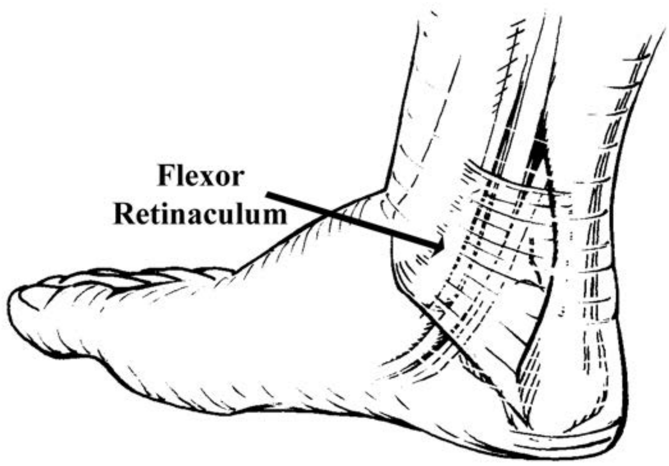


CHAPTER THREE

Peripheral Nerve Entrapment Lower Extremity

03

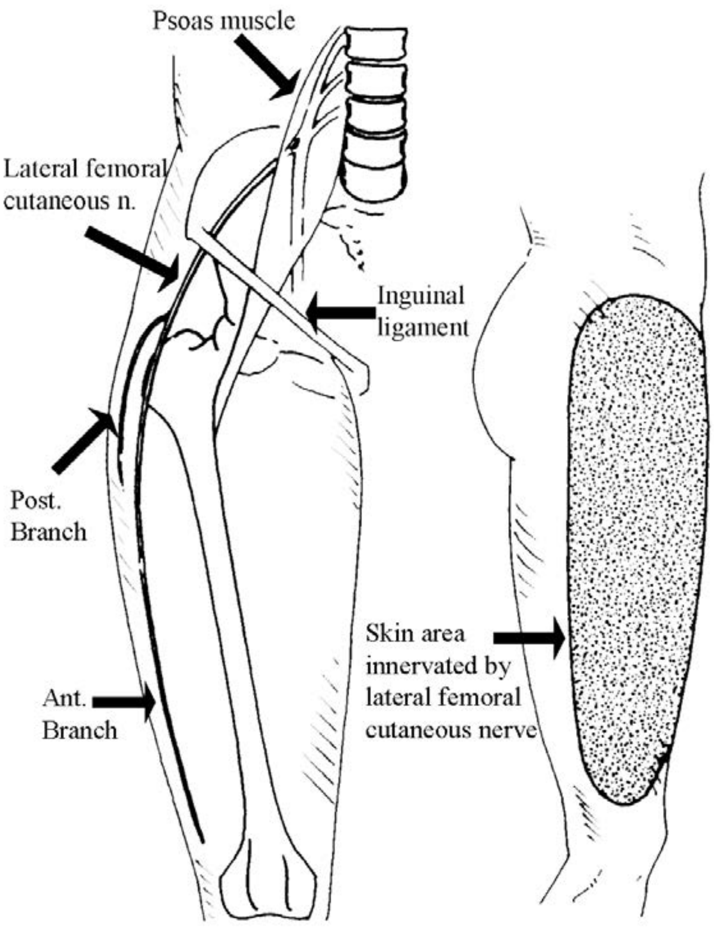
Extraspinal peripheral nerve entrapment of the lower extremity is often misdiagnosed as lumbar or lumbosacral root compression, or a disturbance of the autonomic nervous system. **(Akuthota & Herring, 2009; Russell, 2006; Ruch, 2001; Staal et al., 1999)** Most nerves that go to the extremities have autonomic and afferent fibers, the median and the sciatic nerves particularly. The frequency of radiculopathy and the primary attention that a manipulative physician gives to the spine may preclude consideration of the more distal origins of nerve dysfunction. Further, many physicians — especially those who have been in practice for several years — may not have received adequate education about peripheral nerve entrapment in their initial training.

This chapter emphasizes differential diagnosis of lower extremity peripheral nerve entrapment, with particular attention to the level of involvement and local correction. The condition may be a double crush combination of spinal involvement and a more distal peripheral nerve entrapment. If only the spinal column is corrected, which includes dural tension technique, and the peripheral entrapment is ignored, the patient will improve but the condition will not be completely corrected.

Lateral Femoral Cutaneous Nerve

The lateral femoral cutaneous nerve arises from the 2nd and 3rd lumbar nerves. It is formed in the psoas muscle **(Moore et al., 2009)** and emerges from its lateral border to cross the iliacus and exit the pelvis. The most common point considered for possible entrapment is as the nerve passes between the two slips of the inguinal ligament's lateral attachment to the anterior superior iliac spine, where it exits the pelvis. The nerve is tightly bordered by the tendinous fibers of the inguinal ligament at this point and makes a right-handed bend to change direction from a horizontal course in the pelvis to a more vertical course in the lateral and anterolateral thigh. The lower slip of the inguinal ligament also gives origin to some sartorius fibers. **(Staal et al., 1999; Keegan & Holyoke, 1962)** The nerve may pass in front of or through the sartorius into the thigh. **(Gray's Anatomy, 2004)**

Shortly after it leaves the abdomen, the nerve divides into an anterior and a posterior branch. The anterior branch supplies the skin of the anterior and lateral parts of the thigh to the knee. The posterior branch supplies the skin on the lateral surface of the thigh, from the greater trochanter to about the middle of the thigh. The lateral femoral cutaneous nerve is strictly sensory, giving no motor supply.



Lateral femoral cutaneous nerve.

As the nerve reaches a point just medial to the anterior superior iliac spine where it enters the thigh, it changes its course from nearly horizontal to vertical. This angulation is increased by extension and lessened by flexion of the thigh. In addition to this major point of possible entrapment, other locations may be where the nerve emerges from the psoas muscle, **(Skaggs et al., 2006)** the passage underneath the fascia lata, and the area where the nerve leaves the fascia. **(Staal et al., 1999; Biemond, 1970)** After the lateral femoral cutaneous nerve exits the pelvis at the opening of the inguinal ligament, it is held down as it pierces the fascia lata; thus, movement between the thigh and pelvis can stretch the nerve and increase entrapment at the opening of the inguinal ligament. **(Kopell & Thompson, 1976)**

Entrapment is prone to occur in obesity, with a lax abdominal wall, and in pregnancy. **(Harney & Patijn, 2007; Staal et al., 1999; Stites, 1986)** Diabetics have an increased susceptibility to compression neuropathy, which is especially apparent in this nerve. **(Veves et al., 2002; Staal et al., 1999; Aguayo, 1975)**

Nerve entrapment causes pain called meralgia paresthetica (Barnhardt-Roth syndrome) in the anterolateral thigh. **(Harney & Patijn, 2007; Pearce, 2006)** Interestingly, Sigmund Freud reported that he and one of his sons suffered from the condition. **(Freud, 1895)** The condition affects men more than women due to possible occupational considerations and can also be bilateral in some 25 percent of cases. **(Sunderland, 1978)** It most commonly develops from the nerve's fascial attachment in the thigh pulling the nerve tightly against the opening at the lateral end of the inguinal ligament, **(Staal et al., 1999; Kopell & Thompson, 1976, 1960)** which usually relates to thigh obesity and/or a lateral shift in trunk posture. **(Skaggs et al., 2006; Kopell, 1980)** Generally, meralgia paresthetica develops without prior trauma.

Symptoms consist of increased **(Staal et al., 1999; Aguayo, 1975)** or decreased **(Harney & Patijn, 2007; Kopell & Thompson, 1960)** sensitivity. Rubbing of clothing and other cutaneous stimuli causes a characteristic burning pain on the outer side of the thigh. Pelvic and hip motion, such as walking and running, aggravates the pain. In general, those with meralgia paresthetica have poor posture; consequently, the postural muscles are overactive with prolonged standing, which also aggravates pain. The postural deficiency most common with meralgia paresthetica is lumbar hyperlordosis and a protuberant abdomen. **(Russell, 2006; Skaggs et al., 2006; Massey, 1977; Bellis, 1977)** Applied kinesiology examination usually finds weak abdominal and gluteus maximus muscles failing to provide support to prevent anterior rotation of the pelvis, with subsequent hyperlumbarlordosis. Aggravating the condition will probably be hypertonic lumbar extensor muscles and shortening of the iliopsoas. Sitting and recumbent positions relieve the pain; however, sitting with one leg crossed over the other — especially the ankle on the knee — can exacerbate the pain. Postural shifts by a short leg, whether physiological or anatomical, may be a factor in the condition. Other factors include the muscle fibers from the internal oblique and transverses abdominus muscles originating from the inguinal ligament, or the external oblique muscle inserting into it. **(Keegan & Holyoke, 1962)** Additional causes have been reported, including bodybuilding, **(Szewczyk et al., 1994)** falling asleep in siddha yoga position, **(Mattio et al., 1992)** seat-belt **(Beresford, 1971)** and pocket watch trauma, **(Mack, 1968)** and misplaced injections. **(Ecker & Woltman, 1938)**

Meralgia paresthetica is presented more often to chiropractors than is generally recognized. **(Arcadi, 1996)** In 215 consecutive examinations of patients in a chiropractic office, 12 cases of meralgia paresthetica were diagnosed. The method of diagnosis was "…standard orthopedic and neurologic testing procedures that evaluate the lateral femoral cutaneous nerve territory for superficial tactile sensation, superficial pain, sensitivity to vibration, sensitivity to temperature, and temperature gradient studies." **(Kadel & Godbey, 1983)** The patients had been aware of their condition from two days to 12 years. Five of the 12 had received treatment by the medical profession, with an unsatisfactory diagnosis or results.

The condition should be differentially diagnosed from conditions such as 2nd and 3rd lumbar nerve root compression, appendicitis, spinal cord tumor, colon cancer, and trochanteric or iliopsoas bursitis. **(Staal et al., 1999; Stites, 1986)** Disturbance at the lumbar level is usually associated with diminished or absent patellar reflex and weakness of the quadriceps muscles. Because the lateral femoral cutaneous nerve is purely sensory, there is never weakness or reflex change when disturbance is limited to its entrapment. **(Staal et al., 1999; Aguayo, 1975)** In femoral neuropathy or L2, 3 root lesions, sensory changes usually spread out to extend more anteromedially than in entrapments of the lateral femoral cutaneous nerve where the sensory change is limited to its dermatome. **(Harney & Patijn, 2007; Stites, 1986)**

Staal **(Staal, 1970)** considers meralgia paresthetica rare and suggests that "…one should look carefully for vertebral or disc lesions, intrapelvic anomalies, old operation scars, compression from outside due to clothing or chronic microtrauma: in short for all kinds of local pathology along the course of the nerve, from its beginning until distal to the anterior superior iliac spine. In the majority of the cases no obvious cause will however be found even after thorough investigation." Failure to find a cause for meralgia paresthetica is not consistent with applied kinesiology findings. This may be due to a more functional evaluation of spinal, pelvic, and muscular function with AK methods.

There will usually be pain below and slightly medial to the anterior superior iliac spine, close to the inguinal ligament attachment. To determine the attachment of the inguinal ligament, palpate along the ligament to the bone. A positive indication that meralgia paresthetica is present when deep pressure in this area causes radiation of pain in the skin supplied by the lateral femoral cutaneous nerve. Another source of pain at the anterior superior iliac spine is the origin of the sartorius, especially when there is a category II pelvic involvement. Since skeletal muscular imbalance is often present in meralgia paresthetica, sartorius pain and a category II may be present in combination with lateral femoral cutaneous nerve entrapment. To differentiate sartorius pain, palpate its origin at the anterior superior iliac spine and the upper half of the notch below it. From the sartorius origin, palpate down the sartorius tendon into the muscle, observing for pain; this indicates muscle involvement rather than, or in addition to, nerve entrapment. Usually the muscle will also test weak.

Low back derangement may cause pain in the greater trochanteric region or in the tensor fascia lata. It is distinguished from meralgia paresthetica by the absence of sensory alteration in the skin, and lack of pain on digital pressure to the nerve. **(Skaggs et al., 2006)**

Entrapment of the superior gluteal nerve that supplies the gluteus medius and minimus and tensor fascia lata, as well as the greater trochanteric region and a portion of the hip joint, can cause what has been termed false meralgia paresthetica. **(Staal et al., 1999; Kopell & Thompson, 1976)** In this condition there is pain over the gluteus medius and minimus that radiates down the lateral aspect of the thigh to the knee. Lack of cutaneous sensory findings and no tenderness of the lateral femoral cutaneous nerve differentiate this from true meralgia paresthetica. Structural correction of the spine and pelvis typically corrects the condition.

Body language that first indicates that entrapment of the lateral femoral cutaneous nerve and meralgia paresthetica may be present is the location of the pain, and whether it is relieved in seated or recumbent positions and exacerbated by standing, walking, and hip extension. Hip extension applied as a provocative test for meralgia paresthetica will aggravate the paresthesia and discomfort. **(Russell, 2006; Casscells, 1978)** A therapeutic test that is sometimes done is a local anesthetic injection at the point of probable entrapment. **(Williams & Trzil, 1991; Bellis, 1977)** Williams and Trzil **(Williams & Trzil, 1991)** report on an impressive series of 277 patients for whom only 24 cases required surgery. In a surgical study for the relief of meralgia paresthetica there were findings of constrictive fascial bands around the lateral femoral cutaneous nerve in 19 of 21 cases, indicating that soft-tissue disturbances of the mechanical interface with the nerve the most common etiology. **(Edelson & Stevens, 1994)**

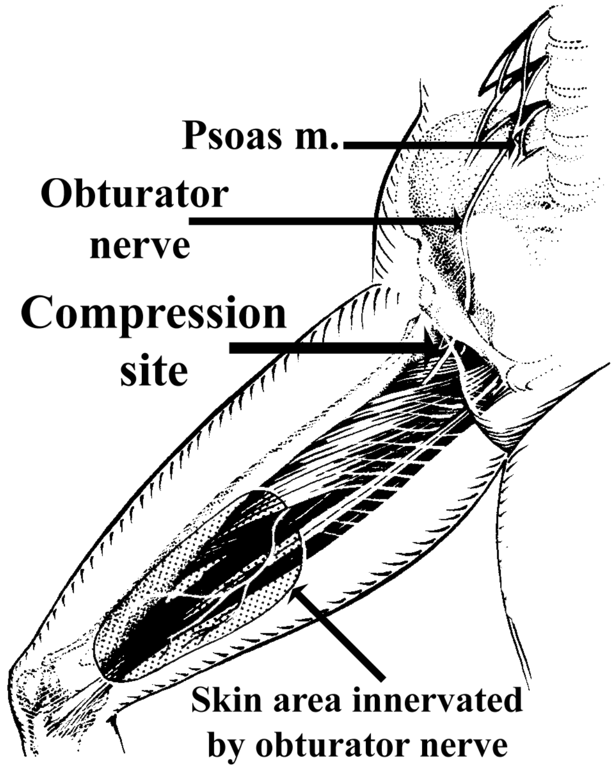
Correction of entrapment of the lateral femoral cutaneous nerve is usually readily accomplished using applied kinesiology techniques. Even without the effective procedures of applied kinesiology, exercises directed toward improvement of muscles that support the pelvis — especially the abdominals — have been shown to be effective in treating this condition. **(Skaggs et al., 2006)** Surgical procedures are usually not done as a method of treatment. **(Aguayo, 1975)** Correction of spinal subluxations has been indicated as a method of treatment. **(Skaggs et al., 2006; Ferezy, 1989; Dunn, 1975)** Obviously, the optimal approach is to evaluate all aspects of the condition and correct any dysfunction that is present.

There is usually a pelvic category I or II and weak abdominal and gluteus maximus muscles. Total postural balance should be evaluated and corrected, as well as modular interaction of PRYT, cloacal synchronization, and dural tension. **(Walther, 2000, 1981)** These techniques restore organization to the muscles and postural balance, and are often important in improving range of motion. A painful myofascial trigger point (MTrP) may be found just medial to the anterior superior iliac spine. **(Biemond, 1970)** Muscle stretch reaction of the hip flexors differentiates this from radiating pain due to pressure on the entrapped nerve. The trigger point is treated with the usual AK methods of percussion, trigger point pressure release, or stretch and spray methods. **(Leaf, 2010; Cuthbert, 2002; Walther, 2000)** A recently published systematic review of the literature on the chiropractic management of myofascial trigger points and myofascial pain syndromes **(Vernon & Schneider, 2009)** reviewed 112 publications and came to the recommendation that moderately strong evidence supports some manual therapies (manipulation and ischaemic pressure) for immediate pain relief for myofascial trigger points. According to Leibenson **(Liebenson, 2007)** the combination of muscular inhibition, joint dysfunction and trigger point activity is the key peripheral component leading to functional pathology of the motor system. In AK, the presence of myofascial trigger points can be identified using the muscle stretch procedure that produces detectible changes in muscle strength on MMT. **(Cuthbert, 2002; Mense & Simons, 2001; Walther, 2000; Goodheart, 1998; Travell & Simons, 1992)** After these techniques have been applied, evaluate hip extension range of motion; if limited, evaluate the hip flexors for muscle stretch reaction and apply either trigger point pressure release, percussion, or stretch and spray technique to obtain improved range of motion. Frequently the percussion and/or trigger point pressure release technique is the appropriate one. This appears to release the fascial pull on the lateral femoral cutaneous nerve that is causing the entrapment at its pelvic outlet. Stretching exercises designed to stretch the iliopsoas and rectus femoris muscles are usually contraindicated since they will cause additional irritation to the entrapped nerve. Generalized body organization or local muscle treatment is usually satisfactory for obtaining effective correction.

