

Diagnosis and Management of Posterior and Plantar Heel Pain in Non-Athletes

Introduction

Heel pain refers to pain or discomfort around the calcaneus or heel pad that most often presents inferiorly or posteriorly.¹ According to existing literature, the risk of developing this condition strengthens with increasing age, body mass index (for sedentary individuals), prolonged weight-bearing activities (often work-related), and running; therefore, heel pain most commonly affects the elderly, obese, and athletes.^{1,2} Heel pain can be debilitating for patients since it often presents during weightbearing—affecting one’s ability to ambulate, perform activities of daily living (ADLs), and ability to participate in various social activities. Causes of heel pain are largely mechanical in nature, but can also result from traumatic, neurologic, arthritic, or other systematic causes.³ Physical therapists have the clinical knowledge of the foot and ankle to help differentiate between causes of heel pain, provide interventions and recommendations to alleviate symptoms, and address the underlying mechanisms that exacerbate the condition. The specific types of heel pain are often categorized in literature by the location or the associated structures affected by pathology, however, this paper will only explore posterior and plantar causes of heel pain. Overall, this paper aims to differentiate between the mechanisms, differential diagnoses, and physical therapy management of posterior and plantar heel pain in individuals uninvolved in competitive athletics, by focusing on two common diagnoses—plantar fasciitis and Achilles tendinopathy.

Pathoanatomy of the Heel

The heel is comprised of the calcaneus, the heel bone that connects to the talus and cuboid bones, and the surrounding adipose tissue that creates a heel pad for protection to the foot during weight bearing.¹ The fat pad of the heel anchors the skin to the calcaneus and serves as a

shock absorber by dissipating forces through the hindfoot during the heel strike phase of ambulation.⁴ Although injury to the calcaneal bone is rare, it can occur as a result of a traumatic fall or excessive weightbearing, resulting a calcaneal fracture or stress fracture, respectively.¹ However, the heel is more often affected by pathology to the soft tissue structures that are connected or close to the area, including tendons (e.g. Achilles, tibialis posterior, and peroneus), plantar fascia, and nerves.¹ The Achilles tendon originates from the gastrocnemius and soleus muscles (responsible for plantar flexion) and inserts medially and inferiorly onto the posterior aspect of the calcaneus.⁵ It is continuous with the plantar fascia, which conversely tightens as tensile loads are applied to the tendon.⁴ The plantar fascia is described as a broad, fibrous band of fascia that originates from the calcaneal tuberosity and expands throughout the plantar surface of the foot.⁶ As the Achilles tendon endures some of the highest tensile loads in the body, it is at a higher risk of developing pathology at the insertion site or the mid-substance of the tendon.^{1,5} However, the heel is comprised of neural tissue that can also present as soft tissue pain.¹ For example, the posterior tibial nerve can become entrapped under the flexor retinaculum and cause burning, tingling, or numbness at the sole of the foot with prolonged standing or increased activity, which describes a condition termed as *Tarsal Tunnel Syndrome*.¹

Differential Diagnosis of Heel Pain

When differentiating between different causes of heel pain, it is imperative to obtain a thorough subjective history of the patient, physically examine the foot and ankle, and utilize appropriate imaging as necessary.⁷ The subjective interview of the patient should acquire details about the pain and symptom characteristics, history of onset (traumatic versus insidious onset), factors that exacerbate or alleviate symptoms, recreational or occupational activities, physical activity levels or recent changes, pain patterns during gait, shoe wear characteristics, and various

comorbidities or previous treatments implemented.^{4,7} However, identifying the anatomic location of pain can be integral in diagnosing the etiology of heel pain.⁷ Therefore, physical examination should include palpation of tendinous insertions, bony prominences, foot and ankle joints, and various soft tissues during rest and weightbearing.⁷ There should also be an assessment of active and passive range of motion of the ankle joint to identify soft tissue tightness or restrictions.⁷ Although not always required, additional causes of heel pain can be ruled out with magnetic resonance imaging (MRI), radiography in weight-bearing, and ultrasonography.⁷ Mechanical causes of heel pain can be described by location, such as plantar, posterior, and the medial and lateral aspect of midfoot; however, this paper will focus on conditions associated with the posterior and plantar aspects of the foot.⁷

