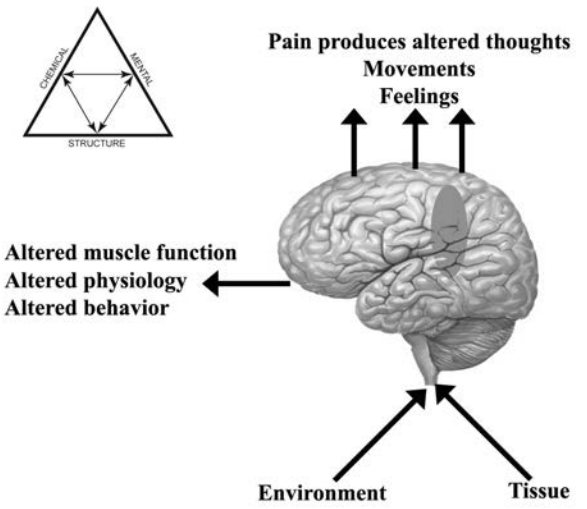
“If you don’t have a leg to stand on, you can’t put your foot down.”

~ Robert Altman



CHAPTER FIVE

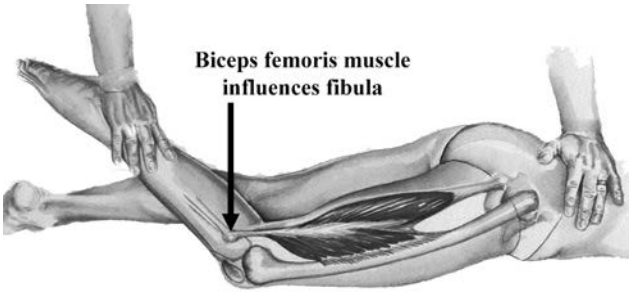
Leg and Ankle

05

This chapter deals with the leg’s anatomy and conditions that develop in the leg and ankle. It excludes ankle anatomy and function, since those are covered in the previous chapter on the foot. In anatomical and functional discussions, the leg is typically considered that portion of the lower extremity from below the knee to the ankle.

The tibia is the second longest bone in the body and with its companion, the fibula, articulates superiorly and inferiorly; in addition, there is attachment along the shafts by the fibrous interosseous membrane. Normal motion between the bones is necessary for proper function of the ankle mortise and action between the bones during gait. The distal end of the tibia is rotated laterally (tibial torsion) compared to its proximal end by about 30 degrees, with this torsion accentuated in Africans. **(Eckhoff et al., 1994)**

The superior tibiofibular articulation is a plane joint with a synovial membrane and fibrous capsule. The tibial articular facet is on the posterolateral aspect of the rim of the tibial condyle, facing obliquely posteriorly, inferiorly, and laterally. The fibular facet is on the upper surface of the head of the fibula, meeting the tibial facet anteriorly, superiorly, and medially. A portion of the biceps femoris tendon inserts into the styloid process of the fibula. The movement of the fibular head is powerfully influenced by the biceps femoris muscle. **(Greenman, 2003; Walther, 1981)**



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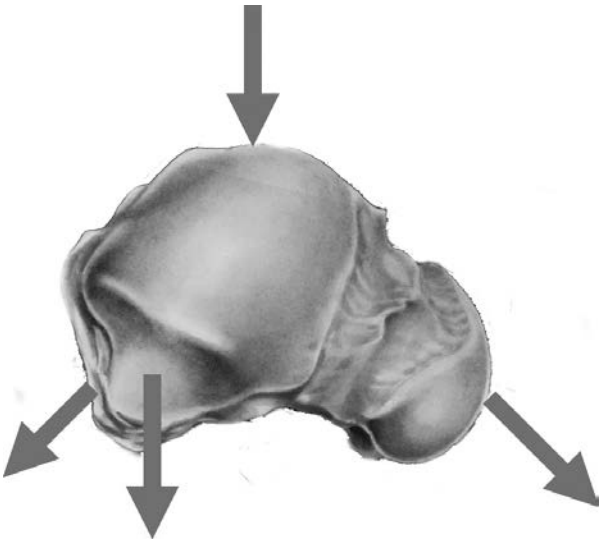
The inferior tibiofibular articulation is a syndesmosis, having no articular cartilage. This permits the talus to distribute loads over the entire foot, and is entirely covered by articular surfaces and ligamentous insertions and has no muscular attachments, giving it the characteristic of a 'relay station'. The body weight is effectively distributed through the talus in the manner. The two bones have no contact with each other, giving flexibility between the internal and external malleoli that is necessary for them to follow the tapered head of the talus.



The distribution of body weight through the talus (after Kapandji, 2010)

Ankle mortise function is discussed in the previous chapter. Briefly, ankle dorsiflexion separates the lateral and medial malleoli to accept the wider anterior portion of the talus body. During plantar flexion the distance between the malleoli narrows. **(Dubbeldam et al., 2010)** The contraction of the tibialis posterior, having its bipennate origin from both the tibia and fibula, causes an approximation of these bones, thus tightening the ankle mortise when the tibialis posterior contracts during plantar flexion. **(Gray's Anatomy, 2007)**

The bones and muscles of the leg are surrounded by a strong fascial sheath called the crural fascia. There are fibrous septa that separate the muscles of the leg into compartments. The anterior compartment contains the tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius muscles. The anterior tibial artery and vein and the deep peroneal nerve are also contained in this compartment. The lateral compartment contains the peroneus longus and brevis muscles; the superficial posterior compartment contains the gastrocnemius and soleus, and the almost vestigial plantaris muscle. The deep posterior compartment contains the flexor digitorum longus, flexor hallucis longus, and popliteus muscles. It also contains the posterior tibial and peroneal arteries and veins and the tibial nerve. A fifth compartment has been described **(Gray's Anatomy, 2007)** that contains only the tibialis posterior muscle. The muscle compartments and their significance are described later.

Ankle

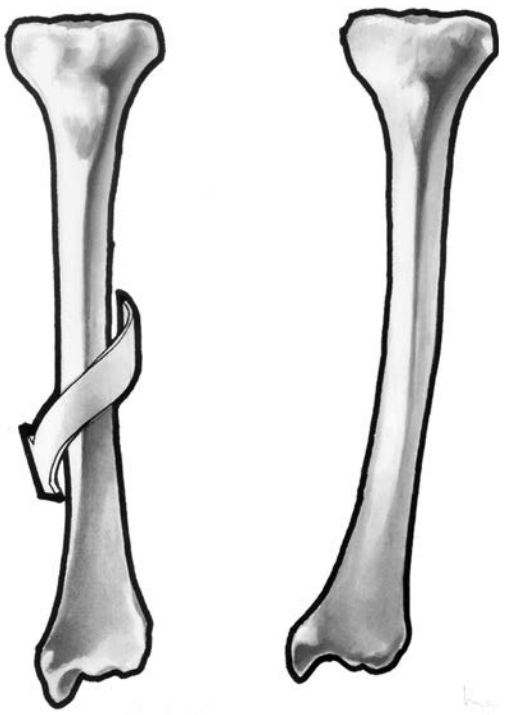
Ankle Joint Strain

The general use of the terms "sprain" and "strain" denotes a sprain as a partial or complete rupture of ligament fibers, **(Dorland's Illustrated Medical Dictionary, 2007)** and a strain as an overstretching or overexertion of some part of the musculature. **(Chaitow & DeLany, 2002; Cailliet, 1997)** In this discussion, "joint strain" refers to mechanical stress between a joint's components caused from remote dysfunctioning forces. As a result of joint strain, muscle strain may develop.

Movement throughout the lower extremity must be properly integrated to allow unstrained action of the joints. Imbalanced function in the lower extremity can result from conditions such as tibial torsion, muscle imbalance, or foot dysfunction. Goodheart and Greenman both observe that recurrent ankle sprains are frequently due to weakness of the peroneal and anterior tibial muscles. **(Greenman, 2003; Goodheart, 1980)**

If increased torsion is transmitted into the ankle or knee from a proximal or distal source, the ankle and/or knee receive greater strain during weight bearing, especially when one walks and runs.

Tibial torsion or hip rotation may be responsible for excessive internal leg rotation. Leg rotation puts torsional forces into the ankle mortise from proximal to distal. On the other hand, extended foot pronation puts torsional forces into the ankle mortise from distal to proximal. The torsion in the ankle puts strain on the ligaments, typically causing tenderness around the malleolus on digital pressure. The tibialis posterior is also strained in its role of



Tibial torsion

maintaining joint congruity by adjusting the width of the ankle mortise. Strain is indicated by tenderness of the ankle ligaments and of the tibialis posterior's belly as well as the muscle's origin and insertion on digital pressure. Under these circumstances the tibialis posterior may test weak, having been stressed and failing in its usual function. Frequently when the muscle is corrected with applied kinesiology techniques, it will not hold the correction when the patient walks or runs. Effective treatment to the remote cause of ankle joint strain may be needed before the tibialis posterior will hold its correction.

When the tibialis posterior muscle continues to function poorly, the overall problem may compound. The tibialis posterior is important in maintaining the medial longitudinal arch when a person walks and runs. **(Neville et al., 2010)** If the arch breaks down, internal leg rotation during the stance phase of walking increases. All causes of increased internal rotation during gait must be determined. Extended pronation, discussed in the previous chapter, is a common dynamic cause of increased internal leg rotation, as are weak external or hypertonic internal hip rotators. **(Gangemi, 2011)** The applied kinesiology method offered by Gangemi for insuring the stability of tibialis posterior muscle should be reviewed. **(See chapter 4)**

Normally the hip rotator muscles are balanced when each foot points laterally, approximately 10° from the sagittal plane. One can observe this muscular balance by pointing the feet directly forward and then standing on one leg only. The body will rotate away from the weight-bearing leg approximately 10° to the neutral position of the hip rotators. **(Jones, 1945)**

When foot pronation is present, its influence on rotation of the lower extremity can be observed by measuring rotation while a person stands on one leg, as indicated above, and then repeating the procedure when padding has been placed under the medial longitudinal arch to properly orient the calcaneus, as indicated by Helbing's sign. If forefoot varus is indicated, it will also be necessary to place a wedge under the forefoot that tapers from medial to lateral. **(Rose, 1962)**

The ankle joint is liberally supplied with mechanoreceptors capable of producing powerful reflex responses from the leg muscles. **(Dananberg, 2007; Goodheart, 1967)** Proprioceptors (mechanoreceptors) are found in the skin, muscles, tendons, ligaments and joints. Afferent fibers from mechanoreceptors converge segmentally on the dorsal horn of the spinal cord. The afferent fibers tend to diverge in an ascending and descending manner, over several segments, synapsing with different neuronal pools and spinal interneurons. This sharing of afference by motor centers has also been demonstrated in the cortex. **(Ginanneschi et al., 2005)** For this reason many synergistic muscle groups share common afferent inputs. **(Luscher & Clamann, 1992; Eccles et al., 1957)** This means that muscle spindle afference from one group of muscles supply the motor neurons in which they are embedded, as well as other synergistic muscles, consistent with Hilton's Law that the nerve supplying a joint supplies also the muscles which move the joint and the skin covering the articular insertion of those muscles.

In this way strain in the ankle from proximal or distal torsion may cause some of the local or remote aberrant muscle function observed in applied kinesiology testing. Subluxations of the talus and proximal or distal tibiofibular articulations may also result. Elimination or reduction of torsion causing ankle strain is usually accomplished by correcting extended foot pronation, pelvic and hip conditions, dural tension, and other modular distortions of the body. **(Leaf, 2010; Walther, 2000)**

Ankle Sprains

The acute trauma of ankle sprains may or may not be treated by an applied kinesiologist, depending on his or her scope of practice. Many prefer to limit practice to treating functional conditions. In any case, dysfunction often remains after the acute injury has healed, leaving the person with a "weak ankle" and prone to further injury.

The expression, "Once a sprain, always a sprain," need not be applicable. Applied kinesiology examination and treatment offer many techniques for returning normal function. Whether the physician treats the acute injury or not it is necessary to understand the mechanics and extent of the original injury when treating residual dysfunction.

Severe ankle sprains are frequently associated with direct trauma, often as a result of athletic endeavors. In athletes, the lateral ankle is the most frequently injured single structure in the body. **(Slimmon & Brukner, 2010)**

The frequency of ankle sprains varies significantly with the sport. Runners have relatively few ankle sprains. They frequently occur when an individual runs at night or otherwise twists an ankle on some substrate factor. **(Slimmon & Brukner, 2010)** Ankle sprains are common in high school basketball. In one study, **(Elkus, 1986)** 70% of eighty-four varsity basketball players from five public high schools had a history of ankle sprain. Of these, 80% had multiple sprains. Sports like football and skiing also have a relatively high incidence of ankle sprains.

There is a constant effort to improve the design of sports equipment to reduce ankle injuries. There has been a dramatic decrease in ankle injuries in alpine skiing as a result of improved slope grooming, a higher level of ski expertise, and better ski equipment. **(Deibert et al., 1998; Leach & Lower, 1985)**

Occasionally various foot articulations can be sprained. These, too, are influenced by footwear and the playing surface. Clanton et al. **(Clanton et al., 1986)** did a study that demonstrates this, revealing that synthetic playing surfaces and lightweight flexible athletic shoes have increased the incidence of metatarsophalangeal sprain. Particularly vulnerable to this trauma are those who have less than 60° dorsiflexion of the metatarsophalangeal articulation, especially of the 1st ray. It is suggested that the joint be protected by more solid shoes or shoes that have less than 6 millimeters descent from heel to forefoot (barefoot running technologies). **(Abshire, 2010)** Thick heels in footwear can result in increased dorsiflexion of the ankle while walking and running, adding more stress to the ankle and throughout the rest of the body. **(Bishop et al., 2006)**

Shoe fit and quality (as discussed in **chapter 4**) is important in reducing the chance of ankle sprain. A shoe that has a small sole as opposed to the individual's foot predisposes one to ankle sprain. **(Paiva de Castro et al., 2010)**

Although playing surfaces, equipment, footwear, and support to the ankle (such as taping) have been studied in depth, little effort has been directed toward intrinsic factors of the injured individual. In sports and everyday living, one must wonder why an ankle is sprained with a routine cut while running or when stepping off a curb. Granted, this type of sprain is usually not as severe as having another player fall on one's leg, causing a deltoid ligament tear. Nevertheless, the almost spontaneous ankle twist in everyday activity is a constant concern to some individuals. One must be able to evaluate, classify, and treat both the severe sprains and the common recurrent "twisted ankle." **(Kohne et al., 2007)**

Types and severity of sprains

In understanding the common types of ankle injuries, it is necessary to observe the following anatomical features. **(Bergmann & Peterson, 2010; Hammer, 1999; Cailliet, 1997)** The lateral malleolus is longer than the medial malleolus. Its distal tip extends further down the lateral articulating surface of the talus, and the bulk of the bone is much less than that of the shorter, bulkier medial malleolus. The tibia and fibula are bound together by the anterior and posterior tibiofibular ligaments, which are thickened expansions of the interosseous membrane that fastens the two bones together throughout their length. The deltoid or medial collateral ligament is heavy and has strong attachments to the internal malleolus, with many bands extending down to unite with ligaments supporting the arch of the foot; thus, in addition to giving medial ankle support it helps support the arch. The lateral collateral ligament has three main branches — the anterior talofibular, posterior talofibular, and calcaneofibular.

Inversion sprain dynamic

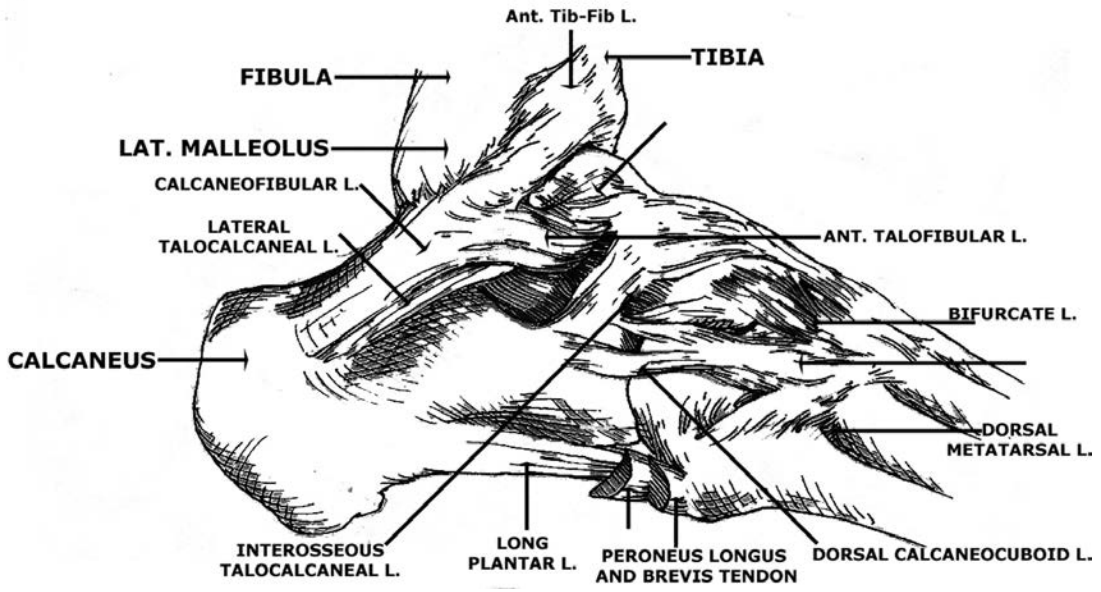
In inversion injuries there is push against the strong, stubby internal malleolus that acts as a fulcrum to cause pull to the lateral ligament, which more easily yields to the force. The ankle mortise separates and causes tearing of the lateral collateral ligament, with the severity depending on the amount of force.

In eversion injuries, the talus again pushes against the strong medial malleolus. Compressive force is applied to the more distal aspect of the lateral malleolus, which is thinner, and the bone yields to the force. At the medial side, the talus rides down on the lateral malleolus and causes a pull against the ligaments, which may tear.

There is a considerable difference in the seriousness of sprains to the medial structures as opposed to the lateral ones. Medial sprain is more serious but less frequent. It is caused by forced pronation, while the lateral sprain is caused by forced supination. Obviously a foot and an ankle with excessive pronation are more likely to suffer a medial sprain, and a supinated one a lateral sprain. **(Slimmon & Brukner, 2010; Gray's Anatomy, 2007)** Extended pronation may also cause a person to be more prone to lateral sprain, with the lateral ligaments sustaining the initial impact; **(Adirim & Cheng, 2003; Michaud, 1987)** this is discussed later.

In a medial sprain the structures torn are the deltoid ligament, anterior tibiofibular ligament, and the interosseous membrane. **(Slimmon & Brukner, 2010; Gray's Anatomy, 2007)** The deltoid ligament is so strong that it may fail to tear, but it avulses from the medial malleolus **(Cailliet, 1997)** or significantly tears with fracture of the fibula. **(Gray's Anatomy, 2007)** The medial sprain is frequently accompanied by a fracture of the medial malleolus, distal fibula, the posterior aspect of the tibia, or a combination of these. The fibula can be fractured almost anywhere along its length; consequently, x- rays should be taken of the leg to the knee. **(Yochum & Rowe, 2004)** The medial sprain usually occurs in athletic endeavors in which the foot is fixed to the ground and the leg is forced medially by another player hitting or falling upon it.

Professional soccer (football) players frequently sustain these types of ankle injuries, graphically displayed by watching popular soccer channels around the world. Ozetkin et al. **(Ozetkin et al., 2009)** collected data on 66 players with severe ankle and foot injuries. The most common diagnosis was ankle sprain (30.3%) with anterior talofibular ligament injury. Most (55%) hindfoot injuries were Achilles tendinopathy with or without rupture. Treatment was surgical in 23 patients (35%). The mean time lost from play for players with severe foot and ankle injuries was 61 days (range 21-240 days); after Achilles tendon ruptures, the mean time lost was 180 days. Injury severity was severe (>28 days lost from play) in 64% patients and moderate (8-28 days lost from play) in 36% patients. Serious ankle and foot injuries in this study resulted in players being out of professional competition for about 2 months.



Lateral foot ligaments

The literature has many classifications indicating the extent of an ankle sprain. In general there are three degrees of severity listed as Grades I through III, or mild, moderate, or severe. The grading may rank the injury by the amount of soft tissue damage, which ligaments are torn, or the amount of motion torn ligaments allow on stress tests.

The lateral ankle sprain is much more common, comprising approximately 85% of ankle sprains. **(Slimmon & Brukner, 2010; Cailliet, 1997; Hertling & Kessler, 1996)** It frequently results from forced adduction and inversion and most often plantar flexion, which make up all the motions of supination. There is a predictable sequence of ligament tearing, **(Slimmon & Brukner, 2010; Levangie & Norkin, 2001)** the extent of which provides one type of grading system. The first to tear is the anterolateral capsule and anterior talofibular ligament, which is a Grade I injury. Progressively, the talocalcaneal ligament — a Grade II injury — is included. **(Merck Manual, 2010; Levangie & Norkin, 2001)** The posterior talofibular ligament may tear, but it is rare.

A classification of soft tissue damage evidence and instability can be given based on this literature. This classification of sprains is as follows: (1) local signs of inflammation with no significant swelling or ecchymosis or joint instability; (2) intermediate as moderate swelling, ecchymosis, and some instability on ligamentous testing but a definite end point on stress examination; and (3) severe, with local signs of inflammation and marked instability of the specific ligaments tested.