Medical approaches have included ultrasound at L2 and where the nerve leaves the pelvis, and high voltage electrogalvanic stimulation. "The medical approach, after attempting to relieve external stress to the nerve, would include mild analgesia or local injection of xylocaine or corticosteroids." **(Stites, 1985)** These procedures are usually not necessary in an applied kinesiology practice. Kopell and Thompson **(Kopell & Thompson, 1960)** have found that stretching exercises to relax the tensor fascia lata and shoe lifts are ineffective. They do, however, state that most often the condition can be corrected without neurolysis.

Obturator Nerve

The obturator nerve arises from the 2nd, 3rd, and 4th lumbar nerves. After forming in the substance of the psoas major, **(Moore et al., 2009)** it passes down the pelvis immediately anterior to the sacroiliac joint. It traverses the obturator canal with the obturator vessels and is the closest to the bone of all of the structures passing through the canal. It divides into an anterior and a posterior branch. The anterior branch supplies the hip joint, adductor longus, gracilis, and usually the adductor brevis. It innervates the skin at the medial portion of the thigh. The posterior branch innervates the obturator externus, adductor magnus, and adductor brevis when not supplied by the anterior branch. There is an articular branch for the knee joint.



Obturator nerve.

Entrapment is at the osteofibrous tunnel of the obturator canal. The tunnel is formed by the obturator muscles covered by the obturator groove of the pubic bone. **(Barral & Croibier, 2007; Kopell & Thompson, 1960)** It may be aggravated by psoas muscle hypertonicity. The obturator nerve is in the upper part of the canal, and the obturator artery is directly beneath it.

Pain from entrapment is located in the groin; it radiates down into the medial aspect of the thigh, which is the obturator nerve's cutaneous distribution. It is typically not relieved by rest. There will probably be adductor muscle weakness. With injury to the obturator nerve, the legs cannot be crossed easily.

Entrapment may be the result of complications of genital or urinary surgery. The edema of osteitis pubis (inflammation of the pubic bone), which often follows surgery, can compress the obturator canal and be the major factor. Kopell and Thompson **(Kopell & Thompson, 1960)** consider that the severe pain of osteitis pubis is from this cause.

Obturator hernia can be a cause of entrapment. Increased pain radiation localized to obturator nerve distribution that develops with abdominal pressure is a positive sign of hernia. **(Staal et al., 1999; Kopell & Thompson, 1976; Staal, 1970)**

The obturator nerve can be injured during difficult labor. Pressure from the fetal head or from forceps may be the injuring factor. **(Aguayo, 1975)** The ileocecal valve dysfunction found in applied kinesiology examination can produce a lymphadenitis in the region that may irritate the obturator nerve. These pseudo-appendix conditions can be effectively resolved with applied kinesiology methods aimed at the ileocecal valve. **(Walther, 2000)**

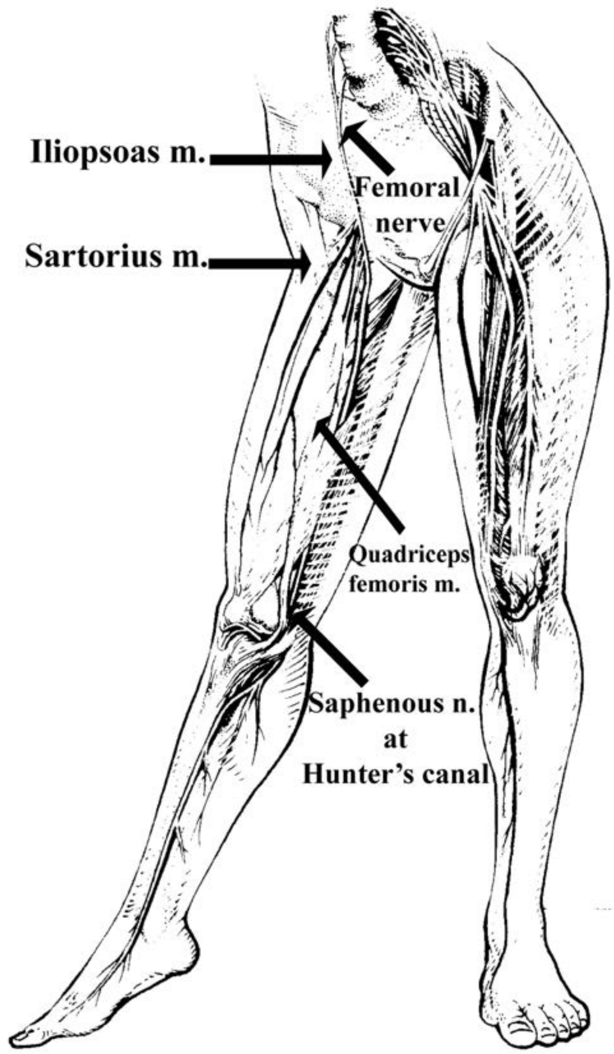
The obturator nerve is formed within the psoas muscle; consequently, hip motions that stretch or contract the psoas may aggravate the pain. The psoas should be evaluated for muscle stretch reaction, which is most easily done by moving the patient close to the edge of the table and dropping the leg over the edge with extension and abduction. Immediately move the leg into position to complete the psoas muscle test. The psoas is also often involved with strain/counterstrain, in which a maximum contraction for three seconds causes a previously strong muscle to weaken. **(Walther, 2000; Chaitow, 2002)** Treat appropriately and balance the pelvis, lumbar spine, and other postural muscles. Isolated weakness of the adductor muscles, with or without sensory changes, is a strong indication of obturator nerve injury. However because the nerve roots making up the obturator nerve also contain fibers to the femoral nerve, particular attention should be paid to the manual muscle test finding of the quadriceps muscle as well as to the patellar reflex; both should be normal in an isolated obturator neuropathy.

Patients who fail to respond to conservative treatment are subjected to an intrapelvic section of the nerve. An extrapelvic section obviously leaves the entrapment neuropathy intact. **(Skaggs et al., 2006; Kopell & Thompson, 1976)**

Lumbar Plexus

Femoral Nerve

The femoral nerve arises from L2, 3, and 4 and is the longest and largest nerve of the lumbar plexus; it runs from the 2nd lumbar vertebra to the big toe. The nerve is formed in and passes through the psoas muscle; **(Moore et al., 2009)** it continues between the psoas and iliacus muscles and travels under the inguinal ligament. **(Biemond, 1970)** After leaving the pelvis, the nerve runs in the psoas fascia **(Mozes et al., 1975)** and comes in close proximity to the femoral head. It is separated by tough, unyielding tissues, including a thin slip of the iliacus muscle, reflected head of the vastus intermedius tendon, psoas tendon, and the hip joint capsule. These structures provide minimal cushioning. Anteriorly, the nerve is protected only by the skin, making it vulnerable to trauma; **(Moore et al., 2009; Kopell, 1980)** this can cause a subperineural hematoma that may need to be surgically removed. **(Kopell & Thompson, 1976)**



Femoral nerve and saphenous branch.

Muscles supplied in the abdomen are the psoas major and minor and the iliacus; in the thigh they are the sartorius, pectineus, and four parts of the quadriceps group. An articular branch supplies the knee.

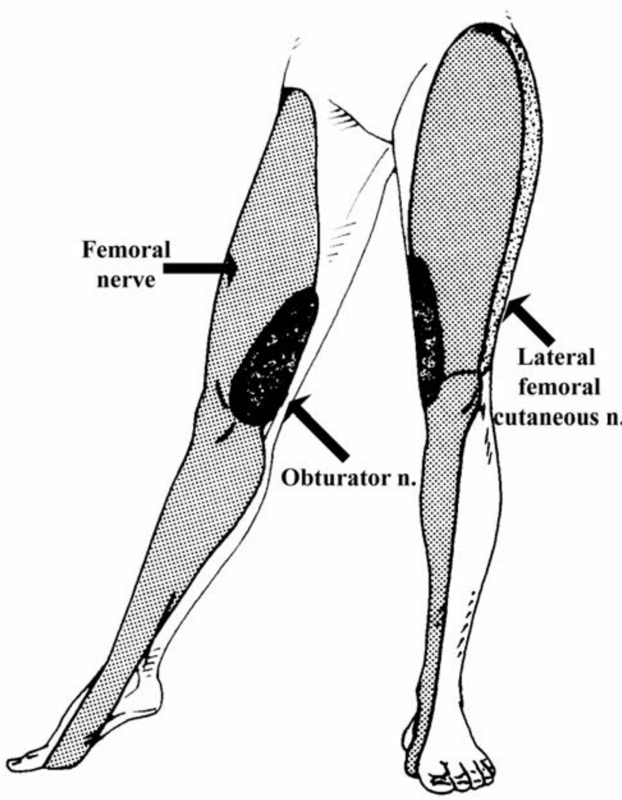
There are cutaneous branches supplying the skin of the medial and anterior surface of the thigh, medial surface of the leg, and — finally — the medial aspect of the foot to the metatarsophalangeal articulation of the big toe. The saphenous nerve is the largest cutaneous branch of the femoral nerve and is its termination. It originates near the inguinal ligament and descends into the adductor or subsartorius canal (Hunter's canal) to run along with the superficial femoral artery. It emerges from the canal approximately 10 cm proximal to the medial femoral condyle. In the leg the nerve is in close proximity to the saphenous vein. The name "saphenous" derives from the Arabic word for visible. **(Mozes et al., 1975)**

Entrapment of the femoral nerve may develop in the pelvis or at the inguinal region. According to Aguayo, **(Aguayo, 1975)** it may be associated with inguinal hernia or adhesions from careless surgery. Johnson and Montgomery **(Johnson & Montgomery, 1958)** state, "The most common cause of femoral neuropathy is involvement of the femoral nerve in the course of abdominopelvic surgery." It may also develop as a result of retroperitoneal hematoma, a complication of hemophilia, or in patients receiving anticoagulants. **(Staal et al., 1999; Aguayo, 1975)**

In femoral neuropathy, there is weakness of the quadriceps muscles and diminished or absent patellar reflex. Complete paralysis of the femoral nerve is indicated by three conditions. **(Russell, 2006; Biemond, 1970)** (1) There is motor loss of the quadriceps muscle. In the event of a high lesion, the iliopsoas function is also lost. (2) There is hypoesthesia (seldom anesthesia) of the anterior and medial aspects of the thigh, following down the tibial aspect of the legs and sometimes extending to the medial margin of the foot. (3) The quadriceps reflex is absent.

Femoral neuropathy can be caused by severe stretching of the nerve, as in accidents that cause extreme extension and abduction of the thigh or by abduction at surgery. **(Russell, 2006; Biemond, 1970)** Surgical accidents frequently follow gynecologic procedures. In more severe cases, the patient's leg collapses under her when first trying to ambulate following surgery. In a less severe condition, the patient feels weakness when attempting to climb stairs, walk up a hill, or with general activity. It is easier to walk backward than forward because the leg can support in a stance stage with accessory muscles; when hip flexion is attempted, the leg tends to collapse. If bracing is not provided, a pronounced genu recurvatum will develop in long–standing paralysis. Even when there is no paralysis of the quadriceps muscles but muscle weakness as identified by applied kinesiology, the patient will stand with the knee hyperextended.

Radiculopathy, neoplasms, abscesses, and other pathology must be ruled out. The metabolic problem often identified with femoral neuropathy is diabetes. Usually diabetic neuropathy is more widespread, but it can be localized to the femoral nerve. **(Veves et al., 2002; Kopell, 1980; Aguayo, 1975)** Possibly the reason the femoral nerve is so vulnerable to diabetic neuropathy is the poor blood supply to the intra–abdominal portion.



Cutaneous supply of lateral femoral cutaneous, femoral, and obturator nerves.

It should be remembered that there is a direct relationship between the ovaries and the femoral nerve. Pathophysiological consequences of spinal dysfunction on the segmentally related visceral organs have been described. **(Leach, 2004)** Lumbar and sacral nerve root compression as the result of lumbar and sacral articular dysfunction and degeneration has been identified as a potential cause of pelvic pain and organic dysfunction (PPOD), a term coined by Browning. **(Browning, 2009, 1988)** The impact of spinal subluxations upon sexual function may be important. **(Rome, 2010, 2009; Nansel & Slazak, 1995)**

Because the femoral nerve is formed in the psoas muscle, its hypertonicity can cause entrapment. Compression of the femoral nerve often causes pain radiation into the groin, which is increased by extension of the thigh. Sensory impairment is over the anteromedial aspect of the thigh, occasionally extending into the leg.

Time is a factor in recovering from surgical complications. Johnson and Montgomery **(Johnson & Montgomery, 1958)** indicate there is generally spontaneous recovery in three to six months, but if there is failure of some degree of recovery within three months, surgical intervention to explore and repair the nerve is indicated.

Femoral nerve involvement of a functional nature can be successfully treated using AK diagnostic and treatment methods, especially when treatment causes an immediate improvement of quadriceps muscle function. Even in the absence of frank radiculopathy, spinal and pelvic manipulation can improve femoral nerve involvement. Several cases successfully treated by such methods both with and without the advantage of AK methods have been reported. **(Picard, 2008; Skaggs et al., 2006; Kaufman, 1997; Kadel et al., 1982)** The addition of evaluation and treatment of psoas muscle weakness or hypertonicity is of primary importance, along with spinal and pelvic function. There is sometimes a tendency to concentrate examination and treatment on the side of nerve involvement. Remember to always evaluate the contralateral psoas and other muscles that may be involved. Hypertonicity of the ipsilateral psoas is frequently secondary to contralateral psoas weakness.

The saphenous nerve distal to its exit from the adductor (subsartorius) canal is superficial and innervates the medial thigh, calf, and foot. **(Russell, 2006; Worth, 1984)** Sensory symptoms of saphenous distribution are rare when the lesion involves the main trunk of the femoral nerve. **(Aguayo, 1975)** The most reliable evidence of saphenous nerve entrapment is point tenderness in which the nerve emerges through the fascia from Hunter's canal. **(Staal et al., 1999; Worth, 1984; Kopell & Thompson, 1960)** This is about 10 cm proximal to the medial condyle of the femur. At this level, palpate posteriorly across the vastus medialis to the edge of the sartorius, the point where the nerve emerges. Since the saphenous nerve is wholly sensory, there will be no muscle weakness that is dependent on its individual entrapment.

Pain is usually present at the knee when there is entrapment of the saphenous nerve. The pain at the medial knee is aggravated by walking, standing, and quadriceps exercise. The tenderness at the medial knee must be differentiated from the tenderness that is present with a weak sartorius and/or gracilis and a category II pelvic fault. Weakness of the sartorius on manual muscle testing is not adequate differential diagnosis, because the sartorius receives its nerve supply from the femoral nerve.

Misdiagnosis of saphenous nerve entrapment is common. The increase of pain on walking may lead to the diagnosis of intermittent claudication with symptoms of fatigue and heaviness of the leg. **(Russell, 2006; Staal et al., 1999)** Saphenous nerve entrapment can simulate hip pain. One patient is described who had a fracture of the femur neck and was operated on for the condition. She suffered pain attributed to hip joint pathology for a full year following the operation. A second operation to free the saphenous nerve from its entrapment point resulted in complete relief from the pain. Likewise, irritation of the infrapatellar branch of the saphenous nerve may cause pain simulating arthritis of the knee joint **(Mozes et al., 1975)** or other knee dysfunction.

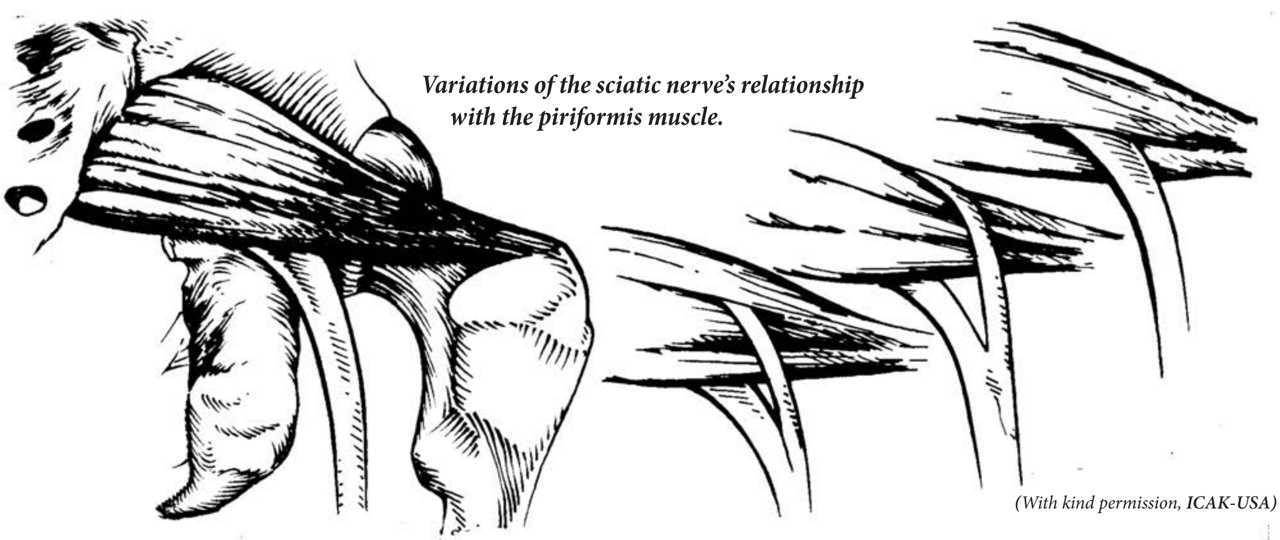
Entrapment of the saphenous nerve is often a sequel to knee surgery. In a study by Worth et al. **(Worth, 1984)** of fifteen entrapments, ten followed knee surgery. Their surgical treatment is neurolysis or neurectomy. Conservatively, repeated injections of Novocain are sometimes effective on a lasting basis. **(Mozes et al., 1975)** This also gives a definitive diagnosis. **(Staal et al., 1999; Kopell & Thompson, 1960)** Other iatrogenic causes of saphenous nerve disturbances are operations for varicose veins or harvesting of the saphenous vein for arterial grafting. **(Staal et al., 1999)**

Applied kinesiology techniques are effective in many cases. There are usually many spinal and pelvic factors to correct including dural tension release. Also, examination and treatment, if necessary, to local muscular imbalance of the hip and knee with particular attention given to fascial release techniques are important.