Mechanical causes of posterior heel pain can include Achilles tendinopathy, Haglund deformity, Retrocalcaneal bursitis and Sever disease.⁷ *Sever disease* is an apophysitis of the calcaneus causing pain in adolescents during a growth spurt or increased physical activity.⁷ Examination may reveal tenderness at the Achilles insertion or pain elicited from passive dorsiflexion or mediolateral compression to the calcaneus.⁷ The age of the patient can help to differentiate Sever disease from *Achilles tendinopathy*, which is characterized by achy and occasionally sharp pain exacerbated by increased activity or direct pressure to the area.⁷ As the most common source of posterior heel pain, Achilles' tendinopathy will be explored in further detail later in this paper. When the fluid-filled sac, or bursa, located between the Achilles' tendon and the calcaneus becomes inflamed, it is diagnosed as *Retrocalcaneal bursitis*.⁸ With this condition, observation and subjective information can reveal swelling, erythema, and pain directly superior to the calcaneus and anterior to the Achilles' tendon that is exacerbated by repetitive or increased activity.⁸ Physical examination may include a tenderness with palpation, a

positive “two finger squeeze test” (which applies compression in the mediolateral direction but anterior to the Achilles’ tendon), and pain with dorsiflexion or “active resisted plantarflexion.”^{7,8} This diagnosis is most common in runners and is associated with inflammatory arthritis and skeletal deformities such as hindfoot varus and a “rigid plantarflexed first ray.”⁸ Lastly, *Haglund deformity* refers to a bony prominence at the superior aspect of the posterior calcaneus that mostly affects middle-aged women.⁷ This skeletal deformity can directly contribute to retrocalcaneal bursitis or Achilles tendinopathy, especially when combined with ill-fitting shoes that increases the pressure to the area that causes further inflammation.⁷ It is important to remember to consider the age and activity levels of the patient to assist with differentiating these conditions. For example, Sever disease is only appropriate for adolescent-aged patients, while Haglund deformity may be a more appropriate cause of retrocalcaneal bursitis in the sedentary individual. Although MRI can be used to assist in identifying a Haglund deformity, it does not change the intervention options for the patient and is therefore unnecessary.⁹

Plantar heel pain affects more than 2 million people each year and costs approximately \$284 million to evaluate and treat in the United States.⁴ Mechanical causes of plantar heel pain include plantar fasciitis, heel spurs, calcaneal stress fractures, nerve entrapment, neuroma, heel pad syndrome, hindfoot deformities (cavus/calcaneus), inflammatory enthesopathy, and partial or complete ruptures to the plantar fascia.^{4,7} A sedentary individual who recently increased their levels of weight-bearing physical activity or ambulates on hard surfaces may acquire a *calcaneal stress fracture*, which develops from an excess, repetitive load to the heel and most often presents “immediately inferior and posterior to the posterior facet of the subtalar joint.”⁷ Physical examination may reveal pain with calcaneal squeeze test and ecchymosis, swelling, and/or point tenderness at the site of the fracture.⁷ Although the pain is initially only present during activity, it

can worsen and progress to being painful during rest without proper treatment.⁷ Therefore, calcaneal stress fractures should be considered when there is an insidious or gradual onset of pain and can be adequately ruled out with imaging.⁷ Sedentary individuals are less likely to sustain a partial or complete *rupture of the plantar fascia* since it is associated with direct trauma or athletic activity.⁴ Similar to other causes of heel pain, excessive and repetitive loads to the fascia can result in subacute degeneration creating pain for the individual.⁴

However, heel pain in conjunction with numbness, tingling, or burning may indicate a neuropathic etiology, such as nerve entrapment or neuromas.^{4,7} *Nerve entrapment* of the posterior tibial nerve branches can occur from surgical injury, trauma, or overuse. Although nerve entrapment often presents unilaterally, it is important to rule out radiculopathy from the lumbar spine (L4-S2) in differential diagnosis.⁷ Similarly, *neuromas* can occur on the tibial nerve branches, mimicking plantar fascia pain with an intensified, “electrical” sensation of tingling or burning radiating through the heel.^{7,10} Neuromas can develop in response to direct or chronic trauma and is also associated with obesity and excessive, repetitive loads, causing an increase in pain throughout the day.¹⁰ If this pathology is suspected or the patient is unresponsive to plantar fasciitis intervention, then the site should be palpated for a lump.⁷ Ultrasound and MRIs can also be used to identify both neuromas and nerve entrapments.^{7,10}