The sinus tarsi syndrome is characterized by pain and tenderness over the lateral opening of the sinus tarsi. The patient has a feeling of ankle instability. This condition is secondary to an inversion strain intense enough to traumatize the interosseous talocalcaneal ligament. The condition is usually prevented by proper treatment of the sprain, with follow-up to obtain proper bone interaction and foot function. When the condition fails to respond to conservative care, surgical excision of the pathological tissue is performed. **(Pisani et al., 2005)**

Examination

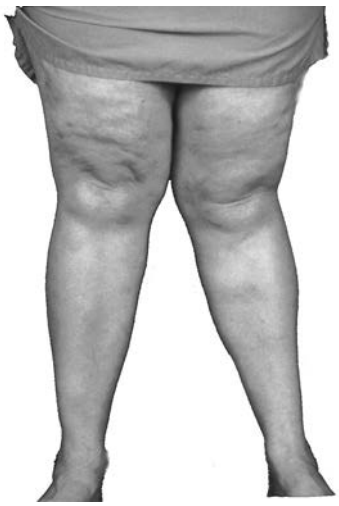
Injury to the ankle is evaluated for severity by areas of tenderness, swelling and ecchymosis, and by motion tests to evaluate ligament integrity. The diagnosis of ankle trauma is much more accurate if it is accomplished within a few hours of the injury. With time swelling becomes more diffuse, and palpation for tenderness is less specific. **(Slimmon & Brukner, 2010; Hertling & Kessler, 1996; Tropp, 1985)**

Examination should be designed to analyze the mechanism of injury and determine the exact structure damaged. If one fails to recognize the extent of injury and treats a severe one as mild, serious consequences may develop; appropriate treatment will not be as effective at a later date. The patient can usually describe how the ankle was twisted, giving an indication of whether there is medial or lateral involvement. Medial sprain will show tenderness under the medial malleolus at the area of the deltoid ligament and over the anterior aspect of the ankle where the anterior tibiofibular ligament and the interosseous membrane are torn. **(Slimmon & Brukner, 2010; Cailliet, 1997)**

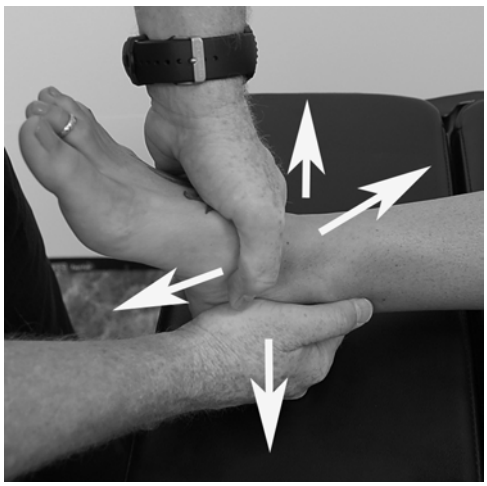
It is very important that the nature of the injury not be underestimated. There may be more extensive ligament damage than is observable by range of motion testing, because pain and swelling may limit the examination. **(Slimmon & Brukner, 2010; Hertling & Kessler, 1996)**

First, examine the uninjured ankle to determine range of motion and its characteristics for comparison. After observing the injured side for gross deformity, swelling, and circulatory disturbances, gently move the ankle passively through range of motion stress tests. It is particularly important to observe for lateral motion, which indicates ligament disruption. If there is no lateral motion observed but there is pain on attempting to elicit it, ligament damage is present but not necessarily disruption.

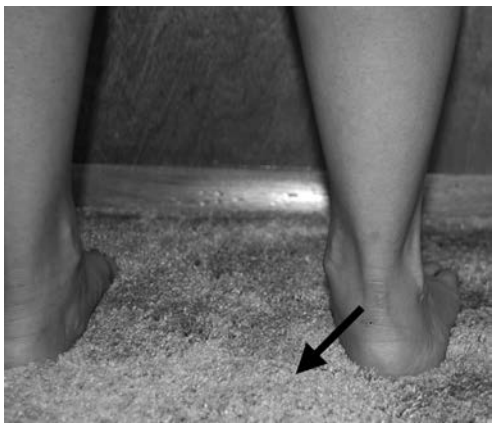
A mild sprain indicates partial tearing of the ligaments,



Severely pronated (knock) knees

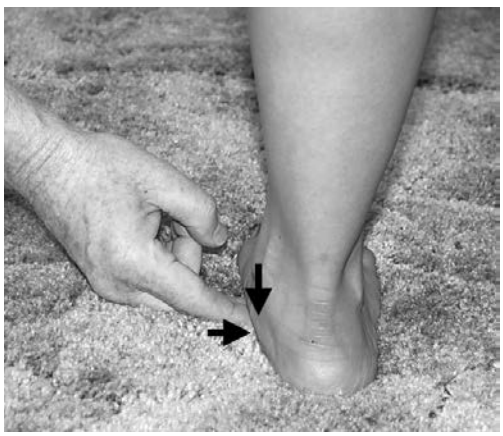


Stress tests

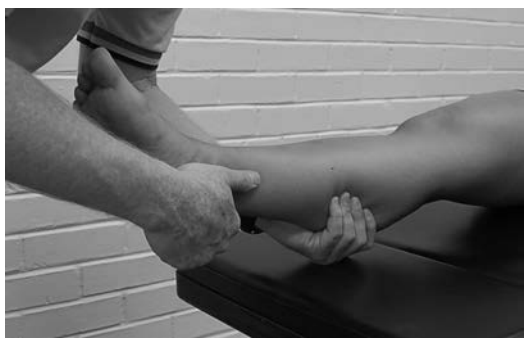


Pronation with curved Achilles tendon

Anterior Drawer sign

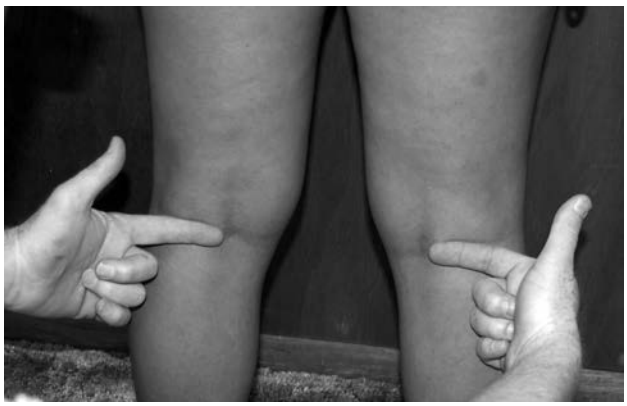


Palpation of collapsed medial arch

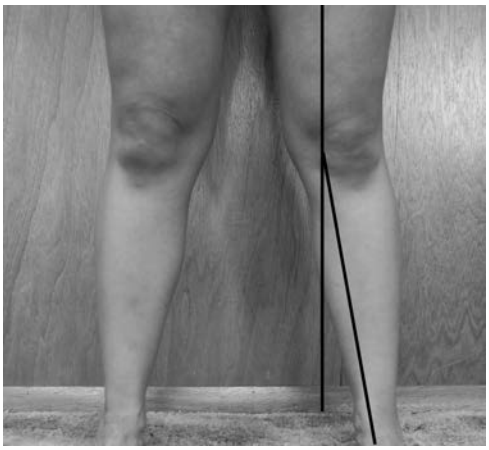


Thompson's squeeze test

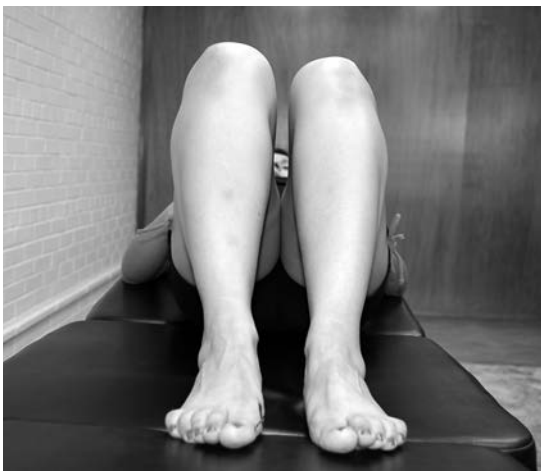
Signs and tests of ankle and leg dysfunctions.



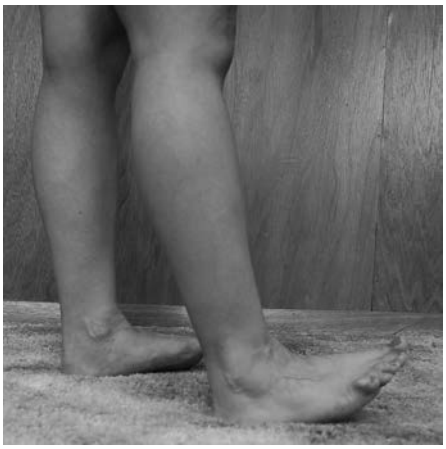
Unlevel knee folds, short tibia and anatomical short leg



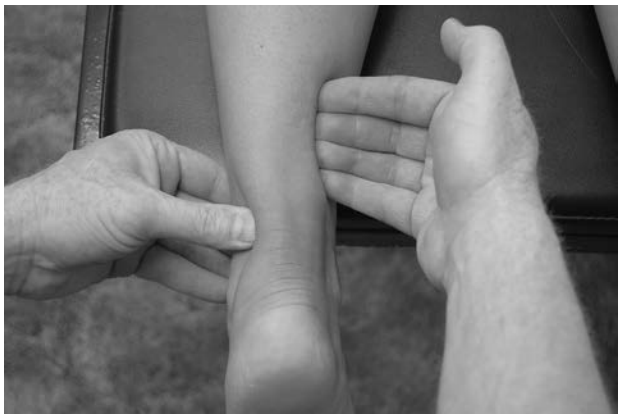
Valgus left knee



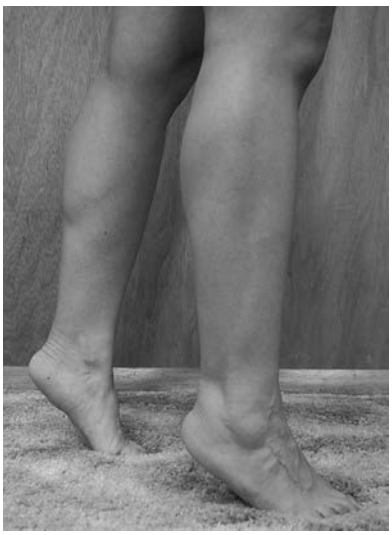
Unlevel knee heights may indicate short tibia and leg



Heel walk for L4-L5 nerve root involvements



Achilles Tendon palpation



Toe walk for S-1 nerve root involvement

Signs and tests of ankle and leg dysfunctions.

but without weakening the ankle structure. There will be local tenderness, swelling, and mild disability. There will be no pain on normal motion, and only a moderate degree when re-applying the stress that caused the injury.

A moderate sprain is an extension of the mild sprain, still without major loss of joint integrity but with greater ligament damage.

A severe ankle sprain is one in which there has been complete loss of ligament integrity. In most cases, this type of injury requires surgical intervention with appropriate non-weight bearing follow-up. This Grade III ankle sprain is suspected when there is 1) a history of a snap or a pop at time of injury; 2) ecchymosis over the lateral ankle and/or hindfoot; 3) tenderness over the anterior deltoid ligament in addition to more severe tenderness over the lateral ligaments.

The severity of a lateral sprain is further indicated by whether it is a single or a double ligament tear. This is sometimes difficult to differentiate since the anterior talofibular and calcaneofibular ligaments are very close in their origin on the fibula. As the ligaments traverse to the talus and calcaneus respectively, they are more easily palpated for differentiation. **(Slimmon & Brukner, 2010; Logan, 1995)**

The integrity of the anterior talofibular ligament can be assessed by the anterior drawer sign. **(Slimmon & Brukner, 2010; Evans, 2008)** As much relaxation of the ankle as possible should be obtained. This can be assisted by having the knee flexed with the patient seated, taking tension off the ankle from the triceps surae. The tibia and fibula are stabilized, and the foot is gently brought forward in the sagittal plane. If the talus moves forward, it indicates sprain of the anterior talofibular ligament. A posterior drawer maneuver is also described **(Slimmon & Brukner, 2010; Evans, 2008)** that tests for integrity of the posterior talofibular ligament; however, it is rarely injured except in cases of complete ankle dislocation.

When there is an actual tear in a lateral ankle sprain, there is greater inversion as the talus separates from the lateral malleolus. In a simple sprain no gap can be palpated, and the talus remains in its normal position. **(Slimmon & Brukner, 2010; Logan, 1995)**

The twisting injury that sprains an ankle can also sprain the ligaments of the midfoot. **(Adirim & Cheng, 2003; Levangie & Norkin, 2001)** This is usually easily differentiated from an ankle sprain by the location of pain, tenderness, and swelling.

Dislocation of the peroneal tendons is not a frequent occurrence. When it is presented in a general practice, it is often misdiagnosed as an ankle sprain. The dislocation usually develops from forced foot eversion with dorsiflexion and contraction of the peroneus longus and brevis. **(Hertling & Kessler, 1996; Martens et al., 1986; Cox, 1985)** This condition is discussed later.

X-ray examination

X-ray examination is important in severe ankle sprains, and it is done in two phases. **(Yochum & Rowe, 2004; Logan, 1995)** First, routine views are taken to rule out fracture. The projections include AP, lateral, and oblique views. If the trauma has caused foot deformity, one or more x-rays should be taken that display the deformity to provide information that might not otherwise be available about the degree of injury. **(O'Donoghue, 1958)** The ankle may then be manipulated into position for the standard views.

The AP view is done with the plantar surface of the foot perpendicular with the film and the leg internally rotated approximately 10° so the line through the malleoli is parallel with the film. This provides a true AP view. **(Yochum & Rowe, 2004; Logan, 1995)**

The lateral view projects transversely through the malleoli. It is usually taken with the lateral malleolus adjacent to the film. This usually causes poor visualization of the ankle joint space. A variation of technique places the medial malleolus adjacent to the film, with the medial knee on the x-ray table. This slightly everts the forefoot and usually provides a clear view of the joint. **(Yochum & Rowe, 2004; Cox, 1985)** The tube is centered on the lateral malleolus.

A common fracture site is the posterior tibial tip; it is demonstrated with slight external rotation in an off-lateral view. **(Yochum & Rowe, 2004; Logan, 1995)**

The oblique view is taken with the intermalleolar line forming an angle of 35-45° with the surface of the film. This view may reveal fractures not otherwise seen. If clinical examination indicates probable fracture and it is not observed on x-ray, additional oblique views should be taken. **(Logan, 1995)**

Fracture of the talus and 5th metatarsal may be present. A talus fracture should be suspected if symptoms persist after the normal healing time for ligamentous injuries. There are two types of fractures of the 5th metatarsal. Less serious is an avulsion fracture of the base of the metatarsal at the insertion of the peroneus brevis tendon. In the immature skeleton, it must be differentiated from the developing apophysis of the metatarsal. The secondary center generally has a longitudinal axis, whereas the avulsion fracture is transverse. **(Cox, 1985)** A much more severe fracture is one of the diaphysis or proximal shaft of the 5th metatarsal.

In medial sprains, x-ray examination should include the leg to the knee because of possible high fibular fracture. Additional views are necessary for thorough evaluation of the ligaments and the bony structure of the ankle and foot. **(Yochum & Rowe, 2004; Logan, 1995)** Bohler's angle for evaluating calcaneal fracture and other specialized views can aid in evaluating ankle and foot trauma.

The second phase of stress view x-ray examination is done if fractures are ruled out. This can usually be accomplished conservatively if done within the first few hours of injury. If pain is severe or there is significant swelling, it is necessary to anesthetize the area, taking the examination out of the conservative realm. **(Yochum & Rowe, 2004; Logan, 1995)**

The uninjured ankle is routinely examined with stress x-rays for comparison. It is necessary to rule out the effect of normal anatomic variation among individual patients. If the opposite ankle has been involved in an earlier trauma, this information will provide only limited comparison. Either the anterior drawer or the talar tilt measurements are adequate to determine whether one or both of the anterior talofibular or the calcaneofibular ligaments have been damaged. If only one set of measurements is done, the talar tilt series is preferred.

An anterior drawer maneuver is positive for a Grade III injury when a ten-pound force applied for two minutes causes displacement of the talus 2 mm greater than the non-injured ankle. **(Smith & Reischl, 1986)** The optimal position of the ankle and foot for x-ray examination for anterior drawer sign is internal rotation and 10% of plantar flexion. This gives maximum displacement if there is an anterior drawer sign from rupture of the anterior talofibular ligament and others. **(Liu et al., 2001)** If the anterior drawer test is positive, the talar tilt test is not done.

Talar tilt is viewed on the mortise view with the foot in inversion stress. As with the anterior drawer view, 10% of plantar flexion is optimal. This relaxes the muscles and allows maximum displacement if ligaments are torn. **(Larsen, 1986)** Comparison with the uninjured side is necessary since there is a wide range of normal motion. An evaluation of normal ankles revealed talar tilts ranging from 3° to 23°. Smith and Reischl **(Smith & Reischl, 1986)** prefer that the physician perform the talar tilt test. An inversion force is applied slowly to avoid peroneal muscle tension caused by pain. "Instability on the talar tilt maneuver is indicated by a 3 mm or greater separation of the lateral tibiotalar surfaces compared to the noninjured side or a 6° or greater tibiotalar angle compared to the noninjured ankle."

When there is a clinically serious ankle injury but normal ankle stress x-rays and a positive anterior drawer stress x-ray, further evaluation with a subtalar arthrogram should be done to evaluate for a subtalar sprain. **(Liu et al., 2001)** This condition is thought to be more prevalent than generally recognized. "Particular attention should be paid to athletes involved in indoor activities, such as volleyball or basketball. They are prone to this type of injury because of the high adherence between playing surfaces and modern footwear. Unrecognized and untreated subtalar sprains can lead to serious consequences, such as chronically painful ankles or the sinus tarsi syndrome. These problems may be avoided only if the exact extent of the lesion is appreciated." **(Meyer et al., 1988)**

Smith and Reischl **(Smith & Reischl, 1986)** find several disadvantages regarding the general use of arthrography. It requires fluoroscopy, there may be an allergic reaction to the iodine dyes, and ankle arthrography may not be able to identify ligament rupture just several days after injury because the capsule seals itself with a weak layer of fibrin. This prevents the escape of dye, and possibly presents a false-negative conclusion in terms of a ruptured ligament.

Trauma to the distal thickened portion of the tibiofibular interosseous membrane may allow separation of the syndesmosis with resulting ankle instability. Tibiofibular widening at the area of the syndesmosis can be evaluated by two methods of measurement. The "clear space" is the distance between the medial border of the fibula and the lateral border of the posterior tibia as it extends into the incisura fibularis. The tibiofibular overlap is the maximum overlap of the distal fibula and the anterior tibial tubercle. The measurement is made 1 cm above the plafond of the tibia. Normal clear space is less than approximately 6 mm on the AP view of the ankle. Normal tibiofibular overlap is approximately 6 mm, or 42% of fibular width. According to Harper and Keller, **(Harper & Keller, 1989)** the width of the tibiofibular clear space appears to be the most reliable parameter for detecting early syndesmotic widening.

Nerve injury accompanying ankle sprain

Severe ankle sprains may be accompanied by a high percentage of peroneal and tibial nerve injuries. Nitz et al. **(Nitz et al., 1985)** did a clinical and electromyographic study to determine the extent of this type of nerve trauma. They found no nerve damage in sprains that only involved the lateral complex. When the deltoid ligament was involved, the peroneal and tibial nerves had 17% and 10% injury, respectively. When the sprain included the lateral complex, deltoid ligament, and distal anterior tibiofibular ligament, 83% incurred posterior tibial nerve injury. They hypothesized that the mechanism of nerve injury was from stretch at the ankle at time of injury. This may cause entrapment of the common peroneal nerve that can be the cause of persistently weak ankles. **(Staal et al., 1999)** There is a poor prognosis for quick recovery when the peroneal and/or tibial nerves are involved. This should be taken into consideration for return to work or sports activities.

Richie, **(Richie, 2001)** in a comprehensive review of functional ankle instability, suggests that the instability is not the result of hypermobility, but rather the diminishment of proprioception, muscle strength, muscle reaction time, and postural control generally. For the majority of patients in Richie's review, the functional instability of the ankle results from a loss of neuromuscular control. "Proprioceptive deficits lead to a delay in peroneal reaction time…Balance and postural control of the ankle appear to be diminished after a lateral ankle sprain and can be restored through training that is mediated through central nervous mechanisms."

After injuries where muscular strength, timing, velocity and control are decreased, the sensory input, central processing and motor output time may all be increased. **(Hurley, 1999)** This may delay the activation of neuromuscular protective reflexes and is another impediment to the shock-absorbing function of muscles, exposing the joint to damage. **(Colledge et al., 1994)** In addition, improvement of neuromuscular functions as a result of applied kinesiology treatment and rehabilitation regimes for the ankle may decrease the reaction times, improve functional joint stability, strength, and proprioception, **(Hurley & Scott, 1998)** which may be important in restoring the shock-absorption capacity of muscle and protect against further ligament and joint damage.

Treatment

Treatment of mild to moderate ankle sprains ranges from **R**est, **I**ce, **C**ompression, and **E**levation (RICE) to surgical intervention for severe sprains. The simplest treatment is by RICE therapy.

- **R**est. Reduce activity and get off your feet.
- **I**ce. Apply ice in a plastic bag or towel over the injured area, following a cycle of 15 minutes on, 40 minutes off.
- **C**ompression. Wrap a bandage around the area, but be careful not to pull it overly tight.

∞ Elevation. Position the foot in an elevated position on a bed, couch or chair, higher than your waist, to reduce swelling and pain.