Piriformis Syndrome

The sciatic nerve arises from L4, 5, and S1, 2, and 3. It is the largest nerve in the body, about 2 cm in diameter. In reality, the sciatic nerve is two nerves, the tibial and peroneal, contained within the same epineurium. In some cases the nerves fail to combine in this manner. **(Prakash et al., 2010; Moore, 2009)** The tibial division originates in the sacral plexus from the ventral divisions of L4, 5, and S1, 2, and 3, and the peroneal from the dorsal divisions of L4, 5, and S1 and 2.

The sciatic nerve usually passes out of the pelvis through the greater sciatic foramen below the piriformis. Here it is flattened or in an elliptical form. Beneath the



Variations of the sciatic nerve's relationship with the piriformis muscle.

*(With kind permission, **ICAK-USA**)*

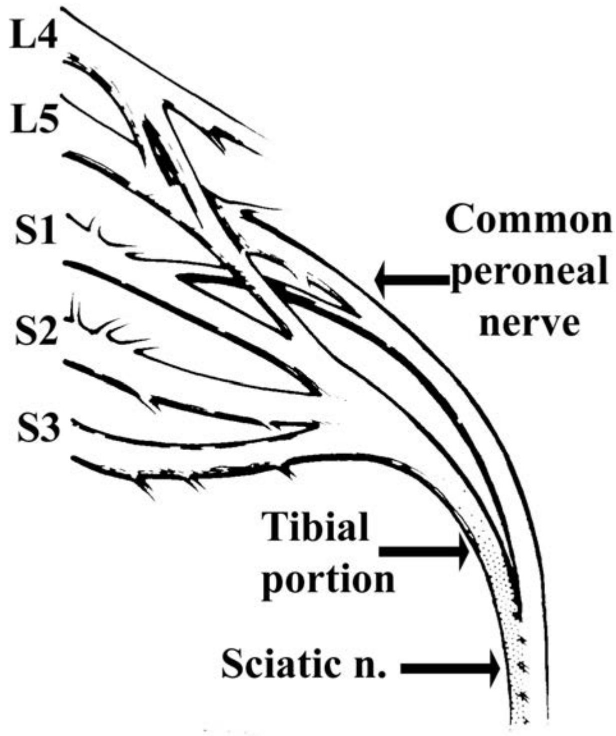
nerve is the obturator internus muscle. The nerve may divide at the piriformis muscle; when it does, it is usually the peroneal trunk that deviates. **(Prakash et al., 2010)** The nerve then passes under the gluteus maximus muscle midway between the greater trochanter of the femur and the ischial tuberosity. It continues to course down the thigh to divide in the lower one–third into the tibial and peroneal nerves.

In a study of 240 cadavers, Beaton and Anson **(Beaton & Anson, 1938)** found 10% variation from the usual nerve passing beneath the piriformis muscle. In 9.2%, the nerve divided; one portion passed through the muscle and the other above or below it. In .8%, the undivided nerve passed through the belly of the piriformis muscle. Travell & Simons **(Travell & Simons, 1992)** review many cadaveric studies on the varying courses of the two divisions of the sciatic nerve, and they list similar proportions to Beaton & Anson.

It is in its relationship with the piriformis muscle that sciatic entrapment neuropathy can develop distal to radiculopathy. If the sciatic nerve passes under the piriformis muscle, it exits the pelvis crossing the greater sciatic notch, where the nerve is flattened and adjacent to the sharp edge of the bone. **(Prakash et al., 2010; Kopell & Thompson, 1960)** When the piriformis is in spasm or shortened, it can cause sciatic compression and perhaps ischemia, especially if a portion of the nerve goes through the muscle. **(Travell & Travell, 1946)**

Travell & Simons **(Travell & Simons, 1992)** also note that the inferior gluteal nerve that innervates the gluteus maximus muscle penetrated the piriformis muscle in 15% of 112 patients, making it vulnerable as well to piriformis entrapment. The inferior gluteal artery and vein cross the sciatic trunk under the belly of the piriformis. Contraction of the piriformis may conceivably produce sustained congestion in the vein and limit circulation from the artery. This may explain the tenderness in the entire piriformis muscle in addition to sciatic pain. It may also cause an ischemia of the nerve contributing to the pain. **(Cox, 1999; Freiberg & Vinke, 1934)** Kendall et al **(Kendall et al., 1993)** report that either a contracted or a stretched piriformis can contribute to sciatic pain.

The piriformis syndrome may be caused by subtle, moderate, or severe trauma. **(Chaitow & DeLany, 2002; Cox, 1999; TePorteen, 1969; Edwards, 1962)** Acute trauma usually relates with a sudden twist of the hip, which is either activated or counteracted by the piriformis. The trauma may be such that it injures the fascia and/or tendons of the rotator muscles. Fascial trauma may cause a disrelation between the muscle and fascial function that requires the fascial release technique of applied kinesiology. **(Walther, 2000, 1981)** The ends of the taut bands of the piriformis muscle are likely to create enthesopathies; for this reason stretching the muscle may further inflame the attachments thereby creating weakness on subsequent manual muscle testing. Immediately after the trauma the muscles become hypertonic but, as the stress continues, the tendons develop enthesopathy. **(Palesy, 1997; Lund et al., 1991)** The enthesopathy causes inhibited firing of the muscles leading eventually to atrophy and eventually fatty infiltration. **(Hodges & Moseley, 2003)** Experimentally induced pain has been shown to change muscle activity, including reflex activity and muscle spindle sensitivity.



Sacral plexus.

(Zedka et al., 1999) The neuromuscular spindle cell or Golgi tendon organ may be injured, causing the muscle to test weak on manual muscle testing. It should be remembered that physical trauma can directly affect receptor axons (articular receptors, muscle spindles and other proprioceptors), **(Lederman, 1997)** and that muscle spindles possess extreme sensitivity, where the muscle spindle reacts to a pull of only 1 gram and a stretch of $1/1000^{th}$ millimeter. **(Korr, 1997, 1979)** Acute trauma often relates with sudden starts or stops, as in playing tennis, or from falls and other direct injuries to the pelvis. A sit–down type fall is often described. **(Robinson, 1947)** Twists that develop in the pelvis, such as entering or leaving an automobile on one leg, can cause strain to the muscle. Basically any type of trauma to the pelvis or buttocks may be responsible for piriformis imbalance.

More subtle etiology of piriformis imbalance can develop as a result of the patient assuming positions for prolonged periods. Usually the problem posture is in a seated position. Sitting cross–legged, especially with the ankle crossed over the opposite knee, shortens the piriformis. When quickly brought out of the position, the nervous system may not have adequate time to adapt. A muscular syndrome, as described by Jones **(Jones, 1981)** in his strain/counterstrain technique, may develop. Sitting on the feet or a foot provides a similar etiology. When driving an automobile, one may put the leg in a twisted position to contact the accelerator with the right foot, or have the left foot in a position to cause strain to the piriformis. Waddell **(Waddell, 1998)** warns that driving and sitting improperly (ergonomic stress positions) for long periods are major causes of back pain. Ergonomic seats, good posture, frequent periods of movement, and optimal tire pressure and suspension systems are important factors for reducing the jarring forces inherent in driving. Imbalance of the piriformis in all these conditions probably relates with muscle dysfunction that can be treated with the strain/counterstrain technique. This type of dysfunction can also develop from rapid trauma. Brugger **(Brugger, 1960)** suggests a postural exercise that eases the stresses of sitting and contribute to neck and back pain. Exercise like this and ergonomic advise should be used for patients whose jobs require prolonged sitting.

Piriformis muscle dysfunction often develops after obstetric or urologic procedures in which the patient is in stirrups, especially when under general anesthesia. **(TePorteen, 1969)** Coital positions are also responsible for piriformis strain. The patient frequently does not relate this to the physician. Goodheart **(Goodheart, 1992)** observes that backache from sexual intercourse probably relates to disruption of the craniosacral primary respiratory action of the pelvis and spine. On inspiration the pubes should drop slightly inferiorly and raise superiorly on expiration. If during the sexual act there is a pelvic thrust by the male during inspiration by the female, the pelvic respiratory action may be disturbed. This disturbance will likely involve the piriformis muscle and its function.

Structural disorganization anywhere in the body, especially in the pelvis and hips, can be the causative factor of piriformis syndrome. Lumbar hyperlordosis with anterior pelvic rotation and hip flexion tightens the rotator group of muscles over the nerve and draws the nerve trunk more tightly against the rim of bone. **(Kendall et al., 1993; Kopell & Thompson, 1960)**

Careful differential diagnosis must be done in sciatic nerve conditions. There is a close symptomatic association in piriformis syndrome, radiculopathy, and sacroiliac subluxations and fixations. **(Travell & Simons, 1992)** There may be a combination of conditions, or symptoms of one being created by another condition.

Piriformis entrapment of the sciatic nerve is not accepted as a common syndrome by all. Dawson et al. **(Dawson et al., 1983)** take the view of surgeons, stating, "It should be mentioned at the outset that sciatic entrapment neuropathy has not been fully accepted as a legitimate syndrome, and in spite of occasional cases it remains difficult to substantiate." It appears that their concern is for total paralysis as a result of sciatic entrapment. They continue, "The sciatic nerve innervates the knee flexors and all the muscles below the knee. Therefore, complete palsy of the sciatic nerve would lead to marked instability of the foot and to severe impairment of gait because of knee and ankle impairment." The entrapment found by AK examination is more subtle, leading to pain and muscle dysfunction that readily respond to the various types of treatment used in applied kinesiology. Piriformis entrapment may also complicate sciatic involvement by contributing to a double crush of the nerve.

Travell & Simons and Steiner et al. **(Travell & Simons, 1992; Steiner et al., 1987)** support the concurrency concept of radiculopathy and piriformis syndrome. These conditions may appear almost identical. There is a consistent absence of true neurologic findings in the piriformis syndrome. Back pain is common to both. Even when there are specific indications of a herniated disc, one should examine for piriformis involvement.

Dawson et al. **(Dawson et al., 1983)** indicate the following in differentiating piriformis syndrome from radiculitis due to a lumbar disc. "Pain on coughing, sneezing, or laughing is a sure indicator of epidural disease and should be inquired for in every case of this type. Most patients with sciatic pain due to lumbar disc disease have monoradicular symptoms. Therefore, the pain generally radiates to the lateral side of the foot and the small toe (S1), to the dorsum of the foot (L5), or to the medial part of the calf (L4). Many patients also have proximal sciatic pain in the posterior thigh and the buttocks, but this has less anatomic or localizing significance." Piriformis syndrome must also be differentiated from lumbar spinal stenosis.

In addition to other differential diagnosis in sciatic–type pain, particular attention must be given to the correlation of the piriformis and sacroiliac syndromes. Vleeming et al **(Vleeming et al., 1995)** and Poole **(Poole, 1985)** assert that many conditions diagnosed as sacroiliac or lumbosacral involvement are, in reality, piriformis syndromes. Vleeming et al. observe that sacroiliac joint pain may not be a local problem "but symptomatic of a failed load transfer system" of the muscles that attach to the sacroiliac joint. Many other muscles are involved in the stability of the sacroiliac joints, including the gluteus medius, multifidus, biceps femoris, transverse and obdominal obliques, gluteus maximus, and latissimus dorsi, among others. **(Harrison et al., 1997)** Various tests of the sacroiliac designed to stretch the ligaments, such as Gaenslen's test, iliac maneuvers designed to stretch the anterior and posterior ligaments, such as Hibbs' test, and others should be analyzed to determine the structure involved in addition to the sacroiliac. Physicians from many disciplines appreciate that maneuvers that move nerves and receptors often reproduce radiating pain. This symptom reproduction has important implications for the diagnosis and management of radiating pain syndromes. Applied kinesiology examination reveals close association between piriformis dysfunction and sacroiliac subluxations and fixations. The concurrent presence of both conditions is common. **(Knutson, 2004; Corwin, 1987)**

The piriformis muscle is important in stabilizing sacral function. Sacral subluxations are attributed to its imbalance. **(Boyajian-O'Neill et al., 2008; Chaitow & DeLany, 2002; Kendall et al., 1993; Travell & Simons, 1992; Retzlaff, 1974; Goodheart, 1972)** A sacroiliac fixation may develop with piriformis hypertonicity and shortening. On the other hand, piriformis spasm can be present as a vain effort of the piriformis to correct pelvic distortion.

Skillern **(Skillern, 1944)** explains the mechanism of piriformis spasm as coming from peripheral irritation caused by minor strain of the sacroiliac joint. Fourteen cadavers were studied by Freiberg and Vinke **(Freiberg & Vinke, 1934)** to establish the relationship of the sacroiliac joints with the sacral and lumbar plexus and the sciatic nerve to the piriformis. There is a very intimate relationship of the branches of the plexus with the blood vessels. They are so closely interwoven that it is difficult to separate them. Some symptoms of piriformis syndrome may occur from local inflammation and congestion caused by the muscular compression of small nerves and vessels – including the pudendal nerve and blood vessels, which emerge at the medial inferior border of the piriformis muscle. **(Moore, 2009)**

Pain associated with the piriformis syndrome is in the area of the femoral head and greater trochanter **(Travell & Simons, 1992; Khoe, 1975)** and may radiate into the inguinal area. There is local tenderness over the piriformis muscle and tendon. The pain radiating into the hip often causes difficulty in walking, and it may radiate down the leg in the form of sciatic neuralgia. There may also be pain at the lateral knee behind the head of the fibula, due to involvement of the common peroneal nerve. Pain is often relieved by a change in position or walking. There is difficulty in sitting, standing, or lying comfortably. One may find the patient walking around the examination room rather than sitting and waiting for the physician. The patient often gives a poor description of the pain and has a difficult time localizing its origin. The symptoms are vague in the area of the posterolateral hip and down the tensor fascia lata muscle. When the physician palpates into the piriformis, the patient usually quickly recognizes that is the source of the pain.

Sciatic notch location.

The pudendal nerve leaves the pelvis between the piriformis and coccygeus muscles. It supplies much of the external genitalia and related peroneal musculature in both males and females. When the pudendal nerve and blood vessels are involved in piriformis muscle entrapment, there may be a lack of genital sensation, pain, incontinence, or impotence. **(Cuthbert & Rosner, 2012; Browning, 2009; Retzlaff, 1974)**

The first phase of examination includes palpation of the piriformis muscle's insertion and around the greater trochanter for tenderness. From this insertion proceed toward the sciatic notch to palpate the piriformis muscle mass. The sciatic notch is located approximately 2" lateral and 1" caudal to the posterior superior iliac spine. **(Lee, 2004; Corwin, 1987)**

In sciatic neuralgia and with other signs of piriformis syndrome, observe for an antalgic position of external thigh rotation and slight hip flexion, which indicates a possible piriformis syndrome. This position takes tension off the piriformis and may thus reduce irritation on the sciatic nerve. **(Chaitow & DeLany, 2002; Freiberg & Vinke, 1934)** This body language position is called the "piriformis sign". **(Foster, 2002; TePoorten, 1969)**

Internally rotating the thigh stretches the piriformis and usually increases the irritation on the sciatic nerve, causing pain radiation. Evaluate the effect of stretching the piriformis with the patient prone, using the lower leg as a lever to internally rotate the thigh. What is often referred to as "tenderness at the sciatic notch" is usually tenderness of the piriformis belly. **(Freiberg & Vinke, 1934)** The Beatty test is useful for diagnosis of the syndrome too. The patient lies on the uninvolved side, and then lifts and holds the superior knee approximately 6 inches off the examination table. If sciatic symptoms are recreated, the test is positive for piriformis syndrome.

The above descriptions are of stretching or shortening the piriformis muscle passively. When the patient actively contracts the piriformis, such as in a manual muscle test, there is external thigh rotation and tightening of the piriformis. This may cause the same type of pain as passively stretching it.

Pain and dysfunction from piriformis nerve entrapment must be differentiated from sciatic involvement at a higher level. The straight leg raise and Lasegue tests can aid in this determination. **(Morris, 2006; Robinson, 1947)** The piriformis muscle is put on stretch after only a few degrees of leg raising. Differentially the straight leg-raising test is usually positive for a pathological disc rather than the first stage of Lasegue's sign before the knee is extended, although both may be present. In a piriformis syndrome, pain is caused by hip movement and stretching of the muscle rather than sciatic nerve stretch. Freiberg and Vinke **(Freiberg & Vinke, 1934)** evaluated straight leg raising on cadavers. When the thigh reached approximately 25–50° of flexion, there was tightening of the sacrotuberous ligament and the piriformis muscle. They point out that in many patients with sciatic pain, straight leg raising is limited at only a few degrees of hip flexion. This is long before the sciatic nerve is stretched by the maneuver, indicating entrapment below the vertebral level.

