TUTORIAL

Diagnosis and Management of the Painful Ankle/Foot. Part 2: Examination, Interpretation, and Management

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■ **Abstract:** Diagnosis, interpretation, and subsequent management of ankle/foot pathology can be challenging to clinicians. A sensitive and specific physical examination is the strategy of choice for diagnosing selected ankle/foot injuries and additional diagnostic procedures, at considerable cost, may not provide additional information for clinical diagnosis and management. Because of a distal location in the sclerotome and the reduced convergence of afferent signals from this region to the dorsal horn of the spinal cord, pain reference patterns are low and the localization of symptoms is trustworthy. Effective management of the painful ankle/foot is closely linked to a tissue-specific clinical examination. The examination of the ankle/foot should include passive and resistive tests that provide information regarding movement limitations and pain provocation. Special tests can augment the findings from the examination, suggesting compromises in the structural and functional integrity of the ankle/foot complex. The weight bearing function of the ankle/foot compounds the clinician's diagnostic picture, as limits and pain provocation are frequently produced only when the patient attempts to function in weight bearing. As a consequence, clinicians should consider this feature by implementing numerous weightbearing components in the diagnosis and management of ankle/foot afflictions. Limits in passive motion can be classified as either capsular or non-capsular patterns. Conversely, patients can present with ankle/foot pain that demonstrates no limitation of motion. Bursitis, tendopathy, compression neuropathy, and instability can produce ankle/foot pain that is challenging to diagnose, especially when they are the consequence of functional weight bearing. Numerous non-surgical measures can be implemented in treating the painful ankle/foot, reserving surgical interventions for those patients who are resistant to conservative care. ■

Key Words: Ankle, Examination, Foot, Midtarsal, Subtalar, Talocrural, Tarsal Tunnel, Sprain, Instability, Tendopathy, Orthotic, Synovitis, Arthrosis

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INTRODUCTION

Diagnosis, interpretation, and subsequent management of ankle/foot pathology can offer the clinician a challenging clinical experience. The ankle/foot complex functions as a symbiotic mechanism comprised of numerous joints that respond to internal and external

forces and constraints. Because of a distal location in the sclerotome and the reduced convergence of afferent signals from this region to the dorsal horn of the spinal cord, pain reference patterns are low and the localization of symptoms is trustworthy. While imaging results can be helpful, the clinical examination prevails in importance to the clinician for accurate diagnosis. Therefore, a thorough history and reliable physical examination should rest at the center of the diagnostic process.

As in other joint systems, primary arthropathies of the ankle/foot are painful disturbances in the joints that develop as result of trauma or disease, such as synovitis, arthrosis, or chondropathy. Conversely, secondary afflictions emerge in tissues adjacent to underlying non-painful joint, capsuloligamentous or muscular structures. In addition, the weight bearing function of the ankle/foot compounds the diagnostic picture. Frequently, limits and pain provocation are produced only when the patient attempts to function in weight bearing. As a consequence, clinicians should consider this feature and implement weightbearing components in diagnostic and management strategies.

EXAMINATION

History

Prior to examining a patient with an ankle/foot disorder, a clinician should take inventory of the patient's history. Age and sex can provide the clinician insight into a patient's disorder, due to association with specific sex and age groups.[1] For example, osteochondrosis (Kohler's disease) of the naviculum and second metatarsal is seen more frequently in males 3 to 8 years of age. One would expect to see Severs disease (apophysitis or avascular necrosis) of the calcaneus most often in pre-adolescents between the ages of 8 and 12 years.[1] In early adolescence, osteochondrosis dessicans occurs mostly between 15 and 25 years of age in the talar dome.[2,3] Juvenile rheumatoid arthritis (JRA) occurs most often in the subtalar joint between 10 and 20 years of age.[4] Numerous systemic diseases, such as ankylosing spondylitis and Reiter's disease, occur more in males between 16 to 35 years old, while chondromatosis (multiple loose bodies) is seen most often between the ages of 35 and 55 years, where the synovial loose body proliferation may be calcified. Traumatic arthritis occurs most often between the ages of 25 and 65 years, resulting in a capsular pattern of the affected joint segment. A capsular pattern is seen in patients over 40 years old due to primary arthrosis (DJD) of the joint, whereas secondary arthrosis (instability, postfracture) is seen in patients under 40 years.

Interpreting the relevance of a patient's ankle and foot problem is assisted by an understanding of neuroanatomy in the region. Accompanied by reduced convergence in the dorsal horn of the spinal cord, a more dense arrangement of sensory fibers lends to increased sensory discrimination and less referred pain when compared with more proximal joint regions.[5,6] Increased sensory discrimination and symptom localization is similar to that in the wrist and hand,[7] allowing clinicians to trust the location of pain generation by region.

The ankle/foot can be divided into regions for the purpose of differential diagnosis. Anterior ankle pain can result from a synovitis of the talocrural joint (TCJ) or anterior talotibial compression syndrome (ATTCS) that emerges as a consequence of a hyperdorsiflexion trauma. Synovitis of the TCJ will produce a diffuse aching compared to the sharp, focal pain associated with ATTCS. In addition, anterior capsular impingement can occur with chronic ankle instability, syndesmosis involvement with inversion/eversion ankle trauma. While several afflictions can produce pain in the posterior ankle/foot region, achilles tendopathies commonly serve as pain generators in the region. Posterior talotibial compression syndrome (PTTCS) can cause focal posterior or posteromedial ankle pain, due to hyperplantarflexion injury and subsequent compression of soft tissue structures between the talus and calcaneus. Moreover, flexor hallucis longus (FHL) tenosynovitis can produce similar symptoms in the region.

Plantar heel pain can result from referred pain in the lumbar region, which can be ruled out through selective dural testing.[8,9] A less common affliction that lends to plantar pain is tibial nerve entrapment at the tendinous arch of the soleus associated with hypertrophy of the popliteus.[10] Plantar fascitis and rupture are common causes of focal plantar medial heel pain, along with heel spurs and tendopathies of the flexor digitorum brevis and adductor hallucis. Additionally, medial heel pain can result from tarsal tunnel syndrome (TTS), subtalar joint arthritis, stress fracture of the calcaneus and posteromedial talar tubercle, and tenosynovitis of the FDL and FHL.[1] Additionally, compression neuropathy of the lateral and medial plantar nerves, calcaneal nerve, and posterior tibial nerve can be ruled out with slight modification of traditional dural testing.[8,9] Finally, triceps surae tendopathy must be considered in cases of medial heel pain.

With plantar flexion inversion ankle injury, the lateral ligaments can generate local symptoms once compromised. Snapping ankle can result from retinacular rupture allowing the peroneus longus and brevis to sublux around the lateral malleolus. In addition, peroneal tenosynovitis can lead to focal swelling and pain on the lateral side of the ankle. Lateral heel pain can result from stress fracture of the calcaneus, sural neuropathy, peroneal retinaculum, and triceps surae tendopathy.[1]

Diagnostic Imaging

Plainfilm radiography can be useful for viewing fracture or syndesmosis injury in the ankle/foot, while it is of little value for examining ligamentous injury. When syndesmosis involvement is suspected, radiographs performed in approximately 15° internal rotation are very specific and reliable.[11] Takao et al. found using computed tomography (CT) clearly allowed for measurement of mortise angle and injury assessment (Takao, 2001).[12] In cases of recurrent ankle instability, subtalar arthrography can be useful in assessing the structural integrity of the joint capsule and lateral ligamentous structures (particularly the calcaneofibular ligament) with a sensitivity of 92% and specificity of 88%.[13] Scintigraphy (bone scan) is very useful for the assessment of stress fractures of the sesamoid bones,[14] as well as the differential diagnosis of bony tumors when used in concert with CT.[15] Conversely, articular cartilage and soft tissue lesions are best visualized using magnetic resonance imaging (MRI).[16]

Clinical Examination

The functional examination of the ankle/foot can be witnessed in Appendix 1. Visual inspection can be informative and should be included as part of the basic functional examination of the ankle/foot. For this, the clinician should observe general skin integrity, color, and texture. Additionally, palpation is performed before and after the clinical examination for the sole purpose of detecting subtle inflammatory responses to testing. During the initial inspection, the clinician should examine the patient's ankle/foot posture and functional biomechanical behavior. Abnormal ankle/foot posture and biomechanical behaviors can be linked to pathology. For instance, a greater risk for stress fractures in the tibia, fibula, and foot has been associated with high longitudinal arch, leg-length discrepancies, and excessive forefoot varus in runners of both genders.[17] In addition, excessive movement and dysfunction of the subtalar joint system can have an effect on overuse injuries in runners[18] and patients suffering from patellofemoral pain syndrome.[19]

Several tests can be performed to assess these behaviors. Calcaneal orientation can be observed in bipedal and unipedal stance in the frontal plane. In unipedal stance, the amount of calcaneal valgus should decrease. If this does not occur, then the clinician should suspect hyperpronation.[1] The dynamic supination test assesses the integrity of the plantar fascia and posterior tibialis tendon by passively dorsal extending the patient's great toe while he or she maintains a unipedal stance. Normally, the arch should increase; however, when plantar fascia is stretched out (as in hyperpronation) little to no change will occur. Additionally, clinicians utilize the naviculum drop test as a definitive test for hyperpronation (see Figure 1).[20] A normal naviculum drop test is less than 3–5 mm, while a positive test is greater than 10 mm.[21] Although this test could indicate a patient's excessive pronation in weightbearing, poor to moderate intertester reliability has been demonstrated for inexperienced clinicians finding subtalar neutral and using the naviculum drop test.[22–24] Poor inter-rater reliability for finding subtalar neutral may be due to the "ideal foot" being non-existent.[25] Moreover, static maximal eversion can serve as a clinical indicator of rearfoot eversion during gait but may not predict other dynamic responses of the rearfoot during gait.[23,24,26]

After evaluating selected postures and biomechanical behaviors in the ankle/foot, the clinician should perform the basic functional examination. During this examination the quantity, quality, and provocative nature of all each test should be evaluated. Active tests in standing begin with ten unilateral toe raises to assess the strength of the plantarflexors and provoke symptoms associated with Achilles tendopathy. Next, bilateral squatting is performed while the clinician observes for one heel to leave the floor earlier than the other. If this occurs, a TCJ hypomobility is suspected. Later, this outcome can be compared to dorsiflexion in NWB. If both are hypomobile, then a true hypomobility is suspected. Conversely, if the NWB dorsiflexion is normal, then a TCJ hypermobility/instability is suspected, respecting the influence of loading on the congruency of the joint and subsequent distorted joint kinematics. Following bilateral squatting, the patient performs a unilateral squat for pain provocation associated with ATTCS[27] or syndesmosis involvement. Liu et al. found a clinical examination demonstrates a sensitivity of 94% and specificity of 75% for the

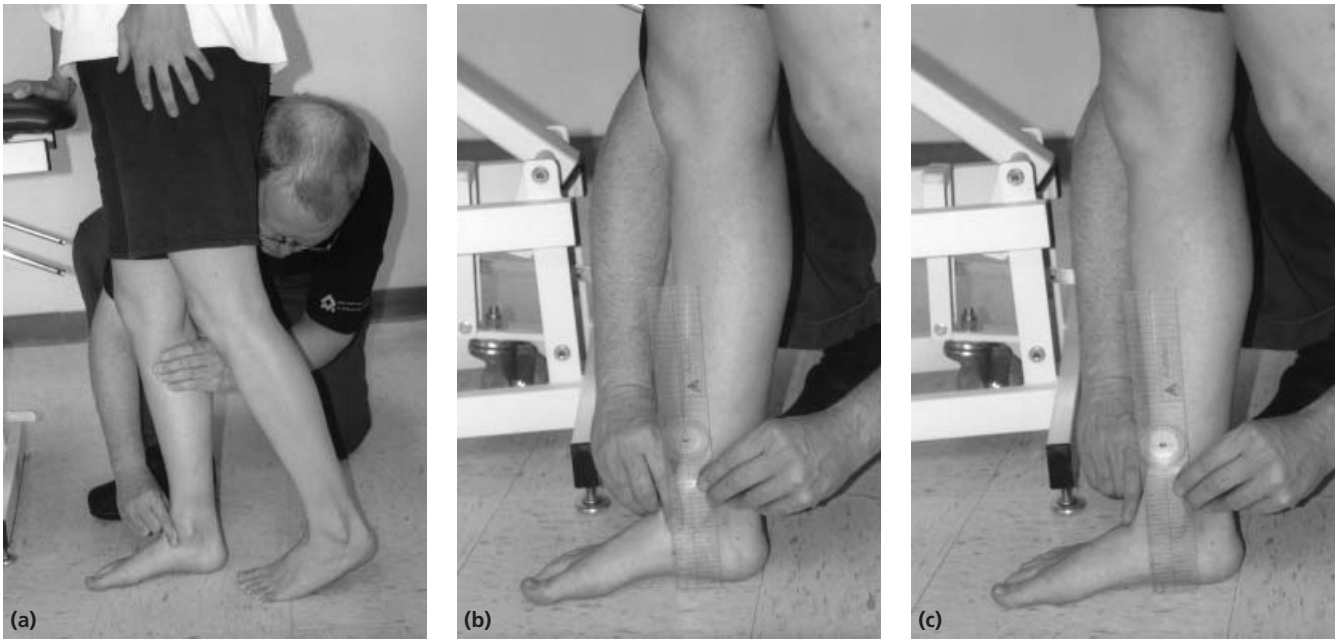


Figure 1. Naviculum drop test. The foot is first placed in subtalar neutral, where the foot is everted and inverted until the medial and lateral talar dome is equally prominent (a). For this test, the change in the distance between the naviculum tubercle and floor is noted when the foot transitions from a subtalar neutral position (b) to a relaxed foot posture (c) in weightbearing.