Initially the patient rests with a compression bandage, usually for 24 hours. Ambulation is then allowed as tolerated with an Unna boot or Elastoplast(R) for 7 to 10 days. **(Safran et al., 1999)** This is usually referred to as mobilization treatment. Care should be taken not to override pain with medication that allows the patient to perform physical activities that would normally be limited by the discomfort. "Injections in these injuries are contraindicated since this can mask significant pathology and may result in a more severe injury." **(Clanton et al., 1986)**

Non-steroidal anti- inflammatory drugs (NSAIDs) do not appear to improve healing rate or effectiveness. A double-blind study of treating ankle sprains with ibuprofen was statistically insignificant over a 28-day period. **(Dupont et al., 1987)** In another double-blind study, Fredberg et al. **(Fredberg et al., 1989)** evaluated ibuprofen in the treatment of acute ankle joint injuries. The subjects had ankle sprain or fracture within 24 hours prior to hospital admission. They were administered a placebo, or 600 mg ibuprofen tablets taken four times daily. There was no reduction in ankle swelling in those taking the medication. The subjects were allowed to take additional analgesic medication if they felt the need. Those taking ibuprofen required as much additional medication as those on the placebo, indicating that ibuprofen has no effect on pain. Their conclusion was that "…the results indicate, no difference between relief of pain and reduction of swelling was demonstrated. We cannot recommend routine treatment with ibuprofen for acute ankle joint injuries." In the treatment of patients with excess inflammation there is an extensive body of literature describing as many as 10,000-20,000 fatalities and 103,000 hospitalizations produced yearly by non-steroidal anti-inflammatory drugs (NSAID) medications, frequently the treatment given for low-back and other joint pains in medical settings. **(Wolfe et al., 1999; Gabriel et al., 1991)** These figures are similar to the number of deaths from HIV and far more than the number of deaths from cervical cancer, myeloma or asthma. If the deaths from gastrointestinal toxicity resulting from the use of NSAIDs were tabulated separately, these would constitute the fifteenth most common cause of death in the United States. **(Wolfe et al., 1999)**

Immobilization treatment consists of casting the ankle, which is usually positioned to bring the torn ligaments together. The cast is left on for 4-6 weeks. When more intensive treatment is desired, as with professional athletes, removable immobilization is applied, such as taping or use of a type of cast brace. This is removed daily for ice therapy initially, followed by contrast whirlpool hydrotherapy. **(Cox, 1985)**

Generally, Grades I and II ankle sprains can effectively be treated by mobilization or immobilization methods. Initial swelling must be controlled by ice, elevation, and pressure bandaging. Healing time is in direct relation to the amount of swelling. The presence of effusion favors the formation of adhesions, which further delay healing. Apart from the toxic nature of NSAIDs, untreated joints have been found to remain in better condition than those treated with NSAIDs. **(Pizzorno et al., 2007; Werbach, 1996)** Protection by wrapping or a cast may be needed for ten days to three weeks, depending on the extent of the damage. **(Hertling & Kessler, 1996)** With strapping and mobilization of lateral ankle sprain where talar tilt is less than 10°, return to full working capacity is expected within one month. In a Grade III sprain, the ankle is often immobilized in a walking cast for six weeks. **(Oztekin et al., 2009; Beskin et al., 1987)** When stress x-rays are negative but clinical examination points to a severe injury, the patient is still managed for a Grade III sprain. **(Oztekin et al., 2009; Hontas et al., 1986)**

Surgical repair of torn ligaments is followed by cast immobilization. There is no common agreement over the indications for surgical repair. The criteria for operative repair are (1) significant talar tilt indicative of a double ligament tear (anterior talofibular and calcaneofibular ligaments), (2) a history of functional instability (multiple sprains) with acute injury, (3) an acute injury with a bony ossicle or avulsion fracture from the lateral malleolus, and (4) associated osteochondral fracture of the talus. Using these indications, 12% of cases resulted in surgery. **(Brand & Collins, 1982)**

The severity of the sprain dictates the intensity of treatment, but there are several considerations that must be made in comparing and determining the type of treatment. These include (1) healing time, (2) stability of ankle after healing, (3) life-style of the patient, (4) cost of treatment, (5) whether surgery can be successfully done even many years after the failure of conservative care, **(Safran et al., 1999)** and (6) avoiding unnecessary surgical complications.

In 1985 Cass et al. **(Cass et al., 1985)** did a long-term follow-up study to determine if ankle stability improves with immediate surgery, as opposed to conservative treatment followed by reconstructive surgery if the conservative treatment fails. They evaluated both the subjective results via patient questionnaire and objective results by clinical examination, stress roentgenology, and biomechanical gait study. A satisfactory result can be expected in 80% of the Grade III patients treated conservatively. Surgical reconstruction can be applied to the remaining 20% at a later time, with the confidence of obtaining patient satisfaction and a reasonable certainty of obtaining a satisfactory objective result as well. Their study does indicate that for those who require high performance and functional perfection of the extremities, such as professional athletes, acute repair gives slightly improved objective functional results. For these individuals, early surgery may be the appropriate choice. Otherwise, they find it is proper to attempt conservative treatment first.

Freeman **(Freeman, 1965)** compared randomly selected patients for effectiveness of lateral ankle sprain treatment done conservatively by strapping and mobilization, immobilization in plaster for six weeks, and suture of the ligament with immobilization for six weeks. In ankles treated by mobilization and immobilization, none displayed more than 8° of relative talar tilt. Duration of disability after strapping and mobilization, immobilization, or surgical treatment was 12, 22, and 26 weeks, respectively. One year after injury, 58% and 53% of patients treated

by mobilization and immobilization, respectively, were symptom-free, but only 25% of patients treated by suture and immobilization had become symptom-free. It is concluded, "For these reasons, and because simple sprains are satisfactorily treated by mobilization, it is suggested that mobilization may be the treatment of choice for most, perhaps all, ruptures of the lateral ligament of the ankle." **(Freeman, 1965)**

Finally, Van der Linden et al. **(2004)** reviewed 292 cases of Achilles tendon rupture treated either surgically or non-surgically (with splinting) for ankle stability. For both groups mean follow-up time was 3 to 6 years. There were 14 re-ruptures, ten after surgical repair and four after non-surgical treatment. In the surgical group there were seven major wound problems, 11 minor wound complications and six patients with complaints related to the sural nerve. In the non-surgical group one patient suffered a pulmonary embolism after a re-rupture, 3 months after the initial rupture. There was no difference in mean ankle score and patient-satisfaction score between groups. Only 52% regained their original sports activity level, slightly better in the surgically treated group. With a non-significant difference in re-rupture rate but relatively more complications after surgical repair, non-surgical treatment is the recommended procedure. With a slightly better recovery of sports activity after the surgical repair however, this might be used as an argument for surgical treatment in young athletes.

Non-surgical treatment is usually the choice method of treatment, even though surgical intervention for some patients may give better results. Martin et al. **(Martin et al., 2007)** offer a number of reasons: (1) surgical complications are avoided, (2) the surgical scar can cause future problems of altered sensibility and traumatic neuroma, (3) surgical intervention can be selected, even several years after the injury if initial results are unsatisfactory, (4) residual symptoms after non-surgical treatment are usually mild and not disabling, (5) recovery time is greater after operation than after strapping, and (6) routine surgical treatment of these common injuries would greatly increase the work of surgical units and overall cost of treatment.

Surgical intervention is usually most appropriate for fractures about the ankle, especially for proper reduction of fractures that include the articulating surfaces. **(O'Donoghue, 1958)** Calcaneofibular ligament injury confirmed by stress x-ray is a good surgical candidate. **(Rijke et al., 1988)**

A medial sprain is potentially much more serious than the more common lateral sprain. Primary concern is whether the deltoid ligament sprain is accompanied by interosseous membrane tearing and diastasis of the distal tibia and fibula. If there is diastasis, it is sometimes treated by closed reduction and casting after 48 hours of ice application and compression to decrease the swelling. Surgery may be necessary to remove particles of the medial deltoid ligament in order to reduce the diastasis. **(McBryde, 2007)**

Children present a somewhat different picture in ankle sprains. There is more elasticity in the ligaments of children than in adults; consequently, stretching injuries are more common than severe tears. When there are small flecks of bone from the tip of the lateral malleolus or, rarely, from the tip of the medial malleolus, there is indication that the bone gave way rather than tearing of the ligament. These lesions can be treated as sprains rather than fractures. Immediate application of cold and compression bandaging are important. Unfortunately, these injuries often occur when there is no supervision and immediate measures are not applied.

Healing time in children is shorter than in adults, probably due more to lack of frank tears rather than the age of the child. **(Adirim & Cheng, 2003)** If there is ligamentous tearing, a below-knee plaster cast is the treatment of choice. Generally, in this age group, the cast can be removed after three weeks, with protection for an additional three weeks by an adhesive wrapping or elastic bandages. Surgery is rarely applicable in a child, with the exception of a severely torn deltoid ligament. In this case there is an increased propensity toward surgery. Young patients have a greater percent of residual ankle stability after conservative care than do older patients. They are good candidates for primary surgical repair, especially those who need perfect ankle function, such as athletes. **(Slimmon & Brukner, 2010; Levangie & Norkin, 2001)**

Tarsal-metatarsal sprain in children is generally caused by forced plantar flexion and inversion. The injury is usually not severe, and compression bandaging with local cold applications and crutches is sufficient treatment.

Rehabilitation

Rehabilitation begins with the initial treatment of an ankle sprain and continues until optimal function is obtained. Failure to accomplish the latter may leave the patient with a propensity to additional ankle sprains. Proper treatment helps eliminate the often-quoted phrase, "Once a sprain, always a sprain."

The body's initial reaction to the soft tissue trauma of a sprain is acute inflammation. This is appropriate in the case of an infection, but it is an overreaction to closed soft tissue trauma; **(Marymont et al., 1986)** it promotes edema, which delays healing. **(Fredberg & Stengaard-Pedersen, 2008)** It should be remembered that chronic inflammation may be aggravated by a number of factors including adrenal stress syndrome, nutritional deficiencies and hormonal imbalance, **(Schmitt & McCord, 2010; Pizzorno et al., 2007)** each of which may be diagnosed using applied kinesiology methods. **(Leaf, 2010; Walther, 2000; Goodheart, 1998-1964)**

The first step of local treatment is to reduce the inflammation by the RICE method. At this stage compressive bandaging is important. Care in application avoids circulatory deficiency, but the patient should be made aware of the signs of circulatory embarrassment and told where to cut the tape or remove an elastic support if disturbance develops.

At the initial examination the patient should be tested for lymphatic function, including neurolymphatic reflexes and retrograde lymphatic technique. Since muscles cannot usually be tested in the acute phase, therapy localizing the reflex is an adequate testing method. Retrograde and anterograde lymphatic is tested in the usual manner **(Walther, 2000; Goodheart, 1998-1964)** to ensure normal lymphatic drainage from the lower extremities.

When myofascia is permitted to remain immobile for

extended periods of time, its ground substance solidifies, leading to the loss of the collagen fibers' ability to slide across one another and this permits the development of adhesions. Akeson et al. **(Akeson et al., 1977)** have shown the changes that occur in connective tissue with prolonged immobilization. In Achilles tendinosis, there is a significant decrease in the total collagen content and altered collagen cross-linking that result in an aberrant collagen network similar to fibrosis. **(De Mos et al., 2007)** With easy exercise, torn ligaments heal to be stronger, larger, and have a higher collagen content than when immobilized. **(Yang et al., 2005)**

Most ankle injuries can be treated with the mobilization method. It is best, if applicable, because of the reasons indicated earlier; also, it provides stress on the ligaments to properly align the developing collagen fiber synthesis in the direction of function. **(Solomonow, 2009; Marymont et al., 1986)** The strength becomes greater than the ligament- osseous junction, whether sutured or not. Movement encourages the collagen fibers to align themselves along the lines of structural stress as well as improving the balance of glycosaminoglycans and water, thereby lubricating and rehydrating the damaged connective tissues. **(Lederman, 1997)** Immobilization has the disadvantage of producing muscle atrophy if done at or shorter than the resting length. There is also a decrease in protein synthesis within six hours of immobilization. **(Amiel et al., 1983)**

Weight bearing is not allowed if there is diastasis of the distal tibiofibular syndesmosis, which should be adequately supported for proper healing; if severe, surgical repair is indicated.

There are numerous methods of providing support to the ankle while healing takes place with mobilization. Figure-of-8 taping combined with basket weave is often used and has been shown effective. **(Hubbard & Cordova, 2010)** A classic type of taping is the Gibney boot. **(Hamill et al., 1986)** More extensive taping is often used for athletes as a prophylaxis, and it is effective in providing support during the healing of a sprain. **(Morrissey, 2001)** More rigid support can be supplied by splinting material (Orthoplast, Johnson & Johnson, Inc.) that is softened in hot water and molded to the patient; it then becomes semi-rigid when cooled. This is combined with foam cushioning and tape to provide a support that allows flexion and extension, but controls supination and pronation. **(Pope et al., 1987; Cox, 1985)** A similar support on a more elaborate basis can be provided by modifying a brace designed for fracture treatment. **(Cooke et al., 2009)**

Rehabilitation time after ankle sprain can be reduced by combining cryotherapy, compression massage, and exercise. In appropriately equipped training facilities, this can be accomplished by a pneumatic boot, which cycles pressure around the ankle and foot at 1.5 pounds, alternating 15 seconds on and 15 seconds off. The water bath is maintained around 37° F., and the athlete exercises the ankle with gentle dorsiflexion and plantar flexion. The movement is allowed because of the anesthetic effect of the cold. The cycling pressure assists the venous system in draining congestion from the injured site to help control edema. In addition, elevation enhances the drainage effect. **(Starkey, 1976)**

Prevention and optimal function

The comment, "Once a sprain always a sprain, need not apply" depends on returning normal function — and perhaps improving it — over that present before the injury. Muscle rehabilitation exercises, discussed later, are standard procedures used by many physicians and trainers, but if the muscles are limited by neurologic factors producing arthrogenic inhibition, **(McVey et al., 2005)** the exercises will probably fail to return optimal function. There are many AK examination and treatment techniques that should be routinely applied in the rehabilitation of ankle sprains to return normal function, and possibly enhance it beyond that present prior to the accident. This phase of rehabilitation is particularly important. Ferguson, **(Ferguson, 1973)** who emphasizes the importance of muscle rehabilitation, indicates that an "…athlete who has sustained a knee or ankle ligamentous tear is 15 times more susceptible to reinjury than one with no previous injury." One must question whether the increased susceptibility is mandated by the injury or is due to inadequate treatment and rehabilitation. The importance of specific muscle and joint impairments to optimal gait and ankle function has been carefully described. **(Santilli et al., 2005; Perry et al., 1986)** The use of the manual muscle test for assessing the specific muscle and joint impairments that have been described in many textbooks regarding ankle function and gaiting adds specificity to the evaluation and treatment of these disorders of the ankle.

Applied kinesiology examination can begin as soon as acute injury healing allows muscle testing. All muscles crossing the ankle are tested and corrected, if necessary, by the usual methods. This may require any of the five factors of the IVF. Particular attention is given to any foot subluxations or fixations. In athletic injuries, the neuromuscular spindle cell, Golgi tendon organ, and cutaneous receptors may need treatment. Do not forget to consider remote possibilities, such as spinal subluxations, meridian involvement, or dysfunction of the cranial-sacral primary respiratory system. Extended pronation is a common problem associated with ankle sprains. Michaud **(Michaud, 1997, 1987)** attributes recurrent ankle sprains to failure of the mid-tarsal locking mechanism during gait, i.e., the foot fails to become a rigid lever during the stance phase as is present in extended pronation. It is corrected in the usual manner, as discussed in the previous chapter. Tarsal coalition should be considered in any active adolescent who has recurrent ankle sprains or strains. **(Clanton et al., 1986)**

When an athlete sprains an ankle when making a sharp turn, an activity frequently done, there is the possibility of a reactive muscle. He may have injured a muscle on a previous play that is now inhibiting an important ankle stabilizing muscle. For example, the tibialis anterior may become hypertonic from injury, and by reciprocal inhibition inhibit the antagonistic peroneus longus and brevis that are necessary for lateral ankle support. **(Santilli et al., 2005)**

Another consideration when there is injury for no apparent reason is the ligament stretch reaction. **(Walther,**

2000) This condition is thought to be associated with adrenal gland stress in applied kinesiology. **(Goodheart, 1976)** When the ligaments of a susceptible joint are stretched, associated muscles of that joint temporarily weaken. **(Sprieser, 2003)** Treatment is directed toward the adrenal glands. This is done nutritionally and by treating any of the implicated five factors of the IVF for the particular patient. **(Walther, 2000)**

Selye points out that an athlete is often under considerable stress during competition, even though this may be eustress. **(Selye, 1974)** If there is an underlying adrenal stress disorder, the ligament stretch reaction may be present and cause the muscle function so necessary to perform optimal joint motion to fail. Deutsch and Durlacher suggest that the ligament stretch reaction is a systemic problem, affecting all the ligaments of the body. **(Durlacher, 1977; Deutsch, 1975)** Sprieser and Blaich report on the specificity of the ligament stretch reaction to the knee in acute and chronic conditions. **(Sprieser, 2003; Blaich, 1980)**

It was suggested as far back as the turn of the century that ligamento-muscular reflexes exist from sensory receptors in ligaments to muscles that modify the load imposed on the ligament and joint. Goodheart first discussed the law of the ligaments in 1973. **(Goodheart, 1973)** Goodheart found that pressure applied to the ends of ligaments towards the belly of the ligament tightens it. The opposite force will elongate the ligament.

It has been shown that ligamento-muscular reflexes exist in most extremity joints. **(Solomonow et al., 1998; Freeman & Wyke, 1967)** The ligaments associated with each extremity are richly endowed with afferents that produce reflex activation of the many muscles associated with the extremity's movement. The muscles therefore are a major component in maintaining the stability of the extremity's ligaments, bursae and capsules. **(Solomonow, 2009)**

Goodheart also suggested that when there is a chronically weak muscle there will usually be a ligament involvement (the ligament is stretched) that provides stability in the same direction as the muscle. Conversely, a stretched ligament will cause a weakness in a muscle that provides stability in the same direction. **(Solomonow, 1987)** For extremity dysfunctions, detection of ligament injuries through these ligamento-muscular reflexes can be specifically assessed with the MMT.

Particularly in over-trained athletes who demonstrate higher cortisol levels as well as adrenal gland enlargement due to hypertrophy and hyperplasia, **(Maffetone, 2010; Stallknecht et al., 1990; Barron, 1985)** the ligament stretch reaction of applied kinesiology may be critical in the prevention of one of the causes of recurrent ligament sprains.

In the unusual case in which muscle(s) fails to respond to applied kinesiology techniques, one should consider possible nerve damage as a result of a severe sprain, as previously indicated.

Exercise

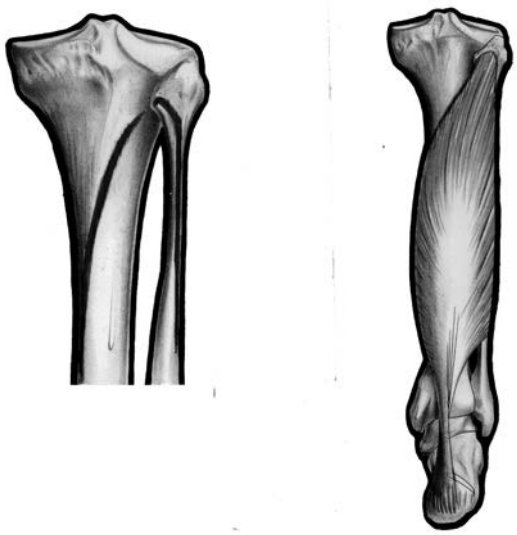
In most cases it is advisable to rehabilitate the muscles with exercise. This begins when AK examination indicates the muscles are functioning normally, and as soon as pain and swelling allow brisk walking, usually just several days into the rehabilitation program. Clinical experience has shown that muscles functioning poorly on an AK examination do not respond well, in general, to exercise. This is because the specific arthrogenic impairments resulting from structural subluxations, fixations, remote factors, and other neurological influences on the muscle are still present following the exercise program. Once the AK examination shows normal facilitation of the muscles in the area, peroneal and ankle dorsiflexor muscle strengthening becomes the foundation of the rehabilitation program, because these muscles are responsible for actively resisting an inversion/plantar flexion injury. **(Smith & Reischl, 1986)**

Several have described the use of thick-walled, non-metallic rubber surgical tubing for rehabilitation exercises. **(Kemler et al., 2011)** The U.S. Naval Academy **(Cox, 1985)** used this method wherein thirty-six inches of 3/16" tubing were used to provide isotonic exercise that increases in resistance as the tubing is stretched. The longer the tubing, the less resistance it provides. Tubing exercise has the advantage of being inexpensive, versatile, and easily carried so the patient can exercise frequently throughout the day.

In addition to the home-made method of simply tying a loop on one end of the tubing, a kit called the "Thera-Ciser" is available from Foot Levelers, Inc. **(Foot Levelers, Inc.)** The kit provides a cloth loop, tubing, and a booklet describing the exercises. For ankle sprains, the anterior and lateral compartment muscles are usually exercised.

The anterior compartment muscles (dorsiflexors) are exercised by anchoring one end of the tubing to a piece of furniture; the forefoot is put through a loop at the other end of the tubing. With the knee in extension, the patient dorsiflexes the foot against the resistance of the tubing through the full range of motion. Resistance is increased by starting the procedure with greater stretch on the tubing.

The lateral compartment muscles (peroneal group) are exercised by everting the foot against the resistance of the tubing. One end of the tubing is anchored with the non-involved foot. The forefoot of the exercised foot is put



Soleus muscle's origin means that stretching the muscle with the knee bent effects it more than the gastrocnemius

through the loop of the tubing, while the heel is kept firmly on the floor to prevent lower leg rotation. The exercise starts with the foot in inversion and slight plantar flexion. The forefoot is abducted against the resistance into eversion and slight dorsiflexion.