Another cause of plantar heel pain is inflammation or atrophy of the fat pad, commonly referred to as *heel pad syndrome*, which produces a “deep, bruise-like pain, usually in the middle of the heel.”⁷ As the fat pad begins to deteriorate, the structural integrity becomes compromised, but evidence is inconclusive as to whether the thickness or elasticity of the fat pad is reduced with aging and increased body weight.⁴ Pain from heel pad syndrome can be elicited with firm palpation to the heel pad, barefoot walking, and ambulation on rigid surfaces or for prolonged

duration.⁷ Conversely, patients who report “throbbing” pain that presents medially on the plantar surface of the heel and is the most intense the first few steps following rest but eases with additional ambulation, may be diagnosed with *Plantar fasciitis*.⁷ Plantar fasciitis is the cause of 80% of plantar heel pain cases and will also be discussed in further detail later in this paper.⁴ Lastly, *calcaneal/heel spurs* is a bony growth of the calcaneal tuberosity that occur in approximately 50% of plantar fasciitis cases.⁷ It is often asymptomatic in the general population and can be diagnosed through radiologic imaging; however, imaging is not required since the indication of heel spurs has no effect on the patient’s intervention plan.⁶

Rare Causes of Heel Pain

There have been a few rare causes of plantar and posterior heel pain reported in literature that can be considered in the differential diagnosis when a patient is unresponsive to treatment—such as Nora’s lesion, calcaneal spur fractures, and heel lipoma.^{3,11,12} For example, one case reports a 48-year old woman referred for a “sharp, stabbing pain” with ambulation, dull aching pain during sitting, swelling of the right foot with no known incidence of trauma, and tenderness with palpation of the calcaneal tuberosity and mediolateral heel compression upon physical examination.¹¹ Following a treatment plan for plantar fasciitis that included stretching of the plantar muscles, nonsteroidal anti-inflammatory drugs (NSAIDs), rest, and protected weightbearing, her symptoms and pain continued to proliferate for the following two months.¹¹ A series of imaging revealed edema and a growing osseous mid-calcaneal lesion that was surgically removed following a bone biopsy that diagnosed it as a benign neoplasm, called bizarre parosteal osteochondromatous proliferation (BPOP), also referred to as Nora’s lesion.¹¹

Rare cases of calcaneal spur fractures resulting from acute trauma are also reported in literature, including the case of a 45-year old woman with right foot pain that restricted her

ability to bear weight.³ Although the area was painful to palpation, there was no indication of inflammation, swelling, atrophy of the heel pad, or range of motion restrictions.³ Once imaging confirmed a “fracture of the inferior calcaneal spur on the right foot,” she was treated with a short leg cast, NSAIDs, cryotherapy, and activity modification.³ Lastly, lipoma is another rare source of heel pain that can present similarly to plantar fasciitis due to sharp, focal pain that increases with activity, aggravation with improper shoe wear, and pain with the first step of the day or after prolonged periods of inactivity.¹² One case study highlighted the importance of exploring alternative diagnoses when symptoms and treatment outcomes are not consistent with plantar fasciitis.¹² For example, following eight months of unsuccessful plantar fasciitis treatment, further imaging of a 64-year-old dancer revealed a “fusiform capsulated lipoma,” described as a benign tumor comprised of adipose tissue, which was surgically removed to ultimately resolve her heel pain with ambulation.¹²

Achilles Tendinopathy

Achilles Tendinopathy is the most common mechanical cause of posterior heel pain and is marked by achy or sharp pain that intensifies with increased activity.⁷ As the tendon sustains excessive tensile loads or direct pressures, the Achilles begins to break down, resulting in acute inflammation (tendinitis) that can gradually progress to diffuse thickening of the associated soft tissues, proliferated matrix remodeling, and a mechanically unstable structure at an increased risk of injury (tendinosis).^{7,13} As an overuse injury, Achilles tendinopathy most commonly affects competitive athletes and runners at a 24% and 40-50% lifetime expectancy rate, respectively; however, sedentary individuals are not exempt from this condition.¹³ Extrinsic risk factors that contribute to Achilles tendinopathy include destructive training habits (e.g. abrupt changes in intensity, duration, and activity level), harsh environmental elements (e.g. rigid running surfaces

or cold temperatures), and inadequate shoe wear.¹³ Conversely, intrinsic factors leading to Achilles tendinopathy include lateral ankle instability, tendinous or foot structure deformities, impaired plantar-flexor strength, and abnormal moving patterns such as impaired dorsiflexion and subtalar joint motions and excessive pronation.¹³ Non-musculoskeletal factors include increased body mass index and associated conditions (hypertension, diabetes, and increased cholesterol), male sex, corticosteroid and fluoroquinolone use, and older age.¹³ Achilles tendinopathy commonly affects middle-age individuals as reductions in blood supply and tensile strength of the tissue occur as a result of aging.¹³