diagnosis of ATTCS, compared with 39% and 50% using magnetic resonance imaging.[28]

Passive TCJ movement is tested in NWB, allowing the talus to rotate in all planes. Talocrural plantar flexion is tested with the knee extended, while dorsiflexion is tested with the knee fully extended, slightly flexed, and flexed to 80°, so to assess the influence of various soft tissue and joint capsule components on motion.[29] Talocrural dorsiflexion is classified as hypomobile when dorsiflexion is less than 4.3°, inflexible at 4.3° to 11.2°, normal when it moves 11° to 25°, flexible if moving 25° to 32°, and hypermobile when movement is greater than 32°.[30]

Testing of the subtalar joint (STJ) is performed in supine for provocation testing and prone for mobility testing. Provocation testing is performed by passively moving the calcaneus into inversion and eversion, while guiding the forefoot with the other hand and completing the movement with overpressure through the calcaneus for pain provocation in both directions. Mobility testing is performed on the patient positioned in prone, with the clinician grasping the distal tibia anteriorly and placing the thumb against the posteromedial talar tubercle. The opposite hand grasps the calcaneus to perform the tilt starting from maximal eversion. Inversion is performed until the posteromedial talar tubercle moves up underneath the thumb. If inversion is continued past this point, the outcomes will be misleading since talar movement will result in increased inversion.[31]

Passive testing of the mid-tarsal joints (MTJ) is performed as a general screen during the basic examination, followed by joint specific testing if limits are seen during the screen. The clinician stabilizes the calcaneus while grasping the forefoot with the opposite hand. Passive DF/PF is tested with the forefoot hand in a direction perpendicular to the dorsum of the foot. Next, inversion/eversion and pronation/supination are performed, comparing the involved side to the uninvolved side.

Passive testing of the first tarsometatarsal (TMT) and metatarsal phylangeal joint (MTP) is important, since it can have a profound impact on TCJ and STJ performance during gait. The first TMT joint is typically involved with hallux valgus deformities, due to hypermobility of the first TMT joint.[32–34] In the literature, testing of the first TMT is performed by stabilizing the second through fifth metatarsals while moving the first metatarsal on the first cuneiform. Using the abovementioned method, intrarater reliability was an average of 0.73 with interrater reliability between 0.09 and 0.16.[32] Results of the Glasoe study do not support manual testing of the first TMT.[32] Normal first TMT

range of motion (ROM) is 10.3° verified by goniometric measurement[34] and radiography.[33] Hallux valgus patients exhibit first TMT ROM of greater than 13° to 14° leading to metatarsalgia and metatarsal fracture.[33,34] An alternative method of testing performed stabilizing the first cuneiform, while the mobilizing hand grasps the proximal metatarsal staying perpendicular to the dorsum of the foot may be implemented. Plantar flexion and dorsal extension are tested for hypo or hypermobility that may be contributing to a focal lesion or ankle/foot pathology. Next, the MTP joint is tested grasping the distal metatarsal and proximal distal phalanx applying a perpendicular force in dorsal or plantar direction.

Testing the ligamentous system of the lateral ankle begins with the clinician grasping the calcaneus with one hand and guiding the forefoot on the lateral side with the other hand. First, the ankle is tested in full plantar flexion, inversion, and supination to stress the anterior talofibular ligament. Second, the ankle complex is tested in 10° plantar flexion, full inversion, and supination to stress the calcaneofibular ligament. Third, full dorsiflexion, inversion, and supination are performed to test the posterior talofibular ligament.[35] In order to test the medial ligamentous system, the hands are switched with the guiding hand on the medial aspect of the forefoot. In full plantar flexion, eversion, and pronation the anterior tibiotalar and talonavicular ligaments are tested. Performing overpressure in 10° plantar flexion, full eversion and pronation tests the talocalcaneal ligament. Lastly, the posterior talotibial ligament is tested in full dorsiflexion, eversion, and pronation.[1]

Isometric resistive tests can give the clinician an index of the patient's ankle/foot strength, as muscle strength imbalance has been associated with risk for inversion ankle sprains.[36] Moreover, specific isometric tests can function as provocation for various tendopathies. Isometric tests should be performed in diagonal directions to load the muscle, musculotendinous junction, and tendon components surrounding the ankle/foot (see Figure 2). Additionally, testing the flexor hallucis longus should be performed 10 times to mimic weight-bearing forces. Clinicians should grasp the great toe and ask the patient to flex the great toe. The extensor hallucis longus can be tested in a similar manner by asking for resisted dorsal extension.[1] The clinician should finish the examination with specific stability testing. These tests evaluate the integrity of the ligaments that stabilize the TCJ and STJ. The review of the test procedures and outcomes can be found in "Lateral Ankle/Foot Pain."

DIAGNOSTIC INTERPRETATION AND MANAGEMENT

Capsular Pattern Limitations

Upon conclusion of the examination, the clinician can proceed with an interpretation of the examination findings. First and foremost, the clinician must pay particular attention to the types of limitations observed in the examination of the patient's ankle/foot. For example, limits in active motion accompanied by normal passive movement should lead the clinician to suspect a central nervous system dysfunction, peripheral nerve injury, or rupture of the involved motion system tendon (as in the case of complete Achilles tendon rupture). If both active and passive motions are limited in the examination, the clinician should discern whether the limit reflects a capsular or non-capsular pattern.[1]

A capsular pattern is a predictable, repeatable, and reliable pattern of passive motion limitation that is unique to each joint system. The limitation is initiated by intra-articular swelling[37] and represents a synovitis or arthrosis within the joint. A talocrural joint capsular pattern is distinguished through a larger limit in plantar flexion than dorsiflexion. The subtalar joint capsular pattern demonstrates itself as a varus limitation, while the midtarsal joints have plantar flexion, supination, and adduction > dorsiflexion > adbuction and pronation limitation.[1] For the capsular pattern of the first TMT joint, flexion is limited greater than extension.

When a capsular pattern is observed in any of the joints of the ankle/foot, clinicians should pay particular attention to the patient's history. As discussed earlier, this pattern of limitation can represent a synovitis, either traumatic or non-traumatic, or arthrosis associated with degenerative changes on the articular surfaces. Traumatic synovitis can be related to a single macrotraumatic event such as falling off a step, or associated with repetitive microtrauma that can occur with high volume training for marathon runners, gymnasts, or cyclists. Patient's can present with a non-traumatic synovitis that is associated with systemic diseases like Rheumatoid Arthritis, Gout, Psoariasis, Reiter's Syndrome, and Systemic Lupus Erythematous (SLE). Furthermore, a capsular pattern may slowly emerge as a result of a primary arthrosis. Finally, a capsular pattern may develop from an earlier accelerated degenerative process within the joint (secondary arthrosis) from a previous intra-articular trauma.[1]

Traumatic synovitis and primary arthrosis can be effectively managed utilizing anti-inflammatory medi-

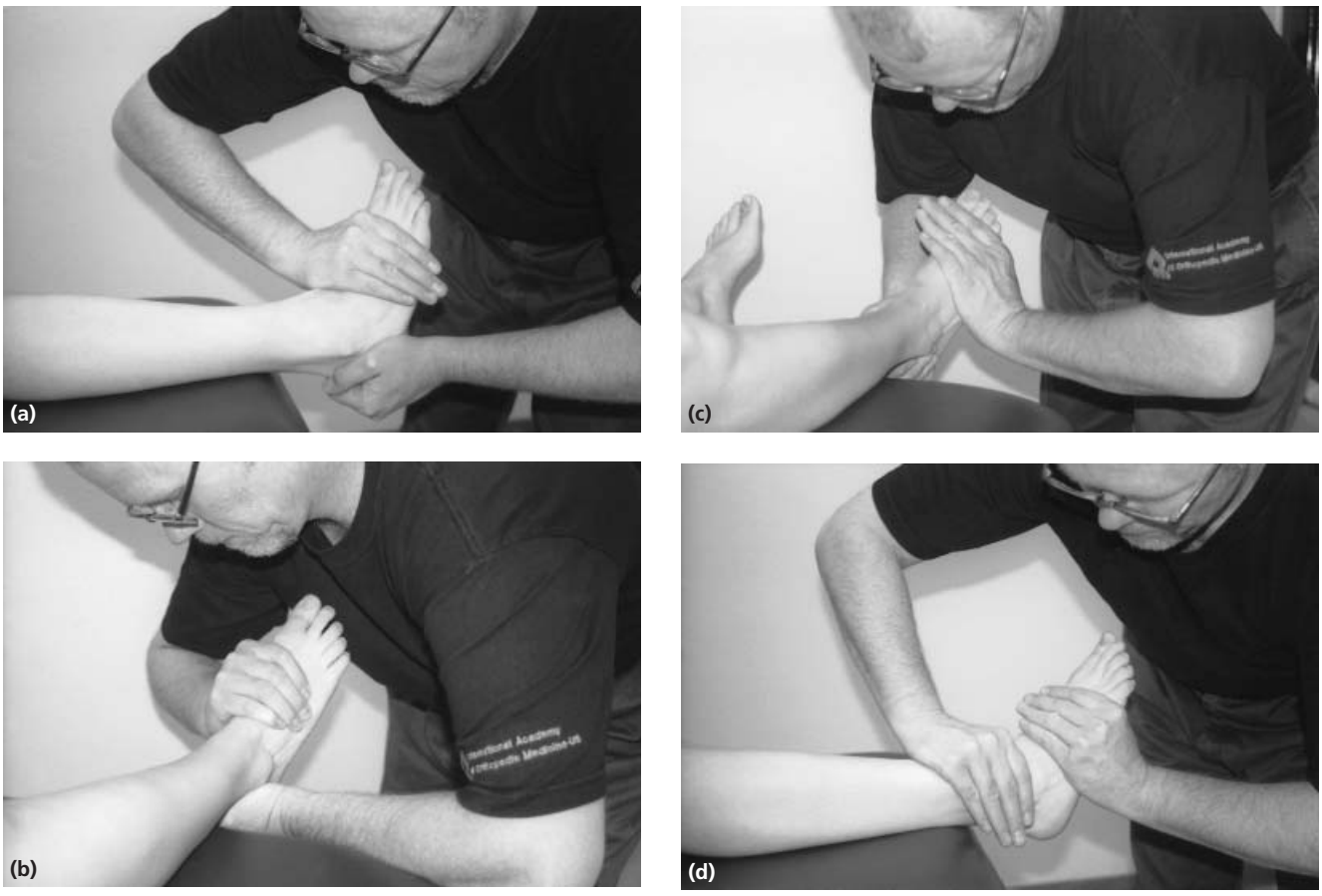


Figure 2. (a) Pain with resisted dorsiflexion and inversion indicates a tendopathy of the anterior tibialis. Without changing hand placements, the foot can passively moved into maximal plantarflexion, abduction, and pronation to stretch the tenosynovium of the anterior tibialis. (b) Resistance to plantarflexion and adduction and supination tests for posterior tibialis, flexor hallucis and digitorum longus, and triceps surae tendopathy. Passive movement into dorsiflexion, abduction, and pronation can stretch these tendons, thereby irritating the tenosynovium. (c) Third, applying resistance to dorsiflexion, abduction and pronation stresses the tendinous unit of the extensor digitorum. Stretching the tenosynovium is accomplished by passively moving the ankle/foot into plantar flexion, adduction and supination. (d) The peroneals are tested through resisted plantarflexion, abduction and pronation, while tenosynovitis can be elicited through passive dorsiflexion, adduction and supination.

cations, joint-specific mobilization (traction and slides/glides), and high repetition, low load exercise programs to assist with decreasing joint inflammation. In more severe or chronic cases, an intra-articular injection may be needed to accelerate the dispersal of inflammatory properties. Management of non-traumatic synovitis consists of treating the underlying pathology (e.g. Reiter's, RA, etc) and encouraging low load joint movement through the patient's available pain-free range of motion.