There will be tenderness of the sciatic nerve in a piriformis syndrome; however, it is often difficult to elicit because of the bulk of tissue covering the structure. When a straight leg raise test elicits pain, lower the leg slightly, just below the point of pain. Internal rotation in this position stretches the piriformis and will increase the pain. There will be relief by passive external rotation. **(Morris, 2006)**

The superior gluteal nerve supplies the gluteus medius and minimus, and the inferior gluteal nerve supplies the gluteus maximus. These nerves originate from the sacral plexus with the sciatic nerve, and travel with it in its short intrapelvic course. Both gluteal nerves leave the pelvis through the greater sciatic foramen, but the superior nerve leaves above the piriformis and the inferior one below it. **(Gray's Anatomy, 2004)**

There is controversy over involvement of the gluteal muscles in a piriformis syndrome. Some indicate that if there is weakness of the gluteal muscles along with others of sciatic distribution, there is indication of spinal or intrapelvic entrapment, such as tumors. When the lesion is due to piriformis entrapment, the gluteal muscles will not be involved; however, there is involvement of the hamstrings and all muscles of the lower leg. **(Boyajian-O'Neill et al., 2008; Aguayo, 1975; Kopell & Thompson, 1960)**

The other side of the question is that with chronicity there may be gluteal atrophy on the affected side. **(Staal et al., 1999; Robinson, 1947)** Because the superior gluteal nerve leaves the pelvis above the piriformis and the inferior nerve below it, it seems that the gluteus medius and minimus will be spared and the gluteus maximus will be vulnerable in a piriformis syndrome. In manual muscle testing one observes a varied involvement of the gluteus medius, minimus, and maximus. In any event, when the piriformis muscle is responsible for gluteal weakness, they strengthen after the piriformis muscle is treated. This seems to indicate that there is a varying morphology of the superior and inferior gluteal nerves in relationship to the piriformis.

Patients with a vascular neuropathy, particularly a diabetic one, may acquire a piriformis syndrome. The symptoms are relatively acute in onset, developing over several days. **(Veves et al., 2002; Dawson et al., 1983)** Pain develops in intermittent claudication with a specific amount of walking, and is eased upon standing, **(MacNab, 1977)** which is opposite that of typical piriformis syndrome. The pattern of pain with activity in claudication is consistent, and inconsistent in neuromuscular, osseous conditions. **(Greenfield & Andersen, 1982)**

In summary, the piriformis syndrome should be differentially diagnosed from iliotibial band contracture, **(Ober, 1987)** lumbar, lumbosacral, and sacroiliac disturbances, hip joint lesion, various types of arthritis, prostate conditions, female organ dysfunction, cystitis, and epidural and pelvic tumors that may be found with CT scan. **(Boyajian-O'Neill, 2008; Cohen et al., 1986)**

The use of specific manual muscle tests can facilitate the diagnosis of the syndrome, and differentiate it from other disturbances to the sciatic nerve. In patients with piriformis syndrome, manual muscle test results will be normal for muscles proximal to the piriformis muscle and abnormal for muscles distal to it.

Specific treatment of the piriformis syndrome is directed toward the piriformis muscles. In addition, total body function must be considered and any pelvic and spinal problems corrected, as well as modular interaction of the body and gait problems, including foot dysfunction. Applied kinesiology could be defined as diagnosis and treatment of disorders of the body within a conceptual basis of the inter-dependency and continuity of all the tissues of the body. For patients with piriformis syndromes, TePoorten **(TePoorten, 1969)** recorded decreased range of motion of the T10-T11 segments, tissue texture changes at T3-T4, pain and decreased range of motion of the contralateral C2 segment, and ipsilateral occipital-atlantal lesion. TePoorten's observations are consistent with the experience of applied kinesiologists.

The piriformis muscles should be evaluated bilaterally for hyper– or hypoactivity. Early in applied kinesiology Goodheart proposed that much hyperactivity or muscle spasm is secondary to weakness of the antagonist muscle(s). This observation has been confirmed by the work of Lund **(Lund et al, 1991),** Wall et al., **(1988)** and Mense and Simons **(2001).** Mense and Simons suggest that the recognition of the muscle weakness caused by myofascial trigger points is often a critical step in the restoration of normal function. In their view other muscles suffer from compensatory overload due to the inhibition created by the myofascial trigger points in the inhibited muscles. Travell and Simons also state that "weakness is generally characteristic of muscles with active myofascial trigger points". **(Travell & Simons, 1983)** When tightness or spasm of a piriformis muscle is present and there is weakness on the opposite side, it is advantageous to correct the weak side first. This is done with the usual subluxation and fixation correction techniques, reflex techniques, cranial–sacral primary respiratory correction, meridian balancing, and sometimes nutritional corrections as used in applied kinesiology.

Berry and Retzlaff **(Berry & Retzlaff, 1978)** describe a method to treat a hypertonic muscle by giving attention to the hypotonic or weak muscle on the opposite side, based on Sherrington's law of reciprocal innervation. **(Denny-Brown, 1979)** When a muscle on one side of the body contracts, its homologue on the opposite side relaxes. Retzlaff and Fontaine **(Retzlaff & Fontaine, 1960)** have described a neuronal mechanism that provides simultaneous excitation of the neurons to one muscle (or group) and inhibition to the contralateral muscle (or group).

These basic principles are used when indirectly treating the hypertonic piriformis syndrome by improving function or stimulating the contralateral (non–affected) piriformis muscle. In addition, the piriformis reciprocal innervation technique (Berry method) **(Retzlaff, 1974)** can be applied. The patient is supine, with knees and hips flexed. The physician stands on the side opposite involvement. The patient abducts the knee of the uninvolved side against the physician. This contracts the non–involved piriformis to send inhibitory impulses to the hyperactive piriformis. The physician monitors the relaxation of the involved piriformis by palpating it, while the patient continues to isometrically contract the non–involved side. The procedure is repeated several times, with a short rest period in between. With each period of isometric contraction by the patient there is usually greater relaxation of the affected piriformis.

There are several applied kinesiology techniques applicable to the piriformis syndrome. The involved side is first tested to determine if the muscle is strong in the clear, and it usually is. If there is weakness on manual muscle testing, treat the muscle with the five factors of the IVF. **(Walther, 2000, 1981)** When the muscle tests strong in the clear, it is further evaluated for muscle stretch reaction by stretching the muscle and then immediately testing to determine if the previously strong muscle weakens. If so, percussion, trigger point pressure release, or stretch and spray methods are indicated. Most often fascial release technique is effective. **(Leaf, 2010; Cuthbert, 2002; Walther, 2000)** Whichever technique is used, internal thigh rotation should be increased.

Further evaluation is done by means of the strain/counterstrain technique. This treatment is often needed when no acute trauma is known to have caused the condition. The neuromuscular condition develops as a result of the individual maintaining a strained postural position of the piriformis for a prolonged period and then quickly moving out of the position. Jones **(Jones, 1981)** relates this to the nervous system's inability to quickly adapt to the change in muscle position.

To test for the need for applying the strain/counterstrain technique, have the patient maximally contract the piriformis, externally rotating the thigh. The contraction is held for three seconds, immediately after which the previously strong muscle is tested for weakening; if it does, strain/counterstrain technique is indicated. This is most easily accomplished with the patient prone. The hip and knee of the involved side are flexed, with the knee resting on the examination table. The tender point in the piriformis is usually located close to the greater trochanter. The thigh is passively rotated externally until the position is found that minimizes the tenderness in the piriformis. Some spinal extension and rotation may be required to relieve the tender point. When the point is found, have the patient maximally exhale while you simultaneously spread the tissue over the tender point with a two–finger contact. Hold this for approximately thirty seconds, after which the patient's involved limb and spine are slowly and passively brought back to a neutral position.

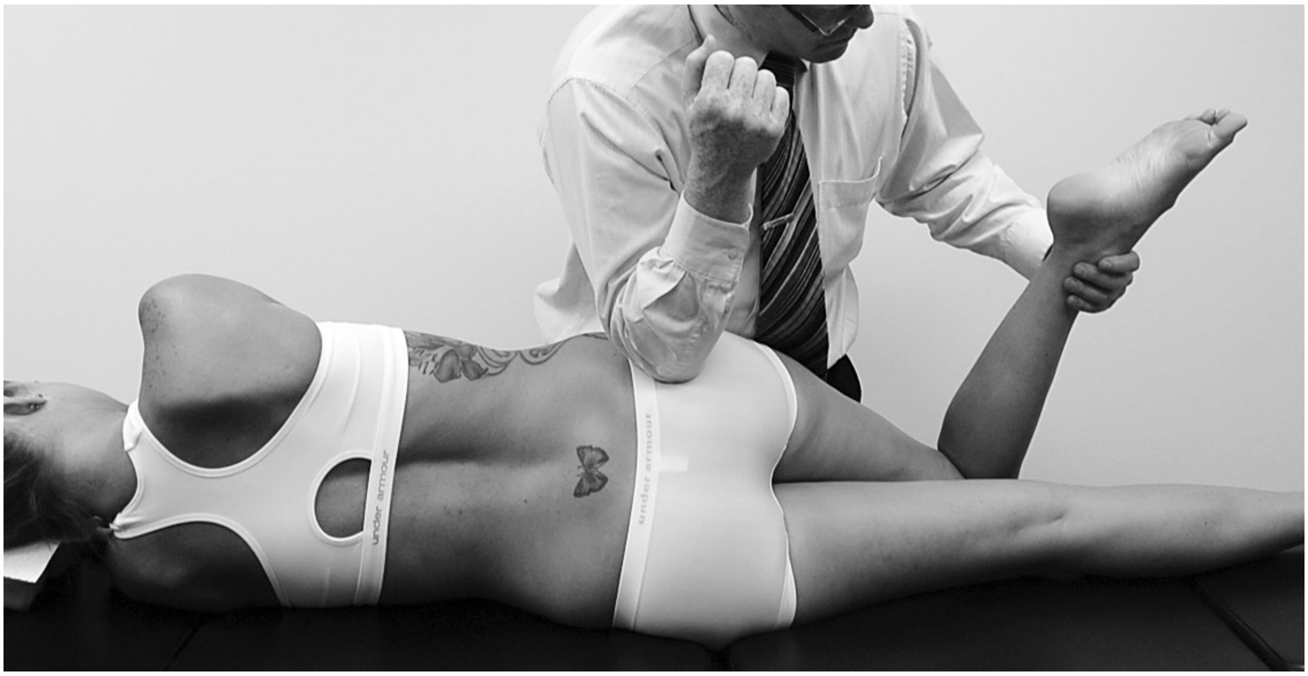
Another method of strain/counterstrain treatment is with the patient prone near the edge of the treatment table so the involved leg drops off the table with hip and knee flexion. The physician supports the patient's leg at the knee and passively directs external thigh rotation and abduction to relieve the tender point. The same exhalation and two–point finger spread over the tender point is done, after which the leg is passively brought back to neutral. Optimal relief of tenderness may not be accomplished in this position because of inability to extend and rotate the spine. Successful treatment by strain/counterstrain technique is indicated when there is no weakening after the patient maximally contracts the piriformis for three seconds.

There are several pressure techniques for relaxing a piriformis muscle. Chaitow & DeLany **(Chaitow & DeLany, 2002)** and Edwards **(Edwards, 1962)** recommend that heavy pressure be applied to the belly of the muscle by the thumbs, thenar eminence, or elbow pressure. The patient lies on the unaffected side, rotated slightly toward the examination table, with slight hip and knee flexion. The physician applies a heavy (40–60 lbs) pressure for approximately ten seconds. The pressure is sustained, not a massaging movement. This is repeated eight to twelve times. The pressure should be heavy enough to stretch fibrous tissue, yet not so heavy that it produces further inflammation and additional fibrositis. Pressure in the piriformis is painful but it does not accentuate the pain the patient is experiencing; rather, it tends to block it. After treatment the patient typically feels better, but the pain will return somewhat within a few hours. Repeated treatments eliminate the condition. People with good muscle physiology and in good health respond more rapidly than those with sedentary habits and poor general health.

A stretching technique described by TePoorten **(TePoorten, 1969)** is also done with the patient side–lying on the unaffected side. The knees are flexed to 90°, with very slight hip flexion. The physician applies pressure to the piriformis with his or her elbow. The contact is just posteromedial to the greater trochanter. The thigh is moved into internal rotation by using the leg as a lever. Simultaneously pressure is maintained on the piriformis while the muscle is thus stretched for one or two minutes and repeated two or three times.

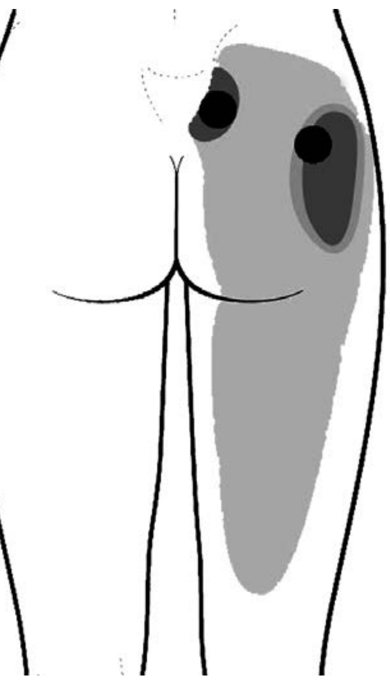
Piriformis muscle stretch reaction

Strain-counterstrain of piriformis muscle



TePoorten's piriformis stretching technique

Another stretching activity described by TePoorten **(TePoorten, 1969)** and Retzlaff et al. **(Retzlaff, 1974)** is to have the supine patient flex his knee and hip to approximate the thigh on the abdomen. Use the leg as a lever to rotate the thigh while flexing and extending the hip and knee. When flexing, rotate the thigh externally; when extending, rotate the thigh internally. A following step is to hyperflex the hip and knee so the thigh approximates the abdomen. The physician uses his shoulder on the patient's knee to adduct it, with passive pressure at the patient's ankle to internally rotate the thigh. Throughout the first and second procedures the patient resists the motion, providing a type of isometric contraction.

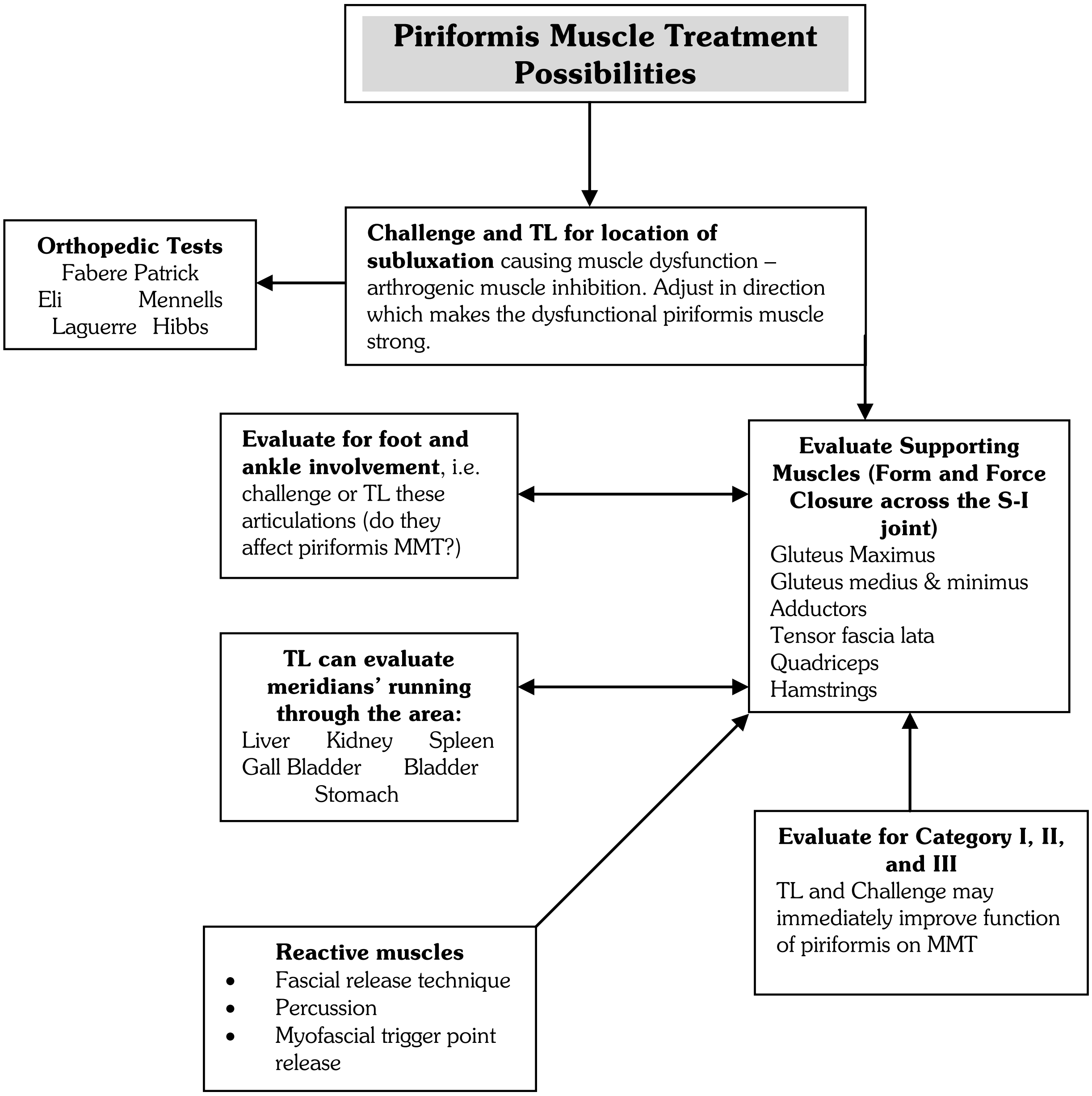


Piriformis MTrP Referred Pain Zone

Quite often the applied kinesiology approaches previously mentioned will correct the condition. Additional help may be obtained by the stretching and pressure procedures. An additional approach recommended by Poole **(Poole, 1985)** that might occasionally be beneficial is treatment of the piriformis spasm done rectally. A finger is inserted into the rectum with the patient side–lying, with the painful side up and the hips and knees slightly flexed. From contact of the coccyx, the flexor surface of the finger moves laterally to touch the anterior surface of the levator ani and coccygeus muscle. The fibers of the piriformis muscle are felt posterior to the sacrospinous ligament and stroked in a lateral motion. There may be extremely sensitive trigger points which, according to Nimmo, **(Nimmo, 1985)** cause the muscle spasm.

Whatever therapeutic approach is used, there should be an increase of passive internal thigh rotation. The standing posture should improve, with reduction of the excessive external leg rotation on the involved side. There should be balance of the piriformis muscle test bilaterally.

