint inflammation and/or pain.

Settled Stage / Mild Condition

Goals: Increase pain-free activity tolerance
Patient education on condition and preventative care

Same as listed above, except

- Therapeutic Exercises
  Develop a home exercise program (HEP) encompassing stretching, strengthening and aerobic exercises (avoiding high-impact sports)
  Start or continue with general conditioning activities such as Yoga, Tai Chi and swimming

- Physical Agents
  Educate on the use of ice and heat as listed above.

- Pain management
  Acetaminophen (as recommended by the College of American Rheumatology)
  Exercise to reduce pain and stiffness at this stage, especially morning pain

- Re-injury Prevention Instruction
  Educate the patient about his or her condition and to avoid symptom producing weight bearing recreational activities or job tasks
  Teach general body mechanics and proper posture to prevent further deterioration of articular cartilage
  Body weight reduction, load-carrying modifications, and assistive device use

Intervention for High Performance / High Demand Functioning in Workers or Athletes

Goal: Optimal functioning at desired work or leisure activities

- Work with patient to select appropriate leisure activities that will not speed up the degenerative process of the hip cartilage

- Perform an ergonomic consult at the patient's job (if possible), or simulate the work environment to recommend joint conservation strategies

Selected References

Brand, R.A., Crowninshield, R.D. The effect of cane use on hip contact force. *Clin Orthop*. 1980;147:181-4.

Chitnavis J, Sinsheimer JS, Suchard MA, Clipsham K, Crr AJ. End stage coxarthrosis and gonarthrosis.Aetiology, clinical patterns and radiological features of idiopathic osteoarthritis. *Rheumatology* 2000;39:612-619

Daly, M.P., Berman, B.M. Rehabilitation of the elderly patient with arthritis. *Clinical Geriatric Medicine*. 1993; 9:783-801.

Deyle, G.D., Henderson, N.E., Matekel, R.L., et al. Effectiveness of manual physical therapy and exercise in osteoarthritis of the knee: A randomized, controlled trial. *Ann Int Med*. 2000; 132:173-81.

Dieppe, P. Management of hip osteoarthritis. *British Med J*. 1995; 311:853-7.

Falconer, J., Hayes, K.W., Chang, R.W. Therapeutic ultrasound in the treatment of musculoskeletal conditions. *Arthritis Care Res*. 1990; 3:85-91.

Hochberg MC, Perlmutter DL, Hudson Jl, et al.: Preferences in the management of osteoarthritis of the hip and knee: Results of a survey of community-based rheumatologists in the United States. *Arthritis Care Res*. 1996 Jun;9(3): 170-6

Lewis, D., Lewis, B., Sturrock, R.D. Transcutaneous electrical nerve stimulation in osteoarthritis: A therapeutic alternative? *Ann Rheum Dis*. 1984; 43:47-9.

Liang, M.H., Fortin, P. Management of osteoarthritis of the hip and knee. *New Engl J Med*. 1991; 325:125-7.

Lorig K, Feigenbaum P, Regan C, et al: A comparison of lay-taught and professional-taught arthritis self-management courses. *J Rheumatol* 1986:13-4:763-767.

Lorig K, Holman H: Arthritis self-management studies: A twelve-year review. *Health Education Quarterly* 1993;20-1:17-28.

Nesher, G., Moore, T.L. Clinical presentation and treatment of arthritis in the aged. *Clinical Geriatric Medicine*. 1994; 10:659-75.

Nevitt, M.C. Definition of hip osteoarthritis for epidemiological studies. *Ann Rheum Dis*. 1996; 55:652-5.

Steultjens MPM, Dekker J, van Baar ME, Oostendorp RAB, Bijlsma JWJ. Range of joint motion and disability in patients with osteoarthritis of the knee or hip. *Rheumatology* 200;39:955-961.

# Manual Therapy for "Hip Capsulitis"

ROM Examination:
- Hip Extension: 1 joint muscles and capsule; 2 joint muscles
- Hip Flexion: 1 joint muscles and capsule; 2 joint muscles and nerves (SLR)
- Hip Internal Rotation: at 90 degrees of hip flexion; at 0 degrees of hip flexion
- Hip External Rotation: at 90 degrees of hip flexion; at 0 degrees of hip flexion
- Hip Adduction

Intervention:

Soft Tissue Mobilization:
- Iliacus (hug knee, fingertips)
- Psoas (same side, flat fingers)
- Post Gluteus Medius (sidelying, elbow)
- Gluteus Max, Quadratus Femoris, Pirifomis (sidelying, knuckles)

Joint Mobilization:
- Anterior Glide w/ strap assistance
- Distraction via strap w/ rotation MWM; w/ flexion MWM

Contract/Relax Stretch:
- Iliacus (Thomas Test position)
- Medial Rotation w/ Hip at 90 degrees (supine)
- Lateral Hamstrings (using PNF pattern)
- Medial Hamstrings (using PNF pattern)

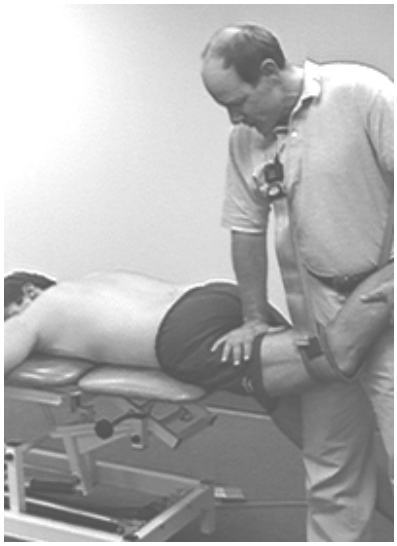
Iliacus

Psoas

Gluteus Maximus/Medius

Hip External Rotators

Impairment: Limited Hip Extension
Limited Femoral Anterior Glide

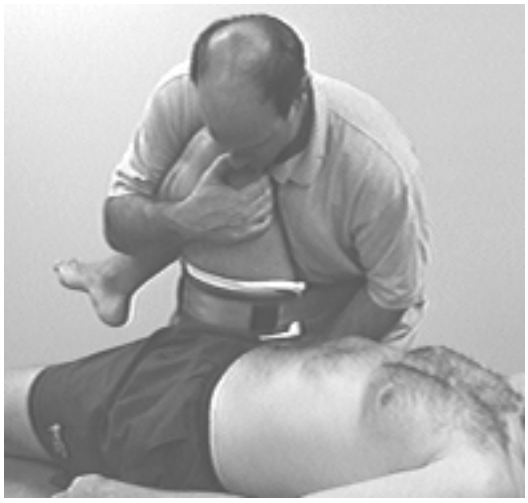


Femoral Anterior Glide

Cues: Position patient so that his/her ASIS's are in contact with the table - with the femoral head/acetabulum distal to the edge of the table
Stand medial to the involved thigh
Use a strap around your 1st rib area and around the distal one-third of the patient's thigh strap
Position strap so that sidebending of your trunk toward the hip slightly increases the patient's hip extension
"Hug" the patient's tibia and ankle with one hand and forearm
Anteriorly glide the proximal femur with the other hand using a sidebend of your trunk

The following reference provides additional information regarding this procedure:
Freddy Kaltenborn PT: Manual Mobilization of the Extremity Joints, p. 181, 1989

Impairment: Limited and Painful Hip Internal Rotation
(at 90 degrees of hip flexion)



Hip Rotation MWM

Cues: Stabilize the pelvis with one hand (stabilize the elbow of this hand against your groin)
"Hug" the involved thigh and leg with the other arm and thorax
Laterally glide the femur using a belt
Sustain the lateral glide while the patient's femur is actively and passively internally rotated – the passive rotation and overpressure is a result of the therapist's trunk rotation
Alter the amplitude and direction of the lateral glide in an attempt to provide a deep, tolerable hip "stretch"

The following reference provides additional information regarding this procedure:
Brian Mulligan MNZSP, DipMT: Manual Therapy, p. 103-104, 1995

# Hip Muscle Power Deficits

*ICD-9-CM codes*: 843.8 Specified sites of hip and thigh strains

*ICF codes*: Activities and Participation Domain codes:

**d4105** Bending (Tilting the back downward or to the side, at the torso, such as in bowling or reaching down for an object)
**d4350** Pushing with lower extremities (Using the legs and feet to exert a force on an object to move it away, such as pushing a chair away with a foot)
**d4351** Kicking (Using the legs and feet to propel something away, such as kicking a ball)
**d4552** Running (Moving with quick steps to that both feet may be simultaneously off the ground)

Body Structure code: **s75002** Muscles of thigh
Body Functions code: **b7300** Power of isolated muscles and muscle groups

*Common Historical Findings*

Pulled" muscle during a quick, eccentric demand of muscle - or - blunt trauma to hip myofascia
Daily activities which stretch or contract the involved muscle are painful

*Common Impairment Findings* - Related to the Reported Activity Limitation or Participation Restrictions:

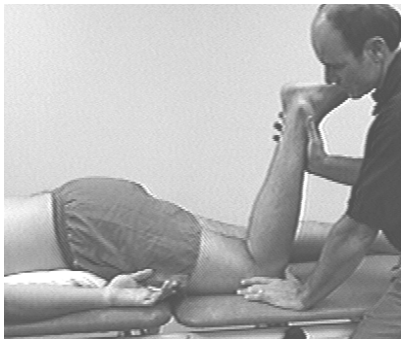
Pain with stretch to the involved muscle
Pain with contraction of the involved muscle
Pain with palpation of the injury site

*Physical Examination Procedures*:

Lateral Hamstring Stretch

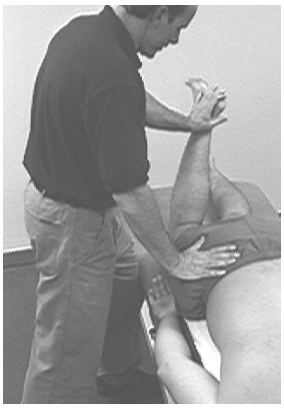
Medial Hamstring Stretch

Performance Cues:
Flexion/adduction movement stretches the posterior lateral part of thigh
Flexion/abduction movement stretches the posterior medial part of thigh