Usually the plantar flexors (gastrocnemius and soleus) need to be stretched rather than exercised. In case strengthening is needed, they are exercised by putting the loop over the distal foot and holding the tube in the hand. The foot is plantar flexed against the tubing resistance. With the knee in full extension, the gastrocnemius and soleus are exercised. With the knee in flexion, the activity is directed more toward the soleus.

With dorsiflexion, the ankle joint mortise has a gradual increase in distance between the malleoli. The average increase in this distance is 1-1/2 mm from full plantar flexion to full dorsiflexion. Even with full plantar flexion, there is no looseness of the talus in the ankle joint mortise. This is attributed to ligament function **(Norkus & Floyd, 2001)** and to tibialis posterior muscle action by Kapandji. **(Kapandji, 2010)** The difference between the transverse dimensions of the anterior and posterior portions of the trochlea of the talus is as much as 4.7 mm. There is less distance change between the malleoli because no point on the tibial articular surface moves along more than one-half the length of the trochlear surface. Close AK examination and correction should be given to the tibialis posterior muscle. When it needs to be exercised, the foot is held in inversion during the plantar flexion exercise described above. **(Ramsak & Gerz, 2002)** More specific tubing exercise is done by starting with the forefoot abducted in full plantar flexion; the heel position is maintained while the forefoot is adducted against resistance.

Proprioceptive training

Prevention of ankle sprains depends on proper organization of the muscles supporting the ankle and foot. Often the cause of the sprain in the first place is poor muscle control when an inversion force is applied to the foot while walking or running. A significant amount of research has shown ankle sprains to be associated with muscle imbalances, most particularly weakness of the dorsiflexors and invertors of the foot. **(Baumhauer et al., 1995)** Chronic ankle sprains have been associated with arthrogenic muscle weakness. **(McVey et al., 2005)**. The arthrogenic weakness of these ankle muscles can produce inhibition of the peroneals **(Santilli et al., 2005)** and the hip abductors **(Zampagni et al., 2009; Bullock-Saxton, 1994)**

Disorganization present before or developed from the current injury must be corrected before function is returned to normal. Clinical experience has shown that foot subluxations are a common cause of neurologic disorganization throughout the body, apparently because improper stimulation to the joint proprioceptors of the foot causes dysponesis. AK examination and correction are the prerequisites to proprioceptive training. The training procedures are enhanced when there are no foot/ankle subluxations or fixations and the muscles perform correctly in an AK examination.

The need for proprioceptive training is indicated by decreased ability to stand on one foot with stability. This often decreases following ankle sprain. Freeman et al. **(Freeman et al., 1965)** suggest "…that functional instability of the foot is usually due in the first place to motor incoordination consequent upon articular de-afferentation…." In particular, the mechanoreceptors in the human foot and ankle (among other receptors) control the instantaneous and qualitatively precise contractions of the calf muscles that must occur if the foot is to remain stable on uneven ground. **(Kavounoudias et al., 2001)**

An objective method of evaluating stability, called stabilometry recording, has been described by Lopez-Rodriguez et al. **(Lopez-Rodriguez et al., 2007)** When there is instability on one foot standing, as indicated by stabilometry recording, there is a significantly higher risk of sustaining an ankle injury than when there are normal stabilometric values. Manipulation of the talocrural articulation improved the stabilometric values and distribution of weight on the feet in these patients. Further evidence of this was determined by Tropp et al. **(Tropp et al., 1985)** when they studied one-legged stability standing in a control group and applied that information to 127 soccer players. The soccer players showing an abnormal stabilometric value ran a significantly higher risk ($p < 0.001$) of sustaining an ankle injury during the following season compared to players with normal values. Players with a history of previous ankle joint injury, but a normal stabilometric value, did not run a higher risk compared to players without previous injury. Furthermore, they did not find persistent functional instability in those who had previous ankle joint injury; when instability was present, it increased the risk of future ankle joint injury regardless of whether the person had sustained a previous injury. This indicates that a person with an ankle joint injury is not destined to instability.

Freeman et al. **(Freeman et al., 1965)** evaluated patients who had recent sprains of the foot and ankle by a modified Romberg test, comparing stability when standing on the injured extremity with the uninjured one. They found that 34% of their patients had an objective or subjective proprioceptive deficit. No deficit was detected in 16%. The remainder were unstable, but the cause could not be determined because of pain or stiffness. Thus in at least 34%, and at most 84% of the patients studied, there was instability due to proprioceptive deficit. They attribute this deficit to causing the symptom of the foot "giving way."

If instability continues after muscles have been tested and corrected by applied kinesiology techniques, the patient can be trained on unstable surfaces such as a balance board, or rocker and wobble boards, and ankle disk. **(Waddington & Adams, 2004)** The balance board is about 15" square; at the bottom, a 2" board is centered to balance on. Several boards with higher balancing points can be used for increasing exercise levels. The ankle disk has a spherical undersurface so that it tips in any direction.

The balance board is generally used first. Balancing anterior to posterior exercises the muscles and develops proprioception in flexion and extension. Lateral balancing effectively improves action of inversion and eversion. First both ankles are exercised together. Balancing is done to bring the edge of the board close to the floor, but to control it from touching the floor by muscle action. Movement of the board is then reversed to bring the opposite edge of the board close to the floor before stopping action. As control is optimized, one ankle at a time can be exercised on the board.

With exercise on the ankle disk, there is greater proprioceptive and muscle demand. This activity exercises one ankle at a time. First the ankle is moved in circumduction both clockwise and counterclockwise, keeping the edge of the disk in contact with the floor. As proficiency is obtained, the movement is done to just keep the edge of the disk from touching the floor.

Exercise with the balancing board or an ankle disk is done for about ten minutes per day. After optimal function is obtained the exercise can be reduced to about five minutes per day, three times a week to maintain performance.

The inversion sprain in athletic competition is most common. It may be reduced by a conditioning program to strengthen the peroneal muscles, and by teaching the players to land with a relatively wide-based stance. The latter places the foot slightly lateral to the falling center of gravity, making an inversion stress on the ankle unlikely. **(Smith & Reischl, 1986)**

The work of Vladimir Janda (with its emphasis on treating muscle imbalance, and particularly muscle inhibition, with physiotherapy) has become popular. Morris and Page have presented the Janda approach (particularly using the rocker and wobble board apparatus) comprehensively. **(Page et al., 2010; Morris et al., 2006)**

It should be noted that in Janda's model of the diagnosis and treatment of muscle imbalances, Sherrington's Law of reciprocal innervation operates primarily in one direction: muscle hypertonicity/tightness/spasm generates inhibition in its antagonists and so spasm is treated first. For this reason muscle spasm and tightness are considered the etiological factors of articular dysfunction. In Janda's approach hypertonic muscles are treated with physiotherapeutic means such as massage, stretching, proprioceptive neuromuscular facilitation, electrotherapy and other methods that do not usually include manipulative therapy. **(Page et al., 2010; Chaitow & DeLany, 2002)** In Janda's classic text on MMT **(Janda, 1983)** there is no mention of spinal or other joint manipulation options for the muscle inhibitions found; nor were correlations observed between muscle inhibitions found on examination and cranial, meridian, nutritional, or psychological treatments.

In Janda's model the inhibited (weak) muscles are treated with exercise, rocker boards, wobble boards, balance shoes, and mini-trampolines among others. The principles of this physical therapy approach to muscular imbalances were based on the work of Bobath and Bobath who developed physiotherapy programs for children with cerebral palsy. **(Bobath & Bobath, 1964)**

Muscle imbalance as conceived by Janda was mainly embraced by the physiotherapy community, though in recent years it has lost some of its popularity to the concept of core function and motor control. **(Wallden, 2007)** One common reason for the decline in interest in muscle imbalance in the physiotherapy and manual medicine communities is that, as with nearly all clinical entities, to find a textbook patient showing the muscle imbalance syndromes described by Janda is less common than finding only a partial case. This makes the diagnosis of muscle imbalance syndromes confusing. Further, the approach to diagnosing a muscle imbalance (for practical purposes) is based primarily on subjective assessment, such as the visual observation of standing posture. Based on this visual diagnostic method the prescription of treatment preferred by Janda – corrective stretching, corrective mobilization, corrective exercises and other nutrition and life style advice – may be somewhat non-specific. Additionally, aside from subjective symptomatology, progress is difficult to gauge with this subjective approach.

The visual diagnosis of a specific muscle's problem is difficult. The different elements within the chain of events of any particular movement that a patient undergoes in front of the examiner occur within a fraction of a second, too rapidly to be accessed separately in the absence of laboratory tools. Dananberg makes this clear in the failure of the great toe to move in the 100 msec time-frame it must during the stance phase. **(Dananberg, 2007)** Visual detection of this muscular impairment is essentially impossible. Therefore, what is actually observed by the examiner is the grand total of how rapidly and smoothly a person can change between two activities.

For these reasons it is strongly urged that to evaluate any muscle imbalance, clinical measurement tools, including but not limited to the manual muscle test, dynamometers, and digital cameras should be used.

Tape and support

Tape and support are used at the ankle for two purposes: (1) to provide compression and support for the injured ankle, and (2) to prevent injury of the normal ankle. **(Hubbard & Cordova, 2010; Morrissey, 2001)** Tape should be applied to the ankle over skin that has been prepared with a tape adherent tincture. Tape adhesion has been improved considerably. Some believe the more complicated woven or basket-weave strapping lasts no longer than simpler types.

There is controversy regarding the use of support to prevent ankle injuries. It seems obvious that ankle support muscles, especially the peroneus group and the tibialis posterior, must be functioning normally and there should be no extended pronation. Simultaneous electromyography and stop-action movies show the peroneus brevis and longus and the tibialis posterior to be the most important stabilizing muscles. **(Langer, 2007)** Langer offers a useful

test to determine if the tibialis posterior muscle is injured. If patients feel pain or an increase in pain when shifting their weight to the affected foot and rising up onto their toes, then it is likely that this muscle is dysfunctional.

Ferguson **(Ferguson, 1973)** points out, "Only the muscle and its tendon attached to the bones can, by contraction, prevent excessive motion of an individual joint." In his paper, *The Case Against Ankle Taping*, he points out that the subtalar articulation acts as a "safety valve" for the ankle joint and knee. A cleated shoe and taped ankle prevent motion at this safety valve. As the trunk rotates with the foot and ankle fixed firmly to the ground, the strain of rotation becomes more manifested in the knee, which is a frequently injured structure in the lower extremity in sports. They cite studies conducted by the New York State Public High School Athletic Association based on 61,000 reports of high school athletic injuries, as well as studies of high school athletes in Philadelphia indicating that in preventing injury a low shoe, disc heel, and ankle wrap (not tape) or a low shoe, short cleat, and no ankle support produced statistically significant lower rates of serious injuries. An extensive study in college football **(Rovere et al., 1987)** showed that laced ankle stabilizers and low-top shoes prevented injury better than tape support. In general, schools that have adopted a policy of non-taping ankles have found satisfaction in the resulting decrease in knee injuries and ankle sprain.

The use of tape as opposed to elastic support is questionable. Rarick et al. **(Rarick et al., 1962)** found that 40% of the net support of taping is lost after 40 minutes of exercise. It would appear that proper shoes, correct muscle and foot function, and elastic support provide the optimal protection against ankle injury.

There is not universal support for non-taping of ankles to prevent sprains. **(Maffetone, 2003)** A study by Garrick and Requa **(Garrick & Requa, 1973)** of 2,562 intramural basketball players indicated that prophylactic ankle taping and high-top shoes provide the best protection against ankle sprains. This group had 6.5 ankle sprains over 1000 player games (6.5/1000). The rate progressively increased with less support: low-top shoes and taped, 17.6/1000; high-top shoes and untaped, 30.4/1000; low-top shoes and untaped, 33.4/1000. There was no evidence produced in this study that taping increased the propensity toward knee strain.

An electrogoniometric method of evaluating motion between the tibia and the calcaneus-shoe complex revealed significant support to the ankle by a closed basket weave with heel locks and a half figure-eight taping technique over the untaped ankle complex. The evaluation was done before and after figure-eight walking exercise, concluding that ankle taping appears to be an effective technique to help prevent ankle injuries. The tape's effectiveness is more dependent on its tensile strength than its adhesive properties. **(Laughman, et al., 1980)** Stress x-ray reveals Gibney tape support significantly reduces talar tilt before and after 30 minutes of exercise, but elastic bandages do not. **(Vaes et al., 1985)**

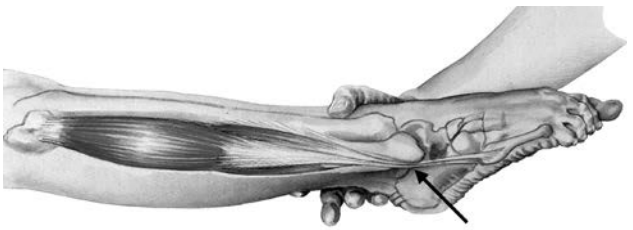
Regardless of one's interpretation of the literature on prophylactic taping, it should be obvious that optimal muscle, foot, and leg function and balance are necessary to reduce the frequency and severity of ankle sprains. The use of taping may have an advantage other than direct support to the ankle. From simultaneous electromyography and stop-action movies, Glick et al. **(Glick et al., 1976)** concluded that "…the advantage of taping is probably attributable to its stimulating effect on the peroneus brevis muscle or in short to a dynamic action." In athletes with optimal lower extremity function tape may not provide the same prophylactic value as in those with dysfunction. **(Stoffel et al., 2010)**

Performance is another factor that may be important in deciding whether to support the ankle with tape. With increased ankle support limiting ROM in the ankle and subtalar joints, performance is significantly and progressively decreased. **(Stoffel et al., 2010)**

Peroneal Tendon Dislocation

The peroneal longus and brevis tendons are located in a common synovial sheath that lies in the retromalleolar groove and is held there by the superior peroneal retinaculum. The strong retinaculum rarely ruptures. It either strips away from the periosteum of the lateral malleolus or avulses a thin cortical shell. **(Ferran et al., 2009; Raikin et al., 2008)** The depth of the groove varies; when it is shallow there is predisposition to dislocation of the peroneal tendons. In this case little or no trauma may be necessary for dislocation. In some cases, the tendons can even dislocate with a squat. **(Martens et al., 1986)**

Traumatic dislocation of the peroneal tendons over the lateral malleolus is an uncommon injury. It primarily occurs with various athletic endeavors, particularly skiing, soccer, and ice-skating. It usually occurs with violent dorsiflexion, followed by strong reflex contraction of the peroneus longus and brevis. This often happens from ski tips digging in, with the skier being pitched forward by the sudden deceleration. **(Ferran et al., 2009; Raikin et al., 2009)** When the ankle is in maximum dorsiflexion, the tendons are "bow-stringed" against the soft tissues, maximizing chance of dislocation. A dislocation of the tendon causes a sharp pain and snapping.



Peroneus longus dislocation

The frequency of peroneal tendon dislocation from skiing has decreased since the introduction of high, rigid ski boots. This boot protects against peroneal tendon injury because edging can be accomplished by the knee levering the rigid boot rather than requiring active foot eversion by the peroneal muscles. **(Oden, 1987)** Even so, there

is a higher incidence of peroneal tendon dislocation in a practice that has a high percentage of skiers.

The condition can be acute or chronic. When acute it is often misdiagnosed as a simple ankle sprain, probably because it is so uncommon. The diagnosis becomes more difficult if there is some delay, with swelling over the lateral malleolus. There is tenderness to digital pressure in the retromalleolar sulcus, slightly more proximal than in the usual ankle sprain. **(Martens et al., 1986)** Tensing the peroneus longus and brevis muscles causes pain. After dislocation the tendons may spontaneously relocate. Putting the patient's foot in the position of injury against resistance will elicit retromalleolar pain and often dislocation. **(Evans, 2008)** It is important to examine the other ankle because a congenital luxation of the uninjured ankle is often found, leading to the diagnosis of the injured ankle. The tendon will frequently dislocate when the patient's ankle and foot are placed in plantar flexion and eversion against resistance. **(Oden, 1987)**

Varus stress x-rays of the ankle reveal no lateral instability and are not particularly painful to the patient. **(Yochum & Rowe, 2004; Marti, 1977)** A chip fracture of the lateral malleolus may occasionally be observed on x-ray. **(Yochum & Rowe, 2004; Logan, 1995)** This increases the possibility of surgery since the tendons cannot be held while the fracture unites. **(Safran et al., 1999)**

In the chronic state, there may be snapping or popping of the tendons with activity. When there are chronic ankle and subtalar problems with lateral ankle instability, peroneus longus and brevis tendon lesions should be considered. **(Sammarco & DiRaimondo, 1988)** Sometimes the peroneal retinacula are relatively weak; with time they tear loose, allowing the tendons to dislocate anteriorly over the lateral malleolus, with full dorsiflexion of the ankle. This may initially be painful and eventually develop to the point that the tendon dislocation and relocation are present with circular motion of the foot. The patient may complain that his ankle keeps "popping out." When the tendons are dislocated, pronation is weak or impossible, and the lateral ankle ligaments are unusually vulnerable to injury. **(Ramamurti, 1979)** Chronic cases often have to discontinue sports activities because of the feeling of the ankle giving way.

Conservative treatment of ankle taping, non-weight bearing progressing to weight bearing and an ice/heat regime is applicable. **(Cox, 1985)** An exception to this may be the professional athlete or anyone who puts excessive strain on the tendons, predisposing to future trauma. Most patients have little functional loss if subluxation persists and open reduction can be done at a later date if necessary. **(Safran et al., 1999)**

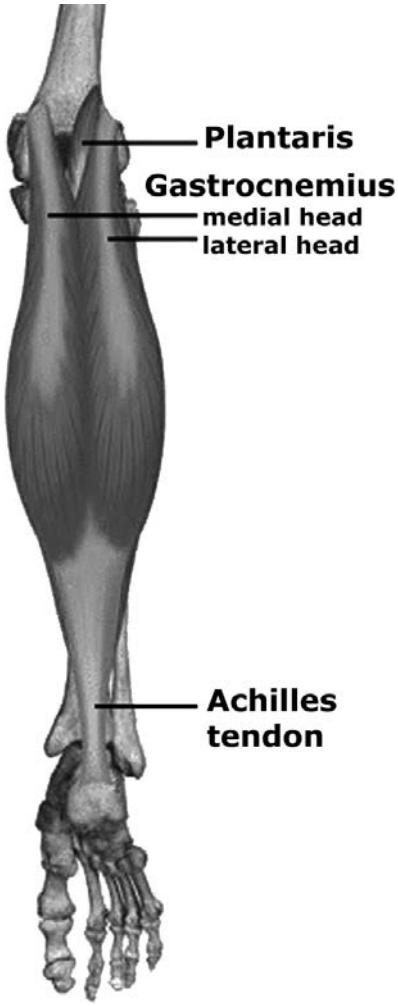
Surgical treatment for recurrent dislocation of the peroneal tendons consists of re-routing the tendons under the calcaneofibular ligament. This surgical procedure is effective in returning the individual to previous athletic activities. **(Martens et al., 1986)** Another surgical method is to deepen the groove, **(Colville, 1998)** the Kelly procedure, which deepens the retromalleolar sulcus by a sliding fibular veneer bone graft. **(McLennan & Ungersma, 1986)**

The peroneus brevis tendon lies behind the fibular malleolus medially and deeper than the peroneus longus tendon. An unusual occurrence is for the peroneus brevis tendon to move laterally and anteriorly around the peroneus longus tendon, but still remain within the tendon's common sheath. This creates a clicking, and it may prevent participation in sports and other activities. This uncommon injury has been successfully treated by surgically interposing two strips of peroneal retinaculum between the two tendons. This is done under local anesthesia so the patient can demonstrate the dynamic activity of the tendons; it also aids in the assessment of the integrity of the reconstruction. **(Safran et al., 1999)**

Achilles Tendon

The Achilles tendon is the thickest and strongest tendon in the human body. It begins approximately in the middle of the leg, extending to attach to the posterior surface of the calcaneus. Contributing to the Achilles tendon are the triceps surae's muscles: the soleus, gastrocnemius, and the almost vestigial plantaris. The soleus contributes muscle fibers almost to the lower level of the tendon. **(Gray's Anatomy, 2007)** The soleus portion of the Achilles tendon twists to insert on the medial portion of the calcaneus.

Gray's Anatomy **(Gray's Anatomy, 2007)** also notes that the Achilles tendon plays an important part in



Muscle insertions onto Achilles tendon

reducing the energy cost of locomotion by storing energy elastically and releasing it at a subsequent point in the gait cycle.

The two heads of the gastrocnemius and the soleus are referred to by some as the triple aspect of the triceps surae. The soleus has been shown to have a bipartite behavior, with the lateral portion active in eversion and the medial portion in inversion, **(Gray's Anatomy, 2007; Levangie & Norkin, 2001)** making what could be called the quadriceps surae. If there is foot or gait dysfunction, Achilles tendon stress can develop as the gastrocnemius and soleus muscles attempt to control foot inversion and eversion.

Clement et al. **(Clement et al., 1984)** point out that the Achilles tendon is surrounded by a peritenon, which is comprised of the epitenon and paratenon. The paratenon functions as an elastic sleeve, permitting free movement against the surrounding tissue while maintaining continuity with adjacent structures. The Achilles tendon receives its vascular supply primarily through the paratenon. There is a region of diminished circulation 2–6 cm above the Achilles tendon insertion. They speculate that reduced vascularity at this area may play an etiological role in rupture of the Achilles tendon.

