

SOFT TISSUE RHEUMATISM AND JOINT INJECTION TECHNIQUES FOR FAMILY PHYSICIANS

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Non-articular musculoskeletal pain can arise as a result of tendinitis, bursitis, nerve entrapment syndromes, myofascial pain and fibromyalgia.

It is important to differentiate soft-tissue rheumatism from other causes of pain arising from the joint. The location of pain and inflammation, and presence of pain only on active range of motion may help to differentiate a soft-tissue problem from a joint problem. X-rays may be helpful by revealing joint problems although it must be pointed out that soft tissue problems can be the cause of pain and disability even when there is an underlying joint pathology.

TRIGGER FINGER

Clinical appearance

A trigger finger is the locking of one or several fingers in flexion. The patient may have to pull the finger to straighten it. The cause is cartilaginous metaplasia at the first retaining pulley (at the palmar aspect of the metacarpophalangeal joint) which normally becomes taut during finger flexion. When 3 or more digits are affected, conditions such as diabetes and hypothyroidism should be considered.

Treatment

Spontaneous improvement occurs only in 20% of cases. If untreated, the process can lead to an inability to straighten the finger and permanent contracture. Trigger finger is treated with corticosteroid infiltrations. With appropriate treatment, including up to 3 infiltrations, the success rate is over 95%. Complications, including

iatrogenic infection or tendon rupture, are rare. Surgery may be required for patients who refuse infiltrations or in whom infiltrations fail.

DE QUERVAIN'S TENOSYNOVITIS

Clinical appearance

This condition is characterized by disabling pain in the radial aspect of the wrist at the base of the thumb. There is swelling and tenderness of the common sheath of the abductor pollicis longus and extensor pollicis brevis. De Quervain's tenosynovitis is particularly common in two settings: in mothers who repeatedly lift infants and young children and in people who use their hands in repetitive activities, such as knitting, sewing, and gardening. A positive Finkelstein's test is characteristic of the condition. The correct way to perform this test is to position the hand in the following manner: In the painful hand, (a) the fully flexed thumb rests on the palm (b) the fingers are curled over the thumb and (c) the wrist is very gently deviated to the ulnar side. Acute pain along the tendon is diagnostic of de Quervain's tenosynovitis. Additional findings can include a diffusely swollen and tender sheath or loculated tenosynovial effusions near the radial styloid.

Treatment

Corticosteroid infiltrations are very effective in de Quervain's tenosynovitis, although the success rate is slightly less than in trigger finger. Surgery is indicated in the event of treatment failures.

GANGLIA

Clinical appearance

Ganglia are ubiquitous uni- or multilocular cystic lesions that arise in paratendinous, pararticular, or intraosseous locations. Ganglia can be confidently

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diagnosed in most cases on the basis of location, typical increase in size and symptoms caused by joint use, and palpatory findings (cystic). Aspiration, which must be made with a large-bore needle, yields a thick jelly-like material. Echography, computer tomography (CT) and magnetic resonance imaging (MRI) facilitate the diagnosis of ganglia that occur in deep and atypical locations.

An imaging procedure should be performed whenever there is doubt about the diagnosis. Because of its low cost and excellent resolution, echography is a useful imaging procedure to investigate soft tissue lesions.

Treatment

Surprisingly, little research has been done on ganglia. There are no published controlled trials comparing different forms of treatment. Aspiration followed by injection of a long-acting corticosteroid is an appropriate treatment. Technical details can be found in the accompanying article. Surgery is generally required for lesions greater than 3 cm in diameter, for anterior wrist ganglia, and for intraosseous ganglia, as well as in cases where there is nerve compression.

TENNIS ELBOW

Clinical appearance

Tennis elbow, or lateral epicondylitis, is common in middle-aged people, most of whom are not tennis players. Tennis elbow results from overuse of the extensor carpi radialis brevis, a muscle that spans the lateral epicondyle, and the base of the third metacarpal, a wrist dorsiflexor. The diagnosis is suggested by the lateral location of the pain, which characteristically affects the soft tissues just

distal to the epicondyle, plus pain reproduction by resisted dorsiflexion of the wrist. Passive elbow flexion and extension are normal. Medial epicondylitis (golfer's elbow) represents the mirror image of lateral epicondylitis.