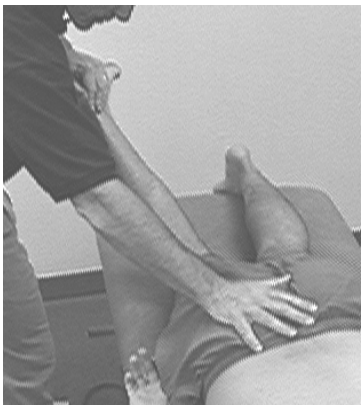


Rectus Femoris Stretch and Resistive Test

Performance Cues:
The amount of contralateral hip flexion/posterior pelvic rotation determines the intensity of the stretch
Flex the knee (slowly) to stretch rectus femoris and vasti muscles
Monitor symptom response and strength of contraction during the resistive test
Prone knee bending also provokes symptoms in the presence of femoral nerve or anterior innominate abnormalities - muscle palpation provocation provides the differential diagnosis

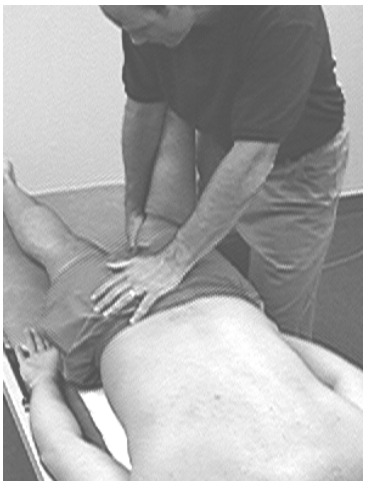


Lateral Hamstring
Resistive Test



Medial Hamstring
Resistive Test

Performance Cues:
For lateral hamstring (biceps femoris) - externally rotate tibia, contact lateral heel, and apply force in a medial direction
For medial hamstrings (semimembranosis and semitendinosis) - internally rotate tibia, contact medial heel, and apply force in a lateral direction
A gentle force will elicit symptoms with moderate tears
Stronger force will be required to elicit symptoms in mild tears and in subacute conditions
Do not expect to be able to totally isolate the medial vs. lateral hamstring contraction – however, symptom responses vary - thus, focusing the palpation examination



Hip Adductor Stretch and Resistive Test

Performance Cues:
Stabilize opposite thigh during application of stretch and resistance
Monitor symptom response and strength during muscle contraction
Adductor tests also provokes symptoms in the presence of pubic symphysis abnormalities - muscle palpation provocation provides the differential diagnosis

## Hip Muscle Power Deficits: Description, Etiology, Stages, and Intervention Strategies

The below description is consistent with descriptions of clinical patterns associated with the vernacular terms **"Hamstring, Hip Adductor, or Hip Flexor Muscle Strain"**

*Description*: Generalized weakness secondary to the damaged muscle's inability to develop tension. Damage presents itself as tears that can occur at any point along the muscle. There are three grades of muscle; Grade 1 - microtearing of the muscle fibers due to excessive tearing, Grade 2 - incomplete tear of the muscle fibers, Grade 3 - complete tear of muscle

*Etiology*: Sudden passive stretch or an activation of the muscle during a stretch (i.e., a forced eccentric contraction) persistent repetitive stress, improper warm-up, fatigue, previous injury and lack of flexibility have been factors correlated with hamstring injury. A slow controlled stretch that continues to an abnormally extreme position can also strain the muscle. Hamstring strains are the most common strain injury in athletes.

Differential Diagnosis for *Hamstring Strains*: Other conditions with similar presentations as hamstring strains are sciatic nerve mobility limitations, lumbar spine disorders (L4-S1), strained popliteus muscle, osteochondroma with bursa formation (one case reported), tendonitis at either origin of the gastocnemius, apophysitis-pain in ischial tuberosity, sprained posterior cruciate ligament, and lesions of the upper tibio-fibular joint. Resisted external or internal rotation can distinguish between a distal biceps lesion or a lesion in the medial hamstrings.

Differential Diagnosis for *Rectus Femoris Strains*: Other conditions with similar presentations as rectus femoris strains are femoral nerve or lateral femoral cutaneous nerve mobility limitations, lumbar spine disorders (L1-L4), sacroiliac joint disorders, hip disorders, and psoas trigger points.

Differential Diagnosis for *Adductor Strains*: Other conditions with similar presentations as adductor strains are pubic symphysis disorders, sacroiliac joint disorders, obterator nerve mobility limitations, lumbar spine disorders (L1-L4), hip disorders, and psoas trigger points.

Prognosis: Complete recover from pain is sometimes difficult. Untreated condition may lead to scaring, fibrosis and shortening of the related myofascia. If effusion occurs close to the adjacent nerve, residual symptoms of nerve tension may be present. A hematoma that is contained within a thigh compartment may lead to a compartment syndrome. The sites affected, in order of incidence, are: injuries muscle belly (most on the musculotendinous junction MTJ), proximal tendon-bone injuries (avulsions) and distal tendon injuries. Ultrasongraphy may be able to detect only half of injuries compared to MRI.

Risk/Predisposing factors: Persistent repetitive stress, improper warm-up, fatigue, lack of muscle strength, previous injury, lack of flexibility, enthesopathy, involvement in high acceleration /deceleration sport activities such as water skating, soccer, rugby or dance.

Prevention: Warm up stretching exercises are frequently recommended. Strengthening exercises tends to alter viscoelasticity and gains in muscle strength may mean losses in flexibility. However progressive eccentric loading exercises are preventive (Askling).

Acute Stage / Severe Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b7300.3** SEVERE impairments of muscle power

- Pain with weight bearing on the involved limb
- Bleeding/eccymosis (i.e., black and blue appearance) if grade 2
- Balled up muscle if grade 3 (which is rare)
- Pain with stretch of the involved muscle (i.e., during a SLR or prone knee bend)
- Pain and weakness with active contraction of the involved hamstring(s)
- Palpable tenderness (i.e., hematoma) at the injury site

Sub Acute / Moderate Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b7300.2** MODERATE impairments of muscle power

- Painful, but strong contraction of the involved muscle(s)
- Palpable soft tissue restrictions at the injury site

Settled Stage / Mild Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b7300.1** MILD impairments of muscle power

- Strong and painfree contraction of the involved muscle(s)
- Potential muscle flexibility deficits of the involved muscle(s)
- Less palpable soft tissue restrictions at the injury site

Intervention Approaches / Strategies

Acute Stage / Severe Condition

Goals: Prevent further injury - protect muscle integrity
Reduce pain

- Immediate Measures: First 24 hours
  Compression taping used with an elastic bandage for 10 minutes followed by mild compression to reduce blood flow
  Ice bandage
  Immobilization
  Crutch use

After the first 24 hours

- Physical Agents
  Non-thermal ultrasound

- Therapeutic Exercises
  Painfree active mobility exercises of the affected muscle (and hip and knee) – consider the use of aqua therapy (i.e., pain free mobility exercises and walking in a pool)

- Re-injury Prevention Instruction
  Perform no movements that cause pain (e.g., streching producing pain should be avoided)
  Crutch training if walking is painful.
  Paresthesias and numbness while exercising should be avoided as well as any numbness produced by the compressive taping or shorts

- External Devices (Taping/Splinting/Orthotics)
  Compression taping using with an elastic bandage – or – wearing compressor shorts

Sub Acute Stage / Moderate Condition

Goals: As above
Optimize tissue regeneration
Restore flexibility, strength, and endurance the involved hamstring

- Manual Therapy
  - Soft tissue mobilization to area of soft tissue restrictions (that may have resulted from excessive scar formation during the fibroplasia phase of tissue healing)
  - Very early intense massage in completely diagnosed cases may contribute to problems such as heterotropic bone formation

- Therapeutic Exercises
  - Utilize stationary bike, walking on treadmill, and active knee flexion in standing for mobility exercises
  - Gentle passive stretching if tolerated without pain and does not worsen the symptoms
  - Painfree submaximal isometric contractions
  - Painfree strengthening exercises, such as low resistance, low load against gravity, prone hip extension, heel slides, scooting with rolling chair

- Re-injury Prevention Instruction
  - Be extremely cautious to proceed exercises and stretching slowly and without pain

Settled Stage / Mild Condition

Goals: Painfree, unlimited walking or long distance running

- Physical Agents
  - Consider applying heat before exercise, and cold after execise

- Therapeutic Exercises
  - End-range passive stretching if tolerated without pain and does not worsen the symptoms
  - Sciatic or femoral nerve mobility exercises
  - Dural (i.e., slump) mobility exercises
  - Progress strengthening exercises, such as partial curl ups, dead bug progression, wall slides, single knee to chest, double knee to chest, increase resistance, increase speed as able, eccentric exercises, seated theraband, modified standing theraband knee extensions

- Re-injury Prevention Instruction
  - Muscles need to be stretched before and after exercise.

Intervention for High Performance / High Demand Functioning in Workers or Athletes

Goal: Return to desired sport or occupation demands

- Approaches / Strategies listed above

- Re-injury Prevention Instruction
  Before returning to sports the patient should be entirely painfree and all additional contributory factors eliminated.
  Grade 2 strains take longer recovery time – 3-4 weeks
  Grade 3 strains may require surgical intervention

Selected References


Askling C, Karlsson J, Thorstensson A. Hamstring injury occurrence in elite soccer players after preseason strength traning with eccentric overload. *Scan J Med Sci Sports.* 2003; 13: 244-250

Baker K, Robertson V, Duck F. A review of therapeutic ultrasound biophysical effects. *Phys Ther*. 2001;81:1354

Brubaker CE, James SL. Injuries to runners. *J Sports Med.* 1974;2:189-198.

Hurme T. Rantane J, Kalimo H.. Effects of early cryotherapy in experimental skeletal muscle injury. *Scand J Med Sci Sports* 1993;3:46-51.