Non-Capsular Pattern Limitations

A non-capsular pattern (NCP) limitation is any limitation other than the capsular pattern.[1] Several combinations of NCP's can occur in the ankle/foot complex, however selected limits occur more frequently. The most common NCP is observed status-post immobilization. Treatment involves passive and active mobilization of the affected joint complex. Loose bodies in the talocrural and subtalar joint will present with a predictable triad of information. First, the patient will complain of pain followed by giving way. Second, the patient will present with a NCP in the affected joint. Third, the patient will demonstrate a pathological end-feel with passive motion testing. Loose bodies may respond to manipulation of the joint attempting to free the loose body into the synovial fluid thereby allowing the synovium to engulf the fragment. However, if the loose body does not respond to conservative interven-

tions, is very large, or numerous, arthroscopic debridment may be indicated.[1]

A NCP can accompany ATTCS. For example, a patient with a true dorsiflexion hypomobility in the talocrural joint can have compression of the talar dome. This may lead to other painful consequences like chronic Achilles tendonitis; however, this affliction is easily rectified through joint-specific mobilization of the talus to restore dorsiflexion.[38] Hypermobility in the talocrural joint can result in aphysiological shearing forces across the talar dome and premature compression of the tibia on the talar dome. Hypermobility of the TCJ is best treated with low-grade high repetition exercises for the peroneal muscle groups and general ankle strengthening, neuromuscular re-education, and bracing or taping. Finally, a loose-body, accumulated scar tissue, hypertrophied synovial tissue, or meniscoid mass in the lateral gutter of the ankle can result in ATTCS or anteroloateral ankle impingement and NCP limitation.[28]

Clinical Biomechanical Management

The ankle / foot complex performs many dynamic functions when in the stance phase of gait, allowing the body to progress forward through space during normal walking. The mobility status of the foot in stance, whether a mobile adaptor or rigid propulsion lever, depends on the position of the STJ. Dysfunction of this mechanism can promote musculoskeletal disorders of the lower extremities.[39] For example, forefoot and rearfoot varus can induce an excessive pronation behavior in landing, which may induce an overuse injury condition.[40]

Appropriate coordination between the motions of the subtalar joint and the knee is critical to attenuation of ground reaction forces in the landing sequence. Prolonged pronation requires increased tibial IR and tibial inclination associated with knee flexion to maintain TCJ congruency. This produces torsional stress, as the lower extremity attempts to extend in gait. The femur internally rotates to attempt to reduce this stress at the knee. This behavior induces maladaptive movements, forces, and loading responses at all of the joint systems in the lower extremity, including the foot and ankle.[41–44]

However, while these mechanical responses are documented, their relationship to the development of overuse injury is controversial. Selected investigators have suggested that this maladaptation can lead to overuse injuries in the lower extremity, such as tendonitis, bursitis, friction syndromes, patellofemoral affliction, periosteal reactions or stress fracture.[18,39,45,46] Williams et al. found that low-arch runners demonstrated a higher incidence in selected overuse injuries, such as tendonitis, medial knee pain and plantar fasciitis whereas high arch runners demonstrated greater incidence of architectural injuries, as well as soft tissue injuries of the lateral knee and foot.[47] Kaufman et al. found that pes planus (an excessively pronating foot) within a shoe was an associated risk factor for the development of overuse injuries in the lower extremity, whereas static or dynamic arch height measures were not.[46] However, Donatelli et al. found that excessive pronation was not a significant contributing factor in the development of overuse injuries in professional baseball players, while Hogan and Staheli found no relationship between arch configuration and pain in the lower extremity.[48–49]

Clinicians have resorted to different strategies for managing biomechanical disorders of the lower extremity, including shoe alterations and or orthotic prescriptions. The use of shoes for managing biomechanical disorders is clinically sensible, due to potential influence shoes have on biomechanical behaviors in the ankle/foot. As a consequence, shoe construction and status may influence the development of overuse injuries in the ankle/foot. Whereas laterally flared shoes do not appear to increase rearfoot eversion,[50] polyurethane with an embedded air cell can potentially be protective against stress fracture.[51] Wilk et al. found that factory defects in a running shoe may contribute to the development of overuse conditions, such as plantar fasciitis.[52] These authors suggested that clinicians should examine the integrity and symmetry of their patients' shoes, including the heel counter alignment, status of adhesion between the outer- and mid-soles, firmness and symmetry of gel or air cells, and shoe position response to downward loading. Moreover, men's versus women's feet are very different in structure and proportion, suggesting an interaction between foot architecture and shoe design.[53] This difference may be found, in part, in the differences in toe box, midsole, and heel design. For instance, Wang found that an increased heel height appears to increase and prolong vertical and posterior-anterior ground reaction forces through the ankle/foot.[54]

Whereas shoes can influence the biomechanical behaviors of the ankle/foot, they may not be sufficient for all management applications. Functional orthotics can serve as a more significant influence on lower extremity biomechanics and subsequent overuse in-

juries. Functional orthotics, constructed semirigid thermoplastic materials, are designed to control the extent of rearfoot motion during the landing sequence.[40] Additionally, orthotics are intended to improve dynamic stability of the STJ by reducing compensatory pronation of the STJ during the landing phase of gait. Investigators have suggested that this is accomplished by reducing maximum calcaneal movements, maximum pronation velocity, time-to-maximum pronation, and total rearfoot motion.[45]

The clinical use of orthotics begins with casting the patient' foot in a subtalar neutral position (previously defined). From the cast a positive plaster mold is formed and used to build posted orthotic devices. The orthotic device is a thermoplastic shell that conforms to the foot's plantar surface from the rearfoot to the sulcus behind the metatarsal heads. The thermoplastic shell is covered on the dorsal surface with a soft, absorbent topcover, while the plantar surface is posted medially to reduce the amount of vertical distance the medial heel must drop through pronation to reach the ground. The orthotics are worn inside the patient's shoe in order to help control rearfoot motion and reduce end-range tissue stresses during a weightbearing sequence. As a consequence, they serve as a means offer relative rest to the tissues in the lower extremity.

Routinely, clinicians use orthotics to post the rearfoot and or forefoot, thus controlling the behavior of the subtalar and midtarsal mechanisms. The extent of medial rearfoot and forefoot posting should be considered for effective clinical applications. Individuals function in a weightbearing position or movement with some degree of pronation.[24,25] Orthotic posting attempts to return an individual's foot back to these norms from an extreme pronatory response by reducing the total amount of pronation that the STJ produces during weightbearing.

It has been suggested that appropriate applications of orthotic devices appear to depend on an accurate attainment of the STJ neutral position. Additionally, the body position of the patient and the clinician appears to influence the reliability of this skill, with prone patient positioning apparently enhancing the reliability.[55] Torburn et al. evaluated the intertester reliability for measuring STJ neutral, full pronation, and full supination with the use of an ELGON. This study demonstrated that the Interclass Correlation Coefficient (ICC) for determining STJ neutral was 0.76. Although end-range inversion and eversion attainment demonstrated lower ICC levels (0.37 & 0.39, respectively), these investigators suggested that accurate and reliable STJ neutral positioning is attainable when appropriate technique and instrumentation are used.[24]

Orthotics have proven useful for reducing symptoms associated with the overuse conditions that arise from over-pronation.[56] However, investigators have disagreed as to the reason why orthotic devices are successful. Studies have demonstrated that semirigid orthotic devices decrease the extremes of pronation in gait,[39,57–59] while Blake and Fergeson found limited use of rearfoot posting orthotics for influencing that same rearfoot motion.[60] Brown et al. measured rearfoot movements via 2-dimensional digital analysis through a custom-made window in the heel counter of experimental shoes. While measuring subjects' rearfoot varus during walking on a treadmill, these investigators observed the lowest maximum pronation and calcaneal eversion with the semi-rigid orthotic condition. They observed no significant differences between shoes-only, over-the-counter arch supports, and custom-made semirigid orthotic devices in terms of maximum pronation, total pronation, or calcaneal eversion. Conversely, the semi-rigid orthotic condition demonstrated a significantly greater time-to-maximum pronation versus the other two conditions, suggesting a rearfoot control value for the semi-rigid orthotic device.[40]

Nawoczenski et al. observed for differences in lower extremity mechanics between an orthotic and non-orthotic condition. They found no significant differences between the orthotic / non-orthotic conditions when measuring the inversion / eversion behaviors of the calcaneus in the frontal plane. However, they observed a significant decrease in total tibial IR during the early stages of stance, where rearfoot motion is most critical for shock absorption and load transfer. The values were, on average, 2° less in IR, or 31% reduction in total IR from heel contact to midstance for the subjects with low arches and 22% reduction for the subjects with a high arch.[45] Although these differences are small in total range, the reductions may be clinically relevant based on the vast number of step cycles a runner takes (200/mile) in a single running episode. Thus, orthotic use may be more important to controlling transverse plane motion of the tibia, with the consequences imposed on the STJ coupling mechanisms versus any changes revealed in the frontal plane. Conclusively, orthotics may reduce the impact of any condition arising from (1) early tibial IR during the landing phase of gait while the knee is remaining in an extended position, and (2) excessive tibial IR coupled with an extending knee

in the propulsion phase of gait. These principles of control benefits associated with orthotic use can be applied to either the low or high arch groups of individuals who suffer from overuse injuries of the LE.

Posterior Ankle/Foot Pain

The achilles tendon mechanism is subject to significantly greater forces without developmental adaptation versus other tendons in the ankle/foot, while maintaining similar biomechanical properties as those tendons.[61] As a consequence, this tendonous system is at risk for developing numerous afflictions, each with a distinctive clinical picture. Trauma is routinely associated with achillodynia, resulting in tendonitis, peritendonitis, tendon tears. Additionally, these forces, coupled with increased age and decrease bone density, could produce achilles avulsion at the tendon bone interface.[62] The clinician should observe for a trauma in the patient's history, as non-traumatic onset of achilles tendopathy strongly suggests a systemic disease such as ankylosing spondylitis.[1,63]

Multiple endogenous and exogenous factors have been identified in the literature that lend to Achilles afflictions. Endogenous factors include a relative avascular zone at 3 to 6 cm proximal to the bony insertion[64] and a potential for stenotic thickening of the vascular intimae, which can lead to a relative hypoxic state in the tendon substance.[65] Other endogenous factors lending to Achilles afflictions include excessive pronation, subtalar mobility disturbances, clinical tibial torsion, high arches and hallux rigidus. Exogenous factors include footwear with insufficient rearfoot control and or hard soles, higher heels that produce increased vertical ground reaction forces at terminal propulsion, drastic training alterations, poor neuromuscular control of the ankle/foot during gait, and inappropriately early return to sport without sufficient training.[54,65]

Different Achilles afflictions present with distinctive clinical features. Insertion tendonitis is witnessed at the connection with the calcaneal tuberosity, where continue around the calcaneus to become confluent with the fibers of the plantar fascia.[1,66] The tendon insertion transitions from collagen to non-mineralized fibrocartilage, then to mineralized fibrocartilage and finally bone, where the inflammatory reaction can be found in the region of the non-mineralized and mineralized fibrocartilage.[67] This condition will demonstrate provocative resistive testing as its most salient feature, whereby the tendon substance is stressed under load. Frequently, however, pain will not be provoked through manual resistance applied to plantarflexion, instead requiring the patient to repeat multiple repetitions of unilateral heel raises up on his or her toes before symptoms are provoked. In addition, the patient may experience discomfort with passive dorsiflexion and may present with swelling at the site of the insertion.

Peritendonitis, also known as pertenonitis, presents with a fusiform swelling in the avascular region of the tendon,[68] due to inflammation and possible fibrotic adhesions between the endotenon and the peratenon.[69,70] This affliction is characterized by early morning pain, stiffness and crepitus,[68] due to the elastic behaviors of the tendon and apparent sheath retraction over the course of the night.[71] The patient's symptoms are most provoked during the basic functional exam with passive dorsiflexion, which produces an irritating rub between the paratenon and deeper endotenon substance. Finally, the clinician can note tenderness on the medial, lateral, anteromedial, anterolateral, or even anterior aspect of the tendon structure.