Treatment

Tennis elbow resolves spontaneously with time and rest of the affected arm. Patients should be given isometric and range of motion exercises for the entire upper extremity, including the shoulder, elbow, wrist, and fingers. Exercises should not create prolonged (more than two hours) post-exercise pain. In tennis players, alteration of technique and revision of equipment may be essential for good long-term results. Medical therapy includes topical and systemic analgesics. Nonsteroidal anti-inflammatory drugs (NSAIDs) can be used often, just prior to the offending exercise. Corticosteroid infiltrations are frequently used in tennis elbow, and at least 50% of patients find immediate relief. However, recurrence is seen in 30% of these patients. About 10% of patients have chronic symptoms despite medical treatment, and repeated corticosteroid injections have been implicated in chronicity. Therefore, more than one injection is ill advised.

SHOULDER PAIN

Particular emphasis should be placed on determining whether paresthesias are present. The common shoulder pain syndromes include rotator cuff tendinitis, subacromial impingement, frozen shoulder and acromioclavicular joint disease.

Rotator cuff tendinitis

Inflammation of the common tendon of the

subscapularis, supraspinatus, infraspinatus, and teres minor may result from microcrystalline deposits (apatites), overuse, impingement on the tendon from above or below, or degenerative changes that occur with aging. Specific shoulder motions are painful, particularly abduction when combined with rotations. Range of passive motion is typically normal. Calcific rotator cuff tendinitis occurs when a pre-existing calcium deposit undergoes resorption, resulting in acute inflammation, extreme pain and loss of shoulder motion.

Subacromial impingement

The rotator cuff tendon, which courses through a narrow space between the acromion and the proximal humerus, may be impinged upon from above or below. Increased tendon bulk acts as a tight-fitting wedge between the two bony boundaries. Impingement symptoms are largely due to the tendinitis. In some cases, there are also symptoms related to the condition that causes the impingement, such as tenderness at an osteoarthritic acromial-claricular joint.

Frozen shoulder

It is important to remember that acromial-claricular frozen shoulder may occur in diabetic fibrosis, paraneoplastic syndrome and occasionally in scleroderma. Frozen shoulder should be distinguished from synovitis such as that seen in rheumatoid arthritis and ankylosing spondylitis, reduced motion caused by inactivity, rotator cuff tendinitis, and posttraumatic and postsurgical capsular retraction with limited joint motion. Because the joint capsule encircles the joint, passive and active shoulder motions are equally lost in all directions.

Differential diagnosis

The single feature that best assists in the diagnosis of shoulder pain is its location. The pain may be located at the top, side, front, back, or axillary sides of the joint. Lateral pain is characteristic of rotator cuff or glenohumeral disease. Superior pain suggest acromial-claricular or sternoclavicular conditions. Anterior pain may be caused by bicipital tendinitis and early frozen shoulder. Posterior pain can be caused by tears in labrum glenoidale and suprascapular neuropathy, and axillary pain of various neural causes. An important maneuver in

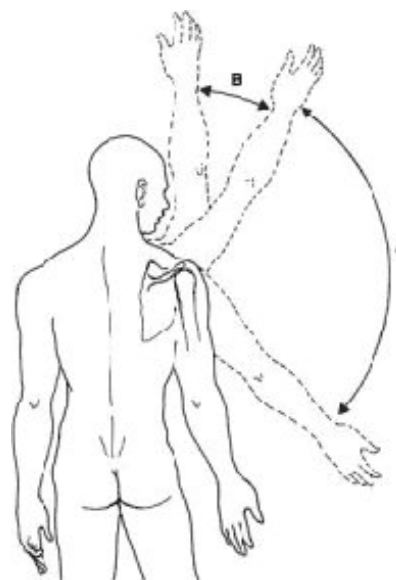


Figure 1: The arc of elevation maneuver. Because the greater tuberosity of the humerus has to clear under the acromion/coracoacromial ligament during abduction, patients with subacromial impingement hurt during arc A. Once the greater tuberosity has cleared, pain ceases in arc B. A similar phenomenon occurs as the arm is brought down: no pain in arc B, pain in arc A, then no pain in full dependency. Because the acromioclavicular (AC) joint has its greatest motion in terminal elevation, patients with AC arthritis hurt in arc B. Finally, when the AC joint causes impingement, e.g., in osteoarthritis, there will be pain in arc A from impingement, plus pain in arc B from stress on the diseased joint.