Jackson, DW, Feagin JA,. Quadriceps contusion in young athletes. *J Bone Joint Surg.* 1973;55-A:95-105

Jarvinen, M. The Effect of early mobilization and immobilization on the healing process following muscle injuries. *Sports Med* 1993;15:78-89

Korberg C, Lew P, The effect of stretching neural structures on grade one hamstring injuries. *J Orthop Sports Phys Ther*. 1989;10:481-487

Koulouris G, Connell D, Evaluation of the hamstring muscle complex following acute injury, Journal of the International Skeletal Society A Journal of Radiology, Pathology and Orthopedics, Skeletal Radiology, August 2003

Kujala U, Orava S, Järvinen, Hamstring Injuries – Current Trends in Treatment and Prevention, *Sports Med* 1997;23:401

MacAuley DC. Ice therapy: how good is the evidence? *Int J Sport Med.* 2001;22:379-384

Magnusson SP, Passive properties of human skeletal muscle during stretch maneuvers. *Scand J Med Sci Sports.* 1998;8:73

Sawyer PC, Uhl TL, Mattacola CG, Jognson DL, Yates JW. Effects of moist heat on hamstring flexibility and muscle temperature, *J Strength Cond Res*. 2003;17:285-290

Thornson O, Hemdal B, Lilija B et al. The effect of external pressure on intramuscular blood flow at rest and after running. *Med Sci Sport Exerc* 1987;19:469-473

Turl SE, George KP. Adverse neural tension: a factor in repetitive hamstring strain? *J Orthop Sports Phys Ther*. 1998;27:16-21.

Verrall GM, Slavotinek JP, Barnes PG, Fon GT, Spriggins AJ. Clinical risk factors for hamstring muscle strain injury: a prospective study with correlation of injury by magnetic resonance imaging. *Br J Sports Med*. 2001;35:435-9. Discussion 440

Webright WG, Randolph BJ, Perrin DH. Comparison of nonballistic active knee extension in neural slump position and static stretch techniques on hamstring flexibility. *J Orthop Sports Phys Ther*. 1997;26:7-13

# Hip Pain

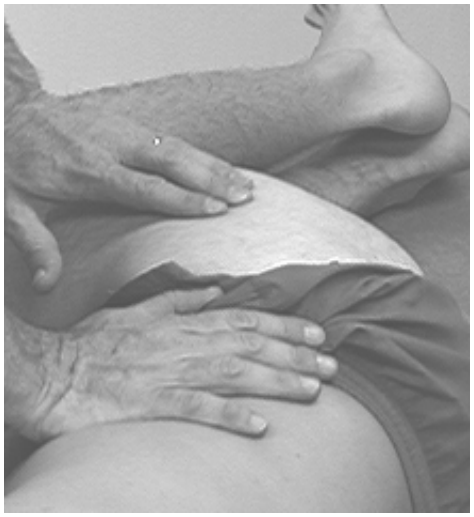
*ICD-9-CM code*: 726.5 Enthesopathy of hip region (enthesopathies are disorders of peripheral ligaments or muscular attachments)

*ICF codes*: Activities and Participation Domain codes:
**d4150** Maintaining a lying position (Staying in a lying position for some time as required, such as remaining in a prone position in a bed.)
Body Structure code: **s75001** Hip joint
Body Functions code: **b28016** Pain in joints

*Common Historical Findings*:
Local pain in lateral buttock area - worsens with laying on affected side

*Common Impairment Findings* - Related to the Reported Activity Limitation or Participation Restrictions:
Symptoms reproduced with palpation over posterior, superior aspect of greater trochanter

*Physical Examination Procedures*:



Trochanteric Bursa Palpation/Provocation

Performance Cues:
Utilize femoral internal and external rotation to assist in localizing trochanter
*Remember - symptom reproduction means reproducing the patient's specific pain complaint*

Hip Pain: Description, Etiology, Stages, and Intervention Strategies

The below description is consistent with descriptions of clinical patterns associated with "**Trochanteric Bursitis.**"

*Description:* Enthesopathy is a general term for disorders of peripheral ligaments or muscular attachments. Thus, trochanteric bursitis is an inflammation of the trochanteric bursa. Anatomically, there are three bursal sacs, associated with the greater trochanter of the femur. The largest is the subgluteus maxiumus bursa and the two smaller are the subgluteus medius and minor bursa (named for their location under each muscle).

*Etiology:* Trochanteric bursitis can be caused by unaccustomed pressure or repetitive microtrauma the trochanteric bursa. The condition is usually insidious but can be sudden if associated with a blow or fall on the affected hip. Potential causes of friction are a tight iliotibial band, tensor fascia lata, or gluteal muscles rubbing over the greater trochanter. Lying on the involved hip on an unusually firm mattress or hard surface for an extended period of time can also cause symptoms. Trochanteric bursitis occurs in all age groups but is most common between the fourth and sixth decades of life. Frequently it occurs in conjunction with arthritis, hip disease and with individuals demonstrating abnormal gait patterns. It is more common in women than men, potentially associated with the predisposition for females to have an increased Q angle, femoral anteversion, or generalized joint laxity. Aggravating activities are activities that require repetitive hip motion and frequent single leg stance such as running, and stair ambulation.

Acute Stage / Severe Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b28016.3** SEVERE pain in joints

- Pain and symptom reproduction with provocatory palpation of the involved trochanteric bursa
- Pain with passive external rotation and abduction and resisted abduction
- Hip abductors may have flexibility deficits – including a positive Ober's sign
- Hip abductors and hip extensors may have strength deficits
- Positive Patrick's - FABER (Hip flexion abduction, external rotation combined movement) test
- Trochanter bursitis often co-exists with gluteal medius muscle strain (tendinopathy)

Sub Acute Stage / Moderate Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b28016.2** MODERATE pain in joints

As above with the following differences:

- The bursa is less provocative with palpation
- Compensatory gait patterns may be exhibited or developing

Settled Stage / Mild Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b28016.1** MILD pain in joints

As above – except the impairments are now minimal

Intervention Approaches / Strategies

Acute Stage / Severe Condition

Goal: Reduce inflammation of the bursa

- Physical Agents
  - Ice packs or ice massage
  - Ultrasound
  - Phonophoresis

- Re-injury Prevention Instruction:
  - Reduce aggravating activities
  - Softer mattress

Sub Acute Stage / Moderate Condition

Goals: As above
Improve hip range of motion and strength

- Approaches / Strategies listed above

- Manual Therapy
  - Attempt to restore normal mobility or symmetry to any impairments of the pelvic girdle or lumbar spine that may be associated with the presenting trochanteric bursitis symptoms

- Therapeutic Exercises
  - Stretching and exercises for muscles with flexibility deficits
  - Strengthening exercises for muscles with strength deficits

Settled Stage / Mild Condition

Goals: As above
Normalize hip range of motion and strength

- Approaches / Strategies listed above

- Therapeutic Exercises
  - Add coordination exercises if any muscle balance deficits are revealed.

Intervention for High Performance / High Demand Function in Workers or Athletes

Goal: Return to occupation or sport

- Approaches / Strategies listed above
- Evaluate and make changes to the training/work program
- Evaluate shoe gear and equipment use
- Add activity specific proprioceptive training
- Patient education/ergonomic instruction

Selected References

Arromdee E; Matteson E. Bursitis: common condition, uncommon challenge. *Musculoskeletal Medicine.* April; 2001. 213-224.

Boyle K. Jansa S. Lauseng C. Lewis C. Management of a woman diagnosed with trochanteric bursitis with the use of a Protronics Neuromuscular System. *Journal of the Section on Woman's Health* 2003;27:(1)12-16.

Browning K. Hip and pelvis injuries in runners. *Physician and Sports Medicine*. 2001;29:23-34.

Fredericson M. Bergman G. A comprehensive review of running injuries. *Critical Reviews in Physical and Rehabilitation Medicine.* 1999;11:1-34.

Haslock R. Trochanteric bursitis-a frequent cause of 'hip' pain in RA: *Ann Rheum Dis.* 1982;41:602-603.

Kisner C; Colby A: *Therapeutic Exercise: Foundations and Techniques 3rd ed*. Philadelphia: F.A. Davis. 1996.

Traycoff RB. Pseudotrochanteric bursitis: the differential diagnosis of lateral hip pain. *J Rheumatology.* 1991;18:1810-1812.

# Hip and Related Lower Extremity Radiating Pain

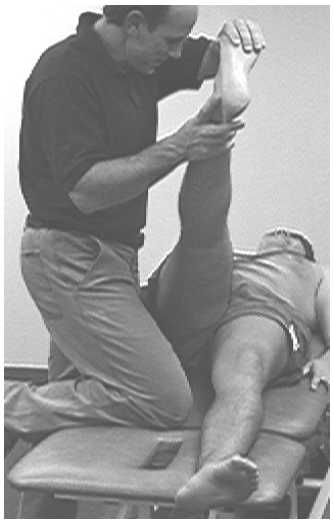
ICD-9-CM codes: 355.0 Lesion of sciatic nerve

*ICF codes*: Activities and Participation Domain codes:
**d4153** Maintaining a sitting position (Staying in a seated position, on a seat or the floor, for some time as required, such as when sitting at a desk or table.)
Body Structure code: **s7408** Structure of pelvic region, other specified
**s7508** Structure of lower extremity, other specified
Body Functions code: **b28015** Pain in lower limb
**b2804** Radiating pain in a segment or region

*Common Historical Findings*
Line of pain in buttock, posterior thigh, and calf; aching in buttock
Symptoms worsen with driving and sitting on involved buttock ("wallet sign")

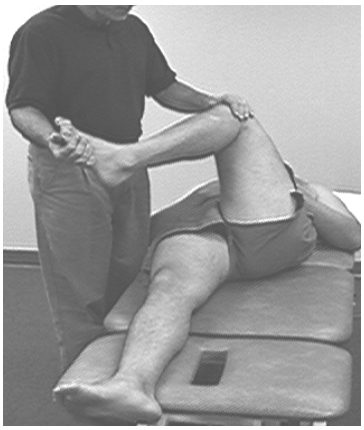
*Common Impairment Findings* - Related to the Reported Activity Limitation or Participation Restrictions:
Symptoms are reproduced with SLR and hip adduction - altered with ankle dorsiflexion and plantar flexion
Symptoms are reproduced with piriformis stretch and palpation/provocation of piriformis

*Physical Examination Procedures*:



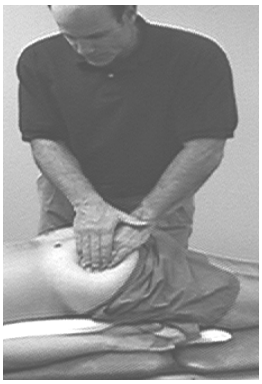
SLR / Hip Adduction

Performance Cues:
Raise leg to first sense of resistance- utilize a combination of additional hip flexion and hip adduction in an attempt to reproduce the reported symptoms - alter the symptoms with ankle dorsiflexion and plantar flexion



Piriformis Stretch

Performance Cues:
Stand on opposite side of involved hip
Flex hip to approximately 90° - fully externally rotate hip - horizontally adduct and flex the hip (at about a 45° angle) simultaneously



Piriformis Palpation/Provocation

Performance Cues:
Stand opposite side of involved hip
Piriformis runs from the anterior part of the sacrum, through the sciatic notch, to the superior aspect of the greater trochanter
Palpate through gluteus maximus on the outer 1/3 of a line from the PSIS to the trochanter
A normal piriformis will be indistinguishable from the surrounding myofascia
Abnormal piriformis hypertonicity feels like a “sausage” and is painful
Careful to not have a false negative as a result of not “sinking” deep enough through the gluteus maximus and surrounding tissues

# Hip and Related Lower Extremity Radiating Pain: Description, Etiology, Stages, and Intervention Strategies

The below description is consistent with descriptions of clinical patterns associated with the vernacular term "**Piriformis Syndrome**"

*Description*: Contracture or spasm of the piriformis muscle causing compression of the sciatic nerve resulting in neuritis of one or both branches of the sciatic nerve. The patient may experience buttock pain with or without lower extremity pain and paresthesias extending to, but rarely below, the knee; as well as tenderness to palpation over the piriformis muscle.

*Etiology:*
Postural Factors: Prolonged or frequent single limb stance, crossed-leg sitting, sitting on one foot, prolonged hip external rotation – such as sleeping with one lower extremity in external rotation, unilateral coxa vara, pronated foot.
Trauma: Femoral neck fracture, sudden starts/stops – such as in playing tennis; direct trauma to the piriformis muscle.
Surgery: May present following a total hip arthroplasty
Diseases: Arthritis, tuberculosis, bone tumors may cause piriformis muscle contractures

Acute Stage / Severe Condition: Physical Examinations Findings (Key Impairments)
*ICF Body Functions codes*: **b28015** SEVERE pain in lower limb

- The reported symptoms are reproduced with a stretch to the piriformis muscle (i.e., hip horizontal adduction, flexion (above $60^{o}$), and external rotation – or with – hip horizontal adduction, flexion (below $60^{o}$) and internal rotation, aka as FAIR)
- The reported symptoms are also reproduced with lower limb tension tests
  Example of lower limb tension tests include:
  SLR with ankle dorsiflexion
  Freiberg's test (passive internal rotation of the hip with the thigh extended in prone)
  Lasegue's sign (hip and knee flexed to 90 degrees in supine and the knee is then extended until pain is reported or knee is fully extended)
- The reported symptoms are also reproduced with provocatory palpation of the piriformis muscle - typically palpated lateral to the sciatic notch to the greater trochanter
- The reported symptoms may also be reproduced with resisted tests to the piriformis muscle, such as the Pace test (resisted active abduction of thighs in seated position)
- The patient may present with any or all of the following biomechanical abnormalities: ipsilateral shortened lower extremity, anterior rotation of sacrum in the contralateral oblique axis (deep ipsilateral sacral sulcus), excessive pronation of ipsilateral foot, in supine- the involved lower extremity may rest in excessive external rotation
- Activities that stretch the piriformis and the sciatic nerve (e.g., exiting our of a car) aggravate the symptoms
- The patient may present with dyspareunia or rectal pain with bowel movements

Sub Acute / Moderate Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b28015** MODERATE pain in lower limb

As Above – except:

- Activities that stretch the piriformis and the sciatic nerve only intermittently aggravate the symptoms

Settled Stage / Mild Condition: Physical Examinations Findings (Key Impairments)

*ICF Body Functions codes*: **b28015** MILD pain in lower limb

As Above – except:

- Activities that stretch the piriformis and the sciatic nerve only aggravate the symptoms after prolonged end-range positions

## Intervention Approaches / Strategies

Acute Stage / Severe Condition

Goals: Decrease pain
Increase hip rotation range of motion

- Manual Therapy
  - Soft tissue mobilization using very slow, inhibitory pressure to the piriformis muscle – followed by gentle contract/relax, manual passive stretching
  - Soft tissue mobilization to areas or potential entrapment of the sciatic nerve of the posterior thigh

- Therapeutic Exercises
  - Instructions in gentle piriformis stretch.
  - Nerve mobility exercises for the sciatic nerve

- Re-injury Prevention Instruction:
  - Modification or avoidance of activities/positions that are aggravating symptoms, such as prolonged sitting with an overstuffed wallet in the back pocket of the involved buttock

Sub Acute Stage / Moderate Condition

Goals: Increase flexibility of piriformis muscle to normalize hip rotation range of motion
Normalize mobility of the sciatic nerve
Normalize pelvic girdle and lower extremity lower extremity biomechanics

- Approaches / Strategies listed above

- Manual Therapy
  Procedures to restore normal pelvic girdle symmetry

- Therapeutic Exercises
  Provide stretching exercises for relevant pelvis and lower extremity tight musculature – typically the hip external rotators and calf muscles
  Provide strengthening exercises for relevant pelvis lower extremity weak musculature – typically the back extensor and hip flexor muscles and hip abductor muscles

- External Devices (Taping/Splinting/Orthotics)
  Foot orthotics may be useful to correct excessive pronation

Settled Stage / Mild Condition

Goal: Return to desired activity or occupation

- Approaches / Strategies listed above

Intervention for High Performance / High Demand in workers/ Athletes

Goals: Return to desired sport or occupation

- Approaches / Strategies listed above

Selected References

Barton PM. Piriformis syndrome: a rational approach to management. *Pain.* 1991;47:345-52.

Beatty RA. The piriformis muscle syndrome: a simple diagnostic maneuver. *Neurosurgery.* 1994;34:512-4.

Benzon HT, Katz JA, Benzon HA, et al. Piriformis syndrome: anatomic considerations, a new injection technique, and a review of the literature. *Anesthesiology.* 2003; 98:1442-1448.

Broadhurst N. Piriformis syndrome and buttock pain. *Australian Family Physician.* 1990;19:1754.

Douglas S. Sciatic pain and piriformis syndrome. *The Nurse Practitioner.* 1997;22:166-180.

Durrani Z, Winnie AP. Piriformis muscle syndrome: an under-diagnosed cause of sciatica. *Journal of Pain Symptom Management*. 1991;6:374-379.

Fishman LM, Dombi GW, Michaelsen C, et al. Piriformis syndrome: diagnosis, treatment, and outcome – a 10-year study. *Arch Phys Med Rehabil*. 2002;83:295-301.

Fishman LM, Konnoth C, Rozner, B. Botulinum neurotoxin type B and physical therapy in the treatment of piriformis syndrome: a dose-finding study. *Am J Phys Med Rehabil*. 2004;83:42-50.

Hallin RP. Sciatic pain and the piriformis muscle. *Postgrad Med*. 1983;74:69-72.

Pace JB, Nagle D. Piriformis syndrome. *The Western Journal of Medicine* 1976;124:435-439.

Retzlaff E, Berry AH, Haight AS, Parente PA, Lichty HA, Turner DM, Yezbick AA, Lapcevic JS, Nowland DJ. The piriformis muscle syndrome. *Journal of American Osteopathic Association*. 1974;73:799-807.

Steiner C, Staubs C, Ganon M, Buhlinger C. Piriformis syndrome: pathognesis, diagnosis and treatment. *Journal of American Osteopathic Association*. 1987;87:318-323.

TePoorten BA. The piriformis muscle. *Journal of American Osteopathic Association* 1969;69:150-60.

**SUMMARY OF HIP DIAGNOSTIC CRITERIA AND PT MANAGEMENT STRATEGIES**

| DISORDER | HISTORY | PHYSICAL EXAM | PT MANAGEMENT |
|---|---|---|---|
| **"Capsulitis"**<br>843.1 onov* = 8 or less<br>mnov** = 16 | Groin ache | ROM deficits<br>End range pain | Rx myofascial ROM deficits<br>(STM, C/R, Ther Ex's)<br>Rx joint ROM deficits<br>(Mob, MWM, Ther Ex's) |
| **"Piriformis Syndrome"**<br>355.0 onov = 4 or less<br>mnov = 12 | Buttock and sciatic pain<br>Sx's increase w/sitting on buttock | SR w/: LLTT,<br>Piriformis stretch,<br>Piriformis palpation | Rx innominate and sacral dysfunctions<br>Rx entrapment<br>(Inhibit Piriformis, Nerve ROM) |
| **"Muscle Strain"**<br>843.8 onov = 4 or less<br>mnov = 8 | "Pulled Muscle" or contusion<br>ADL's which stretch or contract the involved muscle are painful | SR w/: Stretch, contraction, or palpation of the involved myofascia | Allow healing<br>Later, STM and stretching to prevent adhesions |
| **"Trochanteric Bursitis"**<br>726.5 onov = 4 or less<br>mnov = 8 | Lateral hip ache<br>Sx's increase w/lying on the affected side | SR w/palpation or provocation of the Trochanteric Bursa | Patient education<br>(Softer mattress, reduce aggravating activity)<br>Physical agents<br>(US/Phono) |

# Proximal Hip Fracture
# Open Reduction/Internal Fixation and Rehabilitation

Surgical indications and Considerations

*Anatomical Considerations*: The hip is a ball and socket joint with the femoral head aligned towards the pelvic acetabulum. Stability of this joint is achieved by the surrounding ligaments and musculature that attach the pelvis to the femur. The medial and lateral femoral circumflex arteries supply the femoral neck and head with a majority of its blood supply. Fractures of the femoral head pose a threat to the vascular supply of this structure. However, displaced fracture of the femoral neck can occur with no disruption to the blood supply.