Management of achillodynia should include measures that reduce symptoms and inflammation while addressing causative factors and augment collagenation. Transverse friction is recommended at each site for 5–10 minutes, to reduce pain and activate an anti-inflammatory response.[1] In addition, longitudinal friction for 5–10 minutes can be used to activate fibroblasts and promote collagenation. The Achilles mechanism can be stretched with caution, as passive motion can increase symptoms associated with peritenonitis. Previously mentioned causative factors should be addressed when possible, such as excessive pronation (see biomechanical management) and or joint limitations (see management of afflictions with limitation). Finally, eccentric exercises with a substantial load can be incorporated to improve tensile capacity of the collagen and reduce symptoms.[72]

Achilles tendon tears can be sustained during a traumatic incident and can mimic other tendopathies. These lesions appear to be predisposed by degenerative changes in the tendon substance, including cellular variations, vascular changes, collagen disorganization and increased cellularity.[65] As a consequence, the diagnosis of an achilles tear can be elusive, requiring more extensive clinical work-up that includes the clinical examination and imaging (either MRI or ultrasonography). Hartegink et al. reported 100% sensitivity, 83% specificity, 92% accuracy, an 88% positive predictive value and 100% negative predictive value for ultrasonogra-

phy in the diagnosis of achilles tears. In addition, the MRI can be useful for this diagnosis, in concert with the clinical findings of local swelling, painful passive plantar flexion, weak and painful resistive plantar flexion, and pain at the beginning, during and after weightbearing activities.[73] Tears can be partial or complete, where pain characterizes the partial tear and weakness the complete tear.[74]

Management of achilles tears is controversial. Surgical repair entails apposition and primary suturing of the tendon remnants and functional bracing.[75] Postoperatively, Kauranen et al. suggested 2 different rehabilitation options. A more conservative approach includes a short leg cast with the ankle/foot in neutral for 6 weeks that allows full weightbearing after 3 weeks, followed by a gradual return to functional activities. More progressively, they suggested dorsal cast splinting that allows full plantarflexion while restricting dorsiflexion as an alternative to the short-leg cast.[75] Conversely, Aoki et al. allowed early active movement at 1–2 days postprocedure and full weightbearing at 2-weeks out.[76] Speck and Klaue recommended an accelerated rehabilitation program after primary repair. They found that initiating a 6-week program of full weightbearing in a removable ankle-foot orthosis at 24 hours postrepair demonstrated no apparent increased risk of rerupture.[77] Thermann et al. compared primary surgical repair with surgical and non-surgical apposition and bracing. These investigators utilized fibrin glues and bracing to prevent excessive tensile forces on the tendon and reported that, while there was a histological advantage to the non-surgical approach at 2–4 weeks postintervention, the surgical and non-surgical approaches did not differ histiologically at 12-weeks out.[78]

Repetitive impact loading associated with tendon elastic recoil, along with the tendon's previously described relative avascular zone, can place the achilles tendon at risk for developing tendinosis. Here, the tendon degenerates, producing a grayish-brown, mucoid and lipoid degeneration that result in irregular, diffused collagen bundling.[65,68] This degeneration is observed in either the middle 1/3 of the tendon or at its insertion and can produce persistent symptoms that last for months or even years.[79,80] The tendon will demonstrate an asymmetrical, nodular thickening that is more commonly seen in the medial side of the tendon mindsubstance.[68] Once again, provocative resistive tests will be the most salient clinical feature, along with painful passive dorsiflexion. In response, MRI could be useful in differentiating this affliction from other tendopathies, as the tendinosis will demonstrate a signal abnormality in the midsubstance of the tendon.[79] Frequently, tendinosis does not respond to conventional conservative management, with exception to eccentric exercise with a considerable load. Alfredsen recommended loaded eccentric activity with both the knee fully extended and slightly flexed for 3 sets of 15 reps each. These authors suggested that pain and discomfort during the activity is acceptable, as long as it is not severe. The patient is allowed to return to running at 12 weeks postinitiation of treatment, while continuing the exercises 1–2 times per week.[72] However, persistent symptoms may require surgical intervention. Mucoid material should be excised, where up to 50% of the tendon substance can be removed without risk of biomechanical failure.[79,81] Excision should be followed by gait with weightbearing-as-tolerated in a neutral ankle orthosis for 2–3 weeks, followed by strengthening and a return to running after 3–6 months.[79]

Several other afflictions can lead to pain in the posterior heel region, including haglund's syndrome, retrocalcaneal bursitis, posterior talotibial compression syndrome, and os trigonum tarsi. Haglund's syndrome is a condition that can produce posterior heel pain, characterized by painful irritation, swelling, and tenderness at the achilles tendon insertion into the posterior calcaneus.[82,83] Thickening of the Achilles tendon, retrocalcaneal bursitis and subsequent projection causes the characteristic "pump bump" prominence on the heel.[84,85] The condition is caused by compression of the distal Achilles tendon and compression of soft tissue between the os calcis and the posterior shoe counter and is worsened by osseous plantar projections emerging from the calcaneus.[84,86]

Medial Ankle/Foot Pain

Posterior tibialis dysfunction (PTD) is the most common cause of acquired flat foot in the adult population. The acquisition of PTD has many long-term consequences for the tendons, ligaments, TCJ, STJ, midfoot, and hindfoot.[87] Contrary to one's intuition, PTD is not always caused by a trauma.[88] Rather, PTD involves a wide array of etiologies ranging from age-related degeneration, overuse, chronic tenosynovitis, inflammatory arthritides, an association with seronegative spondyloarthropathy,[88–90] and calcification of the tendon.[91] Some patients may have persistent complaints of posterior tibial tendonitis/tenosynovitis due to an accessory navicular bone in either the tendon 3 mm prior to naviculum tubercle or at the tubercle itself.[92] Tarsal tunnel

syndrome (TTS) can result from PTD due to increased eversion stress and tension loading of the ligamentous, vascular, and neural structures contained in the tunnel.[93,94] Patients will present with sharp, shooting pain in the middle and lower thirds of the medial edge of the tibia, posteromedial ankle, and medial arch. Swelling may be present at the posteromedial ankle and the patient may complain of increasing symptoms with standing on toes or with running or other weight-bearing activities.

Investigators have staged PTD, where Stage I involves no clinical deformity, pain and induration along the posterior tibial tendon. Stage II is characterized by a dynamic deformity of the hindfoot, "too many toes" sign, and an inability to single heel rise. Stage III PTD results in a fixed deformity of the hindfoot in valgus without being able to reduce the talonavicular joint, possibly a fixed forefoot supination results to compensate for hindfoot changes, and no evidence of ankle or midfoot disease. Stage IV contains a very small portion of the population suffering from PTD. Examination reveals long-standing fixed deformities as in stage III with the presence of TCJ, STJ, or MTJ synovitis. In some patients, a positive valgus talar tilt will be positive indicating instability from deltoid ligament discongruity.[88,89]

Examination reveals passive eversion of the foot limited and or painful and painful resisted plantar flexion and inversion. If the passive stretch test is positive, a tenosynovitis of the posterior tibialis tendon should be suspected; however, a tendonitis is more likely when resistive testing is most provocative in conjunction with the single limb heel rise test.[1,89,95] The single limb heel rise test is performed with the patient standing next to a wall using a hand for balance. Next, the patient is asked to lift the non-affected foot off the floor and simultaneously rise up onto the ball of the affected foot. If the posterior tibialis is painful or disrupted, the hindfoot remains in valgus or there is an inability to rise onto the forefoot. A simple observation test looks for "too many toes" (observed from dorsal) due to increased hindfoot valgus and increased forefoot abduction.[88,89] The first metatarsal rise sign is another useful test to determine the integrity of the posterior tibialis tendon.[96] Testing is performed by externally rotating the lower leg with the patient in bilateral stance with equal weight bearing (see Figure 3). Clinicians should look for the first metatarsal to "rise" into extension. If this occurs, the posterior tibial tendon has been elongated or partially or completely torn.[88]

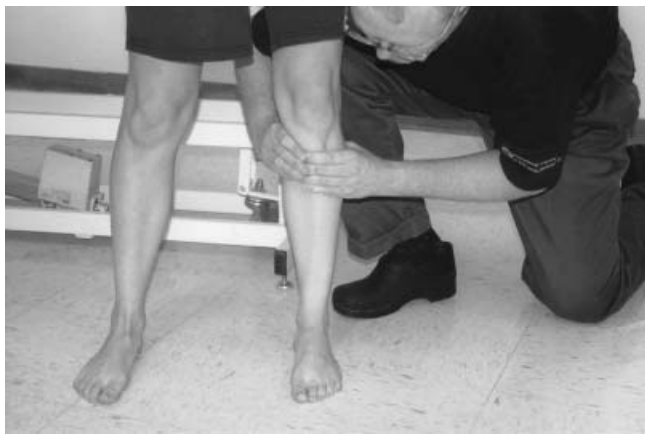


Figure 3. First metatarsal rise sign: The clinician passively externally rotates the tibia while the patient attempts to maintain full foot contact on the ground.

Conservative treatment of PTD can include NSAID's, relative immobilization, local treatments, transverse friction massage (TFM),[1,97] unloaded exercises for the tendon, and orthotic management to control the rotatory behavior of the tibia.[87,89] Injection should be used cautiously as the literature has correlated injection with an increased incidence of posterior tibial tendon rupture.[98] Surgical intervention can include synovectomy, partial tendon debridement (tendonosis or avascular regions), anchoring of tendon insertion to the naviculum tuberosity, tendon transfers, and double or triple arthrodesis.[88,89,95]

Tenosynovitis of the flexor hallucis longus (FHL) is seen frequently in ballet dancers and other sport participants. The focal irritation is found on the posterior side of the talus where the tendon traverses the medial and lateral talar tubercles. Irritation can occur between the two sesamoid bones at the base of the first metatarsal. Upon examination, the patient complains of pain and swelling behind the medial malleolus that is exacerbated with jumping. Resisted flexion of the great toe can be painful; However, passive dorsal extension of the great toe is more painful with possible crepitation. Patients with involvement at the great toe sesamoid bones will have more pain at terminal stance and push off with gait. Treatment can include TFM, gentle stretching, and local or corticosteroid injections.[1] A third possibility is the anomaly known as the accessory flexor digitorum longus (ADFL) that is present in up to 8% of the population. This lesion is seen on MR imaging at the level of the TCJ and distal to where the FHL transitions into tendon and lays beneath the flexor retinaculum with the FHL tendon and traversed into the fibro-osseous

tunnel of the FHL. Pulling on the tendon results in flexion of the lateral toes. Dorsiflexion of the ankle pulls the AFDL into the tunnel, resulting in decreased dorsiflexion of the lateral toes. Examination of the region reveals painful dorsiflexion of the TCJ and great toe, limited dorsiflexion of the lateral toes with the TCJ in dorsiflexion, normal dorsiflexion in the lateral toes when the TCJ is plantarflexed. When an AFDL is the cause and conservative treatment has failed, excision of the accessory muscle typically resolves the patient's symptoms.[99]

With any type of ankle trauma involving forced plantarflexion or dorsiflexion with eversion, the deltoid ligament may be sprained or suffer complete rupture. Many times, a sequelae of events on the medial and lateral side of ankle may occur to include posterior tibialis tendon rupture, lateral malleolar fracture, distal tibial fracture, capsular impingement, impingement syndromes (ATTCS or PTTCS), or talar osteochondritis dissecans.[88] As previously discussed, various ligamentous structures are individually examined through testing in specific prepositions. While each test isolates each ligament of the medial ligament completex, the posterior tibiotalar ligament is rarely involved in pathology. Treatment consists of rest, ice, compression, and elevation (RICE) in the initial 48–72 hours to decrease swelling. Conservative management includes active assisted (AAROM) and active range of motion (AROM) exercises, resistive exercise, neuromuscular re-education, unloading exercises, taping/bracing, local modalities, and TFM.[97] In more chronic cases, when there is no improvement within six visits of TFM, injection into the origin of the involved ligament is indicated.[1]

Impingement on the medial aspect of the ankle is rare when compared to anterior and anterolateral impingement syndromes. However, patients may enter the clinic with persistent medial ankle pain that has been resistant to conservative intervention. Upon clinical examination, patients with anteromedial impingement syndrome demonstrate painful and limited passive dorsiflexion and supination, anteromedial ankle pain, and swelling in some cases. Magnetic resonance arthrography (MRA) may be of benefit in depicting capsular thickening and irregular soft tissue thickening just anterior to the tibiotalar ligament and medial malleolus. Once diagnosed, management is best achieved through arthroscopic debridement of the thickened tissue.[100] Conversely, posteromedial ankle impingement typically arises from a plantarflexion inversion injury whereby the deep posterior fibers of the deltoid ligament are crushed between the talus and medial malleolus. Due to lateral ligamentous injury, posteromedial ankle pain is not the main complaint after injury and many times the symptoms will resolve spontaneously. However, in some cases pain will persist due to thick, disorganized scar tissue impinging between the medial wall of the talus and posterior aspect of the medial malleolus. Patients may present with local induration found adjacent to the posterior medial malleolus, pain, and provocation with digital pressure at the lesion site with ankle inverted and plantarflexed. A differential diagnosis is made through ruling out posterior tibialis tendopathy or tenosynovitis through passive stretch and resistive testing. Treatment can involve local injection and arthroscopic debridement of scar tissue.[101]