There are many terms used in the literature to describe disorders of the Achilles tendon, e.g. tendinitis, tenosynovitis, tendinosis, tendinopathy, rupture, and partial rupture, among others. In general, there are three conditions associated with the Achilles tendon — tendinitis, bursitis, and rupture. The terms used should describe the tissue disturbed and degree of involvement. Tendinitis does not involve the tendon; rather, it is an inflammatory reaction of the paratenon. Since the tendon does not have a synovial sheath, Clement et al. **(Clement et al., 1984)** recommend that the term "Achilles tenosynovitis" be re–classified to recognize the peritenon. Their classification scheme of Achilles tendon disorders is as follows: (1) peritendinitis, (2) peritendinitis with tendinosis, (3) tendinosis, (4) partial rupture, and (5) total rupture. Since the primary vascularization to the tendon is from the paratenon, most of the inflammatory process takes place there. The term "tendinosis" indicates disruptive lesions of the tendon in the absence of peritenon alterations.

Achilles tendinitis

Inflammation of the Achilles peritenon, which will simply be called tendinitis, is the most common cause of chronic posterior heel pain and it is the most commonly reported form of tendinitis. **(Langer, 2007; Levangie & Norkin, 2001)** The etiology of Achilles tendinitis is varied requiring individualized treatment. The condition usually has a gradual onset with pain and swelling along the Achilles tendon, 2–6 cm proximal to the calcaneus insertion. Examination reveals focal tenderness, induration, and frequently crepitus at this area. **(Cailliet, 1997; Logan, 1995)** Chronicity of the condition can develop into tendenosis and eventually partial or complete rupture. The tendon often becomes strained by extended pronation aggravated by a short triceps surae. **(Langer, 2007)** In a study of 1,192 injured runners, there were 20% with Achilles tendinitis. Of the 20%, 71% had pronated feet and 89% had a valgus heel. **(Lutter, 1983)** According to Johansson and Lysholm and Wiklander, **(Johansson, 1986; Lysholm & Wiklander, 1987)** the annual incidence of Achilles disorders is between 7-9% among top-level runners); however, 28% of 694 runners had other injuries as well to the lower extremity. **(Van Middelkoop et al., 2008)**

Training errors for runners and other athletes in their 30s and 40s are major contributory factors. **(Abshire, 2010; McDougall, 2009; Kubo et al., 2007)** Busseuil et al. **(Busseuil et al., 1998)** reviewed the various factors making runners more susceptible to injuries in a comparative study between a healthy control group (n=216) and runners (n=66) suffering from overuse pathologies of the foot and ankle. Specifically, the researchers looked at runners who needed treatment for iliotibial band syndrome, Achilles tendonitis, stress fracture of the tibia, tibial periostitis and plantar fascitis. The significant correlation between these problems and runners with foot dysfunction demonstrates the importance of thorough investigation of the feet and ankles and the rest of the leg and hip in runners with injuries. Their analysis showed that the injured subjects (among numerous other factors) have a more pronated foot than control group subjects. The many factors that come into play for normal foot function are purposely incorporated into the applied kinesiology examination of the feet.

Clement et al. **(Clement, 1984)** evaluated running with slow–motion high-speed cinematography, which revealed a whipping action or bowstring effect of the Achilles tendon with extended pronation. This occurs when the foot continues to pronate as the tibia begins external rotation. At this point, the foot should be supinating and becoming a rigid lever. **(Clement et al., 1984)** They speculate that the combination of torsional forces transmitted through the tendon due to extended pronation, along with vascular impairment, contributes to degenerative changes in the Achilles tendon. In Achilles tendonitis there is a significant decrease in the total collagen content and altered collagen cross-linking producing a dysfunctional collagen network similar to fibrosis. **(de Mos et al., 2007)** Athletic training may cause the triceps surae to become overstrengthened and short, limiting dorsiflexion and contributing to extended pronation. **(James et al., 1978)**

An inflexible midfoot contributes to Achilles strain. In extended pronation, the integrity of the midfoot typically fails. If it doesn't, there is a longer lever arm to put strain into the Achilles tendon. **(Levangie & Norkin, 2001)** In a similar manner, the cavus foot is intolerant of running and even walking. **(Ogon et al., 1999)**

Training errors are responsible for up to 75% of the cases. **(Chang et al., 2010; Van Middelkoop et al., 2008; Clement et al., 1984)** Included are failure to develop triceps surae flexibility, a sudden increase in training mileage, a severe competitive or training session (such as a marathon), sudden increase in training intensity, repetitive hill running, initiating training after an extended period of inactivity, and running on uneven, slippery terrain. The most common cause of chronic heel pain as the result of skiing is Achilles tendinitis. **(Oden, 1987)**

Diagnosis

Careful differential diagnosis is necessary because symptoms of Achilles tendinitis may simulate some of the symptoms of other running–induced injuries, such as tarsal tunnel syndrome, rupture of portions of the triceps surae, tibial stress fracture, tibialis posterior tendinitis, posterior compartment syndromes, retrocalcaneal bursitis, and plantar fascitis. **(Langer, 2007; Levangie & Norkin, 2001)** In children, Achilles tendinitis can be mistaken for Sever's disease. **(Logan, 1995; Ellfeldt, 1983)**

Achilles tendinitis pain associated with running typically occurs early in the run and diminishes, but then it worsens again three to four hours after exercise. The patient may complain of pain in the Achilles tendon when getting out of bed in the morning, but the pain subsides during the day. Normal walking typically does not cause pain, while climbing stairs does. **(Langer, 2007; Ogon et al., 1999; Logan, 1995)**

The symptomatic pattern indicates the severity of the condition. In the earlier stage, pain is present only during strenuous activity, which is usually athletic. As the condition advances, the pain increases during sports activities and may force an athlete to stop or cut down on his activity. There may be pain during normal daily activities. With the advanced stage of tendinosis or partial rupture, there is also pain at rest. Severe morning stiffness indicates an advanced condition. **(Langer, 2007; Nelen et al., 1989)**

The location of pain is 2–6 cm proximal to the Achilles tendon insertion into the calcaneus. When maximum tenderness is on the medial border of the tendon, extended pronation as a contributing factor is indicated. Leaf and Lowdon et al. **(Leaf, 2000; Lowdon et al., 1984)** attribute this to bowing of the Achilles tendon from extended pronation, putting tension on the medial aspect of the tendon.

There may be some soft tissue swelling and/or crepitation with active motion. The Thompson squeeze test can be used initially to quickly test for the integrity of the Achilles tendon. On squeezing the belly of the gastrocnemius and soleus muscles, the foot should plantar flex. Diffuse swelling indicates thickening of the paratenon. With nodular swelling, tendinosis or partial tearing is probably present. **(Nelen et al., 1989)**

Treatment

Conservative treatment consists of immediate withdrawal from activities that put strain on the Achilles tendon. Ice massage and contrast baths usually give rapid relief in the absence of stress to the tendon. Heel pads for cushioning (sponge rubber, Sorbothane) are often used, **(Jorgensen, 1985)** but they have been found insignificant in aiding recovery. **(Lowdon et al., 1984)**

Ultrasound is recommended by some, **(Langer, 2007; Ogon et al., 1999; Schepsis & Leach, 1987)** but Clement et al. **(Clement et al., 1984)** state: "Ultrasound has been used to disrupt adhesions between the tendon and peritendinous sheath. However, because ultrasonic waves may disturb leukocytes in traumatized tendon and potentiate an inflammatory reaction, we feel that the beneficial role of ultrasound in the treatment of peritendinitis is questionable."

Clement et al. **(Clement et al., 1984)** recommend that cast immobilization not be used in Achilles tendinitis, even in stubborn cases. Impaired function of the immobilized joints, tendons, ligaments, and muscles may result.

Transverse friction massage, recommended by Cyriax, **(Nagrale et al., 2009; Chaitow & DeLany, 2002; Cyriax & Cyriax, 1983)** is done by grasping and moving the skin over the injured stretched tendon. This is done without lotion and must be directly over the damaged portion of the tendon across the longitudinal fibers. The lesion typically lies on the medial, lateral, or both borders of the tendon. Care is taken to avoid movement between the therapist's fingers and the patient's skin. Cleaning the patient's skin and the therapist's fingers with alcohol helps remove any oil and prevent slipping. Hammer **(Hammer, 2008)** presents a case report of the successful treatment of Achilles tendinosis, plantar fasciosis, and supraspinatus tendinosis using the Graston instrument. The Graston instrument is used to mechanically mobilize scar tissue, increasing its pliability and loosening it from surrounding healthy tissue. It is hypothesized that for degenerated connective tissue, the Graston instrument re-initiates the inflammatory process by introducing a controlled amount of microtrauma to the affected area.

Some use heel lifts to take strain off the tendon. **(Clement et al., 1984)** If this is done, it should only be during the acute stage. As the acute stage is resolved, it is important to correct extended pronation, if present, and lengthen the triceps surae to obtain adequate dorsiflexion. The proper exercise for gastrocnemius–soleus stretch is discussed in the previous chapter. To the wall lean exercise, Clement et al. **(Clement et al., 1984)** add toe raises by having the patient stand on the edge of a stair and hold onto a handrail for balance; the ankle is moved through its full range of motion. As applicable, shoulder weights are added to increase resistance. Increasing the speed of the heel drop adds an eccentric component to the exercise, as recommended by Stanish et al. **(Stanish et al., 1986)** In addition to having adequate length and function of the triceps surae, one must recognize that the leg functions as a unit. If the hamstrings are also tight, there will be additional tension on the triceps surae complex. **(Myers, 2001; Levangie & Norkin, 2001)**

It is important to correct all aspects of extended pronation. It is the most common factor, along with the short triceps surae, that is present with Achilles tendon disorders.

In most cases, significant reduction of training loads is necessary during rehabilitation. In some cases, it is necessary to discontinue running completely. Under these circumstances, other activities such as swimming or bicycling can be done to maintain the training program. **(Maffetone, 2010; Langer, 2007; Ogon et al., 1999; Schepsis & Leach, 1987)** In cycling, the heel should be put on the pedal to eliminate strain to the Achilles tendon. Clement et al. **(Clement et al., 1984)** recommend continued rest for seven to ten days post–symptomatically, before progressive return to pre–injury activity is attempted. Running should not be resumed until structural balance is

indicated by an applied kinesiology examination. Failure to get adequate rest and obtain structural balance often results in an exacerbation of Achilles tendinitis.

When running is resumed, it should be reduced and done only on soft, smooth surfaces, avoiding hills. Shoes should have a stable, rounded, flared heel and be properly fitted. A sturdy heel counter cannot be overemphasized as a preventive measure. **(MacLean et al., 2008; Krissoff & Ferris, 1979)** A too–rigid sole does not allow the metatarsophalangeal joints to adequately flex; it lengthens the foot's lever arm and increases strain on the Achilles tendon. **(Maffetone, 2003; Levangie & Norkin, 2001; Clement et al., 1984)**

Souza **(Souza, 1994)** reviews the conservative care of various orthopedic conditions in the lower extremities, including disorders involving the Achilles tendon. Co and Pollard **(Co & Pollard, 1997)** describe the care of a patient with heel pain due to Achilles tendonitis, retrocalcaneal bursitis, and partial rupture of the tendon. Brantingham et al. **(Brangingham et al., 1994)** offer an excellent review of the successful chiropractic care (i.e., with 70% reduction of pain) for a patient with Achilles tendinopathy following 3 years of failed medical care. Carter and Carter **(Carter & Carter, 1997)** are advocates for chiropractic care (using a multidisciplinary approach) for patients with Achilles tendinopathy. Gaymans and Till as well as Cook et al. **(Gaymans & Till, 2003; Cook et al., 2002)** investigate the efficacy of manipulating the foot and ankle joint fixations compared to placebo in the treatment of chronic Achilles tendonitis. Kobsar & Alcantara **(Kobsar & Alcantara, 2009)** describe the post-surgical chiropractic care and rehabilitation of a professional ballet dancer following surgical calcaneal exostectomy and debridement with re-attachment of the Achilles tendon. Ramelli **(Ramelli, 2003)** also describes the successful chiropractic post-surgical care of a 25 year-old male to repair a complete Achilles tendon separation.

Recognizing Achilles tendinitis and not trying to "run through" the problem, along with proper conservative treatment, corrects most of these conditions. It is the competitive distance runner who is likely to have surgery for Achilles tendinitis when conservative methods fail. **(McDougall, 2009; Langer, 2007; Ogon et al., 1999; Schepsis & Leach, 1987)** When in doubt about whether one is dealing with peritendinitis or partial rupture of the Achilles tendon, conservative treatment is recommended. If not successful, surgical exploration often reveals a partial rupture. **(Nelen et al., 1989)**

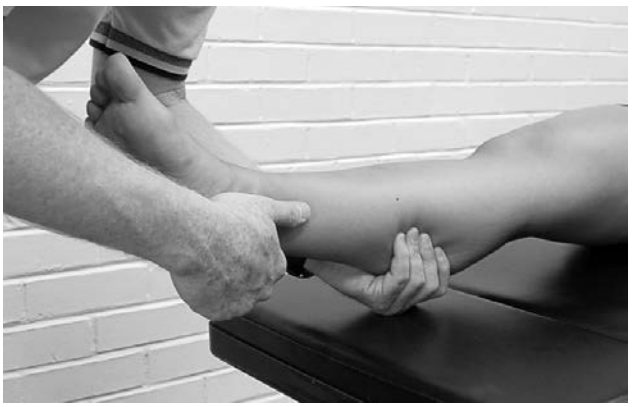
Achilles tendon rupture

Achilles tendon rupture is not often seen in an applied kinesiology practice. The Achilles tendon is the strongest one in the body and only ruptures with degenerative changes, e.g. gout, **(Beskin et al., 1987)** syphilis, training errors, or medication. **(Hess, 2010; Veves et al., 2002)** Other predisposing factors can be prolonged corticosteroid therapy, overuse of performance enhancing drugs (the bodybuilder steroids), and direct trauma to the area. **(Newnham et al., 1991)** The incidence increases if the practice has a large number of middle–aged white–collar workers who engage in part–time athletic activities. **(Kubo et al., 2007; Langer, 2007; Leppilahti, 1998)** It occurs more often in athletes over 30 years of age, less frequently in younger athletes, and rarely in the elderly **(Kubo et al., 2007; Langer, 2007; Ramelli, 2003; Leppilahti, 1998)** unless they pursue past their prime. **(Hess, 2010; Veves et al., 2002)** The vulnerable person is the weekend, but otherwise sedentary, athlete who participates in sports that put a quick impulse stretch on the Achilles tendon. **(Leppilahti, 1998)**

The etiology of Achilles tendon rupture is controversial. **(Hess, 2010; Ramelli, 2003; Veves et al., 2002)** One concept indicates there is repetitive microtrauma in an aging tendon, possibly relating to deficient blood supply. Another theory is that there is an anomaly in the normal inhibitor mechanism of the musculotendinous unit monitoring force to prevent excessive tension on the tendon. Both mechanisms of Achilles rupture may be applicable, with the former relating to an older person's injury and the latter to younger, more active well–conditioned athletes.

One must consider the neuromuscular spindle cell and Golgi tendon organ interrelationship in the control of functional stability of the muscles of the ankle. Afferent information for functional stability of the ankle comes from receptors in the muscle-tendon system. This is in accord with research on other joints. **(Macefield, 2005)**

Murphy provides an excellent review of current concepts of proprioception, but notes that a great deal remains to be learned about proprioception in the human neuromuscular system. **(Murphy, 2000)** The muscle spindles and Golgi tendon organs are two of the most important mechanoreceptors in muscles. The integration of the information coming from the muscle spindle and the Golgi tendon organs in the Achilles tendon complex must be congruent. If they are not, injuries to this complex are likely. **(Loram, 2009)**



Thompson's squeeze test

Steroids, whether injected for tendinitis or administered parenterally for another condition, increase the likelihood of Achilles tendon as well as plantar aponeurosis rupture. **(Brinks et al., 2010)** In one study (n=48), 63% of the subjects had prior corticosteroid injections for tendinitis ruptures. **(Kellam et al., 1985)** Hydrocortisone injected into the tendon causes separation of collagen fibers within 45 minutes. **(Olchowik et al., 2008)** The medication may relieve the symptoms, allowing the individual to increase activity that might lead to a partial or complete rupture.

Steroids also interfere with the healing process. **(Clement et al., 1984)**

The effect of parenteral steroid treatment is emphasized in a case report of a woman who had nine months of steroid treatment for rheumatic fever and experienced bilateral simultaneous Achilles tendon ruptures while simply walking up stairs. Her ankles gave way in an inversion–type mechanism. She experienced no snapping or popping at the time. **(Price et al., 1986)** Hersh and Heath **(Hersh and Heath, 2002)** point out that spontaneous Achilles tendon rupture associated with long-term oral steroid use is not uncommon, particularly in older patients who use these drugs daily to treat systemic diseases. The long-term use of oral steroids poses problems: immunosuppression, cardiovascular disease, depression, insulin resistance, tissue degeneration, mood swings, slow tissue healing and susceptibility to infection have been noted by clinicians managing patients with chronic pain using steroids. **(Wilson, 2002)**

Achilles rupture due to skiing is declining because of the high-ridged boot and multiple mode release ski bindings. **(Oden, 1987)**

Examination

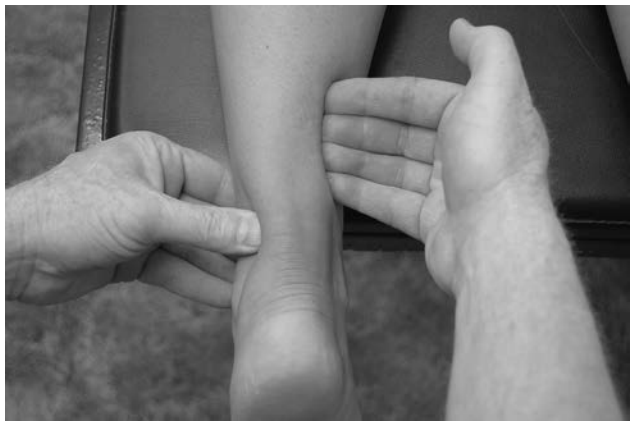
Examination must be done carefully, because up to 25% of ruptures are initially missed. **(Slimmon & Brucker, 2010; Ferran, 2009)** This may be due to insignificant pain. It may initially be a sharp, sudden pain that diminishes to slight or even disappears. In addition, the precipitating violence may be fairly mild. **(Hess, 2010; Hersh & Heath, 2002)** Achilles rupture may be missed when there is other obvious trauma. A case is reported in which the rupture was missed until it was recognized during ankle fracture surgery. **(Martin & Thompson, 1986)**

Partial rupture causes a stabbing, pricking pain with activity; there are localized regions of tendon thickening and nodules. With partial rupture there is some loss of plantar flexion due only to pain. **(Clement et al., 1984)** Palpation may disclose a small gap in the tendon, with a tender, rolled portion of the tendon on the proximal edge. The damage to the tendon may be both transverse and longitudinal. **(Hess, 2010)**

With total rupture there may be no immediate pain, but there is an immediate functional loss. The patient may experience an audible snap or a sensation of being hit in the back of the leg. There is usually no prior indication that the Achilles tendon is vulnerable to rupture. A definite gap may be palpated in the Achilles tendon within 6–12 hours after rupture. After 24 hours, the gap becomes filled with a hematoma and is difficult to distinguish. **(Rolf & Movin, 1997)**

There is only weak plantar flexion provided by the peroneus longus and brevis and tibialis posterior muscles, **(Clement et al., 1984)** with no ability to plantar flex against resistance. This causes a lack of normal push–off during walking. (**Levangie & Norkin, 2001; Thompson & Doherty, 1962**) When the patient's ankle is passively dorsiflexed by the examiner, there is no firm feeling of the Achilles tendon. **(Ramamurti, 1979)**

Because weak plantar flexion is maintained by the tibialis posterior, peroneus longus and brevis, and the toe flexors, **(Travell & Simons, 1992)** diagnosis may be obscured or delayed. **(Cox, 1985)** The Thompson squeeze test is pathognomonic **(Logan, 1995; Turco & Spinella, 1987; Thompson & Doherty, 1962)** of complete rupture of the Achilles tendon. The test can be done with the patient prone, kneeling on a stool, or on all fours with the feet clear of the table or chair. With the patient relaxed, the physician squeezes the calf muscles. This normally creates passive plantar flexion of the foot. Lack of continuity from the muscles to the calcaneus because of Achilles tendon rupture causes no foot movement from squeezing the gastrocnemius–soleus muscles. Contact for the squeeze should be at the bulk of the muscles. When it is applied proximal to the apex of the soleus curve, there is normally no plantar flexion. **(Clement et al., 1984)**



Anterior Achilles tendon can be assessed

Soft tissue changes are observable on x–ray to identify Achilles tendon rupture. **(Slimmon & Brucker, 2010; Yochum & Rowe, 2004; Clement et al., 1984)** A lateral view, with the foot at 90°, should be taken of both the normal and the traumatized foot for comparison. The fat pad triangle between the Achilles tendon and bone is encroached upon by bleeding and edema, causing its sharp border to disappear. Likewise, the fat pad posterior to the tendon is also encroached upon and narrowed. The proximal end of the Achilles tendon may be difficult to identify because of retraction and the surrounding blood and edema. The distal end is usually visible because it is surrounded by the heel fat pad. A bright transillumination light to view the x–ray may help identify the soft tissue structures.