*Pathogenesis:* Femoral neck fractures occur more often in the elderly population secondary to a fall or twisting injury. It is estimated that femoral neck fractures account for approximately one half of the hip fractures in the elderly. These fractures also have a higher incidence in women possibly secondary to osteoporosis being the most significant contributing factor in this type of injury. Femoral neck fractures in the young population (usually in their third or fourth decade) are primarily a result of a high impact, high energy trauma.

*Epidemiology:* Contributing factors that increase the risk of sustaining a proximal hip fracture include, osteoporosis, malnutrition, decreased physical activity, impaired vision, neurological diseases, poor balance, altered reflexes, and muscle atrophy. The number of hip fractures in the United States has doubled from the mid-1960s through the 1980s with 250,000 cases per year. By 2005 it is expected that this number will triple secondary to the increasing number of geriatric patients.

Fractures of the proximal femur are classified by anatomical location. The most common types are:
Femoral neck fracture
Intertrochanteric fracture
Subtrochanteric fracture (typically seen in younger patients secondary to traumatic injuries -
accounting for less than 5 percent of cases in elderly patients)

*Diagnosis:* Diagnosis of hip fracture is often uncomplicated, especially when the bone fragments are displaced

- Radiographs should be ordered for any elderly patient who complains of even mild hip pain after a fall.
  *Note: Nondisplaced femoral neck fractures and fractures of just the acetabulum might not be visible on plain films so further test such as a bone scan or an MRI may be required.*
- Exquisite pain upon weight bearing
- Patient typically present with the fractured leg held in external rotation (frog position)

- The patient's leg may be shortened if the fracture is displaced.
- Key question: Why did the patient fall? Underlying factors may include an underlying infection, myocardial ischemia, or gastrointestinal bleeding, altered medications as well as the contributing factors mentioned above (osteoporosis, malnutrition, decreased physical activity, impaired vision, neurological diseases, poor balance, altered reflexes, and muscle atrophy).

*Nonoperative versus Operative Management:* There are some cases in which nonoperative management is the treatment of choice. Individuals who are non-ambulatory or who are medically unstable may opt for nonoperative management such as traction just long enough to allow for early healing. Traction is followed by bed to chair mobilization. If a nonoperative approach is chosen, patients may have to wait for sufficient bone healing which may take up to 16 weeks before ambulating.

*Surgical Procedure:* It is critical that the open reduction internal fixation (ORIF) be performed as soon after the injury as possible, preferably within 24 to 48 hours in order to prevent disruption on the vulnerable blood supply. Femoral neck pinning consists of placing multiple screws across the fractured femoral neck. The most commonly used technique is a lateral approach, while a percutaneous technique may also be used. The type of procedure is dependent on the fracture and includes the following:

- For nondisplaced or impacted femoral neck fractures an *in situ* fixation with multiple parallel screws or pins is used
- For stable intertrochanteric fractures a dynamic extramedullary fixation with a sliding hip screw and a lateral side plate is used allowing for compression along the fracture site during early weight bearing
- For subtrochanteric fractures a static interlocking intermedullary nail fixation or a sliding hip screw combined with an intermedullary nail is used

There are many types of screws available for this procedure but typically the orthopedic surgeon will use a solid or cannulated screw made or either stainless steel or titanium. In order to minimize postoperative complications, it is recommended that the screws be placed accordingly:

- The pins should be placed within the central two thirds of the femoral head
- The central screw should be placed with the tip of the screw no closer than 5 mm to the subchondral bone
- One inferior screw should be placed along the medial femoral neck
- The inferior screw should exit at or above the lesser trochanter
- Two proximal screws should be placed in a triangular configuration anteriorly and posteriorly (if the screws are placed to close together the strength of the fixation is compromised)

*Preoperative Rehabilitation:* Preoperative rehabilitation is not particularly relevant for patients who have sustained a proximal hip fracture and are status post an ORIF procedure, simply because these fractures are mainly secondary to a trauma such as a fall. Surgery is indicated during the first 24 to 48 hours after the injury in order to prevent any possible or further disruption to the vascular supply. Prior to the surgery certain precautions may be taken to

prevent further injury such as:

- Adequate medication and proper positioning to minimize the patient's pain
- Non-weight bearing status and instructions on the appropriate assistive devise (most likely a front wheel walker for safety considering the majority of proximal hip fracture patient are older adults)
- Instructions/review of post-operative rehabilitation plan

## POSTOPERATIVE REHABILITATION:

Stages of Fracture Healing:

1. Impact
2. Induction
3. Inflammation
4. Soft tissue callus (chondroid)
5. Hard tissue callus (osteoblastic)
6. Remodeling

Possible Signs of Failure of the ORIF:

- Sever groin thigh or knee pain that increased with movement or weight bearing
- Shortening of the involved limb that was not present immediately after surgery
- Constant external rotation of the involved limb
- Positive Trendelenburg sign with weight bearing that does not resolve or significantly decrease with strength training

**Phase I:** Acute Stage/Immediately Postoperatively 1-2 weeks

Goals: Prevent wound complications
Decrease Acute symptoms (control pain and edema)
NWB precautions
Teach self management – especially transfer skills and avoid movements that stress the fracture
or surgery site
Improvement of ambulation skills – using assistive devices as indicated
Facilitation and neuromuscular control of stabilizing muscles (core stability)
Safely perform ADL's
Prevention of DVT's
Prevention of hip flexor contractures

Intervention:

- Ankle pumps
- PROM and AAROM of affected limb

- Passive stretching
- Practice correct posture with transfers and gait training with NWB precautions to prevent development of a faulty hip posture
- Assistive devise training
- Transfer training
- Gait training
- Progress sitting tolerance
- Progress standing tolerance
- Progress walking tolerance with weight-bearing precautions and assistive device

**Phase II:** Subacute Stage: 2 – 6 weeks post-op

Goals: Increase mobility
Increase neuromuscular control on postural muscles
Increase strength and neuromuscular control of affected limb
Learn safe body mechanics
Increase functional skills
Progress weightbearing precautions to touchdown weightbearing and partial weightbearing
Progress walking distance with appropriate assistive devise and weight bearing precaution

Intervention:

- Begin weight bearing activities while following weight bearing precautions
- Progress ambulation to uneven surfaces
- Progress AAROM to AROM of affected lower extremity (no SLR)
- Progress therapeutic exercises by increasing difficulty and /or repetitions preferably at parallel bars
- Functional exercises while focusing on postural control, endurance and time (stairs)
- Progress gait distance while decreasing level of assistance

**Phase III:** Chronic Stage: 6 – 12 weeks post-op

Goals: Increase mobility of hip joint
Increase ambulatory and functional independence with and without assistive devise
Increase dynamic control
Increase cardiovascular endurance
Return to prior level activities
Develop a healthy exercise

Intervention:

- Increase aerobic time and intensity once WBAT
- Balance training

- Ambulation on difficult terrain with decreased level of assistance
- Neuromuscular reeducation for gait sequencing

Selected References:

Schmidt A, MD. Femoral Neck Fractures. http://www.medscape.com/viewprogram/250_pnt

Sherrington C, Lord SR PhD, Herbert RD PhD. A randomized controlled trial of weight-bearing versus non-weight-bearing exercise for improving physical ability after usual care for hip fracture. *Arch Phys Med Rehabil*. 2004;85:710-716.

Penrod JD, Boockvar KS, Litke A, Magaziner J, Hannan EL, Halm EA, Silberzweig SB, Sean Morrison R, Orosz GM, Koval KJ, Siu AL. Physical therapy and mobility 2 and 6 months after hip fracture. *J Am Geriatr Soc*.2004;52,

Egol EA, Koval KJ. Treatment of Ipsilateral Femoral Neck and Shaft Fractures. *Medscape General Medicine*.1999; 1(3)

Linville DA II, MD. Osteoporotic fractures. *Southern Medical Journal*.2002;95(6):588-592.

Ackermann RJ. Medical consultation for the elderly patient with hip fracture. *J Am Board of Family Practice*.1998;11:366-377.

Kisner C, Colby LA. *Therapeutic Exercise Foundations and Techniques*. 4th ed. Philadelphia, F.A. Davis, 2002.

Mohammad MI. Osteoperosis: epidemiology, diagnosis, and treatment. *South Med J*.2000;93:2-18.

# Total Hip Arthoplasty

Surgical Indications and Considerations

*Anatomical Considerations*: The hip joint is a multiaxial ball and socket joint that has maximum stability because of the deep insertion of the head of the femur into the acetabulum. The acetabulum is formed by fusion of part of the ilium, ischium, and pubis. The acetabulum faces anteriorly, laterally, and caudally. Three strong ligaments support the hip: the iliofemoral, the ischiofemoral, and the pubofemoral ligaments. The tensor fascia lata, the gluteus maximus, and the thick condensation of fascia known as the iliotibial band form the outer layer of the muscular envelope. One of these must be split to gain access to the hip joint. Beneath this outer layer, the gluteus medius and minimus muscles and their insertion into the greater trochanter and joint capsule become a focal point of surgical exposure. The nerves of surgical importance during hip arthroplasty include the lateral femoral cutaneous nerve, femoral nerve, superior and inferior gluteal nerves, and sciatic nerve.

*Pathogenesis*: Total hip arthroplasty (THA) is performed most commonly because of progressively severe arthritis in the hip joint. The most common type of arthritis leading to THA is osteoarthritis (degenerative joint disease) of the hip joint. This type of arthritis is generally seen with aging, congenital abnormality of the hip joint, or prior trauma to the hip joint. Other conditions leading to THA include bony fractures of the hip joint, rheumatoid arthritis, avascular necrosis, and the abnormal muscle tone caused by cerebral palsy. The progressively intense chronic pain together with impairment of daily function including walking, climbing stairs and even arising from a sitting position, eventually become reasons to consider THA.

*Epidemiology*: Total hip arthroplasty is a relatively common procedure with more than 100,000 performed annually in the United States. Although most of the procedures are performed in patients older than 65 years, at least 25% are performed in patients younger than 55. The majority of these people have tried and failed to find relief from their symptoms with conservative medical intervention.