establishing the presence of rotator cuff tendinitis and acromial-claricular joint arthropathy is the arc of elevation test (Figure 1).

Treatment

Physical therapy

Broad physical therapy principles apply to shoulder pain treatment. Shoulder physical therapy should begin with pendular exercises during the acute and subacute phases, followed by stretching and strengthening exercises later in the course of the disease. Heating the area with warm packs (or ultrasound) is particularly helpful prior to exercising because it facilitates stretching and provides analgesia.

Medications

Nonsteroidal anti-inflammatory drugs are helpful in rotator cuff tendinitis, including cases of impingement and calcific tendinitis. Corticosteroid infiltrations is a very useful treatment in rotator cuff tendinitis, the initial phases of frozen shoulder, and AC osteoarthritis.

Surgery

Surgery can relieve structural subacromial impingement, which is generally resistant to physical and anti-inflammatory therapy.

HIP PAIN

Clinical appearance

Pain in the hip region may originate in tendinous, bursal, articular, osseous structures or other soft tissues. It may also be due to radiculopathy or peripheral nerve injury, or be referred from elsewhere. The initial assessment should be based on the actual location of pain. Never take for granted that "hip pain" actually originates in the

hip joint! To the presenting compliant "My hip hurts," ask "Where in the hip?" The patient may point to the anterior, the lateral, or the posterior aspect of the joint.

Differential diagnosis

Anterior pain

Pain originating in the hip joint is experienced anteriorly, mainly in the groin and anteromedial thigh. Hip joint disease is usually associated with limitation of motion and endpoint pain. Additional causes of anterior hip pain include iliopsoas tendinitis, capsular stretching and iliopsoas bursitis. Both tendinitis and capsular stretching feature pain on traction, whereas tendinitis also causes pain on resisted motion.

Lateral pain

Diagnosis of lateral hip pain may be difficult. Hip arthritis seldom cause lateral pain alone. Trochanteric bursitis affects the bursa between the fascia lata and the greater trochanter. Bursal inflammation of the bursae underlying the gluteus medius or minimus has also been described. Radiating pain from the lumbar spine is dull and is associated with lumbar/gluteal pain. Neuropathies affecting the subcostal, iliohypogastric and lateral cutaneous nerve of the thigh (meralgia paresthetica) cause lateral pain, paresthesias, and hypoesthesia in the corresponding territories near the iliac crest, the area just below it, and the lateral thigh respectively. Bone pathology in the femoral neck, such as a bone insufficiency fracture, and occasionally aseptic necrosis of the femoral head may cause predominantly trochanteric pain. Also, soft tissue or bone malignancy may affect the trochanteric region, and their presence should be suspected whenever a bulge is felt. Radiographic studies are

negative in routine cases of trochanteric bursitis but are very helpful in defining bone lesions. Echography and MRI effectively detect soft tissue tumors and trochanteric bursitis. Bone scans can detect intraosseous or intraarticular disease.

Posterior pain

Posterior hip pain is less common than anterior and lateral pain. Pain may originate in the lumbar spine, occasionally in the sacroiliac joint, and in some cases in the hip joint, although hip joint disease almost always has coexistent anterior pain. Pain that originates in the ischial tuberosity (ischial bursitis) is aggravated by sitting.

Treatment

The treatment of trochanteric pain caused by trochanteric bursitis involves identification of underlying factors, such as a 2.5 cm leg length discrepancy in favor of the affected side plus ipsilateral iliotibial band contracture, and is key to a successful long-term treatment. In such a patient, a 1.25 cm heel lift should be provided for the short limb, and the patient should be referred to physical therapy for instruction on iliotibial band stretching exercises. Corticosteroid infiltration is an effective mode of therapy. Patients refractory to medical therapy should be evaluated by an orthopedist. A longitudinal split of the iliotibial band, which relieves pressure on the underlying trochanteric bursae, often alleviates the pain.