Achilles tendinopathy can be further categorized based on the affected anatomical location, described as midsubstance or insertional.⁷ *Midsubstance* Achilles tendinopathy occurs 2 to 7 centimeters from the calcaneal insertion and presents with diffuse or localized swelling and pain that most often impairs performance.¹⁴ In contrast, *insertional* Achilles tendinopathy presents at the insertion site of the Achilles' tendon onto the calcaneus and can often cause bone formation and tendon proper calcifications at the site.¹⁴ Patients with insertional Achilles tendinopathy may report pain, stiffness, and swelling.¹⁴ When performing a physical examination on this patient, palpation of the insertion site may reveal a bony spur and pain may present "at the midportion of the posterior aspect of the calcaneus" and elicited with active dorsiflexion.¹⁴ Diagnosis can be confirmed with imaging (e.g. radiography or ultrasound) which may commonly reveal tendon thickening, intratendinous calcifications or tendinous spurring at the insertion site, but is only recommended when the patient is unresponsive to treatment.^{7,15} For the purposes of this paper, interventions will focus on insertional tendinopathies.

Achilles tendinopathy can be treated with activity modifications, heavy eccentric loading, manual therapy, analgesic medications, medicinal injections, orthotic devices (e.g. heel lifts),

extracorporeal shock therapy, nitroglycerin patches, and methods that reduce pressure to the affected area.^{7,14} Activity modifications should be aimed at reducing the load to the tendon and reducing or avoiding activities that exacerbate symptoms.¹⁵ When treating Achilles tendinopathy with eccentric loading, one meta-analysis found that exercises performed at the floor level produces better outcomes than exercises that engaged full range of motion (e.g. calf raises from the floor instead of from a step).¹⁴ For patients with insertional tendinopathies, patients were more inclined to report dissatisfaction with full range of motion exercises than individuals with midsubstance tendinopathy.¹⁴ A 12-week protocol was used in the studies reviewed, consisting of up to three sets of 10 to 15 repetitions of painful, eccentric exercises performed 2 to 3 times per day.¹⁴ In regard to medicinal injections, two studies report promising results but with low-quality evidence.¹⁴ One study evaluated polidocanol injections and while the other evaluated 20 mg/mL of hyperosmolar dextrose guided by ultrasound, finding a mean reduction in the pain 10-point Visual Analog Scale (VAS) at follow-up of 5.9 and 4.1 points, respectively.^{16,17}

Extracorporeal shock therapy is another intervention for treating Achilles tendinopathy that provides low-to-medium energy to the tendon. Evidence reports decreased subjective pain (by a mean of 4.8 points on the VAS pain scale in one study) and high patient satisfaction.¹⁴

Additionally, shockwave therapy provided better outcomes when compared to eccentric loading for insertional tendinopathy.¹⁸ Furthermore, manual therapy for Achilles tendinopathy can include both tendon mobilizations and transverse friction massage to the area.⁷ However, current evidence does not support the use of kinesiotaping, corticosteroid injections, or platelet rich plasma injections for the treatment of Achilles tendinopathy.⁷