Tarsal Tunnel Syndrome

Tarsal tunnel syndrome (TTS) involves the motor and sensory branches of the tibial nerve (L4 to S3) as it travels underneath the flexor retinaculum. Ischemia to the tibial nerve occurs s/p fracture of the medial malleolus, calcaneus, or sustentaculum tali,[102] or by coalition associated with a ganglion between the medial malleolus and the calcaneus.[103] In some cases, a hypertrophic abductor hallucis muscle can cause TTS at the distal aspect of the flexor retinaculum as the tibial nerve divides into medial and lateral branches.[104] The anatomical arrangement of a longitudinal vascular system with bifurcating nutrient vessels dispersing proximally and distally and a lateral plantar nerve receiving a nutrient vessel from the medial plantar artery may predispose patients to TTS with surgical release or chronic inflammation. As discussed earlier, PTD can lead to hyperpronation in the mid-foot and forefoot increasing tension in the tibial nerve and vascular structures in the tarsal tunnel setting up ischemia due to decreased microlymphatic flow to the tibial nerve setting off an inflammatory cascade.[94]

Continuing distally, a "jogger's foot" is typically associated with a medial plantar nerve entrapment due to hyperpronation (eccentric loading) in long distance runners. Compression occurs at the abductor hallucis insertion with the patient complaining of symptoms from the medial longitudinal arch to the toes during and after exercise.[1] A second nerve entrapment involving the lateral plantar nerve can take place just proximal to the previous location at the abductor hallucis insertion. The lateral plantar nerve innervates the flexor digitorum brevis and is typically tension

loaded as it courses from plantar to lateral at the level of the abductor hallucis.[105]

Examination reveals local burning pain at the medial heel. In more severe cases, pain can be referred to the plantar surface of the toes and atrophy of the intrinsic muscles can be witnessed in the foot. Passive eversion and local compression will provoke symptoms. Many clinicians will screen for TTS utilizing a two-point discrimination sensory tool that demonstrates an increased threshold in cutaneous pressure perception versus age-matched controls.[106] Traditional nerve conduction studies have little influence on postoperative outcomes, and generally are insensitive to motor changes (52%) and sensory changes are frequently absent.[107] An alternative nerve conduction technique taken distal to the flexor retinaculum demonstrates improved sensory and motor reliability in the medial and lateral plantar nerves both below and across the flexor retinaculum.[107] A modified SLR test can be used to evaluate the tibial nerve as it courses through the tarsal tunnel (see Figure 4). In general, symptoms will be the worst with entire dural system maximally tension loaded, where the head and cervical spine are flexed forward and the ankle is dorsiflexed with compression at the abductor hallucis. Relief in symptoms should occur with release of compression thereby allowing the irritable focus to move distal or proximal.

Treatment consists of neural flossing (see Figure 5) and local joint mobilization. In addition, joint mobilization (P-A oscillations), soft tissue input, and low frequency TENS is applied to the thoracolumbar juction (T10-L2) to influence the autonomic nervous system to benefit improved healing potential in the nerve. In addition, local treatments using ultrasound and iontophore-

Figure 4. Plantar nerve neural tension tests. (a) Lateral plantar nerve test, starting position: The knee is first flexed and the ankle/foot is passively everted; The medial and proximal aspect of the abductor hallucis insertion is compressed; the lateral four toes are dorsiflexed; (b) Lateral plantar nerve test, test movement: The knee is extended, followed by a chin tuck. (c) Medial plantar nerve test, starting position: The knee is first flexed and the ankle/foot is passively everted; The medial and proximal aspect of the abductor hallucis insertion is compressed; the great toe is dorsiflexed; (d) Medial plantar nerve test, test movement: The knee is extended, followed by a chin tuck.

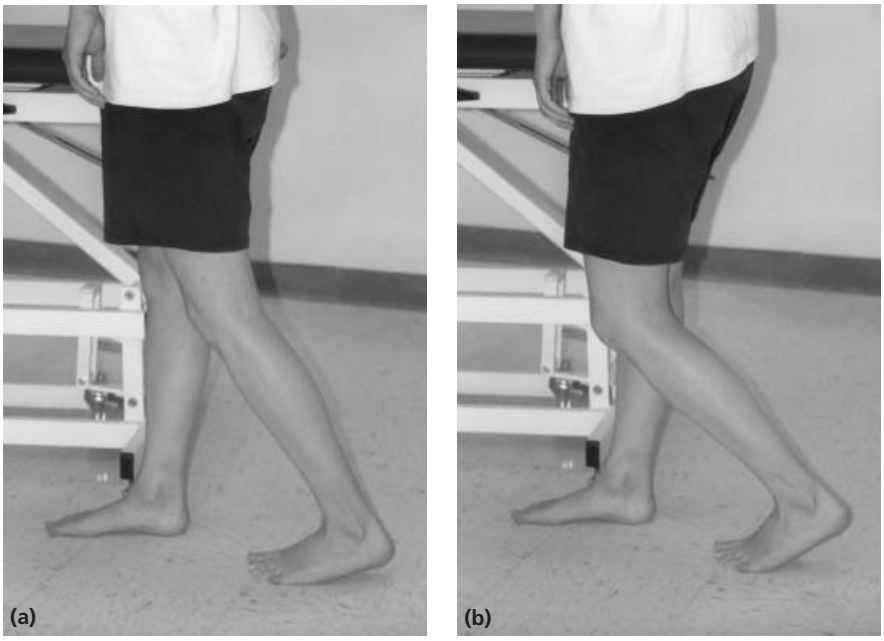


Figure 5. Plantar nerve neural flossing: (a) The patient stands on the nonaffected leg with minimal weight bearing on the affected side. (b) Flossing is performed in a slow rhythmic manner by "coming up on the toes" or plantarflexing the ankle 120 repetitions two times per day or no more than five minutes once per day.

sis above and below the flexor retinaculum may have success although not documented in the literature with TTS. If orthotic treatments are used, a very gentle arch support should be used to avoid compression of the tarsal tunnel. Injections at the tarsal tunnel can temporarily assist with decreasing symptoms and allowing more activity with limited long-term success, however, sympathetic ganglion blocks may have improved long-term success. As a last resort, surgical release of the tunnel can be performed; however, long-term success has not been optimal using traditional approaches. Technique modifications with the insertion of a fat graft between the vessels and the tibial nerve under the flexor retinaculum acting to decompress the nerve has improved the reduction of symptoms associated with TTS.[108]

Plantar Heel Pain

Plantar fasciitis results from repeated microtrauma to the fascia at its origin on the medial calcaneal tubercle with running, walking, and standing.[109–10] Unilateral symptoms are commonplace; however, bilateral symptoms can occur and are typically the result of systemic disease or lumbar radiculopathy. Patients will complain of pain at the plantar medial aspect of the heel that is aggravated by weight-bearing after periods of unloaded rest. Typically, patients will report the greatest pain first thing in the morning during the first several steps of the day that gradually subside with continued activity. Occasionally, patients may complain of pain more distally along the plantar surface of the medial longitudinal arch.[109]

The etiology of plantar fasciitis stems from overuse activity that is detected in the patient history. Several factors (both exogenous and endogenous) contribute to the syndrome. Exogenous factors include such things as footwear and training schedule; whereas, endogenous factors include pes cavus (high arch that shortens the fascia) and pes planus (flat foot that overstretches tissue with activity), and torsional malalignment of lower extremities.[109] For example, a runner may have increased his or her mileage by 20 miles for the week or worn shoes with a flexible sole or limited arch support that contributed to the development of symptoms. Endogenous factors contribute to plantar fasciitis, such as limited dorsiflexion from soft tissue restriction in the posterior muscle groups of the calf and deficits in peak torque.[110]

Far and away, pronation is the most common cause of plantar fasciitis with 81 to 86% of all cases.[109] Differential diagnosis must include heel spurs (ruled out by imaging, examination, and history), abductor hallucis, flexor digitorum brevis (FDB), abductor digiti minimi strains, long plantar ligament sprain, and plantar calcaneal bursitis since all of the above originate or refer pain into the medial heel and plantar surface of calcaneus.[109] Barrett et al. demonstrated a 21% incidence of inferior calcaneal exostosis formation in 200 cadavers whereby 52% of spurs were located within the fascia and 48% superior to the fascia. This may aide foot/

ankle surgeons in the performance of partial and total fascial release.[111] These authors proposed that heel spurs are rarely pathologic and result over time as bony adaptations from soft tissue stresses placed on the medial calcaneal tubercle. In addition, imaging that demonstrates a heel spur is not definitive for the causal factor in heel pain.[109,112] Clinically, a disparity was demonstrated by Shama et al. whereby 1000 patients underwent imaging of the calcaneus and 13% demonstrating a heel spur at the medial calcaneal tubercle. However, only 39% of those with a spur (5.2% of sample) reported ever experiencing subcalcaneal heel pain.[113]

Examination of the plantar fascia includes limited and painful great toe dorsal extension with the ankle prepositioned in dorsiflexion (symptoms worsened when performing the test while weight-bearing), possibly painful resisted toe flexion (since the intrinsic toe flexors originate off the plantar fascia), pain with unilateral heel raises, and painful palpation at the medial calcaneal tubercle.[112] Conservative treatment consists of ice, ultrasound, TFM, NSAID's, and iontophoresis[114] to reduce pain and inflammation.[109,112] Although statistical significance was not attained, Gudeman demonstrated a trend whereby iontophoresis was shown to improve symptoms in patients with plantar fascitis.[114] Additionally, cortisone injection can be used to treat this affliction. However, it has been documented in the literature that out of 765 patients treated with cortisone injection, 51 suffered rupture of the plantar fascia and 44 were correlated to cortisone injection.[115] Finally, reduction of tissue stress can be accomplished through taping, forefoot strapping, and full-length supportive orthoses.[109,112]

While many management strategies have been used, many modalities and treatment interventions have been shown to be unpredictable or minimally effective in the treatment of plantar fascitis.[116] With chronic recalcitrant fasciitis, the use of night splints in combination with traditional treatments has been shown to be effective in managing symptoms.[109,117–120] The last step of conservative management includes the restoration of muscle strength and control.[109,110] Muscle strengthening should focus on the extrinsic plantar flexor and inverter muscle groups as well as the intrinsic muscles of the foot to allow increased control and rigidity at pushoff with running and walking gait.[109,112] When the patient is involved in athletic activities, a gradual return to full weight bearing with running and cutting using an unloading device is beneficial to allow intense training without overstressing the involved tissue.[121] Finally, the most important step in conservative management of plantar fasciitis is an appropriate diagnosis. For example, night splint or orthotic management will increase symptoms in patients suffering from TTS or other nerve entrapment syndromes.