KNEE PAIN

Clinical appearance

Knee pain may be intrinsic or it may radiate from a proximal structure such as the hip or the lumbar plexus. If knee motion is free and painless while the patient is lying on the contralateral side, knee

disease can be safely ruled out. Knee examination should include inspection, range of passive motion, patellofemoral function, stressing of the medial and lateral compartments, and an assessment of joint stability. Inspection may reveal swelling at the suprapatellar pouch and parapatellar gutters indicative of a synovial effusion, prepatellar swelling from prepatellar bursitis, and patellar tendon region swelling caused by pretendinous bursitis, tendon inflammation, fat pad hyperplasia/inflammation, or infrapatellar bursitis. Swelling at the medial or lateral articular lines usually represents a meniscal cyst. A bulge in the popliteal fossa may be caused by a Baker's cyst, but other soft tissue lesions should be excluded.

Palpation is used to confirm or refute the above findings, because fat and synovial proliferation can cause bulges around the knee joint. Identification of focal tenderness may be critical for diagnosis. Classic findings include tenderness at the medial or lateral articular lines in meniscal disease; patellar or tibial insertion of the patellar tendon in calcific enthesopathy and patellar tendon enthesitis, respectively; tibial insertion of the pes anserinus in the anserine bursitis syndrome; and lateral femoral condyle in patients with the iliotibial band syndrome. Valgus and varus stress while flexing and extending the joint will identify unicompartamental abnormalities, such as lateral and medial meniscal lesions. The Lachman test, which can be described as a drawer sign elicited by pulling forward the upper tibia with the knee flexed 20 degrees, is positive in anterior cruciate ligament tears.

Differential diagnosis

Anterior pain

Pain in the anterior knee may be caused by many problems including patellofemoral syndrome

(chondromalacia patella); tender, inflamed medial plica (a vestigial synovial fold that has become swollen by inflammation and fibrosis) which will jump under the examining finger in a medial parapatellar location during flexion and extension movements of the joint; prepatellar bursitis evident as a tender, soft tissue lump in front of the patella. Osgood-Schlatter's disease in pediatric cases results in a tender, hard lump at the tibial tuberosity.

Medial pain

Pain over the medial aspect of the knee is usually caused by a meniscal tear in a younger patient and medial compartment osteoarthritis in patients past the age of 50 years. However, there are other considerations. Anserine bursitis (not truly a bursitis in most instances) hurts on pressure 3-5 cm distal to the medial articular line. Medial collateral ligament bursitis, which overlaps the articular line, hurts more on pressure in flexion when the bursa is exposed than in extension when the sac gets covered by the medial collateral ligament. Meniscal cysts characteristically soften and may become undetectable in full flexion and full extension, while they are hard and prominent when the knee is flexed 30-40 degrees.

Lateral pain

Pain in the lateral knee is seen in the iliotibial band syndrome, a condition that results from the excessive friction of a tight iliotibial band on the lateral femoral condyle. Patients with this syndrome report lateral knee pain while running, going up or downstairs, and bicycling. On examination with the knee in semiflexion, a tender spot is found on the lateral femoral condyle anterior to the band. Tenderness decreases or disappears when the knee is fully extended. Other conditions affecting the lateral knee include

meniscal tears and cysts, as well as the biceps femoris tenosynovitis in which there is tenderness in the posterolateral corner of the joint.

Posterior pain

Pain in the posterior knee may be caused by popliteal (Baker's) cysts, various lesions affecting the popliteal artery, venous thrombosis, hematomas, ganglia, and soft tissue tumors including sarcomas. Foucher's maneuver is very useful in distinguishing popliteal cysts from other mass lesions. When the knee is flexed 30-40 degrees, Baker's cysts become soft or undetectable (a positive Foucher's sign) while other popliteal masses remain unchanged. Echography, with Doppler, is particularly helpful in the analysis of posterior knee pain. Venous disease, popliteal artery aneurysms, solid tumors, and ganglia can be reliably identified by this method.