Plantar Fasciitis

According to evidence, 1 out of 10 people will experience plantar fasciitis his or her lifetime, making it the most common cause of plantar heel pain and the most treated foot condition.^{2,7} Plantar fasciitis is a chronic, overuse injury with symptoms often presenting longer than a year before an individual decides to seek treatment.² The associated “throbbing” pain often presents medially near the calcaneal tuberosity on the plantar surface of the foot.⁷ It is also described as being the most intense during the first initial steps following a prolonged duration of weight-bearing inactivity.⁷ However, this pain tends to lessen with additional ambulation.⁷ The risk of developing plantar fasciitis increases with higher body mass index (BMI), running on rigid surfaces or in spiked footwear, prolonged standing (especially on rigid surfaces), excessive pronation, and limitations in dorsiflexion range of motion.² Obesity is an independent risk factor that is modifiable and prevalent in 70% of patients with plantar fasciitis.⁴ Furthermore, the existence of skeletal malalignment and deformities, such as cavus foot, hindfoot varus, high arch height, and lower medial longitudinal arch height, also increase the risk of developing plantar fasciitis.² Additionally, research suggests positive associations with decreased hamstring flexibility and leg-length discrepancies, which produces plantar fascial pain in the foot of the longer limb.^{19,20} One prospective longitudinal study found that individuals with tight hamstring muscles were 8.7 times more likely to acquire plantar fasciitis due to a “functional biomechanical deficit” created by premature contraction of the posterior muscles resulting in limitations in ankle dorsiflexion.¹⁹ As previously referenced in risk factors, dorsiflexion restrictions places increased tension on the plantar fascia and can ultimately lead to chronic inflammation.¹⁹

Diagnosis of plantar fasciitis should include an assessment of subjective history, an objective evaluation that includes observation and physical examination, appropriate tests, and

diagnostic imaging.² A subjective interview of the patient should reveal the pain characteristics previously mentioned.² Therapists should also inquire about the physical activity habits, work conditions, and footwear that may exacerbate this condition.² Physical examination may reveal painful palpation at the proximal insertion site of the plantar fascia or on the medial plantar surface of the heel, pain or range of motion limitations with dorsiflexion, a positive windlass test, negative tarsal tunnel test, and an abnormal Foot Posture Index score.² The *Foot Posture Index* (FPI-6) is an assessment tool that quantifies the degree of pronation, supination, or neutrally positioned in a positive, negative, or zero value, respectively.²¹ For example, one case-control study found a mean score of 2.4 ± 3.3 in individuals with chronic heel pain, compared to 1.1 ± 2.3 in individuals without chronic heel pain, indicating a more pronated foot.²² The *tarsal tunnel test*, or dorsiflexion-eversion-test, passively places the ankle into dorsiflexion then eversion while extending the metatarsophalangeal (MTP) joints in order to rule out Tarsal Tunnel Syndrome.²³ Conversely, the *Windlass test* is used to rule in plantar fasciitis by passively extending the MTP joints while keeping the ankle neutrally positioned in 90 degrees.²³ Both the tarsal tunnel test and windlass test are considered positive if pain is reproduced in the plantar heel of the patient.²³ The Windlass test elicits the windlass mechanism, which refers to the tightening of the plantar fascia during dorsiflexion of the toes, thus reducing the distance between the metatarsals and calcaneus while placing the medial longitudinal arch in an elevated position.⁴ This mechanism represents the dynamic function of the plantar fascia during weightbearing phases gait.⁴

The American Physical Therapy Association (APTA) published a Clinical Practice Guideline (CPG) that recommends the following interventions with strong evidence for treating plantar fasciitis: manual therapy, stretching, taping, foot orthoses, and night splints.² According

to the CPG, manual therapy can include “talocrural joint posterior glide, subtalar joint lateral glide, anterior and posterior glides of the first tarsometatarsal joint, subtalar joint distraction manipulation, soft tissue mobilization near potential nerve entrapment sites, and passive neural mobilization procedures.”² Taping methods that are most beneficial in treating plantar fasciitis include low-dye taping and calcaneal taping that limits pronation.² A stretching program should include the calf, plantar fascia, and hamstring muscles sustained for a total of 3 minutes and performed 2 or 3 times per day.² Foot orthoses that provide support to the medial longitudinal arch and cushion the heel are also recommended for minimizing pain and increasing function; however, one study found that prolonged use may cause impaired function of the plantar muscles.^{2,24} Lastly, 1 to 3 months of night splint use can be helpful mitigating the pain experienced with the initial steps of ambulation in the morning.² However, there is low-level evidence in support of low-level laser therapy, phonophoresis, ultrasound, rocker bottom shoes, and rotating footwear during the workweek for addressing plantar fasciitis.²

Experts suggest that strengthening exercises of the lower leg should also be implemented in addition to movement training to reduce excessive pronation.² Furthermore, physical therapists are also encouraged to provide patient education on weight loss or to provide referrals for nutritional assistance to an appropriate health professional.² Although corticosteroid injections can provide immediate pain relief, its use can have deleterious effects on the plantar fascia and the fat pad, leading to rupture or atrophy, respectively.⁷ When a patient is unresponsive to treatment, extracorporeal shockwave therapy has also provided good results.⁶ Lastly, two surgical options are reported in literature for plantar fasciitis management—plantar fasciotomy, which directly releases tension of the plantar fascia by dissecting a portion of the

tissue, and gastrocnemius release, which indirectly releases tension in a similar method.⁶ The latter method is indicated for individuals with equinus.⁶