*Diagnosis*

- Patients may limp to reduce the forces on the hip
- Decreased hip range of motion
- Severe pain with activity and at rest that is not relieved by conservative intervention
- Radiographs to determine extent of the degenerative process
- MRI scanning to determine avascular necrosis
- Blood tests to rule out systemic arthritis or infection in the hip

*Nonoperative Versus Operative Management*: Total hip replacement (THR) is used to correct intractable damage resulting in osteoarthritis, rheumatoid arthritis, avascular necrosis, and abnormal muscle tone. Nonelective THR procedures are performed for fractures in which open reduction internal fixation is deemed inappropriate. Contraindications for THR surgery include inadequate bone mass, inadequate periarticular support, serious medical risk factors, signs of infection, lack of patient motivation to observe precautions and follow through with

rehabilitation, and if it is unlikely to increase the patient's functional level. The most common complications following THR surgery include thrombophlebitis (DVT), infection, dislocation, and loosening of the joint. Not all hip conditions require a hip replacement as the initial treatment. There are several alternative treatments to put off replacing the hip as long as possible. Physical therapy, walking aids, medications, injections and activity modification can be used to alleviate pain and improve impairments associated with hip disorders.

*Surgical Procedure*: There are two main types of hip replacements, cemented and noncemented. The type of implant used depends on the surgeon's preference, the individual's age, and activity level. Each implant is made up of two parts: the acetabular component, or socket portion, which replaces the acetabulum; and the femoral component, or stem portion, which replaces the femoral head. A cemented implant is held in place by a type of epoxy cement that attaches the metal to the bone. A noncemented implant has a fine mesh of holes on the surface area that touches the bone. The mesh allows the bone to grow into the mesh and become part of the bone. Many surgeons believe that noncemented femoral components should not have weight borne on them for 6 weeks, whereas cemented femoral components can support weight immediately after surgery. There are various surgical approaches for THA including anterior, anterolateral, posterior, lateral, and combined methods. The posterior approach is probably the most commonly used approaches for THA primarily because it avoids displacement of the abductor mechanism. The posterolateral approach requires precaution instructions that prohibit flexion of the hip past 90 degrees, adduction past the body's midline, and internal rotation of the hip. After an anterolateral THR the patient should observe these precautions and avoid external rotation (especially with flexion). Minimally invasive techniques and mini-incision THR have been pursued by various surgeons to decrease the perioperative complications associated with larger and more extensile approaches and to speed the recovery of patients after THR.

*Preoperative Rehabilitation*

- Educate patient regarding total hip replacement precautions:
  - Do not flex or bend the hip more than 90 degrees
  - Do not twist or pivot on the operated leg
  - Keep legs apart and do not cross them at the knees or ankles
- Help patient become independent in exercise for postoperative phases.
- Instruction in use of assistive devices such as walker and crutches according to the weight-bearing status after a noncemented THR depending on the surgeon's discretion.

## POSTOPERATIVE REHABILITATION

Note: The following rehabilitation progression is a summary of the guidelines provided by Pratt and Gray. Refer to their publication to obtain further information regarding criteria to progress from one phase to the next, anticipated impairments and functional limitations, interventions, goals, and rationales.

**Phase I** Hospital Phase: Days 1-2

Goals: Prevent complications – especially dislocation
Increase muscle contraction and control of involved leg
Help patient sit for 30 minutes

Intervention:

Day1

- Adjust abduction pillow
- Provide patient education regarding total hip precautions
- Begin exercises including: ankle pumps, quadriceps sets, gluteal sets, and upper extremity exercises
- Encourage use of cough and incentive spirometer

Day 2

- Progress exercise program to include heel slides, isometric or active assistive hip abduction and short arc quadriceps sets
- Bed mobility training
- Transfer training
- Gait training as appropriate (using a front-wheeled walker or crutches)

**Phase II**: Days 3-7

Goals: Promote transfers and gait independence
Continue to reinforce THR precautions
Discharge to home

Intervention:

- Continue interventions from phase I with progression of activity as tolerated
- Active range of motion with hip abduction, terminal knee extension, and upper extremity exercises
- Bed mobility training
- Transfer training; initiate car transfers when appropriate
- Gait training; initiate stair training when indicated ("up with good, down with bad")
- Evaluation of equipment needs at home
- Caregiver training

**Phase III** Return to Home (Home Care Phase): Weeks 1-6

Goals: Increase patient independence with gait and transfers
Evaluate safety of home
Plan return of patient to work or previous activities as indicated

Intervention:

- Closed-chain exercises such as heel raises and mini squats
- Cautious stretching of Achilles tendons in the standing position

- Progress from the use of a front-wheeled walker or crutches to single-point cane, this usually occurs 3-4 weeks after surgery (Use of cane is often discontinued after 3-4 more weeks)
- Normalize gait on level and sloped surfaces, jagged sidewalks, curbs, and stairs
- Car transfer instruction and practice

Note: Physical therapy intervention usually ends with the home care phase. Phase IV is for patients with physically demanding lifestyles, patients who have lingering gait problems or did not meet home care status requirements at the time of hospital discharge.

**Phase IV** Outpatient Clinic

Goals: Improve strength, endurance, and balance
Correct gait impairments
Independence with home exercise program

Intervention:
- Strength, endurance and balance training
- Pool exercise
- Stationary bicycling, simulated cross-country skiing, and treadmill (as part of gym program)

Selected References:

Berger RA, Jacobs JJ, Meneghini RM, Della Valle C, Paprosky W, Rosenberg AG. Rapid rehabilitation and recovery with minimally invasive total hip arthroplasty. *Clin Orthop*. 2004;429:239-47.

Bertin KC, Rottinger H. Anterolateral mini-incision hip replacement surgery: a modified Watson-Jones approach. *Clin Orthop*. 2004;429:248-55.

de Pablo P, Losina E, Phillips CB, Fossel AH, Mahomed N, Lingard EA, N Katz J. Determinants of discharge destination following elective total hip replacement. *Arthritis Rheum*. 2004;15:1009-17.

Hanssen AD. Anatomy and Surgical Approaches. In Morrey BF, ed., *Joint Replacement Arthroplasty*. Philadelphia, Churchill Livingstone, 2003.

Jan M-H, Hung J-Y, Lin JC-H, Wang S-F, Liu T-K, Tang P-F. Effects of a home program on strength, walking speed, and function after total hip replacement. *Arch Phys Med Rehabil* 2004;85:1943-51.

Jolles BM, Bogoch ER. Surgical approach for total hip arthroplasty: direct lateral or posterior? J Rheumatol. 2004 Sep;31(9):1790-6.

Pratt E, Gray P. Total Hip Replacement. In Maxey L, Magnusson J, eds., *Rehabilitation for the Postsurgical Orthopedic Patient*. St. Louis, Mosby, 2001.

# Heterotopic Ossification of the Hip

*Pathogenesis*: The basic defect in HO is inappropriate and rapid metaplastic osteogenesis which results in the formation of lamellar corticospongiosal bone. However, both the specific pathophysiology and etiology of HO remain unclear. What induces precursor cells to go through metaplasia and initiate osteogenic activity is still unknown. Similarly the identity of these precursor cells has not been established. Further, it is not known why heterotopic ossification does not materialize in all cases with similar conditions. It seems, though, that there are three requisite conditions for HO to occur. In addition to an osteogenic precursor cell, there must be an inducing agent and a tissue environment that is permissive of osteogenesis.

It appears that non-circulating pluripotent mesenchymal cells are most likely the cells which differentiate to become osteoblastic stem cells. This occurs very soon after injury, within 16 hours after experimentally induced trauma to mice femurs. This suggests that significant pathophysiological events may be occurring in the immediate post-traumatic period, while patients are still receiving acute care.

Two possible candidates which act as inducing agents have been established. Through *in vitro* research it has been established that demineralized bone matrix can induce new bone formation via an osteoinductive protein released from the matrix, now known as bone morphogenic protein (BMP). Additionally, prostaglandins have been implicated as inducing agents.

Certain tissues (spleen, liver and kidney) suppress bone induction while others (muscle and fascia) permit experimental induction of bone. Local factors present within the permissive tissues may further predispose them to HO. These are hypoproteinemia, venous thrombosis or hemostasis, (local) infection, decubitus ulcers and (micro) trauma. Any of these factors may lead to tissue damage and subsequent inflammatory reaction, resulting in edema and tissue hypoxia, further disposing the tissues to heterotopic bone formation. Immobilization can further tissue hypoxia, also contributing to the onset of osteogenesis.

Additional theories of pathogenesis have been put forth to explain neurogenic HO (NH0) as it is known to occur in regions of the body without obvious soft tissue damage, e.g. after spinal cord injury. One theory suggests that damage to the intermedio-lateral sympathetic columns of the traumatized spinal cord might predispose to NHO through autonomic dysregulation. Secondary to an altered balance within the autonomic nervous system, a diversity of metabolic and vascular changes may occur.

The causative agent of heterotopic bone formation has been debated in the literature since the late 1800s. Results from several recent case studies suggest that vigorous passive exercises increase the incidence of HO, but also that a longer time interval between the injury and the beginning of well-dosed passive movement exercises, especially in neurological cases, enhances the risk of HO. These results have also been supported by animal studies. Moreover, biological, biochemical, and biomechanical investigations have established that (micro)trauma and mechanical stress to the musculotendinous apparatus may arise either from vigorous passive exercises or from loss of mobility and muscle imbalance causing peak pressure on soft tissue areas. Mechanical stress causes local microtrauma that may induce ossification either indirectly through an inflammatory response or directly by releasing osteoblast-stimulating factors. In cases of HO accompanied by SCI, the condition for ossification might be further established by the highly “vascular” state of the paralyzed area and the high likelihood of concomitant use of anticoagulants, which may predispose to hematoma and secondary NHO, particularly during the

rehabilitation phase after SCI. Current speculation is that (forced) passive movements following a period of immobilization may easily result in shear and tear of soft tissues leading to an increased risk of developing NHO.

In summary, the common features in all the conditions in which HO is seen are immobilization due to trauma, surgery, or forced therapeutic rest, followed by mobilization with exercise or spasticity. Whether tissues ossify may depend on a fine balance of osteogenic and osteo-inhibitory influences acting both locally and systemically.