Finally, other therapeutic approaches have been advocated. Procaine hydrochloride injection into the piriformis muscle and sacroiliac joint has been recommended by Skillern. In 1947, Robinson **(Robinson, 1947)** recommended section of the piriformis muscle. This was the original publication about the piriformis syndrome reporting about two patients. Although he felt no disability resulted from the surgery, no follow–up reports of this approach have been found. Surgery for this condition is still advocated by some orthopedists **(Staal et al., 1999)** The piriformis syndrome responds well to the conservative approaches described herein.



Peroneal Nerves

The sciatic nerve divides in the lower thigh into the common peroneal and tibial nerves. The common peroneal — the lateral branch — is smaller than the tibial nerve by about one–half. It is derived from the dorsal branches of the ventral rami of the 4th and 5th lumbar and the 1st and 2nd sacral nerves. After passing behind the head of the fibula, close to the tendon of the biceps femoris, it divides into the superficial and deep peroneal nerves. The peroneal nerve, with its superficial and deep branches, supplies the dorsiflexors and everters of the foot, the skin on the lateral portion of the leg and dorsal portion of the foot, a portion of the ankle joint, and the tarsal joints.

Common Peroneal

The common peroneal nerve courses down the lateral aspect of the popliteal fossa, emerging between the biceps femoris tendon and the lateral head of the gastrocnemius. It pierces the fascia to lie against the fibular neck and lies directly upon the periosteum where it is particularly exposed to compression, stretch or other trauma. The nerve is covered by unusually thick fascia as it winds around the lateral surface of the fibular neck, deep to the origin of the peroneus longus. This fascia may be stretched tightly over the nerve to form an osteofibrous tunnel in which the nerve runs for about half an inch. **(Russell, 2006; Marwah, 1964)** There may sometimes be an opening in the origin of the peroneus longus muscle through which the nerve passes. **(Kopell & Thompson, 1976)**

Contributing to the tunnel are the two heads of the peroneus longus muscle that form a bridge under which the common peroneal nerve courses. The bridge is made up of the dense fascia overlying the two heads. The superficial head attaches to the head of the fibula and adjacent tibia. The deep head attaches to the neck of the fibula below the nerve. This has been called the fibular tunnel, where entrapment has been reported. **(Butler, 2000; Schweitzer et al., 1997; Lusskin, 1982; Kopell & Thompson, 1976; Fettweis, 1968)**

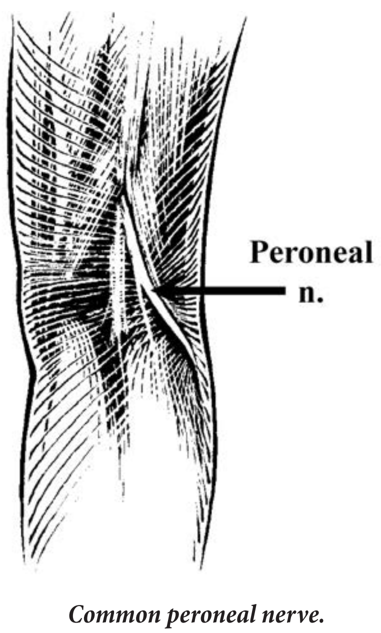
Palpation of the common peroneal nerve can be accomplished by beginning at the apex of the popliteal fossa and following the medial side of the biceps tendon distally and laterally to the posterior head of the fibula. **(Gray's Anatomy, 2004)** Here the nerve can be palpated against the bone. At this point 60% of the nerve is connective tissue and is quite flattened here and will not roll under the fingers as it will at the posterior knee.

Peroneal nerve entrapment usually involves the common peroneal. The extent of the impairments depends upon whether the entrapment involves the fascicles of the deep or the superficial peroneal nerve, or both, and also on the duration of the compression. The common peroneal is vulnerable to compression where it is fixed and angulated at the fibular head and neck. **(Sunderland, 1953)** The etiology relates to its superficial position and lying directly on the bone under the sharp crescentic arch of the tendinous origin of the peroneus longus. The disturbance may result from direct trauma, plaster cast compression, external pressure, fracture of the fibula, or pressure from a neoplasm. Indirectly, osteoarthritis of the knee or platform fracture of the tibia may be involved.

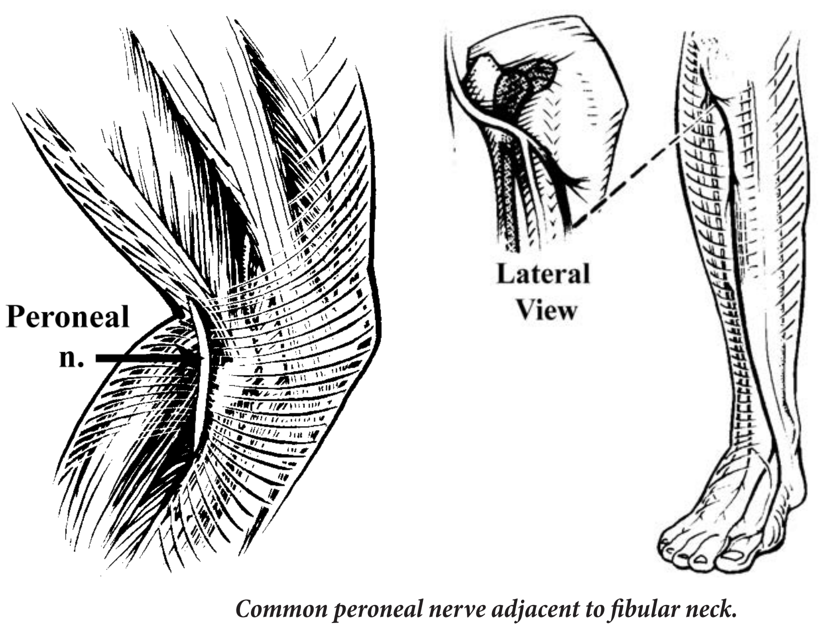
Entrapment of the common peroneal may be due to traction on it, especially from the superficial peroneal nerve. With inversion and plantar flexion of the foot, the nerve is pulled taut against the fascial edge, close to the fibular head. **(Butler, 2000)**

Common peroneal nerve entrapment can develop at the level of the fibular head from habitually sitting with one leg crossed over the other. **(Staal et al., 1999)** The nerve is vulnerable to compression in this position because it is relatively fixed, and flexion of the knee exerts abnormal tension on it. In this position the nerve is also compressed against the neck of the fibula. Both the pressure and the traction can be increased in positions of crouching, squatting, or kneeling. This is particularly problematic if the same position is repeatedly required in the person's occupation. People most vulnerable to this compression are those who are tall, long–legged, and asthenic. **(Nagler & Rangell, 1947)** External pressure caused by casts and tight, high boots may also cause compression of the common peroneal nerve. **(Staal et al., 1999; Mitra et al., 1995; Kopell & Thompson, 1976)** The neurologist Staal **(Staal et al., 1999)** observes that for patients who are bed-ridden or spend many days in hospital "…the common peroneal nerves at the head of the fibula should be protected by soft padding around the head of the fibula. It often proves difficult to convince residents and nurses of the rationale of these measures." Also, for patients with a leg in a plaster case that reaches as far as the head of the fibula or higher, "…the common peroneal nerve should be protected, preferably by an appropriate window in the plaster cast. Such precautions may avert not only pressure palsies but lawsuits. It makes sense to instruct every patient with a leg plaster to check every day if the strength of the toe extensors is still normal; if there is weakness the plaster should be opened."

The initiating injury of common peroneal nerve entrapment may be a sprained ankle. There may be a continued tendency for the ankle to turn over due to muscle weakness. **(Palmieri-Smith et al., 2009; McVey et al., 2005; Sidey, 1969)** Muscular imbalance and structural distortion applicable to applied kinesiology examination and treatment may result from trauma or have an insidious origin. Often attention is focused on the ankle weakness as a supposed ligamentous laxity, yet such laxity is demonstrable in only a few cases. This condition, if allowed to progress, could develop into a complete foot drop. Foot drop is a deceptively simple term for a potentially complex disorder. Foot drop is most commonly associated with intervertebral disc syndromes and radiculopathy, but also with peripheral



Common peroneal nerve.



Common peroneal nerve adjacent to fibular neck.

nerve entrapments, neuropathies, stroke, drug toxicities, diabetes mellitus, multiple sclerosis, peripheral arterial disease or hyperthyroidism, myofascial trigger points in the dorsiflexor muscles of the ankle, among others. **(Brief et al., 2009)** Walking with a more or less complete foot drop leads to the typical stepping gait in which there will be compensatory overaction of the hip and knee flexors to gain clearance for the foot during the swing phase. With moderate foot drop the foot may still land on the heel but immediately afterwards the foot dorsiflexors give way, producing a flat-footed sound. With mild weakness the patient may appear to walk normally to an inexperienced examiner, but the patient is not able to walk on his heels or will swing the foot with less clearance from the floor than on the normal side.

Symptoms of common peroneal entrapment may be both motor and sensory, or either one individually. The most common symptom is pain, which may be severe and disabling, but paresthesia and numbness can also occur. Nocturnal cramping is common, and occasionally there is subjective coldness below the knee and in the foot. **(Staal et al., 1999; Sidey, 1969)** Usually the condition is unilateral, but occasionally it can be bilateral. **(Moller & Kadin, 1987)**

By way of the superficial and deep peroneal nerves the common peroneal supplies the dorsiflexors and everters of the foot, the skin on the lateral portion of the leg and dorsal portion of the foot, a portion of the ankle joint, and the mid–tarsal joints. **(Moore et al., 2009)** When entrapment is at the level of the common peroneal, symptoms and objective tests will be present in the distribution of both the superficial and deep peroneal nerves. Travell and Simons **(Travell & Simons, 1992)** note that when symptoms of neural entrapment of the peroneal nerves are present, the peroneus longus muscle must be evaluated and treated due to its ability to compress neural structures. The ankle jerk is typically normal with disorders of the peroneal nerve. If this reflex is decreased in combination with inhibition of the foot dorsiflexor muscles on manual muscle testing, the disturbance is more likely to be proximal, in the course of the sciatic nerve or in combination with the L5 and S1 nerve roots. A diminished ankle jerk indicates involvement of the S1 nerve root or sciatic nerve. Because sensory deficits in lumbosacral nerve root lesions are vaguely demarcated, they are not very helpful in distinguishing between entrapments of the peroneal nerve and the L5 nerve root. EMG studies are helpful in making this differential diagnosis. **(Staal et al., 1999)** CT or MRI scans may well show a disc protrusion, but such abnormalities are very common in asymptomatic people. **(Jensen et al., 1994)**

Entrapment of the common peroneal nerve may be associated with various types of athletic activities. **(Mitra et al., 1995)** Turco **(Turco, 1987)** comments on factors that are predisposing to this type of involvement. "The peroneal nerve winds around the neck of the fibula and is closely adherent to the bone. The biceps tendon and lateral collateral ligament are lax in flexion, hence stability of the fibular head is correspondingly less in the flexed knee position. Slight rotary and axial motion of the head of the fibula occurs with ankle motion. In some individuals the head of the fibula is more prominent, hypermobile, and laterally positioned. Runners with generalized joint laxity, hypermobility of the proximal tibiofibular joint, and hyperextension of the knees appear predisposed to develop peroneal neuritis associated with overuse." Some athletes with peroneal neuritis have a genu varum that predisposes the nerve to tension. Symptoms may develop only after running or other exercise. In this case, the patient should be examined before and after exercise. **(Leach et al., 1989)**

Under normal conditions, the common peroneal nerve at the neck of the fibula is not sensitive to palpation or to gentle percussion. When there is entrapment, it generally shows a local tenderness. The major symptoms may be summarized as follows: pain, paresis, paresthesia and numbness, and nocturnal cramp. **(Russell, 2006; Sidey, 1969)**

Stretching the nerve usually increases pressure on it at the fibular head entrapment site. Diagnostic stretch may be done by straight leg raise, which may cause proximal and distal pain in the presence of common peroneal entrapment. Inversion and plantar flexion of the foot may increase pain from the nerve being stretched taut against the fascial edge near the fibular head. Pain may not be localized to the distribution of the peroneal nerve. There may also be antidromic impulses causing sciatic nerve pain. **(Mense & Simons, 2001; Kopell & Thompson, 1976)**

There may be post–traumatic autonomic dystrophy (causalgia) in association with neuropathy of the peroneal nerve. When observing x–rays look for osteoporosis, which is associated with causalgia. **(Mense & Simons, 2001; Kopell & Thompson, 1976)** Peroneal nerve irritation may also be associated with a popliteal (Baker's) cyst, peroneal nerve cyst, compression by a sesamoid bone in the gastrocnemius muscle, or a bunching of fibers from an avulsed or ruptured peroneus longus muscle. **(Travell & Simons, 1992)**

Kopell and Thompson **(Kopell & Thompson, 1960)** find that this condition is often misdiagnosed as subtalar traumatic synovitis or continuing subluxation of the talus in the ankle mortise under inversion stress. They recommend treatment with a lateral sole wedge to relax the peroneal musculature and nerve.

Ironically, muscles supplied by the deep and superficial peroneal nerves often test weak with applied kinesiology initially; then after the "weakness" is corrected with the five factors of the IVF, they test positive to the muscle stretch reaction, indicating a shortened muscle. This often occurs with the peroneus longus and brevis. Delay in the peroneal muscle's onset times during contraction has been observed when this muscle is injected with an irritant. **(Richie, 2001)** Generally, the timing of activation of synergistic muscle groups during co-contraction and reciprocal activation of muscles are also affected in injury and pain, and this usually produces muscular inhibition. **(Lederman, 2010)**

The most common finding causing weakness of the muscles is a subluxation of the fibular head on the tibia. In the presence of a weak peroneus longus and brevis, one can challenge the proximal fibula to find the optimal vector that creates strength in the peroneus longus and brevis. The fibular head is usually posterior, and it needs to be adjusted in a generally anterior direction. Take care to determine the exact direction by vector challenge. It is important when evaluating for fibular head dysfunction that the distal articulation of the fibula be carefully examined as well at the ankle joint.

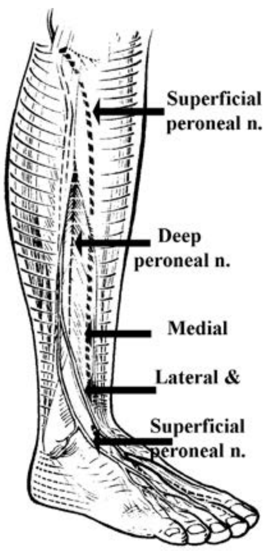
Heidrich **(Heidrich, 1993)** offers a case-report on a

50-year-old physically active male with left leg neuralgia of 3 months duration due to a fibular head subluxation. Symptoms began as a burning sensation that extended from the lateral knee to the dorsum of the foot. Analgesics, anti-inflammatories, physical therapy, and orthopedic treatments provided little relief and the patient was on Percocet® to control pain. Lumbar MRI was negative for discopathy. EMG and nerve conduction velocity tests were positive for left deep peroneal radiculopathy. Orthopedic testing of the knee was unremarkable, with a left genu valgus present. Palpatory pain was elicited at the anterosuperior border of the fibular head. Manual muscle testing showed weakness of the left sartorius, tensor fascia lata, popliteus, anterior tibialis, and peroneus tertius muscles. An AK posterior to anterior challenge of the fibular head produced weakness in a previously strong indicator muscle, indicating an anterior displacement. This displacement was manipulated and fascial release was performed on the tensor fascia lata muscle. Within 9 days there was resolution of the deep peroneal nerve symptomatology.

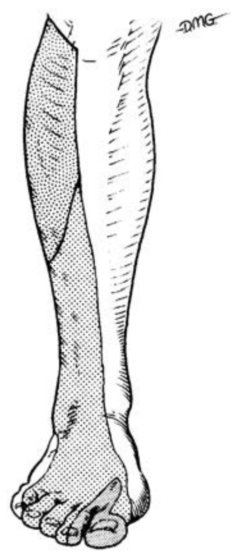
When the peroneus longus and brevis test strong in the clear, evaluate for muscle stretch reaction. When positive, the treatment usually effective is percussion, trigger point pressure release, or stretch and spray methods. **(Leaf, 2010; Cuthbert, 2002; Walther, 2000)** Following treatment for muscle stretch reaction, there will usually be an increased range of motion in foot inversion combined with plantar flexion. There should also be reduction of pain caused by stretching the nerve with inversion and plantar flexion. All muscles of the foot, leg, and knee should be evaluated and corrected as appropriate. There is frequently extended pronation in this condition.

Superficial Peroneal Nerve

The superficial peroneal (musculocutaneous) nerve branches from the common peroneal deep to the peroneus longus. It supplies the peroneus longus and brevis muscles. In the distal lateral portion of the leg, the nerve divides into two branches before or after piercing the deep fascia. It is at the point of passage through the fascia that entrapment neuropathy may occur. **(Chaitow & DeLany, 2002)**



Superficial and deep peroneal nerves



Dermatomes of common superficial and deep peroneal nerves

The medial branch of the superficial peroneal supplies the skin of the medial side of the 1st toe, and the skin between the 2nd and 3rd toes. The lateral branch has dorsal digital branches that supply the skin between the 3rd through 5th toes and the skin over the lateral side of the ankle.

The nerve is strictly sensory from the deep fascial point on; consequently, entrapment at that area can only result in pain and altered sensation. There will be no associated muscle weakness. Digital pressure and palpation of the nerve will probably increase the pain. Antidromic impulses may cause pain in the proximal leg.