Magnetic resonance imaging (MRI) can help diagnose between partial, severe, and complete rupture of the Achilles tendon. It accurately predicts the condition of the tendon found at surgery. **(Tourne et al., 2010)**

Treatment

There is controversy about whether surgical or non–surgical treatment is optimal. **(Ramelli, 2003)** Those in favor of non–surgical treatment, which includes casting for up to eight weeks, cite the incidence of high surgical complications, increased cost, and good results of non–surgical treatment to support their viewpoint. Those in favor

of surgical treatment indicate that the tendon is returned to a more normal length, strength, and range of motion, and the incidence of re–rupture is lower. Complication rate is low, but not as low as with conservative care. Regardless of whether surgical or non-surgical methods treatment is given, Achilles tendons are susceptible to re-rupture. Carden et al's review **(Carden et al., 1987)** of 106 patients with ruptured Achilles tendons showed that in patients treated within 48 hours of injury the result was very similar in conservatively and in operatively treated patients. The incidence of major complications was higher after operation (17%) than in those treated conservatively (4%). However conservatively treated cases have a re-rupture rte of 17.9% compared with a rate of 2.2% in the surgically treated cases.

In a study evaluating results published in the literature in the previous twenty–five years, Wills et al. **(Wills et al., 1986)** found that the rate of re–rupture for surgically treated patients was 1.54%, while for non–surgically treated patients it was 17.7%. Complications as a result of surgical treatment were 20.0%; non–surgical complications were 10%. The surgically treated patients lost thirteen weeks from work, while non–surgically treated patients lost nine weeks. When the re–rupture factor was considered, cost and loss of work advantage for the non–surgically treated patients tended toward balancing.

Molloy and Wood, **(Molloy & Wood, 2009)** in a recent review of the medical literature regarding the treatment of the Achilles tendon, show that tendon re-rupture, sural nerve morbidity, wound healing problems, changes in tendon morphology, venous thromboembolism, elongation of the tendon, complex regional pain syndrome, and compartment syndromes are not uncommon after surgical repair of the ruptured Achilles tendon.

Lea and Smith **(Lea & Smith, 1972)** recommend non–surgical treatment of Achilles tendon rupture. They point out that the tendon has been shown to regenerate itself, and there is a high percentage of complications in surgical repair. They present a study of 66 cases of Achilles tendon rupture treated non–operatively to emphasize the value of non–surgical treatment. The procedure consists of immobilizing the ankle in gravity equinus position in a walking cast for eight weeks. The patient then uses a 2.5 cm heel lift for four weeks, while mobilizing the ankle with gentle resistance exercises that also strengthen the triceps surae. During this period, the patient is advised to avoid sudden jerky movements of the ankle. The results in this series were very good, with re–rupture of the Achilles tendon occurring primarily in cases that were immobilized for a period less than eight weeks. Eleven of the cases were Achilles lacerations, in which the Achilles tendon was completely severed. These had an effective correction similar to the spontaneous ruptures.

Percutaneous repair of Achilles tendon rupture consists of suturing the tendon together without surgically exposing the tendon. It has the cosmetic advantage of not leaving a large scar, but there is the disadvantage of occasional re–rupture over the better results of open repair. **(Nilsson-Helander et al., 2008)**

Surgical repair takes precedence in high performance athletes and those in ballistic sports, e.g. tennis, basketball, racquetball, and volleyball. **(Deangelis et al., 2009; Langer, 2007)** According to Kellam et al., **(Kellam et al., 1985)** non–surgical treatment of Achilles tendon rupture should be considered in a non–athletic patient over the age of fifty because of the high incidence of post–operative wound problems in this age group. In making a decision, an individual should be informed that there is a higher incidence of re–rupture in non–surgically treated Achilles tendon ruptures. In a comparative follow–up study of surgical and non–surgical treatment, 9 of 23 patients treated non–surgically had re–rupture of the Achilles tendon. **(Inglis et al., 1976)** When surgery is to be done, it should not be delayed; follow–up revealed 20% less strength, as tested by the Cybex II dynamometer, when diagnosis and surgical treatment were delayed more than one month.

There are numerous surgical techniques described for repairing a ruptured Achilles tendon. When neglect has caused tendon shrinkage, the surgeon can join the tendon by suturing a supporting substance such as Marlex mesh. **(Ozaki et al., 1989)** The tendon can be supported by suturing in other tendon material, e.g., the peroneus brevis **(Turco & Spinella, 1987)** or plantaris tendon. **(Hohendorff et al., 2009)**

Of the three approaches to Achilles tendon rupture, open surgical repair should be applied to the elite, active athlete to prevent re–rupture; percutaneous repair should be used in the recreational athlete and those concerned with cosmetics; and — finally — inoperative conservative management is suggested for sedentary older patients (over age 50), or chronically ill or debilitated patients. **(Leppilahti, 1998)**

Trauma to the Achilles tendon can be prevented by correcting faulty foot and ankle biomechanics as discussed in this and the previous chapters. It is particularly important that the triceps surae be long enough to provide adequate ankle dorsiflexion. Runners and other athletes should subscribe to a daily program of lower leg strength and flexibility exercise and avoid potentially aggravating training surfaces. The training program should be consistent, without sudden changes in intensity. Athletic and casual shoes should be properly designed and fitted. **(Abshire, 2010; Maffetone, 2010, 2003; Langer, 2007)**

Achilles bursitis

There are two bursae associated with the Achilles tendon. The pre–calcaneal bursa is located between the skin and the Achilles tendon. The retrocalcaneal bursa is between the Achilles tendon and the bone.

The pre–calcaneal bursa is most often involved, and is usually secondary to tight or otherwise ill–fitting shoes. Diagnosis is made by palpating the bursa. The pre–calcaneal bursa is located by lifting the skin posterior to the tendon between the thumb and forefinger. The retrocalcaneal bursa is palpated between the thumb and forefinger anterior to the Achilles tendon. Any palpable thickening or tenderness in either area suggests the presence of bursitis. **(Cailliet, 1997; Logan, 1995)**

Treatment is usually as simple as changing shoe size or style to prevent irritating the bursae. It is rare that a prominence on the calcaneus needs surgical removal. **(Brody, 1980)** This is accomplished by molding the posterior calcaneus. **(Michels et al., 2008)**

Tibialis posterior tendinitis

Tendinitis can develop at the proximal or distal tibialis posterior muscle tendons. When proximal, it is a type of "shin splint" that will be discussed later. Either type is often due to excessive foot pronation that may be observed statically or as extended pronation in mid support. **(Yuill & MacIntyre, 2010; Langer, 2007; Maffetone, 2003)**

Discussion here is limited to distal tibialis posterior tendinitis. The condition can first become evident in poorly conditioned athletes or novice runners running on hard surfaces. It usually occurs during the early portion of a training period. Improper shoes or running on a banked or uneven surface can also be causative. When the condition is not associated with increased activity as in athletics, it is usually insidious in its development and associated with uncorrected extended pronation.

The location of pain in distal tibialis posterior tendinitis is posterior to the medial malleolus, localized along the tibialis posterior tendon, extending to its major insertion at the under surface of the navicular. The pain may also extend proximally along the posterior medial border of the tibia to the origin of the tibialis posterior muscle. Pain is present after long runs, especially on uneven surfaces when there is inadequate foot support. Because of the location of pain along the posterior inferior edge of the medial malleolus, this condition must be differentiated from distal tibial stress fracture. **(Conti, 1994)** Rarely the tibialis posterior tendon can dislocate; it may relate with a history of previous minor ankle trauma. The diagnosis is obvious and the treatment is surgical.

Physical examination may reveal crepitant tenosynovitis in the tarsal tunnel and proximally along the posterior and medial tibial borders. There may be swelling posterior to the medial malleolus. Pain is often present on supination or inversion of the foot against resistance; it is often produced during the manual muscle test. **(Yuill & MacIntyre, 2010; Perry et al., 1986)**

It is important to properly diagnose this condition in its early stages because the tendon may become elongated, leading to the necessity of surgery. When the tibialis posterior fails, extended pronation worsens. This produces a vicious circle, because extended pronation was probably responsible for the tibialis posterior tendinitis in the first place.

Most cases of tibialis posterior tendinitis can be resolved by correcting extended pronation, including any primary factors that are contributing to the condition. If the condition continues without adequate resolution, the tendon can become elongated and cause severe foot distortion. Johnson and Strom **(Johnson & Strom, 1989)** have described three stages of the dysfunction. They consider only the first stage applicable to conservative treatment. In Stage 1 the pain is mild to moderate, and the patient is not incapacitated

A test and a sign are described by Johnson and Strom. **(Johnson & Strom, 1989)** They use the single–heel–rise test to assess tibialis posterior tendon function. Many of the positive characteristics of this test are due to extended pronation. Motion of the foot is assessed as the patient rises on the ball of one foot, with the other foot held off the ground. (While doing this the patient may stabilize against a door or wall for balance.) They state, "The normal sequence for a single–heel rise is as follows. First, the TPT [tibialis posterior tendon] is activated, which inverts and locks the hindfoot, thus providing a rigid structure. Next, the gastrosoleus muscle group pulls up the calcaneus and the heel rise is completed. With elongation of the TPT, however, the initial heel inversion is weak and the patient either rises up incompletely without locking the heel or does not get up on the ball of the foot at all." In Stage 1, the patient is usually able to rise up on the ball, but it will be more painful than the normal side.

In Stage 2, the tendon becomes elongated and foot distortion develops. Pain increases, and the patient finds it difficult to walk and perform normal activities. Swelling and tenderness, located in the same area, increase. The single–heel–rise test becomes more abnormal. The patient stands with his forefoot abducted, giving rise to the "too many toes" sign. This is where the examiner stands behind the comfortably–standing patient and observes for forefoot abduction by counting the number of toes seen on the deformed side. In addition to forefoot abduction, the calcaneus rotates laterally into a valgus position (Helbing's sign).

In Stage 3, the pain may transfer to the lateral aspect of the hindfoot and be located over the sinus tarsi; the hindfoot goes further into a valgus position. The talus impinges on the sinus tarsi, producing the patient's pain symptoms. As this condition progresses into Stages 2 and 3, a surgical tendon transfer of the flexor digitorum longus may be needed to provide stability to the foot and ankle. **(Johnson & Strom, 1989)**

Yuill & Macintyre (**Yuill & MacIntyre, 2010**) present a case report in a 14 year-old soccer player with tibialis posterior tendonopathy who developed right medial foot pain after striking an opponent in the leg while trying to kick the ball 4 months prior to his presentation. Outcome measures of treatment (utilizing Active Release Technique) **(Leahy, 2008)** included subjective pain ratings and manual muscle testing. On initial examination, the tibialis posterior manual muscle test was graded weak. The patient was treated with 4 sessions over 4 weeks with Active Release Technique, particularly over the insertion of the tibialis posterior muscle. Tibialis posterior rehabilitative exercises were also given. After 4 weeks of chiropractic treatment the patient was able to return to playing soccer relatively pain free (1/10 VAS score with jumping and landing), and manual muscle testing of the tibialis posterior showed decreased weakness and improvement.

Shin Splints

The term "shin splints" is misused by most laymen and often by doctors. Common usage indicates any pain in the leg that develops as a result of running, jumping, or other similar activity. There are many factors that can cause this type of pain. An effective therapeutic approach cannot be designed unless the exact cause of the pain is determined, as the etiology of apparently identical symptoms can be considerably different. In addition, there are conditions such as the compartment syndrome, discussed later, that if not treated properly and timely can cause permanent loss of function. Failure to accurately diagnose acute compartment syndrome, and to treat it as a "shin splint" syndrome instead

is a common basis for malpractice litigation. **(Bourne & Rorabeck, 1989)** Estimates have shown that shin splints account for 10-15% of all running injuries and up to 60% of all leg pain syndromes. **(Korkola & Amedola, 2001)**

In addition to being non-specific, the term shin splints is misleading. Dictionary definition **(Dorland's Illustrated Medical Dictionary, 2007)** provides the following definitions for shin: 1) The crest or anterior edge of the tibia, and 2) the anterior aspect of the leg below the knee. This would indicate, then, that shin splints is a pain of the anterior leg. In reality, it can be of the anterior or posterior leg. Ideally, the term "shin splints" should be discarded entirely. **(Galbraith & Lavallee, 2009; McBryde, 2007)** Since the term is in common usage and has been defined in Standard Nomenclature of Athletic Injuries, it will be used here with adherence to the definition. Shin splints is restricted to "pain and discomfort in the leg from repetitive running on a hard surface or forcible, excessive use of the foot flexors; diagnosis should be limited to musculotendinous inflammations, excluding fatigue fracture and ischemic disorder."

There are four factors that must be present for the diagnosis of shin splints. **(Galbraith & Lavallee, 2009; Slocum & James, 1968)**

1) Lesion must lie within the plantar flexors or dorsiflexors of the foot. These include but are not exclusive of the tibialis posterior, soleus, flexor hallucis longus and flexor digitorum longus as plantar flexors, and the tibialis anterior, extensor hallucis longus and the extensor digitorum longus as dorsiflexors.

2) Pain develops with repeated rhythmic activity incurred while walking, running, with possible jumping. Untrained, undeveloped, or poorly functioning muscle, or muscle weakened by fatigue is most likely to be involved. Madeley et al. **(Madeley et al., 2007)** show that athletes with shin splints have significant endurance deficits in the ankle joint plantar flexor muscles, including the tibialis posterior. The applied kinesiology Repeat Muscle Activation Patient Induced testing of the tibialis posterior is commonly positive in these patients. **(Gangemi, 2011; Walther, 2000)**

3) Tenderness is most often at attachment of the muscle to the periosteum of the bone or interosseous membrane. The tenderness is most often accompanied by classical signs of mild inflammation.

4) Conditions resulting from direct trauma or disease are excluded, including stress fractures, compartment syndrome, peripheral arterial disease, peripheral nerve entrapments, medial tibial syndrome and soleus syndrome producing periostitis. **(Gerow et al., 1993)**

Shin splints result from abnormal stress upon the soft tissue structure in the anterior or posterior muscle group. The stress results from running or otherwise using the leg in such a manner as to repeatedly activate the muscles. The condition develops with less exogenous stress when there is muscular dysfunction or structural imbalance. The endogenous and exogenous stresses combine to create the condition. The strain or stress can be localized to the muscle itself, as well as the attachments of the muscle to bone, in which case a periostitis develops. **(Gerow et al., 1993; Subotnick, 1991, 1975)** The tight, aching feeling develops, then, from tendinitis, myositis, and/or pereostitis, which should all be evaluated during examination.

During the phases of gait, the anterior muscle group, which includes the tibialis anterior, extensor digitorum longus, and extensor hallucis longus, comes into play at heel strike to decelerate the rest of the foot coming into contact with the ground. Thus, if there is hard heel contact with a solid substrate, the muscles become more active and thus increase the strain. There is continued action of these muscles to prevent the forefoot from slapping the ground.

Normally the anterior muscle group is active during the swing phase, toe-off, and heel contact. When the muscles must continue to be active during the stance phase of gait, they are active during much of the total cycle, thus being overactivated and becoming vulnerable to inflammatory reactions. Edema develops which cannot escape from the basically closed anterior compartment, and aching and pain result.

Athletic training often develops an imbalance of function between the anterior and posterior groups, with the anterior group being weaker. Balance must be regained between the muscles. This often requires stretching of the posterior group to regain adequate dorsiflexion of the foot.

Shin splints of the posterior compartment involve the tibialis posterior, soleus, flexor digitorum longus, and the flexor hallucis longus muscles. This is particularly true for those with mild claw toe deformity. **(Garth & Miller, 1989)** These muscles are active during the stance phase from just after heel contact to just prior to heel-off. If the foot fails to resupinate from its pronated stance at the proper time, the muscles are overstressed, setting up posterior shin splints. The tibialis posterior is especially stressed under this condition because its activity is attempting to stabilize the ankle mortise as well as support the medial longitudinal arch. The tibialis posterior muscle may have tendinitis in the tarsal tunnel, and myositis of the total muscle.

The soleus muscle has become more strongly implicated in shin splints by Michael and Holder **(Michael & Holder, 1985)** particularly when the heel is in a pronated position. From evidence produced by 3-phase radionuclide bone scanning, they describe a characteristic scintographic appearance implicating the soleus muscle attachments as stress areas.

The mechanism of soleus stress appears to relate with pronation where the calcaneus is in a valgus position. The soleus has been shown to be a bipartite muscle that has a medial and a lateral division. **(Moore et al., 2009)** The medial head of the soleus is an inverter of the calcaneus. When an individual with extended pronation runs or walks, the medial head of the soleus appears to attempt to counteract the excess heel eversion, thus putting extra strain on origins of the soleus. This is supported by electromyographic activity of the medial soleus muscle when the heel is passively everted in individuals with shin splints but not with normal subjects.

The role of extended pronation being contributory to shin splints is emphasized in a study by Viitasalo and Kvist, **(Viitasalo & Kvist, 1983)** which revealed an increased Achilles tendon angle (Helbing's sign) in athletes who are prone to shin splints over a normal control group. While running, they had an increased Achilles tendon angle at heel strike and throughout the pronation stage of the stance phase of gait. The shin splint group also had an increase in the passive movement of inversion to eversion.

In studying shin splints in the young athlete, Jackson et al. **(Jackson et al., 1978)** found a common type of shin pain to be localized along the distal posterior medial tibia. It is often a specific response of the bone to stress. In a study of 40 young athletes with leg pain, they found 26 stress reactions of the posterior medial aspect of the distal tibia, with the rest distributed among typical stress fracture, musculotendinous inflammations, and other categories. This makes the stress reaction to the distal tibia the most common source of disability in competitive young runners. The pain generally begins gradually and increases with increased activity.

Diagnosis

In early shin splints, symptoms are minimal and well-defined. By the time the individual seeks a physician's help, the condition is often advanced with signs and symptoms much more vague, making localization and differential diagnosis more difficult. **(Galbraith & Lavallee, 2009; Slocum & James, 1968)** Shin splints must be differentially diagnosed from stress fractures, acute and chronic compartment syndromes, fascial hernias, tenosynovitis, and chronic interosseous membrane strains with or without exostosis formation. In addition, specific disease processes, which have distinctive diagnosis such as cellulitis, chronic osteomyelitis, thrombophlebitis, intermittent claudication, infective and varicose pereostitis, and tumors must be considered.

The pain of a stress fracture is usually located along the medial and lower third portion of the tibia. The pain is usually localized in the bone itself and is very painful to palpation of the bony surface around the fracture site; edema and warmth may also be present. X-rays may not detect the stress fracture for several weeks. Treatment for this kind of stress fracture is rest and reduced weight-bearing stress.

Travell and Simons **(Travell & Simons, 1992)** describe a medial tibial stress syndrome which is related to tension placed upon the periosteum of the tibial cortex which may result in its separation. The distal half of the medial tibia will exhibit localized pain at the sites of the overstressed muscular insertions. Pain often extends to a larger area than that found in stress fractures. **(Edwards & Myerson, 1996)**

Palpation determines the area of maximal tenderness, which is either localized to the bone, the muscles, or the tendon. Often bony tenderness is just distal to the muscle insertion (there are no muscle insertions on the posterior medial tibia in the distal one-third of the leg). Stressing the musculotendinous unit, as in a muscle test, shows inflammation or tear in that unit when there is increased pain. Differentiating between motion or weight bearing aggravating pain helps to distinguish between musculotendinous inflammation or involvement of the bone itself. There may be soft tissue swelling in a bony reaction, which should not be confused with inflammation of an overlying tendon. **(Jackson et al., 2001)**

Shin splints can be classified as to severity of pain. Grade 1, pain after athletic activity; grade 2, pain before and after athletic activity, does not affect performance; grade 3, pain during and after athletic activity, affects performance; grade 4, pain so severe the athlete is unable to compete.

Treatment

Treatment should not begin before ischemic conditions, stress fractures, and other non-related conditions are ruled out. The acute condition can be treated by reduced or changed physical activity, and ice before and after any running. The condition is frequently made worse by attempts to stretch the affected intercompartmental musculature. **(Schafer, 1986)** Additionally, it is often important that the examination should occur after the patient has exercised enough to reproduce the symptoms.

Various therapeutic efforts for shin splints are put into perspective by Andrish et al. **(Andrish et al., 1974)** They studied 2,777 first-year midshipmen at the United States Naval Academy using four prophylactic programs in an attempt to prevent shin splints during training. For control, these groups were compared with the men in the usual physical education program. The various approaches were as follows. 1) They used a heel pad in an attempt to decrease the amount of violent muscle pull on the tibial muscle origins and to decrease the amplitude of the stress applied to the tibia. 2) Heel cord stretching exercises were used because observation was that the majority of shin splint victims have tight heel cords. It is not specifically stated that the heel cord stretching exercises were started at the same time as training. 3) There was use of a heel pad and heel cord stretching exercise. 4) A graduated running program instituted for two weeks prior to pursuing the normal physical education routine. None of the prophylactic approaches were effective in preventing shin splints. In fact, the study groups had more incidences of shin splints than the control group.

Those who developed shin splints in this program were assigned to one of five treatment programs. All treatment programs included no running until pain-free. Additionally, there was 1) ice applied to the affected area three times a day; 2) same ice regime and ten grains of aspirin four times daily for one week; 3) same ice regime and 100 mg phenylbutazone four times a day for one week; 4) same ice regime and heel cord stretching exercises; 5) a short walking cast for one week. The group who received only no running until pain-free statistically demonstrated a significant advantage over the other regimes. Additionally, in order of descending effectiveness were rest, ice and phenylbutazone; rest, ice, and heel cord stretching exercises; rest, ice, and aspirin; and, finally, as the worst, casting.

Andrish et al. **(Andrish et al., 1974)** concluded their report with, "Finally, the fact that twice as many of those with shin splints had no previous training immediately before entering the Naval Academy agrees with the commonly held belief that shin splints tend to be more prevalent in the unconditioned athlete or recruit."