Clinical Implications

Physical therapists can be useful in diagnosing heel pain and providing conservative options for treatment, but this requires obtaining a thorough patient history and performing the components of physical examination mentioned in this paper. For the sedentary individual, it is important to provide patient education on the importance of progressive physical activity following periods of inactivity, wearing shoe wear that provides adequate support for their unique movement patterns and skeletal alignments, reducing body mass index, and avoiding rigid surfaces in order to minimize the risk of heel pain. It is also imperative to assess for factors associated with chronic heel pain such as alterations of rearfoot, midfoot, or forefoot alignment as a result of compensated strategies to offload the heel during ambulation,²⁵ (psychosocial conditions (e.g. depression, anxiety, stress, kinesiophobia, and catastrophizing),²⁶ and impaired function of plantar flexor muscles with prolonged use of foot orthoses.²⁴ However, it is of utmost importance to consider, address, and provide the appropriate management for underlying conditions that can exacerbate heel pain or cause it reoccur.

APPENDIX A- Anatomical Review of the Heel

Figure 1: Pathoanatomy and common sources of heel pain

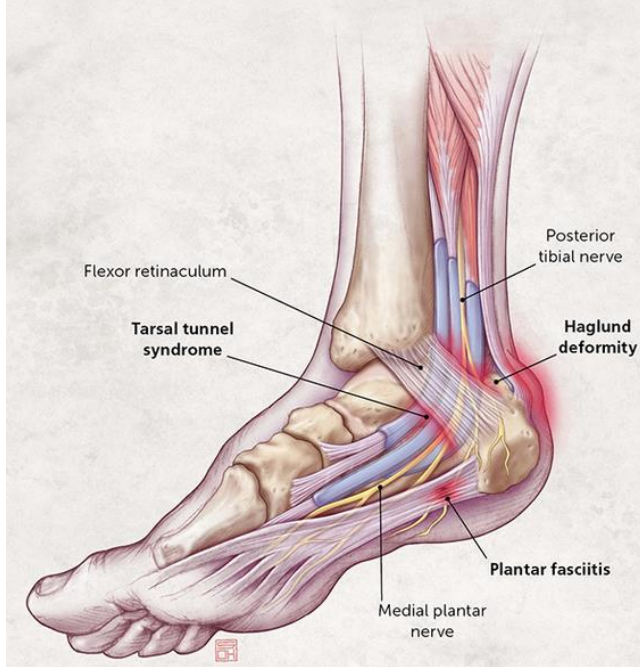
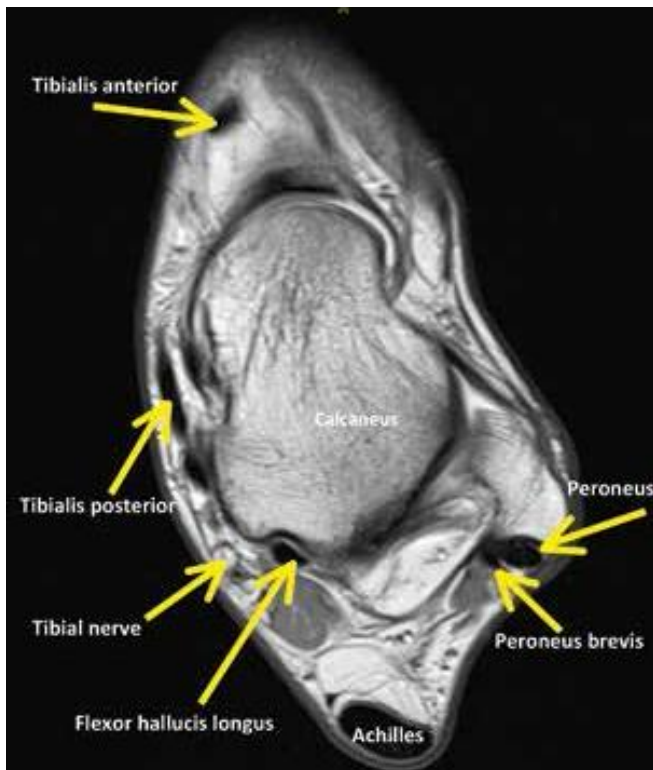