Surgical intervention should only be performed as a last resort after all conservative intervention has failed. Endoscopic or open fasciotomies that are partial or complete can be performed. Open fasciotomy involves a 3–6 cm cut along the plantar medial surface of the heel. Spurs are generally removed along with the partial or complete release of the plantar fascia.[109] Patient response to treatment has been good with pain relief ranging from 74 to 90% using both open and endoscopic procedures.[109,122,123] Endoscopic releases result in earlier return to function, however, when spur removal is needed it is generally not performed using this technique.[109] Perelman suggested that patients wear orthotic supports after the surgery.[123] Consequences that result from a complete release of the plantar fascia include increased flexibility and decreased support of the medial longitudinal arch. This can lend to decreased efficiency with both walking and running gait patterns and increased risk of overuse injury to ligamentous and tendinous structures passing under the medial longitudinal arch.[124]

Lateral Ankle/Foot Pain

The lateral ankle and foot can develop a variety of lesions to the architectural, capsuloligamentous, musculotendinous and neural structures. These lesions most frequently arise as result of inversion trauma, lending to both acute and chronic consequences. Approximately 50% of all acute injuries at ankle are related to inversion trauma. Incidence is activity-dependent, where 82% of all volleyball injuries, 79% of all basketball injuries, and 70% of all football and racquetball injuries are related to inversion trauma. However, one must consider the risk factors associated with inversion trauma. Beynnon reported numerous risk factors associated with inversion trauma, including increased rearfoot eversion, tibia varum and participation in soccer for females, versus increased inversion talar tilt for the males. Conversely, Bennyon found that general laxity, anatomical foot type, limb dominance, postural sway and lower extremity reaction times do not increase risk for inversion trauma. Moreover, investigators have reported that strength and flexibility do not increase risk.[125,126]

Inversion trauma is a type of lesion that emerges as result of unexpected directional changes and or rapid shear force development with the ankle in compromising positions, accompanied by inadequate.[127] The incidence of inversion trauma appears to be related to ankle position.[128] While the degree of inversion at touchdown during a forced landing does not appear to have a considerable influence on ankle sprain occurrence, the degree of plantar flexion does, where increased ankle plantar flexion at touchdown increases susceptibility to subsequent sprain. This influence of ankle position on injury appears to be related to the protective activity of the peroneals. The ability of the peroneal muscles to actively protect the lateral ankle/foot during a high-speed inversion can be compromised, worsening at greater inversion rates and when the foot is positioned in greater degrees of plantar flexion.[129] This peroneal inadequacy appears to be related to a disparity between the time required for the ankle/foot to reach maximum inversion (apx. 40 ms)[127] and the time required for spinal and cortical motor centers to activate a protective increase in peroneal torque (90–170 ms).[130]

As a consequence of traumatic inversion, selected capsuloligamentous structures can be compromised, lending to joint laxity and clinical instability in the talocrural and subtalar joint systems.[131] The anterior talofibular, talocalcaneal and posterior talofibular ligaments can be injured during inversion trauma, contributing to progressive laxity and instability in the talocrural joint.[132,133] The anterior talofibular ligament that reinforces the anterolateral capsule is the most frequently injured ligament in the ankle.[133] While trauma to anterior talofibular ligament does not alter subtalar joint motion,[132] inversion trauma to the talocalcaneal[134] and subtalar interosseus[135] ligament systems appears to increase subtalar laxity.[131] Moreover, inversion trauma can compromise the calcaneocuboidal and bifurcate ligaments, lending to complex instability and persistent symptoms.[136,137]

Various imaging techniques have been implemented in the diagnosis of lateral ankle injuries. Plainfilm imaging has been traditionally used for the diagnosis of lateral ligament injury and subsequent ankle laxity. More recently, the utility of such practices have come into question from clinical and economic standpoints. Clinicians may argue that imaging is most useful in detecting fracture associated with inversion trauma. However, Smith et al. found that the use of radiographic examination for the differential diagnosis of inversion trauma-related disorders can be reduced if specific criteria are incorporated during the examination. They suggested that patients are best suited for radiographic examination when they present with tenderness on the dorsum of the foot, impaired weight-bearing ability, and recentness of injury (less than 12 hours earlier). These criteria were significantly associated with the presence of a fracture, whereas swelling was not.[138]

Other imaging techniques have been implemented in the diagnosis of lateral ligament injuries, including arthrography, MRI, MR arthrography, and ultrasonography. It has been suggested that arthrography can serve as a gold standard for early diagnosis of lateral ankle injuries, due to excellent imaging quality and intertester reliability.[139] Numerous investigators have reported the use of MRI for the diagnosis of these injuries, with mixed results. Investigators have suggested that MRI is a non-invasive, accurate technique for the examination of normal and injured lateral ligaments in the ankle.[140,141] Kreitner et al. reported that MRI could be useful for grading lateral collateral ligament injuries as well as monitoring the ligament healing during non-surgical treatment.[142] Other investigators have questioned the utility of MRI for the diagnosis of lateral ligament injuries.[143] However, the patient's ankle position during the MRI examination may influence image quality and utility, as Farooki et al. found that the ATFL was best visualized during MRI when the ankle was pre-positioned in 20° plantarflexion.[144] In addition, the value of the MRI may be best appreciated for the diagnosis of associated syndesmosis lesions,[145] subtalar laxity,[146] anterolateral talocrural joint impingement,[147,148] peroneal tendon lesions[149,150] and lateral talar process fractures.[151]

Investigators have suggested that diagnostics associated with lateral ligament injuries can be enhanced through the use of MR arthrography. This imaging technique can serve to improve a clinician's understanding of the stage and extent of ligament injury as well as serve as a guide in identifying those patients at risk for developing chronic instability.[152,153] While MR arthography could be very useful in diagnosing lateral ligament injury, controversy exists over the value of ultrasound for similar diagnostic evaluation. Gruber et al. reported that the reliability of diagnostic ultrasound for diagnosing lateral ligament injury is poor,[154] whereas MIlz et al. reported the opposite, suggesting that ultrasonography can be useful in detecting lateral ligament injury.[155]

While imaging techniques could serve to enhance the diagnosis of lateral ankle disorders, they can be expen-

sive and periodically uninformative or misleading. In response, the clinician should use the clinical examination in the diagnostic process as a precursor to imaging studies. However, the value of a physical examination for the detection of lateral ankle ligament injury has been questioned.[156] In addition, patients can present to the clinic with a history of functional instability in the absence of detectable mechanical laxity in the lateral ankle.[157] Yet, other investigators have reported 84% specificity and 96% sensitivity of delayed physical examination for the presence or absence of a lesion of an ankle ligament, respectively.[158,159]

In concert with these findings, tests have been suggested for identifying laxity in the ankle, including the anterior drawer and talar tilt tests. The anterior drawer test has been utilized for identifying anterior laxity in the talocrural joint associated with lesions to the lateral ligaments. For this test the examiner stabilizes the talus and attempts to translate the mortise posteriorly while the foot is planted on the mat (see Figure 6).[1] Investigators have observed changes in anterior drawer test outcomes with changes in the position of the ankle/foot. Hollis et al. found that laxity observed during the drawer test decreased when the ankle is prepositioned in dorsiflexion.[134] Bahr et al. reported that the anterior drawer exerted the greatest force on the ATFL when the ankle was positioned in 20° of plantarflexion, whereas the CFL received the greatest force when positioned in 10° dorsiflexion.[35]

The talar tilt test has been used in concert with the anterior drawer in the identification of lateral ankle laxity. For this test the examiner stabilizes the mortise and attempts to tilt the talus medially, thus testing the ability of the lateral ligaments to constrain talar inversion. Gaebler et al. found that the talar tilt test is not useful for identifying specific lateral ligament pathology, but is reliable for detecting complete double ligament ruptures (ATFL and CFL) when the tilt is 15° greater than the uninjured side. Moreover, the diagnostic utility of this test may be best appreciated when test outcomes are interpreted in concert with the drawer test outcomes, especially when increased internal rotation is observed during plantar flexion movement testing (plantar flexion-internal rotation coupling).[140] The outcomes with each of these tests could be useful for differentiating between isolated ATFL injuries versus double ligament injuries (ATFL and CFL). They suggested that isolated ATFL tears would present with increased drawer test movement accompanied by unremarkable talar tilt and coupling behaviors, whereas all three tests will be abnormal when both ligaments are involved. In response, clinicians should perform both tests to enhance clinical diagnostic accuracy.[160]

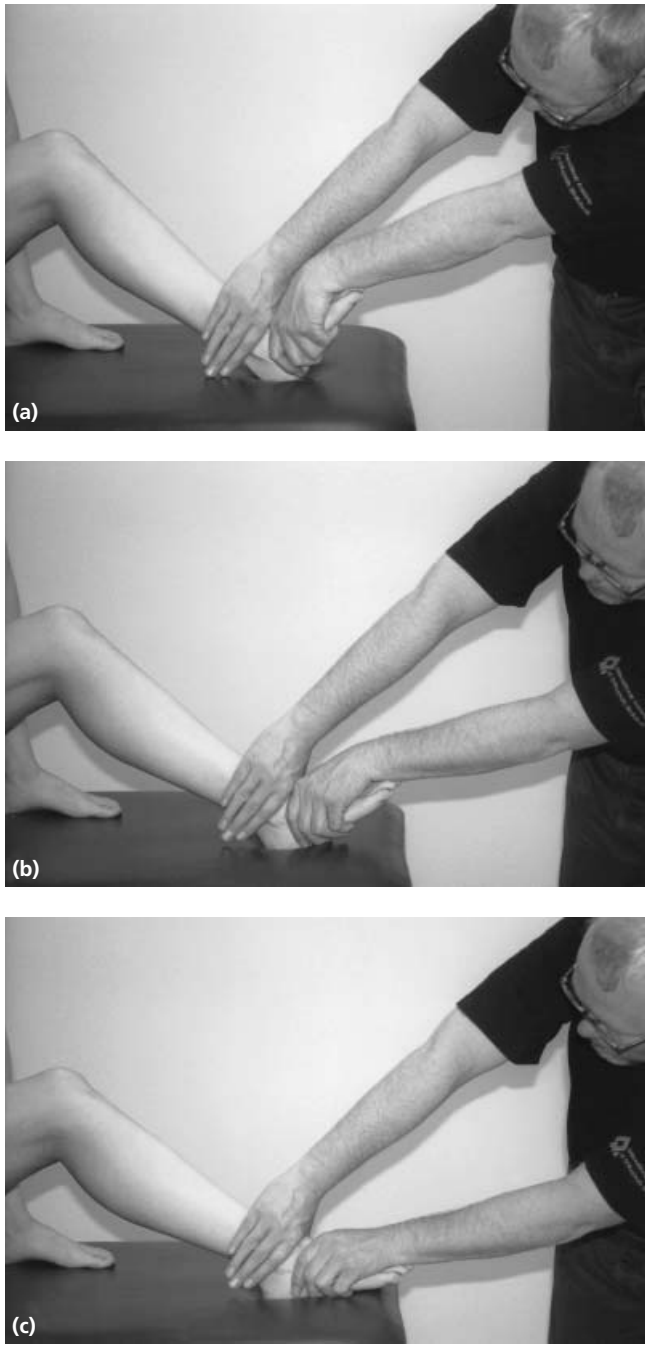


Figure 6. Anterior drawer test of the talocrural joint: (a) Test in full dosiflexion: The talus is stabilized while the ankle is fully dorsiflexed and the mortise is translated posteriorly; (b) Same test with the ankle positioned in 10° plantarflexion; (c) Final stage of the test performed with the ankle positioned in full plantarflexion.

It appears that the physical examination is the strategy of choice for diagnosing lateral ligament injuries and that additional diagnostic procedures, at considerable cost, provide no additional information of clinical consequence. In attempting to classify various ligament injuries about the lateral ankle, classification systems can help to describe the degree of ligament involvement. Inversion trauma has been graded, where grade I includes a sprain of the anterior talofibular ligament (ATFL) without any disruption, grade II produces a partial rupture of the lateral capsule and an isolated rupture of the ATFL, and grade III results in a total rupture of the lateral capsule, ATFL, and calcaneofibular ligament (CFL), occasionally accompanied by trauma to the posterior talofibular ligament (PTFL). However, authors have suggested that delayed physical examination at 5 days after the injury leads to higher sensitivity and specificity for the detection and classification of ligament injuries about the ankle.[161]

Different grades of inversion trauma present with different clinical presentations. Grade I sprains present with ankle pain localized to the anterolateral talocrural region, a "local egg" of effusion of delayed onset in the same region, normal ligament laxity testing, and unsupported ambulation. These traumas are suitably treated with measures to reduce inflammation and swelling, along with functional neuromuscular training. Grade II sprains present with complete ankle pain, a "horseshoe" of swelling of delayed onset that surrounds the lateral malleolus, normal laxity testing, and supported ambulation with weightbearing as tolerated. These lesions can be managed with similar measures as the Grade I sprain, with greater emphasis on improving motor control strategies. Grade III (and IV) sprains cannot bear weight on the involved foot and demonstrate diffused swelling of immediate onset, hemarthrosis with ecchymosis along the lateral foot border, and positive laxity testing.[1] While conservative measures have been suggested for these lesions, they may merit surgical intervention.