Although the mechanisms underlying the development of HO are still not fully elucidated, numerous risk factors have been identified. These can be grouped according to three parameters: patient-related, injury-related, and treatment-related factors. Patient-related factors include male gender (although this may be more a factor of the higher incidence of SCI occurring in the male population), a history of HO after previous hip surgery, bilateral hip disease, hypertrophic osteoarthritis with massive acetabular osteophytes, poor preoperative range of motion at the hip in total hip replacement, fever at the time of surgery, and the presence of other bone-forming disorders such as diffuse idiopathic skeletal hyperostosis, ankylosing spondylitis, and Paget disease. Incidence of HO has no relationship to racial group.

Injury-related risk factors include trauma and full-thickness burns. Treatment-related factors include revision arthoplasty, tissue hypoxia, circulatory stasis, postoperative immobilization with limitation of joint movement, bone demineralization from prolonged bed rest or assisted ventilation, postoperative dislocation of a hip prosthesis in the first week after surgery, postoperative fever lasting more than 5 days, and postoperative hematoma.

Finally, there are specific risk factors for NHO with spinal cord or brain injury. In NHO accompanied by spinal cord injury four risk factors have been identified, including age (older than 30 years), completeness of injury, presence of spasticity, and coexistence of pressure ulcers. These risk factors appear to be additive. The greater the number of risk factors, the higher the incidence of the disorder. Also, HO is less likely to occur in patients whose neurological level of lesion is in the lumbar region. In traumatic brain injury heterotopic ossification seems to be related to the severity of the injury, coma lasting greater than 2 weeks, proximity of fracture to long bones and significant spasticity. HO is rarely seen in flaccid limbs.

*Epidemiology*: Occurrence rates of HO vary depending on the associated injury. In patients with spinal cord injuries, HO is found in 20-30% of cases; however, limitation of joint motion occurs in 18-35% of these cases. Following closed-head injuries the incidence of HO is 10-20% with approximately 10% of these patients demonstrating severe restriction of joint motion (not limited just to the hip). Lower rates are reported among CVA patients. In the traumatic brain injured and the spinal cord injured populations, studies have shown that in those diagnosed with HO, 10% to 16% progress to complete joint ankylosis. After total hip replacement, HO has a reported incidence of 8-90%, although it is clinically significant in only 2-7%. Following internal fixation of acetabular fractures, up to 50% of patients develop HO and in nearly 50% of these cases not treated prophylactically, the HO causes disablement.

Isolated HO can occur at any age but is rare in very young children. Posttraumatic HO is, not surprisingly, most common in young athletic persons. Neurogenic HO is less common in children than among adults and although the clinical symptoms in children are similar to those reported in adults, spontaneous regression of neurogenic HO is more often reported in children and young adults than in older adults.

*Diagnosis:* As stated earlier, secondary to early induction of osteoblastic stem cells, the initial pathophysiological events setting up the possibility of heterotopic bone formation may occur in the immediate post-trauma period. However, the onset of actual heterotopic bone formation usually occurs approximately 2 weeks after injury, although symptoms may not be noted for 8 to 10 weeks. HO is primarily diagnosed based on clinical signs and likelihood. Symptoms similar to those in trauma, inflammation, or tumor occur in the early stages and include peri-articular swelling, erythema, localized tenderness and increased temperature. Additionally, sudden decrease in range of motion is the most common finding and is often the earliest sign of HO. Fever and malaise may also be present. Pain may or may not be present, depending on whether sensation is intact. In patients with NHO, limb spasticity may be enhanced. These early symptoms must be differentiated from arthritis, thrombophlebitis, DVT, cellulitis, soft-tissue hematoma, complex regional pain syndrome and soft-tissue tumor. Thrombophlebitis or DVT may be the most difficult differential diagnosis. Upon visual inspection, the edema of thrombophlebitis/DVT is less localized and more generalized in the lower limbs than in HO. A definitive differentiation can only be made, however, through the use phlebography or ultrasound, especially during the early stages and when it is too early to find radiological evidence of HO.

Laboratory results may show a transient depression in the serum calcium level 1 week after initial insult and an acute elevation in the serum alkaline phosphatase (SAP) level in as little as 2 weeks after insult and on average 7 weeks before the first clinical signs of HO become apparent. Elevated SAP presumably reflects intense osteoblast activity in the local lesions of heterotopic ossification and is associated with clinically significant heterotopic ossification. SAP consists of a series of iso-enzymes which are sensitive, but non-specific indicators of HO. When new bone is actively deposited, the SAP levels are elevated. As soon as the ossification process has stopped, the enzyme levels return to normal. Since SAP levels are non-specific in that they are found in various tissues, they only become strong indicators of HO when they are associated with the clinical signs previously described.

Three-phase 99m Technetium bone scan is the current “gold standard” for earliest detection of HO. In the early stages of HO, the bone formation mainly consists of osteoid that shows a high uptake of osteotropic radionuclides which is readily detectable by bone-scanning. The first phase of the three-phase scan is the period immediately after the intravenous injection of the radionuclides and detects areas of increased blood flow, which is an early indicator of an inflammatory process (the dynamic blood flow phase). The second phase (the static blood pool phase) identifies areas of blood pooling several minutes after the injection. The third phase (the static bone phase) determines the degree of osseous uptake of the labeled radionuclides several hours after the injection. The first two phases of the bone scan are able to detect HO as early as 2.4 weeks after injury and are followed by a positive static bone phase 1-4 weeks later. Compared to plain radiography, bone scanning is a more sensitive diagnostic test for early HO, but radiography is more specific. A disadvantage of the bone scan is its low specificity leading to potential difficulty in discriminating HO from other inflammatory, traumatic, or degenerative processes of the skeleton, e.g. fracture, bone tumor, metastasis, or osteomyelitis which all show increased osteoblastic activity.

Ultrasonography is gaining ground as a preferred initial imaging modality for patients with clinically suspected HO and for differentiating HO from DVT, a developing pressure sore, infection, or tumor. The unique pathological evolution of the ossification can be monitored by sequential sonographic assessment. Although ultrasound can be highly specific and sensitive for

diagnosing HO, these benefits depend strongly on the experience of the radiologist.

Tissue diagnosis by biopsy is not done because of the risk of exacerbating the disorder by resection of an immature lesion. Similarly, CT scanning and MRI are rarely used in the early detection of HO but can be of great benefit in developing a plan for surgical resection of HO.

*Surgical Procedure*: Surgical resection of HO is often a difficult procedure and is considered only in refractory cases not satisfactorily relieved with conservative methods. Indications for surgery include joint immobility causing difficulty in patient positioning, hygiene, ambulation or daily activities; ankylosed joints resulting in pressure sores or skin breakdown; reduction in spasms; and conditions in which HO contributes to peripheral neuropathy.

Traditional thought is that the surgery must be delayed until the bone scan ratio is at steady state and serum alkaline phosphatase (SAP) returns to a normal level, which is usually 12-18 months after injury. More recently, several investigators have published good results of early wedge resection of HO that has not reached maturity. Wedge resection of HO is recommended in lieu of wide resection as the latter often leads to extensive bleeding, wound infection, prolonged immobility and in rare instances necessitates hip disarticulation or higher level amputations. Other risks associated with resection include fracture, ankylosis, pressure ulceration and painful arthrosis or pseudarthrosis. These complications are not uncommon and carry high morbidity.

Success of wedge resection appears to be somewhat dependent on location of the HO. Anterior bone resection usually meets with excellent results for improving hip ROM. Medial bone resection also provides good results with ROM. Early percutaneous or open adductor myotomy and obturator neurectomy are useful in maintaining hip abduction. The posterior bone formation about the hip is the most serious. Since hip flexion contractures are almost always present, resection of posterior ectopic bone may not always be helpful. In this case, a proximal femur extension osteotomy is performed to obtain more range.

A controversial alternative to surgical resection is forceful manipulation under anesthesia. It has been found to be particularly useful in HO with brain injury. Its efficacy for enhancing range of motion versus aggravating and contributing to the HO process is still being studied. It is performed as the heterotopic bone is growing to maintain range and to prevent ankylosis. Repeated manipulations may be warranted if neurologic status improves. If it does not, joint ankylosis may be inevitable. Joint manipulation may also be desirable to place the affected joint in a more functional position when ankylosis is inevitable. Postmanipulation care includes range of motion, use of a continuous passive motion (CPM) machine and 24-hour positioning regimens.

*Preoperative Rehabilitation*: Since surgical intervention is chosen only in refractory cases, rehabilitation consists of efforts to avoid surgery and is considered as a first line treatment modality. Rehabilitation efforts have included monitoring patients for signs suggestive of HO, administering range-of-motion exercise, intervention in seating systems and bed positioning, turning, the use of physical agents, early mobilization of patients, and patient and/or family education.

Through daily hands-on interventions with patients at risk for HO, physical therapists may be the first to become suspicious of the possible onset of HO. In fact, in a retrospective study of 1209 patients with SCI, it was primarily physical therapists that were first to suspect the onset of HO. Physical therapists should observe at-risk patients for decreased range of motion

and for the other symptoms suggestive of HO previously mentioned: peri-articular swelling including the distal leg in HO at the hip, erythema, warmth, localized tenderness, pain and increased spasticity. Early observation of these signs may assist in the early diagnosis of HO, thereby increasing the ability to manage it pharmacologically, without eventual surgical intervention.

There still is no conclusive evidence based on random controlled studies in humans regarding the appropriateness of range of motion exercises to prevent HO. In fact, most recently, van Kuijk and Silver emphasize the power of clinical observation in determining the benefit of range-of-motion exercises to prevent HO, despite the paucity of evidence-based support. From these observations they make strongly stated recommendations that early range-of-motion exercises are beneficial in the prevention and treatment of HO. Some have even called range-of-motion exercises "the mainstay of HO prevention." These exercises are believed to work by inhibiting fibroblast activation and, thereby, preventing muscle contractures. When gentle and cautious passive movements of the large peripheral joints are started and maintained from the day of spinal cord injury, it is believed that the joint capsules are kept as supple as possible, muscles will not easily shorten and contractures will not readily develop, so that HO may be prevented. On the other hand, if a paralyzed extremity is not moved immediately after onset of injury, exactly the reverse occurs. Joint capsules, muscles, ligaments and fascia shrink and adhesions develop between various soft tissue compartments. Once these changes have occurred, subsequent passive movements result in shear and tear of the soft tissue and if this is large enough, it triggers an abnormal process of repair.