Treatment

In addition to specific measures for the diagnosed condition, all patients with a painful knee should be instructed on isometric quadriceps exercises. The use of a cane on the opposite side helps relieve pain and provides stability and safety while the condition improves. Depending on the cause of the pain, local or systemic analgesics or NSAIDs may be used. Both warm packs and cold compresses are analgesic, the effect varying with the individual patient. Prepatellar bursitis is treated in a similar fashion as olecranon bursitis, although it is generally more problematic. Septic cases often require hospitalization, catheter or surgical drainage, and parenteral antibiotics for a few days until the process is controlled. At this point, oral antibiotics may be started and continued to complete 2-3 weeks of therapy.

In some of the regional syndromes, one or more corticosteroid infiltrations will eventually be required. Anserine bursitis, medial collateral ligament bursitis, and the iliotibial band syndrome respond particularly well to corticosteroid infiltrations. Technical aspects of this procedure are discussed in the accompanying article. On the other hand, the medial plica syndrome, tight lateral retinaculum, and meniscal pathology including ruptured menisci and meniscal cysts are amenable to arthroscopic surgery. Baker's cysts almost always reflect intraarticular pathology that results in excess synovial fluid production and distention of a pre-existent communicating gastrocnemius-semimembranous bursa. They are best treated by addressing the knee problem that causes the bursal distention. Provided that infection has been ruled out, knee drainage followed by intraarticular corticosteroids is an effective method to reduce a symptomatic Baker's cyst and avert the risk of rupture. Long-term treatment is aimed at the underlying knee process.

POSTERIOR HEEL PAIN

An enlarged superficial bursa can be readily identified superficial to the Achilles tendon, which is best determined while the tendon is tense. Thickening and tenderness may be felt at the insertional or noninsertional Achilles tendon. The presence of nodules at the noninsertional area (in the absence of nodular rheumatoid arthritis and tophaceous gout) suggests partial rupture and is therefore a warning sign for a complete rupture. The extent and severity of the lesion can be fully disclosed by echography or MRI. Insertional tendinitis can

be diagnosed clinically. A lateral x-ray of the heel may show an intrabursal effusion characteristic of retrocalcaneal bursitis.

Treatment

Superficial bursitis requires better shoes in the long run, but NSAIDs may be used temporarily. Noninsertional tendinitis patients should be referred to an orthopedist with broad experience in foot pathology, as tendon debridement or repair may be indicated. Insertional tendinitis in the spondylarthropathies usually responds to systemic treatment. Refractory cases may be treated with a walking cast. Corticosteroid infiltrations in the retrocalcaneal bursa are very effective treatment but care must be taken not to infiltrate the tendon, which could lead to tendon rupture. Ancillary measures for all types of posterior heel pain include the use of heel lifts to decrease traction on the Achilles tendon and gently performed tendon stretching exercises. Also, the use of a night splint that holds the foot at 90 degrees has been recommended for pain relief.

PLANTAR HEEL PAIN

Clinical appearance

The pain is maximal when the patient first stands in the morning and tends to decrease with walking. There are 4 clinical contexts in which the symptom develops. Fat pad failure is seen in obese patients with attrition of the plantar fat pad, in patients with a thin and flabby plantar pad, and as an iatrogenic condition in patients who sustained multiple plantar corticosteroid infiltrations for heel spurs. "Plantar fasciitis" is seen in runners with interstitial fascial rupture, in patients with flat, pronated feet in whom a collapsed longitudinal arch stretches the fascia, and in patients with

spondylarthropathy and enthesitis at the calcaneal insertion. The tarsal tunnel syndrome results in plantar pain and paresthesias. It is due to a pressure neuropathy of the calcaneal branches of the posterior tibial nerve or the first branch of the lateral plantar nerve. Both are more likely to occur in patients with flat, pronated feet from excessive pressure on the medial edge of the heel. Stress fractures of the calcaneus and calcaneal cysts can also cause plantar heel pain.