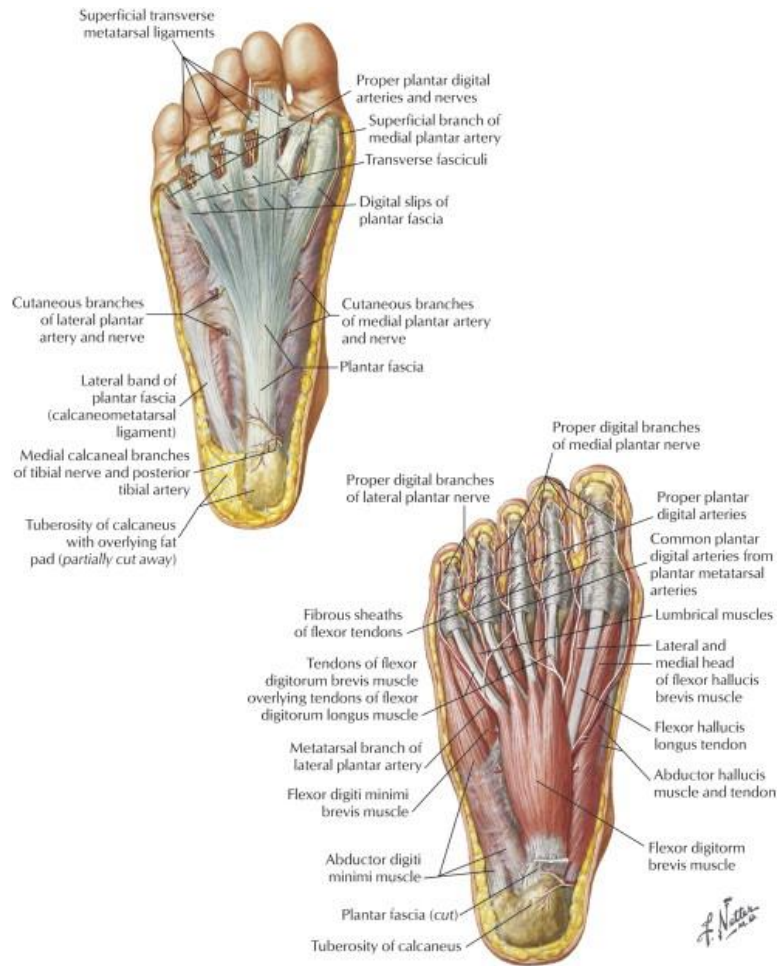
Illustration by Steve OH. Reprinted from Tu P. Heel pain: diagnosis and management. *Am. Fam. Physician* 2018;97(2):86-93.

Figure 2: Neuroal and tendinous structures surrounding the calcaneus.



Reprinted from Rio E, Mayes S, Cook J. Heel pain: a practical approach. *Aust Fam Physician* 2015;44(3):96-101.

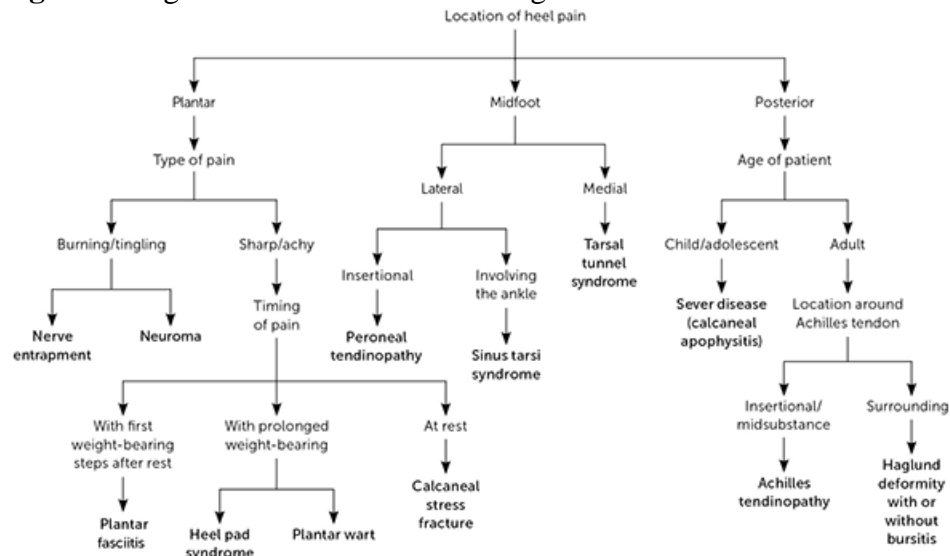
Figure 3: Plantar Fascia and Hindfoot Anatomy