A relatively small percentage of inversion traumas result in an increase in loss of function. In addition, the degree of joint laxity and or lower extremity flexibility does not necessarily relate to the occurrence of inversion sprain.[128] As a consequence, conventional clinical measures may not completely represent the functional status of patients with varying lesions of the lateral ankle. In response, De Bie et al. proposed a functional scoring mechanism that scores the patient on five axes, including pain, dynamic stability, gait pattern, weight bearing status, and swelling. A functional score greater than 40 suggests that no extensive clinical treatment is necessary, only requiring lateral support (taping or bracing) and home exercise. Moreover, a score greater than 40 reflects a better prognosis, with the patient's return to full activity within two weeks postincident. Conversely, a score less than 40 merits regular clinical management for several weeks, accompanied by slower recovery and return to activity.[162]

Management of inversion trauma is stage-dependent.[1] When the patient is in the acute inflammatory stage, the patient should implement relative rest, where they ambulate and function without pain. The ankle should be immobilized in a relative fashion, allowing pain-free movement and encouraging dorsiflexion to neutral (such as with an air stirrup brace). The lower extremity should be compressed from toes to the knee to reduce swelling, along with elevation when not active and periodic manual lymph drainage. Ice for 15 minutes every hour can be used for the first 24 hours to reduce the swelling. Finally, oscillatory grade I and II joint mobilization can be used every other day at the talocrural joint, as a statistically advantaged improvement in movement and pain reduction has been demonstrated with this strategy.[163]

During the proliferation stage (from 4 to 12 days postincident) where collagen repair is underway, increased activity can be initiated with increased lateral support during functional activities (taping or functional bracing). Lateral support, while not completely eliminating inversion movement, appears to restrict the extreme of ankle motion and shorten the reaction time of the peroneal muscles in protecting the lateral ankle.[164] While both tape (Zonas, Leukotape, Jaylastic)[165] and bracing (Push, Kallassy, Swede-O, DonJoy ALPII)[166,167] have been effectively used for lateral support,[166,168,169] the techniques may differ in terms of motion constraint, human performance, and comfort. Cordova et al. suggested that bracing appears to provide superior constraint to inversion over taping, while taping produces a greater limit in dorsiflexion.[170] In addition, several investigators have suggested that tape loses a considerable degree of stiffness and constraint to motion over time as an individual exercises,[168,169] whereas a similar affect was not seen in braced ankles.[171] While Burks et al. found that performance in numerous functional activities (vertical jump, broad jump and sprint) was inhibited by both taping and bracing,[166] Pienkowski et al. did not find any significant influence on similar measures (vertical jump, standing long jump, cone run and shuttle).[172]

Deficits in control accompany inversion trauma, including vibration and two-point discrimination deficits,[173] changes in inverter reflexive latencies,[174] proprioceptive deficits,[126] balance deficits,[173,175,176] increased lateral foot loading responses[177] and altered hip muscle recruitment strategies.[178] Progressive exercise can be initiated during the early remodeling stage of healing (12 to 21 days), where emphasis on neuromuscular control is emphasized.[175] This form of training can improve proprioception,[175] reflexive lower extremity muscle activity,[179,180] and balance.[175,180–182] Exercises, performed in the closed chain, include unipodal stance on the ground, ankle disc, air squab, inversion boards, minitrampoline and angled aerobic step; as well as walking on uneven surfaces, pedalo reciprocating device, and uneven mobile walkways (such as a bin of rubber balls covered by a thick material)[175] Unipodal standing balance activities should be accompanied by two thin strips of tape starting proximal to the lateral malleolus and coursing around the malleolus to the lateral plantar foot (see figure 7), as this technique statistically reduced postural sway and enhanced recovery of dynamic balance over similar exercise without the tape.[182] Furthermore, emphasis can be placed on strengthening of the peroneals, so to increase passive stiffness and enhance passive constraint to inversion through hypertrophic change. Finally, an increase emphasis on return to sports can be initiated after 21 days postincident, progressing to functional activities that include jumping, shuttle run, and cariocas.

When more severe ankle inversion trauma does not sufficiently respond to conservative management and patients cannot return to activity, then surgical measures can be incorporated.[183] Various different reconstruction techniques have been used, including primary ligament reconstruction[184,185] and various tenodesis techniques using the peroneus brevis tendon as a graft material.[134,185] While tenodesis procedures appear to reduce laxity associated with lateral ligament injury,[134] investigators have reported in long-term follow-up studies that patients receiving tenodesis demonstrated greater incidence of laxity and degenerative changes, as well as reduced sports activity levels, versus those treated with primary reconstruction.[185,186]

DiGiovanni reported several associated injuries that accompany lesions to the lateral ligaments after inversion trauma. These include ankle synovitis, posterior talotibial compression syndrome, talar osteochondral lesions, talar lateral process fractures, and syndesmotic

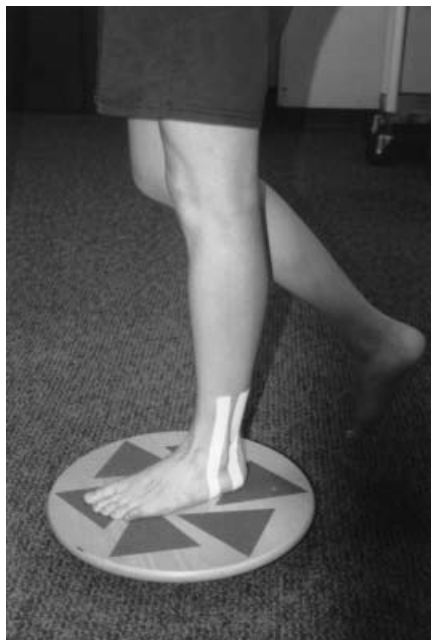
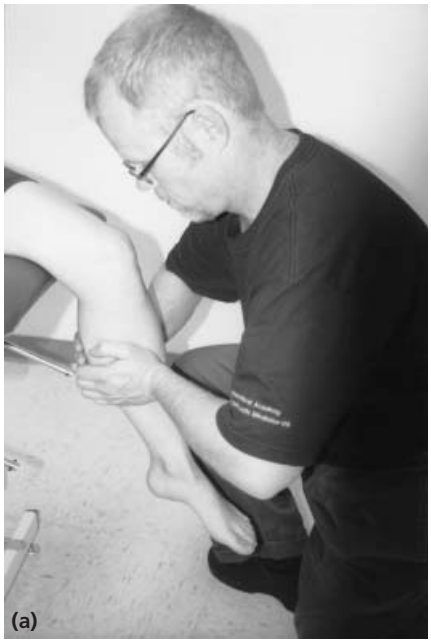


Figure 7. Unipodal standing balance activities accompanied by two thin strips of tape starting proximal to the lateral malleolus and coursing around the malleolus to the lateral plantar foot.

lesions.[137] Talar osteochondral lesions can occur on the posteromedial (rarely symptomatic) and lateral talar (often painful) dome.[187] Surgical management for these lesions includes curettage, drilling, excision, grafting and or transplantation. Talar lateral process fractures are linked to compression accompanying inversion and can be misdiagnosed as chronic lateral ankle sprain.[188] An MRI is best suited for making this diagnosis.[151] Syndesmotic lesions (high ankle sprains)[167] can accompany lateral ankle sprains, especially when more severe.[145] Magnetic Resonance is highly sensitive for identifying syndesomitic lesions.[189,190] In addition, clinical tests have been proposed for testing the syndesmosis, including the squeeze test for acute lesions[191] and the lateral gapping test for subacute lesions (see Figure 8).[167,192] These lesions are best treated with modalities, splinting, and non- to partial-weightbearing crutch ambulation, followed by a more gradual return to weightbearing and functional activities.[167] Lateral support, and neuromuscular control exercises will be incorporated, expecting a longer recovery time versus conventional lateral ankle sprain.[193]

Several conditions can accompany and or mimic a lateral ankle sprain, including impingement lesions, peroneal retinacular compromise, chronic ankle instability, peroneal tendon afflictions, and neural lesions.[194]



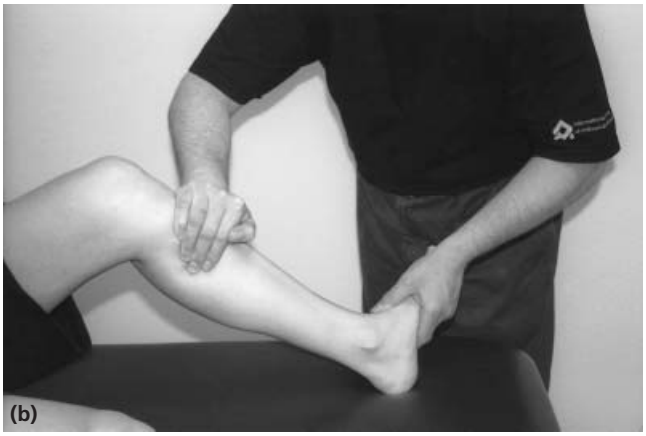


Figure 8. Syndesmosis Tests: (a) Squeeze test for acute lesions: The clinician places the stabilization hand to the anterior medial proximal 1/3 of the tibia and squeezes the fibula in an anterior medial direction; (b) The lateral gapping test for subacute lesions: The clinician provides stabilization to the anterior lateral edge of the proximal 1/3 of the tibia. The ankle is fully dorsiflexed and the clinician uses the foot as a lever to rotate the foot and laterally gap the syndesmosis.

Anterolateral impingement can occur in the presence or absence of lateral ankle instability. Chronic lateral ankle instability allows the mortise to translate posteriorly when the ankle/foot is weightbearing. As a consequence, when the individual attempts dorsiflexion, the anterior inferior tibiofibular ligament is impinged between the anterior edge of the mortise and the talar dome.[195] Conversely, impingement can occur in the absence of instability.[196] Here, a previous talocrural synovitis lends to eventual anterior capsular thickening and ultimate impingement with dorsiflexion in weightbearing.[148] Clinicians will be able to visualize a soft tissue signal mass in the anterolateral gutter of the ankle with MR imaging.[148] In either case the patient complains of pain with closed chain dorsiflexion. The former cause should be managed with stabilization and bracing, while the later can be treated with injection and or surgical excision.

Peroneal retinacular compromise can accompany lateral ankle instability ("lateral snapping ankle").[1] As a consequence, patients can develop overuse tendon reactions as the peroneal tendons repetitively snap over the lateral malleolus. Tendon afflictions can accompany or mimic the pain associated with chronic lateral ankle instability. Less severe lesions, including insertion tendonitis and proximal or malleolar tenosynovitis, can produce persistent lateral ankle pain. Tenosynovitis of either the peroneus longus or brevis is frequent in the proximity of the lateral malleolus and will be most painful during the examination when the ankle/foot is passive dorsiflexed and inverted. Conversely, insertion tendonitis of the peroneus brevis at the base of the 5th metatarsal will be most painful with diagonal resisted plantarflexion/eversion.[1] Both can be treated with iontophoresis, transverse friction, and gentle stretching. In addition, a local infiltration of anesthetic agent and long acting steroid is recommended at the insertion of the peroneus brevis. The injection should be followed by 7–10 days of rest with reduced load, so to avoid any catabolic reaction at the tendon insertion. Finally, management should include previously discussed stabilization measures of the tendopathies accompany chronic instability.

More profoundly, the peroneal tendons are at risk for partial or complete failure. Relative avascular zones found in the tendons lend them the partial or complete tearing.[197] Lateral ankle instability places both longus and brevis tendons at risk for tearing. The peroneus longus tendon demonstrates tearing at the midfoot as the tendon courses around the cuboid.[150] The peroneus brevis tendon is at risk for longitudinal tears when exposed to the posterior distal lateral edge of the lateral malleolus in response to lateral peroneal retinacular laxity.[149,198] Surgical interventions can include tendon repair, retinacular tightening, and previously discussed ligament reconstructions.[198]

Peripheral nerve lesions can produce lateral ankle pain. The branches of the superficial peroneal nerve can become irritated after inversion trauma, resulting in

radiating pain and or sensory changes on the dorsum of the lateral foot.[199,200] This lesion can be provoked through neural tension testing where the clinician performs a modified straight leg raise with the ankle/foot positioned in plantarflexion and inversion. The provocation of a painful nerve is confirmed when the provoked symptoms are changed when the neck is flexed while the modified straight leg raise position is maintained (see Figure 9). In addition, the sural nerve can be irritated by external pressure against the nerve,[201] after achilles tendon rupture or repair,[202,203] or after inversion trauma, resulting in posteror lateral ankle pain. This affliction can mimic peroneal tenosynovitis[204] and is provoked with a modified straight leg raise procedure similar to the superficial peroneal nerve, only with the ankle/foot positioned in dorsiflexion and inversion (see Figure 10). Both conditions can be effectively treated with infiltrative adhesiolysis and neural flossing (see Figure 11).