Alternate physical activity, such as biking or swimming, can be used to maintain cardiovascular proficiency. Lutter **(Lutter, 1983)** states, "Approximately five weeks healing time is necessary to return to normal running from an injury of shin splints."

Most often, pronation will be present and should be treated as indicated under that subject. All muscles of the leg, ankle, and foot should be evaluated and treated with applied kinesiology methods if necessary. Particular dysfunctions must be determined in the tibialis posterior

muscle, which is associated with many types of leg, knee, foot, and ankle dysfunction. Inhibition of this muscle results in excessive pronation because the tibialis posterior is important in maintaining the medial longitudinal arch when walking and running. **(Hamilton, 1985)** The muscle is also a primary one for athletes who must rise upon their toes. Maffetone **(Maffetone, 1999)** notes that tibialis posterior inhibition may be followed by reciprocal tightness of the gastrocnemius and soleus muscles, producing "posterior shin splints". Pincer palpation, muscle stretch reactions, neurolymphatic reflexes associated with the adrenal gland, and other findings will often be found in this muscle in such cases. **(Leaf, 2010; Walther, 2000)**

Bandy **(Bandy, 1978)** offers the hypothesis that pronation is the primary cause of posterior shin splints. Pronation in this model causes stretching beyond the capabilities of the myofascia of the tibialis posterior muscle and this produces weakness followed by tearing in the muscle during physical activity. Bandy finds no more than 30% of the athletes he has treated with inhibition of the tibialis posterior muscle when tested in the clear; however after the muscle stretch reaction (indicating fascial tension in these cases), the muscle then becomes inhibited on MMT. When he treats the fascia of this muscle, using the origin-insertion technique taught in applied kinesiology, the pain from the shin splints in these athletes is relieved quickly. Heavy pressure to the "knot" or "wrinkle" in the fascia near its attachment to the tibia is effective in these cases. This approach combines the methods of Ida Rolf with Dr. Goodheart effectively. **(Rolf, 1977)**

A momentary overload that causes rupture (tearing) of the tibialis posterior muscle or its tendon has been described. **(Holmes et al., 1990)** This kind of micro-avulsion injury is what the applied kinesiology origin-insertion technique was designed for originally. Microavulsions for the pectoralis major, **(Marmor et al., 1961)** peroneal, **(Davies, 1979)** gastrocnemius, **(Froimson, 1969; McClure, 1984)** plantaris, and extensor digitorum longus **(Perlman & Leveille, 1988)** muscles has also been described. As with a muscle bruise, microavulsion in a muscle is generally treated manipulatively to reduce inflammatory reaction and then promote healing. It is rarely repaired surgically. **(Mense & Simons, 2001)** In the case of a ruptured tendon especially in the ankle region, however, surgical repair may be critically important to avoid muscle imbalance and serious disturbance of ambulation.

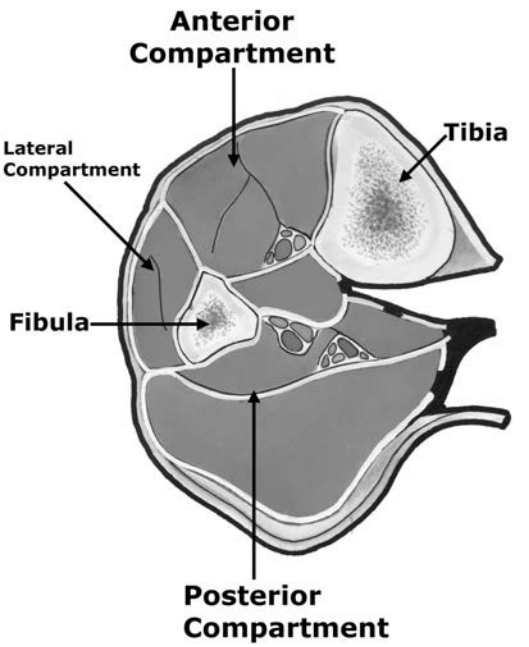
Mense and Simons **(Mense & Simons, 2001)** note that "muscle spasm as the result of pain in the same muscle is an exceptional occurrence." Because the number of specific muscles that may be producing pain and joint dysfunction in "shin splints" is large, specific manual muscle tests that isolate the muscles of the anterior and posterior compartments of the calf are critical. Reduced muscle strength has been suggested to be a surrogate marker for the progression of shin splints in some patients. **(Wilder & Sethi, 2004)** Differential diagnosis of the specific muscle(s) producing the problem is essential; and specific MMT of each of these muscles is possible with AK methods.

Poor gait activity, modular interaction, or dural tension may be causing increased stress during the gait cycle. A graduated training program should be developed for the athlete, as shin splints are frequent in the early phases of the training season. The best method of dealing with shin splints is treating dysfunctions of the foot, ankle, knee and leg before the athlete begins training. The technique described by Bandy and used by the authors is effective in treating shin splints that are symptomatic, but treatment is more painful. The use of the Percussor instrument is often effective for treatment of this pathophysiological disturbance as well. **(IMPAC, 2012)** The importance of inflammation in cases of shin splints has been reviewed by Krenner; **(Krenner, 2002)** chiropractic management of this factor in a patient with medial tibial stress syndrome was successful. Comprehensive AK treatment approaches for two cases of shin splints have been reported as well. **(Rogowskey, 1990; Boven, 1988)**

Compartment Syndrome

The leg has five compartments that are non-yielding in character. A compartment syndrome appears when a muscle enlarges, usually with exercise, to the point that the pressure increases within the compartment to interfere with tissue perfusion and neurovascular function to the muscle or muscles in the compartment enclosure. Edema within the compartment, hematoma or infection are additional reasons for this syndrome. **(Gerow et al., 1993)** The pressure can rise to a critical state where there is neurovascular ischemia with paralysis and necrosis following if left untreated in the early stages. It can be a surgical emergency to preserve muscle function. This is done by fasciotomy to relieve pressure within the compartment.

The compartments are closed spaces with the exception of perforation of the fascia by blood vessels and nerves and the exit of tendons distally. There are 46 compartments in



Compartments of leg

the human body with 38 of them in the extremities. While compartment syndrome may occur in a number of areas, over 80% of them occur in the lower extremities. **(Gerow et al., 1993)** Early descriptions **(Carter et al., 1949; Mavor, 1956; Bradley, 1973)** were of the anterior compartment, followed by descriptions of the lateral compartment **(Reszel et al., 1963)** and posterior compartment which was subdivided into deep and superficial compartments. **(Mense & Simons, 2001; Subotnick, 1991)** A fifth compartment has also been described, containing only the tibialis posterior muscle. **(Gerow et al., 1993; Travell & Simons, 1992; Rorabeck, 1986)**

Fascia is firmly attached proximally at the knee. Distally, the three sets of retinacula strengthen it. The interosseous membrane, bone, and septa complete the compartments into closed spaces with the exception of perforation of the fascia by blood vessels and nerves and the exit of the tendons distally. Bradley **(Bradley, 1973)** draws a fascinating analogy that "This anatomical situation would seem to be analogous in many respects to the cranial vault and its contents." Maykel, incidentally, reports on the successful treatment of 12 cases of compartment syndromes of the stomatognathic system using applied kinesiology methods. **(Maykel, 2002)**

The tibialis posterior compartment is described by Davey et al. and confirmed by Travell & Simons **(Travell & Simons, 1992; Davey et al., 1984)** Follow-up studies have confirmed this fifth compartment. **(Pell et al., 2004; Levangie & Norkin, 2001; Edwards & Myerson, 1996)** The initial study was done by injecting radiopaque dye into the tibialis posterior muscle of amputated legs. The dye failed to traverse the osseofascial boundaries of the tibialis posterior muscle, even though the leg was massaged for several minutes prior to roentgenographs being taken. In cadavers they injected lubricating jelly into the tibialis posterior muscle to increase the pressure and then did a fasciotomy, revealing that the pressure was reduced with the procedure. Finally, in human studies, they did before and after pressure measurements in the tibialis posterior muscle and found it to be independent of the deep posterior compartment.

The superficial posterior compartment contains the soleus and gastrocnemius muscle bellies. The deep posterior compartment encloses the posterior tibial, popliteus, flexor hallucis longus, and flexor digitorum longus muscle bellies. The ability of the examiner to specifically isolate these muscles during manual muscle testing examination gives a distinct advantage in the assessment of these compartments and their functional status.

Compartment syndrome etiology can be grouped into three areas. **(Levangie & Norkin, 2001; Platzer, 1992; Bradley, 1973)** Group 1 is idiopathic, associated with strenuous muscle activity and often referred to as the exercise form, or exertional compartment syndrome. **(Edwards & Myerson, 1996)** Group 2 is traumatically induced, including fractures, sprains, and other injuries of the soft tissue. Group 3 is interruption of major vascular supply, including vascular injuries, disease, and thromboembolism. In other than the vascular group of compartment syndromes, the dorsalis pedis pulse is most often present. Group 1 must be differentially diagnosed from shin splints, which is often loosely termed, not being limited to musculotendinous inflammation. Another common cause of a compartment syndrome is external compression of the leg from a cast or anti-shock trousers. **(Lutz & Goodenough, 1989)**

Lueck and Ray **(Lueck & Ray, 1972)** point out that skeletal muscle is able to produce energy during periods of anoxia by the anerobic cycle. This creates an oxygen debt that is replenished later by the local factors that increase local blood flow. However, if this is not replaced, the high concentration of lactic acid and anoxia lead to muscle necrosis. Mense and Simons **(Mense & Simons, 2001)** show that after approximately 4 minutes of hypoxia, muscle spindle discharge become irregular and then stop suddenly. The muscle spindle discharges recovered approximately 3 minutes after restoration of the oxygen supply. But after a second period of hypoxia, the discharge from the muscle spindles failed sooner and the recovery was incomplete. Apparently, the muscle spindle receptor was damaged by the prolonged hypoxia.

When there are multiple muscles in a compartment, they are not all involved to the same degree. In the anterior compartment this appears to be the result of differential strain to the muscles and the variable blood supply. During exercise, greater stress is placed upon the tibialis anterior muscle and the extensor hallucis longus and extensor digitorum longus muscles. The development of muscle necrosis appears to be individual muscle ischemia. Muscles that have the poorest blood supply and are most active during exercise are most involved. **(Mense & Simons, 2001)**

There is no consensus of the exact etiology of compartment syndrome. Pell et al. and Brody **(Pell et al., 2004; Brody, 1980)** associate lateral compartment syndrome with extended pronation in runners with excessively mobile ankles. The pain is in the general lateral ankle area, and the individual often reports his ankle "gives out." It is known that muscles are more active in extended pronation than in the normal foot. **(Snook, 2001)** Correction of extended pronation and its associated muscle dysfunction can be a step toward preventing compartment syndrome. Bradley **(Bradley, 1973)** considers that compartment syndrome is "…but one stage in the continuum of muscular disorders….Until the critical point for the microcirculation is surpassed, lesser and more common forms of this disease are produced."

Travell & Simons **(Travell & Simons, 1992)** suggest there is a strong possibility that in the muscles prone to developing compartment syndrome, myofascial trigger points may make a significant contribution.

The mechanism of injury frequently reported by patients and the literature is a swelling of overworked muscles after unaccustomed heavy exercise or activity. **(Logan, 1995)** This produces a loosening of muscular fascia from the periosteum of the tibia and/or fibula, producing excessive inter- and intra-compartmental pressure. It appears that almost anyone with sufficient exercise can be a candidate for a compartment syndrome. It can appear in an individual's early athletic training, or in the highly trained athlete.

Edwards and Myerson **(Edwards & Myerson, 1996)** describe exertional compartment syndrome in which the tissues confined in the 5 compartments of the leg are adversely influenced by increased pressure which alters circulation and the viability of the tissues. The increased osmotic pressure and muscle swelling raises

the intra-compartmental pressures. Swelling and pain may produce sensory deficits or paresthesias and motor weakness or paralysis related to ischemic changes within the compartment. With particular emphasis on the anterior compartment, the first motor signs are loss of strength in the extensor hallucis longus and tibialis anterior muscles. **(Gerow et al., 1993)** Characteristically a chronic anterior compartment syndrome demonstrates weakness of the dorsiflexors of the foot, **(Gibson et al., 1986)** making the use of the manual muscle test a valuable tool in the early stage of this syndrome. Pressure on the nerves within the compartment may result in sensory disturbances as well as motor loss which, in severe cases, may result in foot drop. Onset is gradual and associated directly with the intensity of the exercise that the subject undergoes and is usually relieved by cessation of the activity and rest.

Examination for exertional compartment syndrome must therefore take place after the patient has exercised. Muscle weakness will be evident and tenderness and paresthesias to light touch will be present in the involved muscles in severe cases. Neural and arterial occlusion may produce serious complications; assorted diagnostic tests must be conducted for differential diagnosis, with measurement of intra-compartmental pressure a necessity to confirm the diagnosis of exertional compartment syndrome. **(van den Brand et al., 2005; Edwards & Myerson, 1996)**

Muscle trauma can cause an acute compartment syndrome. Straehley and Jones **(Straehley & Jones, 1986)** report on a fifty-one year old male who felt a "pop" in his calf while playing softball and running bases. Increasing pain required examination within four hours, which revealed elevated compartmental pressure. Fasciotomy was performed. A large tear in the gastrocnemius was identified. Eighteen months post-surgery, there were no significant problems with the leg. Surgery in this case revealed specific muscle trauma as cause of the problem. They go on to point out that "the fact that many of the chronic exertional problems occur in well-trained athletes suggests that muscle hypertrophy may act to decrease the relative space available for the obligatory muscle swelling that occurs following exercise." This has been observed from continuous pressure monitoring of the anterior compartment in race walking, revealing that pressure rises as speed is increased. **(Straehley & Jones, 1986)**

Although compartment syndrome is relatively rare, it is mandatory that it be considered in differential diagnosis of leg pain. The acute syndrome is a surgical emergency. Delay in appropriate treatment allows muscle necrosis and permanent impairment to develop. Early muscle damage occurs within four to six hours, and significant irreversible damage will be present in eighteen hours. **(Pell et al., 2004; Lueck & Ray, 1972)** 90% of patients will make a full recovery within 24 hours if the ischemia is present for less than 4 hours; if ischemia lasts up to 8 hours, only 50% will make a full recovery. **(Gerow et al., 1993)**

Differential diagnosis includes cellulitis, thrombo-phlebitis, tibial stress fractures, osteomyelitis, tenosynovitis, and the group that constitutes shin splints (musculotendinous inflammations). A key diagnostic factor is that in these inflammatory or infectious conditions, a true motor weakness, muscular paralysis, or sensory loss may not always be found. **(Pell, 2004)**

Chronic Form

Compartment syndrome is characterized symptomatically by pain upon exertion. The activity is usually considerable, such as when marching or running. Ordinary walking can sometimes initiate pain, which may be burning, cramp-like, piercing, or contracting. It usually disappears after a few minutes of rest. During the painful period, active dorsiflexion of the ankle is often difficult. The pain resembles the often loosely diagnosed condition of shin splints.

Objective findings may include tenderness and tautness over the compartment. Fascial defects were found in nearly 60% of a group (n=61) studied by Reneman. **(Reneman, 1975)** Muscle abnormality is found in a majority of patients with compartment syndrome where fasciotomy is performed; suggesting that conservative management of chronic compartment syndrome should focus on muscle dysfunctions. The dorsalis pedis pulse is palpable with disturbance of any of the compartments. **(Moore & Friedman, 1989)**

An important diagnostic method is measuring the intracompartmental pressure during exercise. Several methods for doing this have been reported. **(Shadgan et al., 2008)**

Acute Compartment Syndrome

In the acute form there is a progressive course, and structures within the compartment are subject to complete or partial destruction. In the chronic form the intermuscular pressure returns to the initial value fairly quickly after exercise (9-15 minutes). In the acute form the increased pressure remains for a much longer period of time. **(Reneman, 1975)** Pain usually starts immediately after intensive use of the lower limbs. Active or passive movement of the foot increases the pain. Generally the severe pain comes only after the cessation of the physical activities. In the acute form of the anterior compartment syndrome, loss of motion and motor impairment varies from slight limitation of toe and ankle extension to complete foot drop. Eversion of the foot may be limited or impossible, and there may be involvement of the deep peroneal nerve causing weakness of ankle or toe extension. The pain of a compartment syndrome may become severe during the night. This is thought to relate with the diminished blood flow that occurs during rest.

Reszel et al **(Reszel et al., 1963)** point out that "the swelling and tenderness of the leg, fever, and the normal arterial pulses rarely make the first examiner suspect ischemia. The initial diagnosis in practically all cases is cellulitis or thrombophlebitis."

The acute syndrome must be treated as soon as possible by fascial release or there may be permanent damage to the muscles in the compartment. In the acute form, the blood flow is severely disturbed, and the syndrome leads to necrosis of one or several muscles in the majority of cases. Reneman **(Reneman, 1975)** states, "It is not known why patients with the compartmental syndrome of the leg had increased or total intermuscular pressure at rest or higher values than normal upon exercise. It is difficult

to understand why in one patient the total intramuscular pressure diminishes soon after exercise (chronic syndrome) whereas in the other it remains above the critical level for a long period of time (acute syndrome)."

Femoral or popliteal artery entrapment may simulate a compartment syndrome. **(Pillai et al., 2008; Bell, 1985)** Pillai et al. and Bell report on cases of popliteal artery entrapment syndrome simulating chronic compartment syndrome. The pain of the two conditions is caused by a muscle ischemia after exercising. At rest there are no positive physical findings. Differential diagnosis in this case was done by measuring the compartment pressure and by an arteriogram. The arteriogram is done after exercise creates pain. In this case report the arteriogram was negative in a neutral position, but showed much narrowing with the foot dorsiflexed. This evaluation can also be done on a non-invasive basis by Doppler ultrasound.

Treatment

Treatment of the acute compartment syndrome is fasciotomy: there is a limited time span in which it can be done and save muscular function. Damage to the muscle begins within four to six hours and is irreversible in eighteen hours. **(Mense & Simons, 2001; Edwards & Myerson, 1996)** Even with fasciotomy, the literature does not report a very good success rate. **(Wall et al., 2007; Bradley, 1973)**

Generally, the condition is initially viewed with an effort toward conservative care. Mubarak et al. **(Mubarak et al., 1989)** warn against unnecessary fasciotomy because it creates muscle weakness. Garfin et al. **(Garfin et al., 1981)** demonstrated this in dogs, showing a 15% muscle strength loss after fasciotomy, but the effectiveness of the surgery was shown by a 50% intracompartmental pressure drop. Deep but careful massage (fascial release, percussion, connective tissue techniques to the area) plus specific stretching for the restricted fascial compartments are good initial therapeutic choices. Manual muscle tests will effectively guide the clinician toward the specific myofascial dysfunctions in this complex scenario.

If applied kinesiology treatment methods are applied to an area which is particularly tense, painful or restricted, the clinician may choose to utilize applied kinesiology approaches which release excessive tone; or one of several versions of strain-counterstrain which releases excessive tone and modifies pain; or myofascial release methods such as the fascial flush, percussion, muscle spindle cell, proprioceptive taping or Golgi tendon organ technique; or if joint restriction is involved and on challenge of the articulations around the compartment proves to be a primary factor in the dysfunctional state of the soft tissues, then an HVLA thrust to a remote area causing the local muscle dysfunction should be employed.

The methods of applied kinesiology assessment and treatment have seamlessly merged with a variety of other methods, techniques and modalities providing the modern clinician with an abundant set of resources with which to handle both acute and chronic somatic dysfunctions.

Fasciotomy in cases of compartment syndrome should only be done after objective tests reveal an abnormal intracompartmental pressure elevation. **(Wall et al., 2007; Mubarak et al., 1989)** Post-operative care involves keeping patients off their feet and their legs elevated following surgery for 2-3 days. Most patients are walking unassisted 2 days and may run or jog in 3 weeks. **(Konstantakos et al., 2007)**

Time is of the essence in this condition. When the patient is seen early, before there is any loss of muscle function, it is reasonable to try to alleviate the pressure by rest and elevation and applied kinesiology treatment. However, with evidence of increasing intracompartmental pressure or with the appearance of a neurologic or muscular deficit, it is recommended that the compartment be explored as early as possible. The muscular deficit present in an acute compartmental syndrome places the applied kinesiologist in an excellent position to recognize the possibility of elevated compartment pressure. Muscles that test weak with manual muscle testing should respond to the usual applied kinesiology techniques. If the muscle or nerve is ischemic, there will be no response, giving a positive indication of compartment syndrome possibility.

Restless Leg Syndrome (RLS)

Restless legs syndrome (RLS) is a common cause of severe insomnia. The treatment of insomnia, when successful, is one of the most satisfying clinical successes in an applied kinesiology practice. "How are you sleeping?" is one of the questions we will often ask new patients. We have found over the years that if patients cannot get a good night's sleep, they are not going to fully recover their health.

Insomnia is a term used to describe the more than 80 million Americans who routinely have trouble falling asleep or staying asleep. For anyone who has ever experienced a few sleepless nights in a row, a feeling of desperation sets in as you struggle to function during the following day. Between 30-40% of the U.S., European, and Japanese populations over the age of 15 reports they've experienced insomnia at least occasionally, **(Ohayon, 2010),** with higher mortality in men related to sleep disturbances. **(Rod et al., 2011)**

It is likely that more than 100,000 motor vehicle crashes are caused annually in the United States by driving while drowsy. Major disasters such as Three Mile Island, Exxon Valdez, Bhopal and Challenger were all officially attributed to sleepiness-related impaired judgment in the workplace. **(National Commission on Sleep Disorders Research, 1992)**

RLS is a neurological sensory/movement disorder affecting 5-15% of the general population. **(Phillips et al., 2000)** A survey conducted in May 2005 of some 1,500 adults reports almost 10 percent of the respondents suffered from restless legs syndrome, and more women than men have it. **(Guillemiault & Framherz, 2005)** Up to 65% of patients with RLS use complementary and alternative medicine. **(Cuellar et al., 2004)** For this reason it is essential that primary contact practitioners be aware of this condition to insure its efficient management. **(Stupar, 2008)**

RLS is characterized primarily by a vague and difficult-to-describe unpleasant sensation in the legs. This discomfort appears primarily during periods of inactivity, particularly during the transition to sleep in the evening.