Inversion or plantar flexion trauma stretches the superficial peroneal nerve as it passes through the fascia, or at the nerve's anchor at the fibular neck. This type of trauma often causes a weakness of the peroneus longus and brevis. **(Leach et al., 1989; Kopell & Thompson, 1976)** Applied kinesiology challenge will usually identify a fibular head subluxation relating with common peroneal nerve entrapment. This seems to perpetuate the weakness of the peroneus longus and brevis, making the individual subject to the recurrent trauma of "twisting an ankle," usually in the direction of inversion. When the fibular head is adjusted, the peroneus longus and brevis will usually immediately test strong. Effective treatment of this articulation upon global muscle and joint dysfunctions have been recorded. **(Zatterstrom et al., 1994)**

Metatarsalgia of the 2nd and 3rd metatarsals may develop as a result of poor function of the peroneus longus muscle. Part of its insertion is to the base of the 1st metatarsal. Weakness causes a failure of the 1st metatarsal to flex during the last phase of stance, shifting weight to the 2nd and 3rd metatarsals. **(Dananberg, 2007; Lusskin, 1982)**

When there is structural imbalance, the fascia lata may be tight and, because of its continuity with the deep fascia of the leg, cause tension on the portion of the fascia through which the superficial peroneal nerve passes. Here again we see the continuity of fascia throughout the body. Kopell and Thompson **(Kopell & Thompson, 1976)** point out that fasciotomy of the fascia lata has been done in the past with some success in reducing irritation of the superficial peroneal nerve. They do not recommend a revival of the procedure; they just note the mechanism. Staal et al. **(Staal et al., 1999)** recommend surgery for the peroneal nerve only in three instances: 1) penetrating trauma, 2) with local mass lesions, and 3) with compartment syndromes. A tight fascia lata can be identified by Ober's test. **(Ober, 1987)** The tensor fascia lata should be tested for muscle stretch reaction and treated with percussion, trigger point pressure release, or stretch and spray methods if positive. **(Leaf, 2010; Cuthbert, 2002; Walther, 2000)** This will often eliminate a positive Ober's test.

The dorsal cutaneous nerves of the superficial peroneal nerve can be compressed by tight shoes. This is especially a problem with running shoes **(Abshire, 2010; Maffetone, 2010; Krissoff & Ferris, 1979)** and with ski boots. **(Whitesides, 1982)** This is corrected by a larger shoe, a shoe that laces over a wider portion of the foot, or one that has fewer eyelets (three or four) that do not cross over the area of entrapment. One can also skip the eyelets over the area of entrapment when lacing the shoes.

Deep Peroneal Nerve

The deep peroneal (anterior tibial) nerve begins at the bifurcation of the common peroneal nerve between the fibula and the proximal part of the peroneus longus muscle. It passes down the leg between the extensor digitorum longus and the tibialis anterior to the inferior extensor retinaculum.

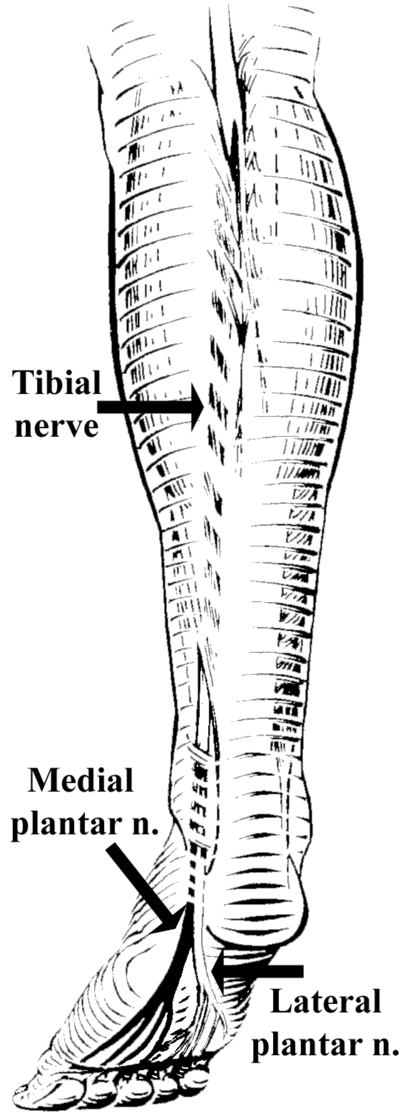
In the leg the deep peroneal nerve supplies muscular branches to the tibialis anterior, extensor hallucis longus, extensor digitorum longus, and peroneus tertius. Deficiency of these muscles is caused by entrapment at the common peroneal nerve or higher. There is rarely an involvement of the deep peroneal nerve itself to affect these muscles. The deep peroneal nerve also has an articular branch in the leg to the ankle joint.

Entrapment of the deep peroneal nerve may occur in the anterior tarsal tunnel. This is a deep passage on the dorsum of the foot covered by the inferior extensor retinaculum, which extends from the lateral to the medial malleolus. After passing through the tunnel, the nerve divides into medial and lateral branches. The medial branch supplies the skin between the 1st and 2nd toes and the 1st dorsal interosseous muscle. The lateral branch innervates the extensor digitorum brevis and the lateral tarsal joints. **(Moore et al., 2009)**

The nerve on the dorsum of the foot, especially over the tarsal bones, is subject to trauma that can be caused by a striking blow or ill–fitting shoes. Trauma that stretches the nerves, such as severe plantar flexion and/or inversion, can also cause neuropathy locally in the foot or further up in the peroneal nerve. An increase of pain is present with stretching of the nerve by forced plantar flexion of the foot and toes. This is not specifically a lesion of the peroneal nerve in the foot, because stretching applies traction in which the common peroneal nerve is anchored to the fibular neck. When only the deep peroneal nerve is involved, there will be dorsiflexion weakness but strength on eversion. In addition, there will be sensory loss between the 1st and 2nd toes. Pressure over the area of suspected neuropathy should cause both local pain and reproduction of the radiated pain.

Tibial Nerve

The tibial nerve is the larger terminal division of the sciatic nerve. Its source consists of the 4th and 5th lumbar and 1st, 2nd, and 3rd sacral nerves. As it descends the leg it is well–protected by muscle. **(Moore et al., 2009; Russell, 2006)** It descends through the popliteal fossa and continues in the leg with the posterior tibial vessels to enter the tarsal tunnel. The nerve splits into the medial and lateral plantar nerves within the tarsal tunnel 93% of the time and proximal to it the other 7%. It also gives off the calcaneal nerve, the origin of which varies. **(Andreasen Struijk et al., 2010)** Prior to the tarsal tunnel there is minimal or no incidence of peripheral nerve entrapment because of the excellent protection provided the nerve by the muscles.



Tibial Nerve.

When there is entrapment of the tibial nerve in the leg, its source is often a deep posterior compartment syndrome. **(Lusskin, 1982)** Compartment syndromes are associated with muscle activity in which the contents of the compartment enlarge past the closed compartment's ability to yield. An acute compartment syndrome can be a surgical emergency; it must be differentially diagnosed from other conditions.

Tarsal Tunnel Syndrome

The first description in the literature of the tarsal tunnel syndrome was in 1962, when two operative cases were reported. **(Lam, 1967)** Lam indicates that in the early investigation of the carpal tunnel syndrome, advanced cases showed marked motor and sensory disturbance; at operation, pathological changes of the median nerve were evident. Tarsal tunnel syndrome is also analogous to the carpal tunnel because the tunnel contains tendons and blood vessels together with the tibial nerve. Lam states in his paper of 1967, "Nowadays the carpal tunnel syndrome is sufficiently well recognized to ensure that most cases are treated before this stage is reached. The same pattern of events may evolve in the case of the tarsal tunnel syndrome." Unfortunately, there are still a large number of people with unrecognized entrapment at the tarsal tunnel. Many physicians overlook the tarsal tunnel syndrome. **(Hudes, 2010)** Alshami et al. **(Alshami et al., 2007)** and Keck **(Keck, 1962)** note that tarsal tunnel syndrome is frequently diagnosed as acute foot strain or plantar fascitis. In applied kinesiology experience, many patients who have previously been unsuccessfully treated for foot dysfunction are found to have functional tarsal tunnel syndromes. This allows the plantar intrinsic muscles to become weak and causes increased development of extended pronation affecting total body function. **(Mondelli et al., 2004)**

The term "functional tarsal tunnel syndrome" was used above to differentiate from frank pathological entrapment of the tibial nerve, which may require surgical decompression. The functional tarsal tunnel syndrome highlighted in this discussion is the type of nerve entrapment that causes dysfunction, yet will respond and return to normal with conservative treatment. Since Goodheart's **(Goodheart, 1971)** introduction of tarsal tunnel treatment into applied kinesiology, many have been treating it successfully. **(Hudes, 2010; McDowall, 2004; Hambrick, 2001; Cuba et al., 1995)** With an increased awareness of this type of tarsal tunnel syndrome we may yet treat these conditions before marked motor and sensory disturbance with pathologic change develops.

Anatomy

The tarsal tunnel is a superficially located osseous tunnel behind and below the medial malleolus and covered by the flexor retinaculum (laciniate ligament) with the bones making up the base of the tunnel. The tibial nerve passes through this osteofibrous passageway with the tendons of the tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles, each within its own synovial sheath. The other components of the neurovascular bundle are the posterior tibial artery and vein. **(Gray's Anatomy, 2004; Goodgold et al., 1965)**

The flexor retinaculum extends between the malleolus and the medial side of the calcaneus but has several deep fibrous septa that blend with the periosteum of the calcaneus. The neurovascular bundle in the tarsal tunnel is often attached to some of these septa, rendering itself more liable to minor degrees of traction on movement of the foot. **(Henricson & Westlin, 1984)**

Vascular supply to the nerve may have a bearing on its susceptibility to compression. As in the carpal tunnel syndrome, the median nerve in the wrist and the posterior tibial nerves have better arterial supply than the ulnar and lateral tibial nerves. The ulnar and lateral tibial nerves rarely have "spontaneous" compression symptoms, though they run through osteofibrous tunnels. The median and tibial nerves have a much more common incidence of "spontaneous" compression. Their ample arterial blood supply may make them more susceptible to the effects of localized vascular insufficiency. Lam **(Lam, 1967)** states, "When exploring the posterior tibial nerve one is struck by the density of the areolar tissue binding the structures under the retinaculum, and by their relative lack of mobility — compared with the extreme mobility and lack of adherence of the median nerve in the carpal tunnel. Hence even slight degrees of compression, possibly caused by oedema following minor strains, may produce vascular insufficiency locally and render a nerve lesion more likely."

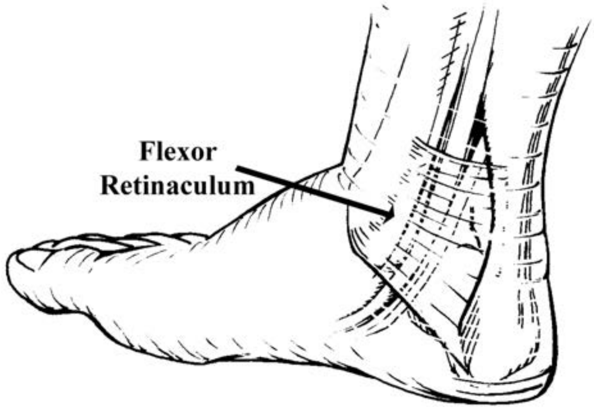
It is suggested by Mense and Simons, and Butler **(Mense & Simons, 2001; Butler, 2000)** that sensory symptoms in nerve compression syndromes are partially due to arterial insufficiency. More slowly occurring motor paralysis is thought to be due to later structural changes produced within the nerve, and the paralysis is less likely to benefit from decompression. Manipulative treatment is known to have an effect on the diameter of arteries and veins. **(Rome, 2010, 2009)** It is important therefore to make the diagnosis and treat the patient before the onset of demonstrable motor involvement resulting from ischemia. Fluid flow through a blood vessel is strongly affected by small changes in the diameter of the vessel, demonstrating how even a small swelling or compression around the neural tissues will severely reduce the flow of blood and lymph in the area. **(Zink, 1977)**

In Lam's **(Lam, 1967)** early series of ten cases surgically treated, one case had enlarged tortuous veins within the tarsal tunnel. There was no other demonstrable pathology in any of the cases. This points out the necessity in an applied kinesiology examination of determining whether actual paralysis develops in the muscle from nerve impingement, or whether there is functional weakness as observed in the manual muscle test. This is readily determined by challenge and therapy localization. If one is unable to return muscle function to normal, as observed by manual muscle testing, surgical intervention may be necessary. Hoskins et al. **(Hoskins et al., 2006)** and Denny–Brown and Brenner **(Denny-Brown & Brenner, 1944)** have shown that when peripheral nerve entrapment is treated in its early stages, it is reversible.

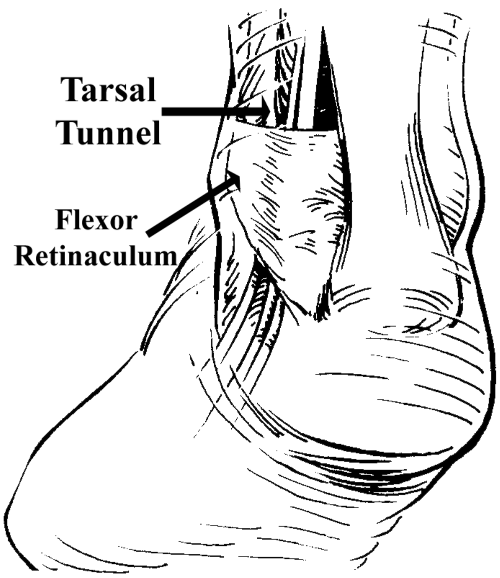
Extended pronation **(See chapter 4)** is a factor in nearly all cases of tarsal tunnel syndrome treated by applied kinesiologists. **(Leaf, 2010; Goodheart, 1998)** Because of the posterior movement of the calcaneus on the talus during extended pronation, the flexor retinaculum is stretched. In extended pronation there is intermittent compression, then release, then compression, then release of the laciniate ligament on the posterior tibial nerve. **(Rosson et al., 2009; Carrel & Davidson, 1975)**

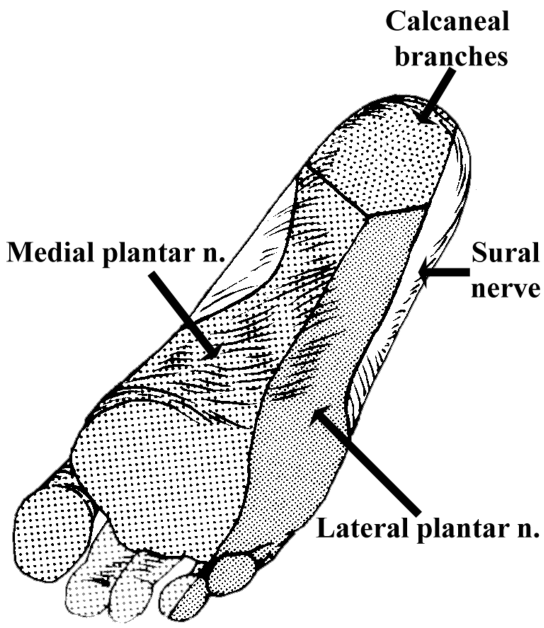
Symptoms

Tarsal tunnel nerve entrapment can be discovered by an astute examiner when the patient has no complaint of foot or ankle dysfunction. As discussed in **Chapter 4**, foot dysfunction plays a major role in total body organization.

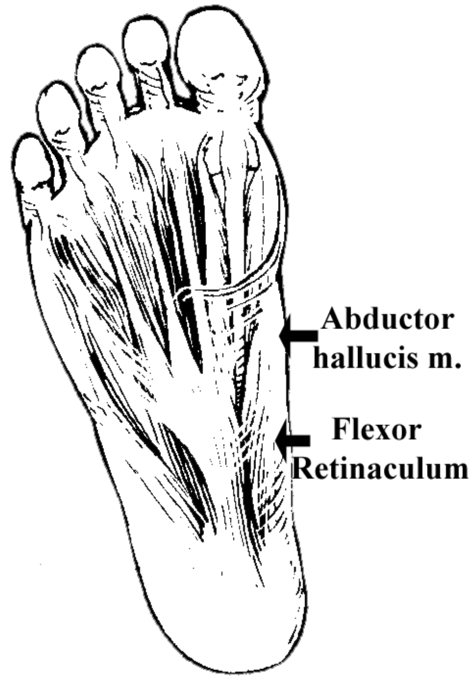


Tarsal tunnel - medial & porteromedial niews





Dermatomes of plantar surface of foot



Plantar view

Recognizing an asymptomatic tarsal tunnel entrapment is just as important as examining and finding the cause of the painful foot.