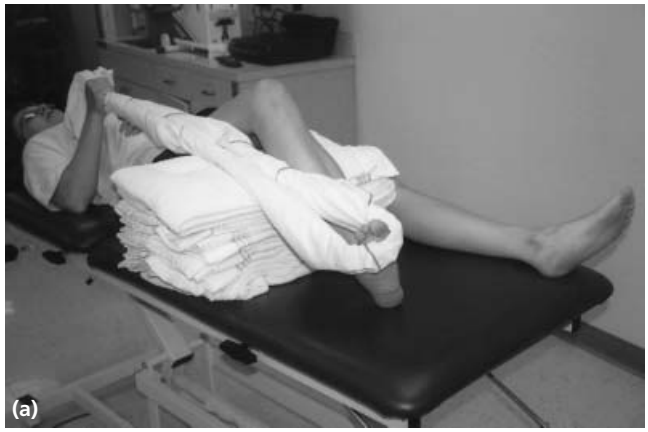
One final affliction associated with the lateral ankle/foot is sinus tarsi syndrome. In the absence of true lateral extrinsic ligament compromise, a patient can suffer from persistent lateral pain, prolonged peroneal reaction times, and a consequential "feeling of instability." This condition is associated with persistent inflammation residing the sinus tarsus and elongation of the cervical ligament within the space.[205] This affliction is best treated with previously discussed neuromotor training. If persistent, then an invasive procedure could be incorporated. Anesthetic agents and long acting steroids have been injected into the sinus tarsus.[1] Alternatively, a radiofrequency thermocoagulation (RFTC) lesion could be used on the lateral terminal branch of the deep peroneal nerve, which innervates the sinus.[206]

Figure 9. Superficial peroneal nerve neural tension test. (a) Starting position: The knee is first flexed and the ankle/foot is passively plantarflexed, adducted and supinated; (b) The knee is extended and the leg is lowered to the mat; (c) The leg is raised in the fashion of a straight leg raise. The neck can be flexed to observe for changes in the provocation.

SUMMARY

Diagnosis, interpretation and subsequent management of ankle/foot pathology can be challenging to clinicians. A sensitive and specific physical examination is the strategy of choice for diagnosing selected ankle/foot injuries and costly additional diagnostic procedures may not provide additional information for clinical diagnosis and management. Because of a distal location in the sclerotome and the reduced convergence of afferent signals from this region to the dorsal horn of the spinal cord, pain reference patterns are low and the localization of symptoms is trustworthy. Effective management of the painful ankle/foot is closely linked to a tissue-specific clinical examination. The examination of the ankle/foot should include passive and resistive tests that provide information regarding movement limitations and pain provocation. Special tests can augment the findings from the examination, suggesting compromises in the structural and functional integrity of the ankle/

Figure 10. Sural nerve neural tension test. (a) Starting position: The knee is first flexed and the ankle/foot is passively dorsiflexed, adducted and supinated; (b) The knee is extended and the leg is lowered to the mat; (c) The leg is raised in the fashion of a straight leg raise. The neck can be flexed to observe for changes in the provocation.



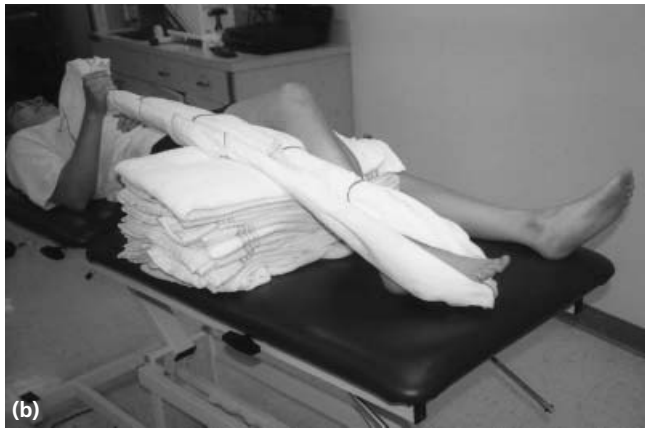


Figure 11. Neural Flossing for the superficial peroneal nerve: (a) Starting position; (b) finishing position; The opposite order could be implemented for the sural nerve.

foot complex. The weight bearing function of the ankle/foot compounds the clinician's diagnostic picture, as limits and pain provocation are frequently produced only when the patient attempts to function in weight bearing. As a consequence, clinicians should consider this feature by implementing numerous weightbearing components in the diagnosis and management of ankle/foot afflictions. Limits in passive motion can be classified as either capsular or non-capsular patterns. Conversely, patients can present with ankle/foot pain that demonstrates no limitation of motion. Bursitis, tendopathy, compression neuropathy, and instability can produce ankle/foot pain that is challenging to diagnose, especially when they are the consequence of functional weight bearing. Numerous non-surgical measures can be implemented in treating the painful ankle/foot, reserving surgical interventions for those patients who are resistant to conservative care.

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QUESTIONS

1. Your patient, who is a runner presents to the clinic with lateral ankle foot pain that demonstrates greatest provocation produced with resisted 3-D position during resisted plantarflexion, abduction, and pronation. All other tests are less provocative. What affliction do you suspect?
   a. Achilles peritenonitis
   b. Peroneal tendonitis
   c. Peroneal tenosynovitis
   d. Tibialis anterior tenosynovitis
   e. Tibialis posterior tendonitis
2. You are testing your patient's anterior talofibular ligament for laxity. Which movement will best test this ligament?
   a. Passive abduction and pronation in full dorsiflexion,
   b. Passive abduction and pronation in full plantarflexion
   c. Passive adduction and supination in 10° plantarflexion
   d. Passive adduction and supination in full dorsiflexion
   e. Passive adduction and supination in full plantarflexion
3. Your patient reports sharp shooting pain in the medial heel region (occasionally lateral heel) that occurs occasionally with weightbearing activities. Immediately after the pain, the lower extremity feels as though it will give-way. Upon examination, you recognize that the subtalar joint demonstrates noncapsular pattern limits in the direction of pronation with a hard end-feel accompanied by a soft, springy end-feel in passive supination. The pain is variable and unpredictable. From this finding, you suspect which of the following?
   a. Flexor hallucis longus tenosynovitis
   b. Plantar fasciitis
   c. Subtalar joint arthrosis
   d. Subtalar joint loose body fragment
   e. Tarsal tunnel syndrome
4. All of the following are considerd to be factors that lend to achilles tendopathy, except for:
   a. Clinical external tibial torsion
   b. Excessive subtalar pronation
   c. Increased vascular supply to the mid-tendon
   d. Stenotic thickening of the vascular intimae
   e. Subtalar joint mobility disturbances
5. Which of the following test procedures would be most provocative for a patient suffering from Achilles insertion tendopathy?
   a. Manual, resisted ankle/foot dorsiflexion in supine
   b. Manual, resisted ankle/foot plantarflexion in supine
   c. Multiple unilateral heel raises in standing
   d. Passive ankle/foot dorsiflexion in supine
   e. Passive ankle/foot plantarflexion in supine
6. Your 27 y.o. female patient presents with posterior heel pain that occurs when she comes up on point during her ballet dance routines. She reports a history of previous multiple ankle sprains on the same side and demonstrates no limitation of movement in the clinical examination. The only two tests that provoke her symptoms are full unilateral heel raises (which are not provocative when performed to submaximal range) and passive ankle/foot plantar flexion in nonweight bearing. All imaging is negative. Which of the following afflictions do you suspect?
   a. Achilles partial tears
   b. Flexor hallucis longus tenosynovitis
   c. Achilles tendinosis
   d. Peroneal tenosynovitis
   e. Posterior talotibial compression syndrome
7. Your patient presents with a history of falling flat on his left foot from a height of 6 feet. Now he complains of medial ankle and foot pain. Upon examination, you notice that he demonstrates a dynamic rearfoot deformity, where his calcaneus pronates and the arch falls flat with weightbearing. In addition, you note "too many toes". The most provocative test in the examination is a diagonal resisted plantarflexion, adduction and supination. What affliction do you most suspect?
   a. Stage I achilles insertion tendinitis
   b. Stage I Flexor hallucis longus dysfunction
   c. Stage II extensor digitorum dysfunction

d. Stage II tibialis posterior dysfunction
e. Stage III peroneal tenosynovitis

8. Tarsal tunnel syndrome can be caused by all of the following, except for:
   a. Hypertropic Extensor Hallucis Longus
   b. Malleolar or navicular fracture
   c. Plantar nerve neural ganglion
   d. Subtalar hyper-pronation
9. Management of tarsal tunnel syndrome can include all of the following, except for:
   a. High arch orthotics
   b. Local joint mobilization
   c. Low frequency TENS
   d. Neural flossing
   e. Surgical release
10. According to the literature, which of the following non-surgical measures has proven most effective in the management of recalcitrant plantar fasciitis?
   a. Facial stretching
   b. Foot taping
   c. Full length orthotics
   d. Night splints
   e. Ultrasound
11. Risk for ankle/foot inversion trauma could increase in selected athletes because of all of the following, except for:
   a. Exagerated tibia varum
   b. General laxity
   c. Increased rearfoot eversion
   d. Inversion talar tilt
   e. Participation in soccer
12. Your patient reports a history of plantar flexion-inversion trauma to her right ankle. She presents with a horseshoe of swelling about the lateral malleolus, normal laxity testing, and ambulation without support. The most provocative tests in the examination are passive inversion while the ankle is positioned at both full and 10° plantar flexion, as well diagonal passive dorsiflexion, adduction and supination. Which affliction do you suspect?
   a. Grade 1 inversion trauma with partial peroneal tears
   b. Grade 1 inversion trauma with peroneal tenosynovitis
   c. Grade 2 inversion trauma with peroneal insertion tendinitis
   d. Grade 2 inversion trauma with peroneal tenosynovitis
   e. Grade 3 inversion trauma with peroneal insertional tendinitis
13. A grade II plantarflexion inversion trauma will involve all of the following ligaments, except for:
   a. Anterior talofibular ligament
   b. Calcaneofibular ligament
   c. Posterior talofibular ligament
   d. Spring ligament
14. Your patient presents with a recent history of plantarflexion inversion trauma. During the examination, you are able to provoke a sharp pain in the anterior ankle region with a lateral gapping test, where you use the dorsiflexed ankle/foot as a lever to attempt external rotation of the patient's lower leg while stabilizing the tibia. This test is indicative of:
   a. Grade 1 plantarflexion inversion trauma
   b. Grade 2 medial ankle sprain
   c. Subacute syndesmosis lesion
   d. Subtalar joint sprain
   e. Tarsal tunnel syndrome
15. A modified straight leg raise with the ankle/foot prepositioned in plantarflexion, adduction, and supination is provocative for entrapment of which nerve?
   a. Medial plantar nerve
   b. Saphenous nerve
   c. Superficial peroneal nerve
   d. Sural nerve
   e. Tibial nerve

ANSWERS

1 b
2 e
3 d
4 c
5 c
6 e
7 d
8 a
9 a
10 d
11 b
12 d
13 d
14 c
15 c

Appendix A: Clinical Examination of the Ankle/Foot

Chief Complaint:

Screening Tests in Weightbearing
- Bipedal Squat
- Unipedal Squat for ATTCS
- Unipodal Toe Raises

TaloCrural Joint
- Passive Dorsal Extension
- Passive Plantar Flexion

Subtalar Joint
- Passive Varus in supine for provocation
- Passive Valgus in supine for provocation
- Passive Varus in prone for mobility
- Passive Valgus in prone for mobility

Midtarsal Joints
- Passive Dorsal Extension
- Passive Plantar Flexion
- Passive Abduction
- Passive Adduction
- Passive Supination
- Passive Pronation

Lateral Ligament Tests
- Passive Adduction, Supination in Full Plantar Flexion
- Passive Adduction, Supination in 10° Plantar Flexion
- Passive Adduction, Supination in Full Dorsal Extension

Medial Ligament Tests
- Passive Abduction, Pronation in Full Plantar Flexion
- Passive Abduction, Pronation in 10° Plantar Flexion
- Passive Abduction, Pronation in Full Dorsal Extension

Diagonal Tests
- Resisted Dorsal Extension, Abduction, Pronation (for Extensor Digitorum)
- Passive Plantar Flexion, Adduction, Supinatio(stretch to ED)
- Resisted Plantar Flexion, Abduction, Pronation (for Peroneals)
- Passive Dorsal Extension, Adduction Supination (stretch to Peroneals)
- Resisted Dorsal Extension, Adduction Supination (for Tibialis Anterior)
- Passive Plantar Flexion, Abduction, Pronation (stretch to TA)
- Resisted Plantar Flexion, Adduction, Supination (for TP, FDL, FHL)
- Passive Dorsal Extension, Abduction, Pronation (stretch to TP, FDL, FHL)
- Appendix A: Clinical Examination of the Ankle/Foot (cont'd)

Instability Tests
- Anterior Drawer in Plantar Flexion
- Anterior Drawer in Neutral
- Anterior Drawer in Dorsal Extension
- Relocation Test for anterior instability
- Syndesmosis Squeeze Test
- Syndesmosis Lateral Gapping Test