Treatment

Plantar heel pain should be treated with nonspecific measures, along with treatment aimed at the underlying condition. A heel cup, a firm plastic device that squeezes the plantar fat pad at its edges and increases its thickness, helps most patients. Anti-inflammatory drugs help, particularly in patients with spondylarthropathy. Achilles tendon and plantar fascia stretching exercises are useful once the condition becomes inactive and may help prevent recurrences. Corticosteroid infiltrations can be quite helpful in patients with an inflammatory cause. Because the procedure is technically difficult and very painful, it is generally reserved for those patients who have not benefited from a more conservative program including NSAIDs in full doses for 6 weeks. Additional measures that have been reported to be useful include a night splint that holds the ankle at 90 degrees and dexamethasone iontophoresis. Laser therapy was found ineffective in a well-designed controlled trial. Because spurs do not cause the pain in noninflammatory cases, they should not be surgically removed. Plantar fasciotomy is a salvage procedure for severe, long-standing cases. There is a partial collapse of the longitudinal arch following this procedure.

Injection techniques:

- κ Infection is a very rare complication of corticosteroid infiltrations. Hollander has reported an overall rate of infection of 1/14,000 infiltrations. Sterile technique is recommended.
- κ Tendon rupture following corticosteroid infiltrations may occur from collagenous weakening caused by the glucocorticoid, especially if the preparation is injected directly into the tendon rather than the tendon sheath. Skin atrophy, hypopigmentation, fat pad atrophy, and tendon rupture are generally related to poor technique (too large a volume or dose, incorrect needle placement) with leakage of corticosteroid into the subcutaneous tissue or direct injection into the tendon. Multiple injections (>3) in the same site should be avoided. It is prudent to have a 4-week gap between injections in the same site.
- κ Fainting is uncommon and is related to apprehension and pain! Excessively anxious patients should be injected while lying down even if the target is the upper extremity. A rare, but potentially severe, lethal systemic effect of corticosteroid infiltrations is anaphylaxis (6). Therefore, epinephrine should be at hand when performing an infiltration.
- κ The most common (seen in up to 40% of patients) systemic effect is facial flushing. This is a benign transient symptom that can last for a few hours but may be quite distressing for the uninformed patient.

Shoulder joint injection

- κ Posterior approach: The anterior landmark is the tip of the coracoid process, which can be identified 2 cm medial to the AC joint and 2 cm distal to the clavicle. The posterolateral landmark is 1 cm below and 1 cm medial to

the posterior angle of the acromion. needle is advanced from the posterolateral landmark toward the coracoid. The needle should be felt to slip into the joint space in which it can move about freely.

Subacromial bursa

- κ Entry is posterolateral, aiming the needle anteromedially under the acromion with the tip directed to the undersurface of the acromion.

Trigger finger

- κ The entry should be 5 mm distal to the proximal palmar skin crease for the index, and 5 mm distal to the distal palmar skin for the long and ring fingers. The nodule should be palpable. The needle is then withdrawn 2-3 mm, and the patient is asked to very gently flex and extend the distal phalanx practiced prior to the (this should be procedure) to ascertain lack of tendon engagement.

Trochanteric bursa

- κ First, the tender spot at the posterior corner of the greater trochanter is identified by ascending palpation along the femur. The 1½ inch or longer #21 or #22 needle is inserted vertically down to periosteum. Larger volume of lignocaine and steroid required.

Knee injection

- κ The needle is inserted horizontally under the patella between the midpoint and upper pole. Once the needle is intraarticular, which can be ascertained by aspirating synovial fluid, the corticosteroid or corticosteroid/lidocaine mixture is instilled.

Anserine bursa

- κ Infiltrate the area of tenderness by introducing the needle directly into bursa at the medial aspect of the tibia 5 cm below joint line.

- o **GENERAL RULES:** Dosage of triamcinolone
- o **Large joint: 20-40 mg 1-2 cc lignocaine**
- o **Small joint: 10-20 mg 1/2-1 cc lignocaine**
- o **Tendons/bursae: 5-20 mg 1/2 cc lignocaine**

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