Once heterotopic bone begins to form, the role of physical therapy for HO management remains even more controversial. Secondary to the inflammatory component of HO, many argue that the therapist should not attempt to mobilize the involved joint directly during the active stage of HO, as this might cause further inflammation or trauma and so increase the amount of bone formation. Based on clinical evidence, Chantraine and Minaire recommend that mobilization of those with SCI should be replaced by placing patients in alternating positions of $90^{o}$ of the hips and knees and that the position be changed every three hours to maintain sufficient range of motion at both joints for functional independence. Once the diagnosis of traumatic heterotopic ossification becomes definitive, it is recommended that active range of motion exercise be continued within the pain-free range.

With regular turning and correct positioning, spasticity is greatly reduced and the development of pressure sores prevented. Thus two significant, known risk factors can be eliminated if an appropriate turning and positioning program is carefully followed. Prone positioning relieves pressure from the most common sites of decubitus ulcer formation and diminishes the risk of flexion contractures of the hips, thus reducing the likelihood of HO developing on their anterior aspect. It can also help to inhibit extensor spasticity.. Sitting the brain-injured patient out of bed regularly may also help to reduce spasticity. From extensive rehabilitation experience, Davies asserts that it is extremely rare to find HO in brain-injured patients who have been sat out of bed while still in coma and stood with the help of knee-extension splints.

For optimal reduction of spasticity and prevention of decubitus formation, the patient should be turned at least every two hours. When working with comatose patients, at least two caregivers are required to turn adult patients in order to eliminate all danger of traumatizing the limbs. Turning such a patient alone may easily result in a limb being pulled into an extreme range of motion resulting in minor trauma which is possibly a contributory factor in HO. When

the patient is being turned, the limbs should be flexed in order to shorten the lever arms and then gradually eased into the corrected position once in side-lying.

Finally, the importance of early mobilization in a wheelchair cannot be stressed enough. In the case of a complete neurological lesion when no recovery of lost function can be expected, patients should be allowed out of bed as soon as the pain at the site of spinal fracture and the condition of concomitant injuries no longer preclude mobilization. In those with incomplete spinal cord lesions, mobilization should begin as soon as the danger of increase in collapse of a crushed vertebral body or of dislocation is over. Early wheelchair mobility decreases the duration of immobilization which is believed to be a primary risk factor of HO.

Chantraine and Minaire report that various physical agents have been tried, including ultrasound, iontophoresis, and cryotherapy, all as adjunctive therapy against inflammation. However, they give no evidence of the efficacy of these modalities.

Once hip range of motion becomes limited and an increase in spasticity secondary to neurogenic HO develops, the patient's positioning may be compromised. The physical therapist should evaluate and recommend changes to seating (if the patient mobilizes in a wheelchair) and to positioning in bed (for bedridden patients) to prevent the risk of pressure sores and related pain complaints. The decreased range of motion at one hip causes body weight to be unequally distributed between the ischial tubera, making it possible for pressure ulcers to evolve usually on the side contralateral to the hip affected by neurogenic HO.

Finally, patients at risk for HO need to be educated about the possible complication of HO and its indicators. If the condition develops, patients need to be informed thoroughly about the condition and the various means of treatment. A safe passive or active-assisted ROM program needs to be presented to the patient and family members to prevent loss of motion, contractures, and possible loss of function. Family members or other caregivers must be educated to avoid overly aggressive ranging of the patient's limbs in order to minimize the possible risk of initiating HO.

## POSTOPERATIVE REHABILITATION

Little mention is made of post-surgical care in the literature other than the administration of disodium etidronate and prophylactic antibiotics given pre-operatively and post-operatively for approximately 7 days or NSAIDs and/or radiotherapy. These therapies are warranted after surgery, as surgically resected HO has a high rate of recurrence. Post-operative radiation has been reported to decrease the recurrence of HO after surgical resection in SCI patients, but no controlled studies are available. Both radiation and indomethacin seem to be effective in preventing HO occurrence after total hip replacement.

When mention is made of rehabilitation, however, there is little agreement about when it should be initiated and what it should include. Furthermore, evidence-based research regarding post-surgical rehabilitaton of HO at the hip is non-existent.

In one surgical case study of HO at the hip with concomitant SCI, the physician advocated an intensive rehabilitation program post-operatively to prevent recurrence of the ossification by unopposed spasticity of the patient's hip extensors. No description was given, however, of the interventions comprised by the recommended rehabilitation program.

In another series of hip resections performed on patients with paraplegia, strict timing

and cautious use of movement exercises were employed. No passive movements were allowed during the first 14 days following resection. From day 15 forward, continuous passive motion was used to achieve suitable flexion for wheelchair mobility ($70^{o}$ to $90^{o}$). Passive motion exercises were continued once patients were mobilizing themselves in wheelchairs.

One author advocates for ROM exercises to begin as early as 48 to 72 hours after surgery in patients with HO associated with burns. Another suggests ROM exercises should begin one week after surgical resection of NHO associated with traumatic brain injury. Neither cites evidence-based research to support these recommendations.

According to Anderson with regards to surgically resected HO with brain injury, passive ROM and CPM should also be initiated 3 to 5 days postoperatively to keep the joint surfaces lubricated. When the patient is cleared by the surgeon for weight-bearing activity, motor re-education and strengthening in function should begin. Commonly, functional hip ROM is obtained after surgical resection; however, it is not uncommon for the patient to revert back to compensatory postures and movements after the bone has been resected. Motor re-education and functional strengthening are critical so that the patient may become more efficient with movement and function. Anderson also states that 24-hour positioning is important for maintaining hip range of motion. Although Anderson provides the most detailed guidelines for post-operative care, no evidence-based research is provided to support these recommendations.

Selected References:

Lal S. Heterotopic ossification. In: Green D, ed. *Medical Management of Long-Term Disability*. Boston:Butterworth-Heinemann, 1996:95-113.

van Kuijk AA, Geurts ACH, van Kuppevelt HJM. Neurogenic heterotopic ossification in spinal cord injury. *Spinal Cord*, 2002;40:313-326.

Damanski M. Heterotopic ossification in paraplegia: a clinical study. *The Journal of Bone and Joint Surgery*, 1961;43 B:286-299.

Singer, BR. Heterotopic ossification. *British Journal of Hospital Medicine*. 1993; 49:247-255.

Riklin C, Baumberger M, Wick L, et al. Deep vein thrombosis and heterotopic ossification in spinal cord injury: a 3 year experience at the Swiss Paraplegic Centre Nottwil. *Spinal Cord*. 2003;41:192-198.

Crawford CM, Varghese G, Mani MM, Neff JR. Heterotopic ossification: are range of motion exercises contraindicated? *J Burn Care Rehabil.* 1986;7:323-327.

Michelsson JE, Granroth G, Andersson LC. Myositis ossificans following forcible manipulation of the leg. *Journal of Bone and Joint Surgery*. 1980; 5:811-815.

Izumi K. Study of ectopic bone formation in experimental spinal cord injured rabbits. *Paraplegia*. 1983;21:351-363.

Smoecx M, De Muynck M, Van Laere M. Association between muscle trauma and heterotopic

ossification in spinal cord injured patients: reflections on their causal relationship and the diagnostic value of Ultrasonography. *Paraplegia*. 1995;33:464-468.

van Kuijk AA, Geurts ACH, van Kuppevelt HJM. Letter to the editor. *Spinal Cord*.2003;41:423-424.

Schafer SJ, Schafer LO, Anglen JO, Childers M. Heterotopic ossification in rehabilitation patients who have had internal fixation of acetabular fracture. *Journal of Rehabilitation Research and Development*. 2000; 37: 389-393.

Goldman J. Heterotopic ossification in spinal cord injuries. *Physiotherapy*.1980; 66:219-220.

Anderson D. Management of decreased ROM from overactive musculature of heterotopic ossification. In: Montgomery J, ed., *Physical Therapy for Traumatic Brain Injury*. New York: Churchill Livingstone, 1995:79-97.

Blount PJ, Bockenek WL. Heterotopic ossification. In: Frontera WR, Silver JK eds., *Essentials of Physical Medicine and Rehabilitation*. Philadelphia: Hanley & Belfus, Inc., 2002:569-574.

Garland DE. Heterotopic ossification in traumatic brain injury. In: Ashley MJ and Krych DK, eds. *Traumatic Brain Injury Rehabilitation,* Boca Raton: CRC Press, 1995:119-129.

Guo Y, CollacoCR, Bruera E. Heterotopic ossification in critical illness and cancer: a report of 2 cases. *Arch Phys Med Rehabil*. 2002;83:855-859.

van Kuijk AA, van Kuppevelt HJM, van der Schaaf DB. Osteonecrosis after treatment of heterotopic ossification in spinal cord injury with the combination of surgery, irradiation, and an NSAID. *Spinal Cord*. 2000;38:319-24.

Freebourn TM, Barbert DB, Able AC. The treatment of immature heterotopic ossification in spinal cord injury with combination surgery, radiation therapy and NSAID. *Spinal Cord*. 1999;37:50-53.

Jamil F, Subbarao JV, Banaovaac K, et al. Management of immature heterotopic ossification (HO) of the hip. *Spinal Cord*. 2002;40:388-395.

Garland DE, Razza B, Waters RL. Forceful joint manipulation in head-injured adults with heterotopic ossification. *Clinic Orthop.* 1982;169:133-8.

Silver, J. Letter to the editor. *Spinal Cord*.2003; 41:421-422.

Ellerin BE, Helfet D, Parikh S, et al. Current therapy in the management of heterotopic ossification of the elbow: a review with case studies. *Am J Phys Med Rehabil.* 1999;78:259-271.

Davies PM. *Starting Again: Early Rehabilitation after Traumatic Brain Injury or Other Severe Brain Lesion*. Berlin: Springer-Verlag, 1998: 361-381.

Chantraine A, Minaire P. Para-osteo-arthropathies. *Scand J Rehab Med*. 1981;13:31-37.

Meiners T, Abel R, Bohm V, et al. Resection of heterotopic ossification of the hip in spinal cord injured patients. *Spinal Cord*. 1997;35:43-45.