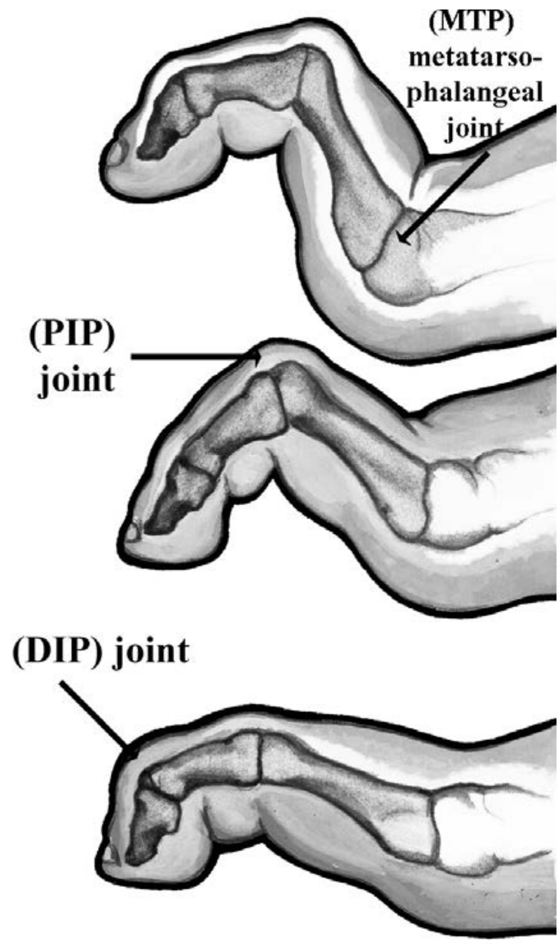
Patients' complaints of tarsal tunnel syndrome include burning pain and paresthesia in the toes and along the sole of the foot. This may cause "burning feet" or the "restless legs syndrome." **(Staal et al., 1999)** One study reports that in 43% of cases the pain is more severe at night. **(Mondelli et al., 2004)** Moving the limb, getting out of bed, or hanging the limb over the edge of the bed may provide relief. **(Langer, 2007)** Transitory nerve ischemia or compression may be relieved by massage or walking. **(Barral & Croibier, 2007; Edwards et al., 1969)**

Proximal pain, tingling and numbness may radiate up the leg from a tarsal tunnel syndrome and is seen in approximately 30% of cases and is called the Valleix phenomenon. **(Lau et al., 1999)** It may simulate a disc problem, peripheral vascular disease, or neuritis. Sometimes the pain is attributed to an existing condition, such as diabetes or peripheral vascular disease, when the problem is really an unassociated tarsal tunnel syndrome. This error frequently occurs with older patients. This is why tarsal tunnel syndrome may be under-diagnosed because it can be difficult to differentially diagnose it from other conditions of the foot. For these reasons, specific as compared to group manual muscle tests are an important advantage in the diagnosis of specific articular-muscular impairments. **(Leaf, 2010; Walther, 2000; Goodheart, 1998; Kendall et al., 1993)**

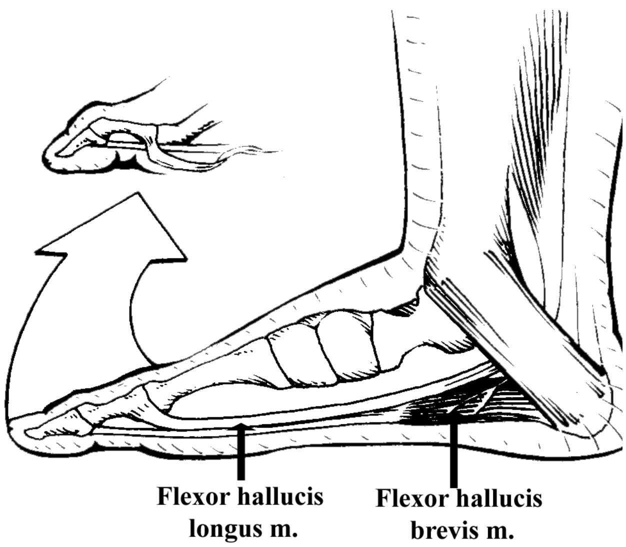
Many aspects of an examination may appear normal when there is entrapment at the tunnel. There may be normal dorsalis pedis and posterior tibial pulses. Skin color, hair distribution, and capillary circulation may also appear normal. **(Keck, 1962)**

With chronicity the plantar muscles of the foot may be atrophied, giving an appearance of a high arch. There is indication in the literature that sensory deficit develops prior to motor deficit; **(Lusskin, 1982)** this does not agree with applied kinesiology findings nor with more recent neurophysiological research. When muscles are painful, fatigued, or injured, there is an inhibition of muscle strength, timing, and a decrease in their endurance. **(Racinais et al., 2008; Mense & Simons, 2001)** In any exercise inducing muscle damage, decreased neural drive to the muscles is thought to be an attempt of the neuromuscular system to protect the muscle-tendon unit from additional damage. **(Nicol et al., 2006; Strojnik & Komi, 2000)** For this reason muscular imbalance is thought to be a primary reflection of the functional state of the neuron; this is particularly true because of the extreme sensitivity of the muscle spindle cells, where a muscle spindle reacts to a pull of only 1 gram and a stretch of $1/1000^{th}$ millimeter. **(Korr, 1997, 1979)** This makes the muscular system and the manual muscle test an extremely sensitive organ and tool.

One often finds severe plantar muscle atrophy with no sensory deficit or hyperesthesia. **(Mondelli et al., 2004)** The patient with tarsal tunnel entrapment will have weak plantar muscles, and the long toe flexors will probably be strong. This, of course, is because the long muscles receive their nerve supply prior to the tarsal tunnel, and the intrinsic muscles after the point of entrapment. With this muscle imbalance, the patient will have hyperextension at the metatarsophalangeal articulations and hyperflexion at the interphalangeal articulations, giving a claw–like appearance to the toes, which is often called "claw toes." **(Lau et al., 1999)** This is because the long muscles insert into the distal phalanx, and the intrinsic muscles into the intermediate phalanx. When the individual stands or walks, one can see the distal phalanx gripping the substrate with elevation of the proximal phalanges. There will often be calluses on the distal ends of the toes.



*(With kind permission, **ICAK-USA**)*



Insertion of muscles to the toes.

Claw Toes

3

Etiology

Often there is no known etiology for functional tarsal tunnel syndrome. Space–occupying lesions such as ganglions or tumors are rare. **(Taguchi et al., 1987)** Even in a surgical series of sixteen cases reviewed by Edwards et al., **(Edwards et al., 1969)** eight of the cases were "spontaneous" entrapment, that is, no space–occupying lesion was found. Five were post–traumatic fibrosis due to fracture, three had accessory or hypertrophied abductor hallucis muscle, and one was due to tenosynovitis.

Tenosynovitis within the tarsal tunnel can develop in runners, **(Abshire, 2010)** from trauma, **(Kopell & Thompson, 1976)** or from infectious processes. **(Kopell, 1980)** The latter, in effect, causes a space–occupying lesion to produce pressure on the nerve. There will be crepitation at the tunnel and severe pain on digital pressure over the tendons.

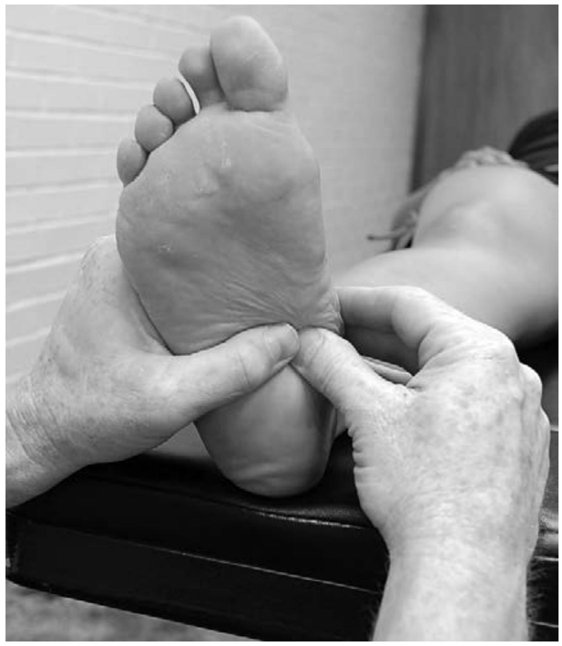
Circulation problems may be responsible for tarsal tunnel syndrome. Peripheral nerves in a diabetic are more easily affected by compression force than are normal nerves. **(Veves et al., 2002)** Venous engorgement of the tunnel can develop as a result of proximal venous obstruction or valvular deficiency. **(Goodgold et al., 1965)** Some of the pain present with an obvious venous stasis or thrombophlebitis may be caused by encroachment on the posterior tibial nerve rather than pressure from the vein distension alone. **(Alshami et al., 2008)**

Any condition that creates fluid retention such as the ileocecal valve syndrome can be the final factor that causes entrapment at the tarsal tunnel. Helm et al. **(Helm et al., 1971)** studied 164 pregnant women to determine if there is an increased incidence of tarsal tunnel syndrome during pregnancy. They found 56.1% had abnormal nerve conduction through the tarsal tunnel. Twenty of the subjects were studied six weeks postpartum. Thirteen of these had abnormal conduction studies of the tibial nerve during the course of pregnancy, and twelve (92.4%) reverted to normal values at postpartum follow–up. Of the subjects with abnormal studies, 69.5% had some complaint of leg cramps, burning of feet, tingling, numbness or pain during the pregnancy.

Tarsal tunnel syndrome must be differentially diagnosed from interdigital neuritis, dropped metatarsal heads, plantar calluses, arch strain, various types of arthritis, tenosynovitis, peripheral neuritis, peripheral vascular disease, and various causes of sciatic pain. **(Staal et al., 1999; Kopell & Thompson, 1976; Aguayo, 1975; Lam, 1967; Goodgold et al., 1965)** Antidromic impulses can cause tenderness along the entire sciatic nerve. The pain may simulate root pain of spinal origin. **(Russell, 2006; Staal et al., 1999; Ricciardi–Pollini et al., 1985)** Moloney **(Moloney, 1964)** reports on a case where three spinal surgeries were done without permanent improvement of pain until the tarsal tunnel was diagnosed and operated, which eliminated the painful condition. Patients who have a systemic propensity toward peripheral entrapment may have a history of problems in other areas such as a previous carpal tunnel syndrome. **(Mondelli & Cioni, 1998; McGill, 1964)**

Examination

When there is frank entrapment of the tibial nerve in the tarsal tunnel, the Tinel sign is sometimes present over the tunnel or the medial arch. One study reports that Tinel's sign is positive in only 67% of cases. **(Mondelli et al., 2004)** The Valleix phenomenon or nerve trunk tenderness may be present proximal or distal to the area of entrapment. **(Lau & Daniels, 1999)** Palpation over the retinaculum may reveal tenderness or a small fusiform swelling of the nerve. **(Barral &**



Abductor hallucis muscle palpation

The neurodynamic test places the entire neuraxis under tension. The tibial & peroneal nerves' tension is felt at the head of the fibula.

Croibier, 2007) Hypo– or hyperesthesia may develop in the tibial nerve branches (medial and lateral plantar nerves), or the calcaneal branches. Sensory disturbance should only be in this distribution, and it should not affect the dorsum of the foot except over the distal phalanges of the toes. There may also be loss of two–point discrimination. Pressure on the calf of the leg created by inflating a sphygmomanometer cuff may reproduce symptoms on the affected side more quickly than in a normal foot. **(Butler, 1991; Cipriano, 1985; Henricson & Westlin, 1984)**

There must be careful differentiation in cases of leg pain. In a series of thirteen patients, seven of whom were operated and four being treated conservatively but expected to go to operation, Mann **(Mann, 1974)** found over half the patients had pain radiating up the medial side of the calf but not past the knee. In one patient the sharp pain in the plantar aspect of the foot could be reproduced by straight leg raising and dorsiflexion of the patient's foot.

Maneuvering the heel into a valgus position narrows the tarsal tunnel and may increase pain; on the other hand, maneuvering the heel into a varus position may reduce pain. **(Butler, 2000; Staal et al., 1999; Kopell & Thompson, 1976)** Neurodynamic testing **(Butler, 2000, 1991)** employs this type of nerve tension testing. When a neurodynamic tension test is positive (i.e. pain or other sensations result from one or another element of the test – for example, the initial position alone, or with 'sensitizing' additions) it indicates that there exists abnormal mechanical tension (AMT) somewhere in the continuous nervous system, but not that this is necessarily at the site of reported pain. For more detail of this assessment and treatment approach, the books by Butler **(Butler, 2000, 1991)** are recommended.

Patients may not complain of muscle weakness in a tarsal tunnel syndrome; **(Aguayo, 1975)** however, muscle deficiency will be found if carefully examined for by inspection, palpation, and specific muscle testing and AK sensorimotor challenges. The abductor hallucis located along the medial longitudinal arch is inspected and palpated for atrophy, along with the other intrinsic muscles of the foot. The ability to flex the toes at the metatarsophalangeal articulation is evaluated by muscle testing. There is no weakness of toe extensor muscles.

The flexor digitorum longus and brevis and the flexor hallucis longus and brevis should be evaluated. The long muscles will have improved function over the short ones. Challenge, usually directed to the calcaneus, may improve the function of the intrinsic muscles. There will be positive therapy localization over the tarsal tunnel at the area of entrapment, and probably at the subtalar articulation, making an immediate and non-invasive diagnosis of this disorder readily available to the clinician.

Electrodiagnosis should be done to determine nerve conduction in cases that are unresponsive to conservative care; however, there may be false–positive findings from these studies. Gatens and Saeed **(Gatens & Saeed, 1982)** studied the adductor hallucis, extensor digitorum brevis, 1st dorsal interossei, and abductor digiti minimi in seventy individuals with asymptomatic feet. They found that 38.6% had at least one of the four muscles examined showing abnormal potentials. They concluded that using abnormalities in intrinsic foot muscles by needle EMG as a diagnostic criterion could be misleading. An interesting question regarding studies like these is what an applied kinesiology examination would have found in the individuals with positive tests. Functional problems are often found in asymptomatic feet. **(Abshire, 2010; Maffetone, 2010)** It is possible in studies like these that the positive findings were in feet that were functionally inadequate but asymptomatic. If the muscles of the foot and ankle are dysfunctioning, the lower legs, knees, thighs, hips, lower back, shoulders, neck and head are also disturbed. **(Maffetone, 2010; Dananberg, 2007; Chaitow & DeLany, 2002; Walther, 2000; Travell & Simons, 1992)**

Treatment

Most cases of tarsal tunnel syndrome respond well to the conservative approach of applied kinesiology. If there are neoplasms or other space–occupying lesions that are irreversible with conservative care, surgical intervention will be necessary.

Most often accompanying and frequently the precipitating factor for tarsal tunnel syndrome is extended foot pronation. The first effort toward correction is to examine for and correct extended pronation, which includes any subluxations of the foot and other foot dysfunction. Intrinsic muscles of the foot may not be corrected until specific adjustments are made for the tarsal tunnel because their nerve supply is being interfered with. **(See Chapter 4)** One of the most common forms of muscle inhibition comes from joint subluxations, commonly called arthrogenic weakness. Even non-noxious stimulation of the joints of the foot and ankle can elicit strong inhibition of the muscles of the foot and ankle. **(McVey et al., 2005)** There are few better options for patients with joint subluxations of the foot and ankle affecting the tarsal tunnel than specific manipulative joint correction. Non-manipulative treatments for the arthrogenic weakness of muscles will be less effective and more time-consuming when joint disturbances are present.

After correcting pronation, challenge the calcaneus in its relationship to the talus. The calcaneus will usually be subluxated posterolaterally, with its posterior surface somewhat superior. There are several methods for adjusting the calcaneus; it can be done with the patient supine or prone.

With the patient supine, the physician stands at the patient's feet facing the foot to be corrected. To adjust the calcaneus, the physician grasps the posterior superior surface of the bone solidly with his or her hand. The left

Calcaneus adjustment

Pain from calcaneus malposition & foot pronation.

hand makes a broad contact across the dorsal surface of the foot. A broad contact is important to avoid creating a subluxation with this stabilizing and controlling hand. The correction is an extension thrust directed toward moving the posterior surface of the calcaneus in a generally inferior anterior direction. The vector of force is determined by challenge. It usually requires moving the inferior portion of the calcaneus medially. It is often necessary to have the patient hold the side of the examination table to prevent slipping, especially if the table is covered with tissue paper. There is often an audible release of the calcaneus; however, it is not necessary for an adequate correction.

Another method is to flex the prone patient's knee to 45°. The physician stands on the side to be corrected. To correct the right calcaneus, the physician's right hand cradles the superior posterior aspect of the calcaneus between his thumb and forefinger. The other hand cradles the dorsum of the foot. The thrust is directed to the calcaneus as previously determined by challenge. Usually the thrust is directed to move the posterior inferior aspect of the calcaneus in an anterior, inferior, and medial direction.

Successful adjustment of the calcaneus and pronation correction are indicated by relief in the painful area inferior to the medial malleolus. Goodheart **(Goodheart, 1971)** states that the disappearance of this diagnostic feature is essential. It may be necessary to adjust the calcaneus several times to completely eliminate this pain.

Leaf **(Leaf, 2010)** points out that the navicular bone is a key in the correction of tarsal tunnel syndrome and other disorders of the foot. The navicular is controlled by the tibialis posterior and when the navicular drops inferiorly from tibialis posterior inhibition, the alignment and dynamics of the rest of the foot is disturbed. As the navicular descends it fails to support the talus and thereby causes the calcaneus to shift posteriorly. The posterior calcaneus that results produces increased pressure on the flexor retinaculum. The initiating disturbance in this sequence is the inferior navicular and it must be corrected.

When tarsal tunnel syndrome is associated with tendinitis, as from jogging, it is treated with ice. **(Redwood & Cleveland, 2003)** Correction of any foot, leg or other problems associated with the running pattern should be done to prevent recurrence.

The intrinsic plantar muscles should be individually evaluated. They often require treatment to the muscle proprioceptors, or need origin–insertion technique, or fascial release. The muscles are often exquisitely tender in the area requiring treatment. It may be of value to use the percussor or vibrator unit for treatment. **(IMPAC, 2011)**

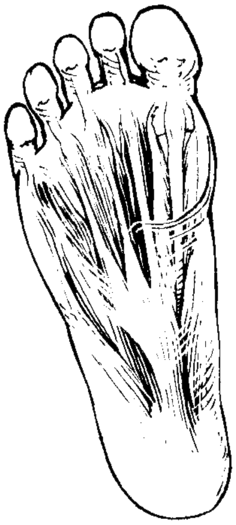
It is very important to follow up with proper treatment of the extended pronation. Failure to adequately stretch the triceps surae if necessary, adjust subluxations or fixations, or to provide proper footwear will result in failure to permanently correcting the tarsal tunnel syndrome.