Reprinted from Rosenbaum AJ, DiPreata JA, Misener D. Plantar heel pain. *Med Clin North Am* 2014;98(2):339-352. doi:10.1016/j.mcna.2013.10.009.

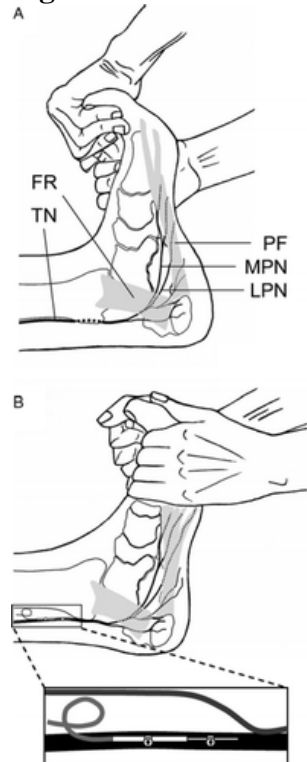
APPENDIX B—Diagnosing Heel Pain

Figure 1: Algorithm for Differential Diagnosis of Heel Pain Based on Location



Reprinted from Tu P. Heel pain: diagnosis and management. *Am. Fam. Physician* 2018;97(2):86-93.

Figure 2: Tarsal Tunnel (A) and Windlass Test (B)



Tarsal Tunnel Test (A)

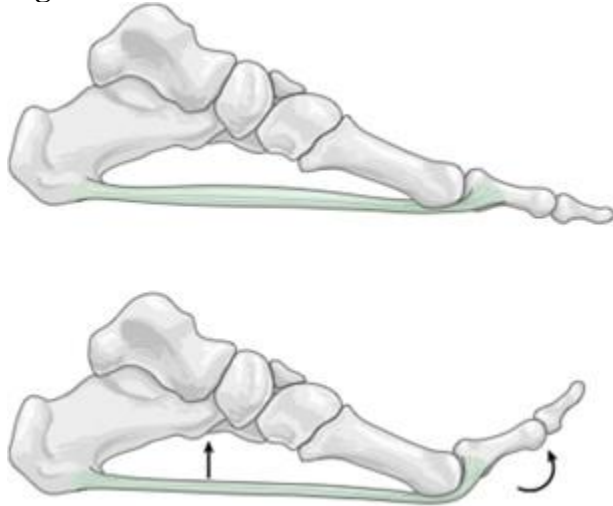
The *tarsal tunnel test*, or dorsiflexion-eversion-test, passively places the ankle into dorsiflexion then eversion while extending the metatarsophalangeal (MTP) joints in order to rule out Tarsal Tunnel Syndrome.

Windlass Test (B)

Windlass test is used to rule in plantar fasciitis by passively extending the MTP joints while keeping the ankle neutrally positioned in 90 degrees.²³ Both the tarsal tunnel test and windlass test are considered positive if pain is reproduced in the plantar heel of the patient.²³

Reprinted from Alshami AM, Babri AS, Souvlis T, Coppieters MW. Biomechanical evaluation of two clinical tests for plantar heel pain: the dorsiflexion-eversion test for tarsal tunnel syndrome and the windlass test for plantar fasciitis. *Foot Ankle Int.* 2007;28(4):499-505. doi:10.3113/FAI.2007.0499.

Figure 3: Windlass Mechanism



The Windlass test elicits the windlass mechanism, which refers to the tightening of the plantar fascia during dorsiflexion of the toes, thus reducing the distance between the metatarsals and calcaneus while placing the medial longitudinal arch in an elevated position.⁴ This mechanism represents the dynamic function of the plantar fascia during weightbearing phases gait.⁴

Reprinted from: *From* Greisberg J. Foot and ankle anatomy and biomechanics. In: DiGiovanni CW, Greisberg J, editors. Core knowledge in orthopedics: foot and ankle. Philadelphia: Elsevier; 2007

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