Patients often have difficulty in describing the unpleasant sensations; they rarely use conventional terms of discomfort such as 'numbness, tingling or pain,' but rather bizarre terms such as 'pulling, searing, drawing, crawling, shimmering or boring,' suggesting that RLS sensations are unlike any experienced by unaffected individuals. These distressing sensations are typically relieved only by movement or stimulation of the legs. It is often difficult for the patient to describe because there is no correlating sensation in normal physiology. It is not the same as the leg going to sleep, such as when sitting for a prolonged period with the knees crossed. It will often be described as a creeping sensation, or that there are small worms in the muscles. The legs may feel tired, heavy, and weak. A common corollary is that the patient feels a need to move his legs. Often he will get up several times during the night to walk around, jump, massage the legs, and otherwise try to obtain relief from the condition. In severe cases, there may be great restriction on the amount of sleep obtained.

"Burning feet" and the "restless legs syndrome" may be caused by entrapment of the tibial nerve at the tarsal tunnel. Symptoms from this condition develop from walking or prolonged standing, and may possibly persist during bedrest. The pain sometimes radiates from the foot to the ankle, or even into the calf. **(Staal et al., 1999)**

The Restless Legs Syndrome Foundation puts the condition this way:

> "Restless Legs Syndrome is an overwhelming desire to move the legs usually caused by uncomfortable or unpleasant sensations in the legs. The sensation can occur during periods of inactivity and become more severe in the evening and night. RLS may often cause difficulty staying or falling asleep, which leads to tiredness or fatigue. Up to 8% of the US population may have this neurologic condition. Many people have a mild form of the disorder, but RLS severely affects the lives of millions of individuals." **(http://www.rls.org)**

Many different techniques have been found by patients for relief: walking about, stomping the feet, rubbing, squeezing or stroking the legs, taking hot showers or baths, or applying ointment, hot packs or wraps to the legs. Although these treatments are effective while they are being performed, the discomfort usually returns as soon as they become inactive or return to bed to try to sleep. The motor restlessness often appears to follow a circadian pattern, peaking between midnight and 4:00 am. **(Trenkwalder et al., 1999)** The prevalence of depression and anxiety found associated with RLS is felt to be secondary to the RLS. The relationship between RLS and insomnia generally and psychiatric conditions are bi-directional: depression may cause insomnia, and insomnia may cause depression. **(Picchietti & Winkelman, 2005)**

The movement can happen just at night, when the jiggling may drive a spouse or significant other up the wall, and/or it can occur during the day, when it will drive everyone else up the wall with the incessant tapping or twitching. In a small percentage of people, the sensations of restlessness and twitching may also be experienced in the arms. The automatic jerks of the legs can disturb their sleep enough to produce a feeling of fatigue the morning after but not enough to fully awaken them. The sleeper's mate, however, is usually fully aroused by their partner's unintended kicks. Both of these people will be in your office asking about this condition together, and the tired expression on their faces and the circles around their eyes can be a clue for you to investigate this problem in a patient.

Many patients with RLS take tranquilizers, muscle relaxers, and over the counter sleep aides to get them to sleep. But most people who use these medications never go into deep restorative sleep, the deep delta-wave sleep that allows the body to get its physiological rest and restorative repair.

One of the most obvious and immediate effects of RLS and its associated insomnia is the increased risk of accidents. As reported in Business Week, "Studies show that someone who has been awake for 24 hours has the same mental acuity as a person with a blood alcohol level of 0.1, which is above the legal limit for driving in most states." **(Business Week, 2004)** But when you consider someone who is a health care worker, pilot, or law enforcement officer, the effects can be catastrophic. Some 39% of health-care workers report that they've had a "near miss or accident" at work due to fatigue in the last year. Further, sleep disorders cost the nation about $45 billion every year in lost productivity, health care and motor vehicle accidents. **(Business Week, 2004)**

Recent studies suggest there may be a susceptibility gene locus in RLS, which would explain why RLS is often familial. **(Desautels et al., 2005)** RLS is commonly seen in pregnancy, hemodialysis or peritoneal dialysis for renal failure and iron-deficiency anemia. Its relationship with iron metabolism abnormalities has led to studies indicating that RLS is associated with abnormal iron metabolism within the central nervous system; **(Allen & Earley, 2001)** the iron deficiency may even require treatment when ferritin levels are normal. **(O'Keeffe, 2005)**

Studies relating this problem with nutritional deficiencies of riboflavin, niacin, and B6 have not been explored in the literature. A search of PubMed showed 10,789 papers on riboflavin; 5,591 papers on niacin; 5,968 papers on niacinamide; and 9,070 papers on vitamin B6. A series of searches was performed, using the terms "restless legs syndrome," "nocturnal myoclonus," "riboflavin," "niacin," "niacinamide," and "vitamin B6". None of these papers explicitly correlated these vitamins with RLS.

One of the conclusions of a case series report by Cuthbert **(Cuthbert, 2007)** was that a nutritional relationship between RLS and these three vitamins may be present.

Recent functional neuroimaging studies have identified thalamic, red nucleus and brainstem involvement in the generation of periodic limb movements in patients with RLS. One PET study found reduced dopamine 2 binding in the caudate and putamen. A subcortical origin of RLS

is supported by transcranial magnetic stimulation studies and the successful treatment of the patient cohort in this case series, i.e. RLS has physiological causes . **(Tergau et al., 1999)**

Demonstration of a continuous hypersensitivity to pinprick, but not to light touch, confirms a sensory component to RLS and may explain the efficacy of AK, nutritional, and opiate medications in RLS. **(Stiasny-Kolster et al., 2004)** Patients with RLS will often report that walking, rubbing or massaging the legs, or doing deep knee bends or finger to floor stretches can bring relief, but only briefly. This suggests that there is a structural component to RLS as well.

Medical Treatment of RLS employs a variety of medications. Generally, they choose from dopaminergics, benzodiazepines (central nervous system depressants), opioids, and anticonvulsants. Dopaminergic agents, largely used to treat Parkinson's disease (like Sinemet and Levodopa), have been shown to reduce RLS symptoms and are considered the initial treatment of choice. Good short-term results by treatment with levodopa plus carbidopa have been reported, although patients usually develop augmentation, meaning that symptoms are reduced at night but begin to develop earlier in the day than usual. Dopamine agonists such as pergolide mesylate, pramipexole, and ropinirole hydrochloride may be effective in some patients and are less likely to cause augmentation. The dopaminergic anti-Parkinsonian medications are a particularly common treatment medication now. **(Desautels et al., 2005)**

Benzodiazepines (such as clonazepam and diazepam) may be prescribed for patients who have mild or intermittent symptoms. These drugs help patients obtain a more restful sleep but they do not fully alleviate RLS symptoms and can cause daytime sleepiness. Because these depressants also may induce or aggravate sleep apnea, they should not be used in people with this condition.

For more severe symptoms, opioids such as codeine, propoxyphene, or oxycodone are prescribed for their ability to induce relaxation and diminish pain. **(Limbaugh, 2003)** Side effects include dizziness, nausea, vomiting, and the risk of addiction.

Anticonvulsants such as carbamazepine and gabapentin (Neurontin) are also considered useful for some patients, as they decrease the sensory disturbances (creeping and crawling sensations). Dizziness, fatigue, and sleepiness are among the possible side effects.

The medical and drug company literature shows that no one drug is effective with RLS. What may be helpful to one individual may actually worsen symptoms for another. In addition, medications taken regularly may lose their effect, making it necessary to change medications periodically.

For one of the patients in the study by Cuthbert, **(Cuthbert, 2007)** Sinemet produced urinary incompetence and systemic muscle weakness as well as blurred vision, light-headedness and depression. These kinds of side effects from the medications patients are taking are not uncommon in our experience. 6 of the patients treated successfully with the applied kinesiology protocols in the study by Cuthbert were taking Sinemet and Levodopa (for Parkinson's disease), or muscle relaxers, sedatives, or painkillers for control of this condition. Drugs designed for the treatment of other diseases (Parkinson's disease and seizures) for the treatment of the RLS is not uncommon, and these are examples of a worrisome tactic of Pharmaceutical Companies called "Off-Label Marketing". **(Critser, 2005)**

On May 5, 2005, the FDA approved the first ever drug for treatment of restless legs syndrome: ropinirole (Requip).

The side effects were announced:

> **"Possible side effects:**
>
> SIDE EFFECTS that may occur while taking this medicine include feeling of warmth, dry mouth, sweating, weakness, fatigue, dizziness, drowsiness, lightheadedness, stomach pain, heartburn, gas, nausea, or vomiting. If they continue or are bothersome, check with your doctor. CHECK WITH YOUR DOCTOR AS SOON AS POSSIBLE if you experience swelling of ankles, feet, or hands; unusual fatigue or tiredness; unusual pain; unusual muscle movement; loss of appetite; fast or irregular heartbeat; falling asleep during daily activities; mental or mood changes; impotence; trouble breathing; sore throat; or vision changes. CHECK WITH YOUR DOCTOR IMMEDIATELY IF YOU EXPERIENCE fainting, or chest pain. An allergic reaction to this medicine is unlikely, but seek immediate medical attention if it occurs. Symptoms of an allergic reaction include rash, itching, swelling, severe dizziness, or trouble breathing. If you notice other effects not listed above, contact your doctor, nurse, or pharmacist.
>
> **Drug interactions**
>
> Drug interactions can result in unwanted side effects or prevent a medicine from doing its job. Use our drug interaction checker to find out if your medicines interact with each other. ADDITIONAL MONITORING OF YOUR DOSE OR CONDITION may be needed if you are taking ciprofloxacin, medicines for anxiety (such as diazepam), medicines for mental or mood problems (such as risperidone), medicines for depression (such as fluoxetine), digoxin, theophylline, levodopa, estrogens, phenothiazines (such as chlorpromazine), butyrophenones (such as haloperidol), thioxanthenes (such as thiothixene), or metoclopramide."

The cost of Requip is $219.96 for 100 tablets (approximately 2 months supply).

Non-pharmacological approaches include decreasing caffeine and alcohol intake, education on sleep hygiene and relaxation methods, moderate exercise and nutritional supplements. **(Stupar, 2008)**

AK treatment for RLS

There is no laboratory test that can identify RLS, and the condition cannot be diagnosed by the physician other than by symptoms reported by the patient. This suggests a

functional problem (one of the 5-factors of the IVF) may be the root cause of the disorder.

In each of the cases successfully treated in a case-series report by Cuthbert (n=23), **(Cuthbert, 2007)** pelvic subluxations and weakness of pelvic and leg muscles on MMT were found. Several patients were obese with an anterior sacral base subluxation that increased the lumbo-sacral lordosis. Each of the cases in the series of patients with RLS responded to chiropractic treatment and Cataplex G ™ **(Standard Process Labs, 2012)** vitamin supplementation (a single pill-form combination of riboflavin, niacin, B6, folic acid, PABA, choline, inositol, biotin, and betaine), and this combination of treatment elements was the common factor among all the cases successfully treated (n = 23) for RLS. **(Table 1)**

As part of a thorough whole-body examination, most of the muscles in the body were tested, and the relationship of muscular inhibition on MMT to the patients' primary complaints was explained. Because AK examinations and treatments are interactive, the patient is educated about the relationships between the different areas of the body and how one dysfunction may be creating problems elsewhere. During the course of treatment of patients who presented with more conventional chiropractic problems, the irritation of the RLS was revealed.

The commonality among all the patients with RLS was the finding of facilitation on MMT of inhibited muscle(s) in the legs with oral nutrient testing of Cataplex G. **(Standard Process Labs, 2011)**

Another important part of the successful treatment of RLS in these patients was the reduction of pain in the legs that was present in 14 of the patients. **(LBLP in Table 1)** Treatment of this factor by standard AK methods to the 5-factors of the IVF, including cranial, pelvic category, vertebral, and muscular corrections to the structures found disturbed in the patient proved successful in reducing the low back and leg pain in these cases. In terms of the structural factors, there was no one recurring specific found in a review of these cases with RLS. The heterogeneity of the chiropractic presentation – each patient showing unique and peculiar features – was the rule here.

Eight of the patients who had problems with insomnia, previous to or independent of their RLS, were treated using standard AK evaluation procedures for insomnia problems. **(Walther, 2000)** The patient would be tested in the examination room with the lights off, and the factor that corrected the global inhibitions on MMT with the lights off would be treated.

Additionally, patients who only had trouble falling into sleep often responded to supplementation with Calcium Lactate (SP); **(Goodheart, 1998)** patients who had trouble staying asleep and awoke frequently without cause often responded to Cataplex B (SP); patients with depression, irritable bowel syndrome, and insomnia often improved with 5-HTP (NW) supplementation. **(Nutri-West)** (5-HTP should be taken with 4 ounces of fruit juice, as insulin is required to carry the 5-HTP past the blood brain barrier.) These are consistent findings in an applied kinesiology practice, and were a part of the improvement in the overall picture of some of these patients who had the RLS also.

Twelve of the patients in this case series showed signs and symptoms of adrenal stress disorder (postural hypotension, paradoxical pupillary reaction, Rogoff's sign, weakness of the sartorius, gracilis, or tibialis posterior muscles that showed strengthening upon TL to the relevant reflex, the ligament stress reaction, etc.). In our practice, nutritional support and lifestyle counseling is frequently used for support of the hypoadrenic and/or overstressed patient.

According to several authorities -- Dr. Royal Lee in *Vitamin News* from 1934 and 1952; Dr. George Goodheart in his articles on vitamin B deficiencies; Dr. Wally Schmitt in *Compiled Notes on Clinical Nutritional Products* (1990); and Dr. Philip Maffetone in *Complimentary Sports Medicine* (1999) -- the signs of a vitamin "G" deficiency (or riboflavin, niacin, and B6) include:

1) Cardiovascular -- tachycardia, extra ventricular beats, increased 1st heart sound with a long silence after 1st sound (increased time of diastolic ventricular filling), angina pectoris, and pre-myocardial infarction

2) Psychological -- Excessive worry, apprehension, moodiness, depression, suspicion

3) Digestive -- Insufficient stomach acid production and excess alkalinity, spastic gall bladder

4) Liver -- Cirrhosis of the liver and loss of fat metabolism activity, deficient formation of Yakitron, a physiologic anti-histamine

5) Neurological -- Insufficient acetylcholine activity and cholinesterase activity (for breaking down acetylcholine and for recycling choline), restless, jumpy, or shaky legs, body or limb jerks upon falling asleep, can hear heartbeat on pillow

6) Skin and mucous membranes -- Cheilosis (cracking at corners of mouth), friable skin, especially on face and neck (when shaving), bright red tongue tip, strawberry tongue (purple), loss of upper lip (thin upper lip), irritated mucous membranes of the rectum, vagina, and conjunctiva (frequent tears), excessive oil on face and nose, roughness, cracking and exfoliation of the soles of the feet, and psoriasis

7) Visual -- Burning or itching of eyes, photophobia, blepharospasm, blood shot eyes due to capillary engorgement, seeing only parts of printed words (circumcorneal injection), pallor of the temporal half of optic disc, transient ischemia of retina – like looking through a fish bowl

8) Endocrine – excess estrogen and menstruation, cystic mastitis or gynecomastia, premenstrual tension, and excessive adrenal function.

Dr. Royal Lee, an excellent chronicler of deficiency signs and symptoms in relation to vitamins and minerals, listed the following deficiency signs for the B vitamins that make up the Cataplex G formula, **(Lee, 2007)** which may explain why "Cataplex G" was of value in the cases of RLS reported:

Signs and symptoms for vitamin B2 (riboflavin) deficiency:

a) Myelin degeneration
b) Incoordination
c) Loss of strength in arms or legs
d) Central neuritis, symptoms resembling degeneration of spinal cord

Signs and symptoms for vitamin B3 (niacin or nicotinic acid) deficiency:

a) Myelin degeneration (motor and sensory)

Table 1:
Restless Legs Syndrome Patient List (n=23)

Patient Age and Sex	Problems with Insomnia? (I) Adrenal Stress Disorder? (ASD) Low Back or Leg Pain? (LBLP)	Intensity of RLS: Severe Distracting Moderate Duration of RLS pre-treatment?	Treatment time until resolution of RLS (days)	Did RLS return? (# Months): Maintenance dose of Cataplex G needed? (Yes: No)
80; M	I, LBLP	D: 7 years	12	12: N
50; F	I, ASD	D: (lifetime)	7	17: Y
63;M	LBLP	S: (lifetime)	15	N: N
61; F	I, ASD	M: (3 years)	13	N: Y
77; F	ASD, LBLP	D: (10 years)	4	Every 6 months: Y
69; F	ASD, LBLP	S: (10 years)	8	N; N
68; F	I, LBLP	S: (lifetime)	11	Every 4 months: Y
49; F	ASD	S: (10 years)	11	Every 2 months: Y
64; F	I, ASD	D: 5	6	N: Y
62; F	ASD, LBLP	M: (12 years)	8	N: N
68; F	I	D: (6 years)	7	N: N
47; F	ASD	D: (lifetime)	10	N: N
57; M	LBLP	M: (20 years)	40	N: N
44; F	ASD	S: (10 years)	9	N: N
80; M	LBLP	S: (2 years)	12	N: Y
			This patient resolved his RLS with Cataplex E2)	
73; M	LBLP	D: (1 year)	5	Every 4 months: Y
61; M	ASD, LBLP	D: (3 years)	2	Every 4 months: Y
70; F	ASD	S: (2 years); also in arms	4	N: Y
54; M	LBLP	D: (5 years)	24	N: N
66; F	I, ASD, LBLP	S: (10 years)	15	N: N
63; F	LBLP	D: (lifetime)	4	Every 2 months: Y
80; M	I	S: (10 years)	28	N: Y
93; M	LBLP	S: (6 months)	3	N: N

b) Headaches, dizziness, insomnia, depression and impairment of memory
c) Burning hands and feet
d) Pain in calves
e) Numbness and weakness of extremities
f) Difficulty in walking
g) Absent knee jerk

Signs and symptoms for vitamin B6 (pyridoxine) deficiency:

a) Severe sensory neuritis
i. Numbness and tingling in hands and feet
ii. Hyperesthesia

In the early investigations of the vitamin B/G complex, the investigators called vitamin B/G an anti-neuritic vitamin. All nerves require B/G vitamins for normal function. Pyridoxine (B6), riboflavin (B3), and folate are especially important. Supplementation should also include B12, biotin and pantothenic acid.

As an example of this fact: inositol has been used in Europe for the past 30 years to lower cholesterol and improve nerve function in diabetics. **(Bloomgarden, 1997; Mayer & Tomlinson, 1983)** Because of its role in cell-membrane function, it has also shown beneficial effects for depression and general neurological function.

Pyridoxal phosphate (vitamin B6) has been implicated as critical in lipid metabolism because its deficiency causes myelin degeneration in man. **(Sauberlich & Canham, 1980)** Impairment of temperature perception is present in a high percentage of RLS patients, and the sensory deficits are at least in part caused by small nerve fiber neuropathy. **(Schattschneider et al., 2004)**

Practically all of the neurotransmitters in the brain are metabolized with the aid of vitamin B6, including dopamine, norepinephrine, serotonin, GABA, histamine, acetylcholine, insulin, growth hormone, follicle-stimulating hormone, luteinizing hormone, aldosterone, glucagon, cortisol, estradiol, testosterone, and epinephrine. Vitamin B6 is also required for the conversion of tryptophan to niacin. Serotonin, a critical factor for normal sleep patterns, is derived from pyridoxal-5 phosphate and 5-hydroxytryptophan, and so another possible improvement in nighttime RLS may have an explanation.

Since dopamine is synthesized with the aid of vitamin B6, and dopaminergic drugs like levodopa and dopamine agonists are the first line of medical treatment choice in idiopathic RLS, the effect of Cataplex G may have another corroboration. However with the use of dopaminergic drugs, augmentation and rebound phenomena are consequences of this type of medical treatment, and must be carefully monitored in long-term treatment.

Niacin has long been used for cardiovascular conditions, especially those involving lipid metabolism. It may also help Raynaud's disease (excessive blood vessel constriction due to cold and symptoms of intermittent claudication) caused by insufficient blood supply to the calf muscles while walking.

According to Carlton Fredericks, Ph.D, "Niacin restores the electrical charge to red blood cells so they don't aggregate and thereby are able to pass through small blood vessels in single file." Deficiency of niacin has been reported to cause weakness, dry skin, lethargy, headache, irritability, loss of memory, depression, insomnia, delirium, and disorientation. Unless the nerve damage or degeneration has gone too far in cases of RLS, the administration of these nutritional factors for healing of the nerve provided quick relief.

Numerous studies have shown that the elderly (18 of the patients in the Cuthbert study were members of the 'senior set') are usually deficient in the nutrients contained in Cataplex G, as well as others. **(Lowenstein, 1982)**

Further study of the relationship between dopamine, central nervous system and vitamin B metabolism will be of great interest and value to neurophysiologists, neurochemists, neuropharmacologists and, of course, to patients with RLS. In the 23 patients with RLS successfully treated in the report, nutritional supplementation was a critical factor.

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