When conservative treatment fails, surgical release is often effective **(Rosson et al., 2009; Murphy & Baxter, 1986)** if proper diagnosis has been made. Surgical release may find a small tag or tags of tissue **(Lam, 1962)** often secondary to trauma. **(Edwards et al., 1969)** At the point of entrapment there may be enlargement of the tibial nerve, **(Keck, 1962)** with poor nerve conduction with electrical stimulation past the point of constriction. In a group of sixteen patients who required surgical decompression, Edwards et al. **(Edwards et al., 1969)** found that "Local injection of cortisone produced only transient, if any, improvement. Shoe modifications provided no improvement and arch supports always increased the severity of symptoms." Burning feet from tarsal tunnel entrapment do not improve with vitamin treatment. **(Babbage, 1965)** When there is a sensation of burning feet without the nerve entrapment, they often respond to vitamin B–complex. **(Cuthbert, 2007; Patrick, 2007)** Goodheart recommends vitamin B complex for burning feet; and burning feet accompanied by swelling in hot water needs thiamine. **(Goodheart, 1991)**

Plantar Nerves

The posterior tibial nerve branches into the medial and lateral plantar nerves and the calcaneal nerve in or around the tarsal tunnel. The medial and lateral plantar nerves arise within the tarsal tunnel 93% of the time and proximal to it the other 7%. The origin of the calcaneal nerve varies. **(Moore et al., 2009)** A portion of the calcaneal branch usually pierces the laciniate ligament. The other portion, which is the sensory supply to the skin inferior to the calcaneus, traverses the tunnel. Where the abductor hallucis muscle originates on the calcaneus, there are two openings through which the plantar nerves pass. The calcaneonavicular ligament is above the medial plantar nerve. Above the lateral plantar nerve is the origin of the quadratus plantae muscle on the calcaneus. These two structures form superior boundaries for the foramen and are locations for entrapment neuropathies. Excessive pronation stresses the nerve in the opening, bringing the superior boundaries down against the nerves. **(Langer, 2007; Lau & Daniels, 1999)** Since the medial and lateral plantar nerves go through individual openings, each can be subject to isolated entrapment neuropathy. **(Rosson et al., 2009; Staal et al., 1999)** Thus entrapment can be of an individual plantar nerve, of all three nerves at the tarsal tunnel, or at a higher level.

The calcaneal nerve is purely sensory; the medial and lateral plantar nerves are mixed. **(Moore et al., 2009)** They correlate with the median and ulnar nerves of the hand. The medial plantar nerve innervates the three–and–one–half medial digits, and the lateral plantar nerve the one–and–one–half lateral digits. The lateral nerve passes under the



Intrinsic plantar muscles.

flexor digitorum brevis and supplies the interosseous muscles and the adductor hallucis. The medial nerve innervates the abductor hallucis, flexor digitorum brevis, flexor hallucis brevis, and lumbrical muscles. The medial and lateral plantar nerves terminate as the interdigital nerves.

Extended pronation tightens the medial and plantar nerves against the calcaneonavicular ligament or the fibrous–edged openings of the abductor hallucis muscle. **(Kopell & Thompson, 1960)** In addition, extended pronation is conducive to narrowing of the tarsal tunnel. In both cases, the extended pronation must be corrected to obtain relief with conservative care. The exact location of entrapment is academic unless it is the type requiring surgical release; otherwise treatment is similar for either level of entrapment.

Pain from entrapment neuropathy of the plantar nerves is in the distribution of the nerves. There is tenderness at the point of nerve entrapment. This is located one finger width below and three fingers anterior to the medial malleolus in an adult. Pressure at this location should cause radiation to the forefoot in a painful condition. **(Russell, 2006; Cailliet, 1997)**

If there is sensory disturbance of the skin of the heel the entrapment site is higher, probably involving the tarsal tunnel. **(Hamilton, 1985)** If the heel is not involved in a tarsal tunnel entrapment, it is due to the calcaneal branch arising proximal to the tunnel. **(Staal et al., 1999)**

The more common cause of heel pain is plantar fascitis, which must be differentiated from entrapment of the calcaneal branch of the tibial nerve. In plantar fascitis there is tenderness of the fascia and its attachment to the calcaneus. In nerve entrapment the tenderness is over the medial anterior part of the heel pad, the anterior calcaneal branch of the tibial nerve, and the origin of the abductor hallucis muscle. X–rays of the calcaneus are negative. Stretching of the adductor hallucis and contraction of the flexors increase the pain; it decreases when the muscles relax. **(Travell & Simons, 1992)** Lusskin **(Lusskin, 1982)** discounts the frequency of heel pain being due to plantar spur, placing more interest on nerve entrapment. Applied kinesiology experience concurs with this, finding that functional peripheral nerve entrapment, foot strain, calcodynia (painful heel), and irritation of the plantar fascia are all part of the complex of extended pronation and general foot strain. **(See Chapter 4)** However, there can be frank neuropathy accompanying the more common structural strain cause of pain. **(Russell, 2006)**

Kopell and Thompson **(Kopell & Thompson, 1976)** describe interesting "…cases of a burning, causalgic type of foot pain." Characteristics of the condition are severe pain on standing or walking (to the point of almost complete disability), moderate hallux rigidus, atrophy of medial foot muscles, anteromedial plantar navicular pain, medial first toe hypoesthesia, and a groove on the first cuneiform observed on x–ray. The groove results from the tibialis anterior tendon wearing down the bone. Overactivity of the tibialis anterior was found to be a natural response of muscle activity to counteract extended pronation. With the extended pronation, entrapment of the plantar nerves developed with the subsequent symptomatic pattern. If conservative treatment was ineffective, they found it necessary to do a neurolysis. "The nerve was usually found to lie within a tough, constricted, fibrous sheath with barely enough room for its passage."

Entrapment of the medial plantar nerve can develop as a result of extended pronation while running for long distances. Rask **(Rask, 1978)** emphasizes the role of extended pronation in running problems by stating, "A fibromuscular tunnel for the nerve is formed by the abductor hallucis muscle and eversion of the foot brings the medial plantar nerve into stretch against the tunnel whose superior surface is the navicular tarsal bone." Entrapment has been found to occur to the medial plantar nerve just distal to the tarsal tunnel, at the entrance of the fibromuscular tunnel behind the navicular tuberosity. **(Oh & Lee, 1987)** It is at this point that there will consistently be tenderness on digital palpation when this condition is present. Often there is a burning heel pain associated with the condition. Rask **(Rask, 1978)** relates this to reflex excitation of the medial calcaneal branches that have their origin higher in the posterior tibial nerve. He applies the term "jogger's foot" to this condition.

It is probable that many applied kinesiologists have unknowingly effectively treated entrapment neuropathy of the plantar nerves. The therapeutic approach to two commonly treated conditions is similar to the therapeutic approach for the nerve entrapment. First is acute foot strain where pain is attributed to strain on the calcaneonavicular ligament and plantar aponeurosis. The second is painful heel, which is attributed to strain on the origin of the intrinsic plantar muscles at the calcaneus and the plantar aponeurosis attachment to the calcaneus. Both of these conditions require correction of foot subluxations and extended pronation, as well as returning all muscles to normal function using applied kinesiology techniques. If there is neuropathy of the plantar nerves as they pass under the calcaneonavicular ligament and the quadratus plantae muscle, an arch support may aggravate the condition. **(Kopell, 1980)** If it is impossible to maintain subluxation corrections, that is, the patient loses the correction as soon as he walks, it may be necessary to temporarily use tape support to the foot. The tape can supply a wider support than the arch support, preventing direct pressure on the area of nerve entrapment. As the condition is corrected, the patient may no longer need arch supports to maintain the correction, or he will be able to tolerate them if needed.

The medial and lateral plantar nerves terminate as the interdigital nerves. They pass over the transverse metatarsal ligament on the plantar surface, and then angle to reach the dorsum of the foot. If the toes are hyperextended at the metatarsophalangeal articulation, the nerves may become irritated and cause pain between the toes. There is increased pain on digital pressure between the toes as opposed to over the metatarsal heads, as in metatarsalgia. This very often happens as a result of wearing high–heeled shoes, or sitting on one's haunches with the toes extended. **(Cailliet, 1997)**

Plantar Interdigital Neuralgia (Morton's Neuroma)

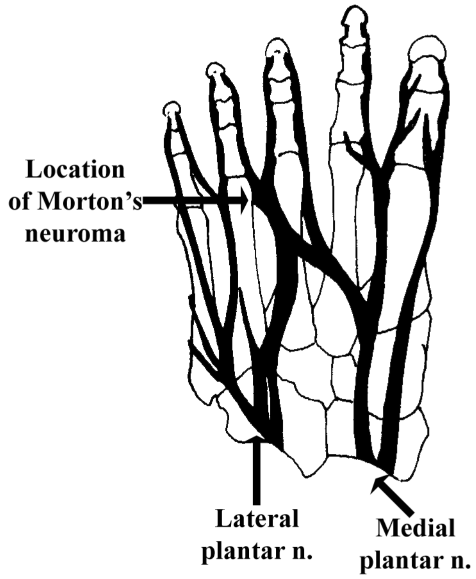
Plantar interdigital neuralgia (Morton's neuroma) is a type of peripheral nerve entrapment not regularly included in an overview of the subject. **(Langer, 2007)** Often misdiagnosed, it may be mistaken for other causes of pain in the forefoot **(Staal et al., 1999)** or for other causes of metatarsalgia.

Plantar interdigital neuralgia was originally described by Thomas Morton in 1876. The condition has been known as Morton's toe, Morton's neuroma, Morton's neuritis, Morton's disease, Morton's affection of the foot, metatarsal neuralgia, and plantar interdigital neuroma. Care should be taken to avoid confusing it with the condition described by Dudley Morton in 1927, which is the 1st metatarsal insufficiency syndrome discussed in **Chapter 4**. The term "plantar interdigital neuralgia" or "neuritis" is chosen here because there is evidence that a true neuroma is not often present in this condition. **(Staal et al., 1999; Viladot 1982; Lusskin, 1982)**

Morton's original treatment for the condition was excision of the 4th metatarsophalangeal joint. In 1891 Bradford **(Bradford, 1891)** produced a paper on "Morton's Affection of the Foot" in which he stated, "It is somewhat singular that an affection which is not infrequent, in these days of thorough investigation of all ailments, should have attracted but little attention either in the researches of surgeons or of neurologists. The cases are usually classed among the ill–defined hysterical or nervous affections, and not thoroughly investigated…." It is interesting that with the passage of time there is still not complete agreement about the etiopathology and optimal treatment for the majority of these cases. Some look at the condition as a neuroma **(Betts, 1940)** with the recommendation of surgical excision, **(Turek, 1984)** while others take the position of microtraumatism to the nerve that walking brings about **(Viladot, 1982)** and treat the condition, in most cases, conservatively. **(Govendor et al., 2007; Brantingham et al., 1994)**

Anatomy

Plantar interdigital neuralgia is a lesion of one of the interdigital nerves of the foot, which arise from the medial and plantar nerves, at the point where these course between the heads of the metatarsal bones, just before they divide into two digital nerves. These lesions are found mostly between



Anastomosis between medial and lateral plantar nerves.

the 3rd and 4th metatarsal bones, particularly in women. **(Staal et al., 1999)** The 2nd cleft is occasionally involved. The condition has been attributed to the anatomical characteristics of the nerve in the involved individual.

Walther points out **(Walther personal communication, 2007)** that Viladot **(Viladot, 1982)** describes an anastomosis between the medial and lateral plantar nerves that is not illustrated in most general anatomy texts. In this case there is an enlargement of the nerve at the common site of Morton's neuroma. He proposes that this may "…explain why it is precisely this nerve that is the most sensitive to the microtraumisms that it receives from walking, especially when it is caught between the metatarsal heads." Turek **(Turek, 1984)** disagrees with this, stating that the digital nerve between the 3rd and 4th toes "…lies in relation to the plantar surface of the transverse ligament and therefore cannot be compressed between the metatarsal heads."

Symptomatic Pattern

The description of plantar interdigital neuroma has been mostly consistent, from Morton's **(Morton, 1876)** early description to the present. In the next major description, Bradford **(Bradford, 1891)** stated, "The symptoms can be removed by kicking off the boot and squeezing the forepart of the foot, especially if the heads of the middle metatarsals are at the same time pressed upwards by the finger. When this is done, a kind of grating is again felt together with a sharp twinge of pain, but almost immediately afterward the pain entirely ceases."

Betts **(Betts, 1940)** subscribes to surgically removing a neuroma. His description parallels many aspects of the early descriptions. A synopsis of his well–known paper is that neuritis of the 4th digital nerve is caused by a neuroma. It is initially characterized by an attack of severe pain while walking with shoes on. Relief is obtained by removal of the shoe, compression of the foot, and flexion of the toes. Upon examination, deep pressure over the site of the nerve does not produce acute pain, only tenderness. This correlates with relief being obtained by compression of the foot, and indicates that stretching of the nerve rather than compression initiates the attacks. The sharp pain of the acute attack is often followed by numbness and tingling. The acute pain may be diffuse, which also applies to the numbness and tingling after the attacks. A dulling sensation is frequently present adjacent to the sides of the 3rd and 4th toes. Passive movement of the foot and toes in various positions does not usually cause any pain. The condition usually occurs in what appears to be a generally healthy foot, perhaps with slight clawing of the toes. It does not occur in the weak, flat foot with poor circulation and calluses under the heads of the middle metatarsals. **(Betts, 1940)**

The fact that squeezing the metatarsals together relieves the acute attack, and that digital pressure over the site of disturbance does not elicit acute pain, differentiates this condition from intermetatarsal bursitis. Not all authors agree with this. Krissoff and Ferris **(Krissoff & Ferris, 1979)** state that transverse compression of the metatarsal heads causes pain. They further recommend confirming the diagnosis with lidocaine (xylocaine) ingestion to determine if it eliminates the pain. It is difficult to see how either of these approaches differentiates the condition from intermetatarsal bursitis.

The pain is not always localized. It can cause a radiation that is sciatic–like, radiating from the outer side of the foot into the calf and posterior thigh. **(Cohen, 1952)**

Many authors concur that this is not a true neoplasm; **(Subotnick, 1991; Viladot, 1982; May, 1976)** rather, it is a traumatic neuritis with proliferation of connective tissue elements.

Some surgeons move immediately to surgery following positive diagnosis of Morton's neuritis, **(Keh et al., 1992; Turek, 1984; Betts, 1940)** while others prefer a conservative approach, reserving surgery for unresponding cases. **(Maffetone, 2010; Logan, 1995; Subotnick, 1991; Viladot, 1982; Whitesides, 1982; Wickstrom & Williams, 1970)** Additionally, a subsequent neuritis may form after surgery and the permanent numbness of the toes may be very troublesome to the patient. In any event, there a re times when the patient is a poor surgical risk and conservative treatment is the preferred choice, regardless of the surgeon's usual preference. This is usually when there is poor vascular supply to the extremity. **(Veves et al., 2002)**

Treatment

Once plantar interdigital neuralgia has been diagnosed, further evaluation of the rest of the foot and factors influencing it must be made. Frequently extended pronation or some other factor increases the strain throughout the foot. Correcting remote factors enhances the opportunity for successful conservative care. Particular attention should be given to gait and foot function.

Evaluate the patient closely for subluxations or fixations throughout the foot. Make certain there is adequate range of motion between the metatarsal heads by evaluating with a scissors–type motion palpation. Use the rehabilitation exercises and procedures discussed in **Chapter 4**, and particularly evaluate footwear for tightness in the toe–box. If the patient has to buy new shoes to meet your requirements, be certain that the toe–box is wide enough, because often as corrections are obtained the forefoot widens considerably. **(Leaf, 2006)**

Specific padding designed to spread the metatarsal heads can be applied for plantar interdigital neuritis. To locate the position for the pad, press with the index finger between the metatarsals just behind or at the heads into the affected interspace. Continue changing the pressure and vector until the location is found that maximally spreads the toes. **(Langer, 2007; Ball & Afheldt, 2002)** A triangular foam adhesive rubber pad approximately 1/4" thick, 3/8" to 1/2" wide, and 3/4" long is applied to that location by the pad's adhesive and additionally held in place by tape. A small pad can be placed between the toes to additionally provide spreading action. It should be attached to only one of the toes.

Each patient must be evaluated to determine what structurally sound portion of the forefoot may be used to help transfer weight away from the involved area. Often an L–shaped pad **(described under foot support in Chapter 4)** can be applied to the 1st metatarsal to transfer some of the weight of the metatarsal arch to that ray. Determining the exact pad location to separate the metatarsals and to shift weight to another area is often a time–consuming process. Adequate examination time should be allowed, or the patient should be scheduled at the end of the day so the procedure is not rushed. When pads are applied, it is important to evaluate the individual with applied kinesiology techniques to determine that the padding is not creating a neurologic insult that causes muscles to weaken when standing or walking.

Its important to note that placing orthotics in shoes may result in the shoe not fitting properly, usually too tightly; this is one of the most common causes of plantar interdigital neuritis in the first place. In this case the shoe must be modified or a different shoe used altogether. Unless this is done, many people with orthotics supports have ill-fitting shoes. **(Maffetone, 2003)** The same AK procedures are used here as when evaluating an individual in the various weight–bearing modes.

Determining whether a correction in the foot is effective and whether the correction endures is one of the major advantages of using applied kinesiology as an adjunct to the diagnostic approach for peripheral nerve entrapments in the feet. For example when subluxations and muscular or ligament injuries in the feet have been corrected but the problem returns immediately when the patient walks, there is an indication that something is wrong in the individuals gait mechanism which may include the feet or muscles of ambulation as well as the torquing patterns within the body. Without the weight-bearing evaluation of manual muscle testing in AK, how would a physician who had just corrected a foot dysfunction recognize if there was a persisting disturbance (unless the patient complains of pain immediately upon walking)?

With the complexity of symptoms on display in the typical patient with lower extremity dysfunction, including pain and dysfunctional soft tissues, joints, etc., where would it be most appropriate to evaluate causes and initiate treatment? The manual muscle test identifies the dysfunctional tissues and the process of therapy localization and challenge allows for the identification of the precise articular correction that will change that finding with a rapidity and certainty that palpation alone cannot provide.

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