PLANTAR FASCIITIS

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Wood in 1812 first described plantar fasciitis and attributed it to tuberculosis. Plantar fasciitis has been called the painful heel syndrome, subcalcaneal bursitis, subcalcaneal pain, medial arch sprain, stone bruise, calcaneal periostitis, neuritis, subcalcaneal spurs, and calcaneodynia.1-3 In this article we offer a means for serial specific evaluations and treatment (Fig 1).

Plantar fasciitis may present as a single entity or as a “final common pathway” due to other disorders.4-6 These other disorders may be due to overuse, biomechanical derangement of the foot, nerve entrapment, inflammatory arthritis, or even stress fractures.1,4-7,22-25 Snook and Chrisman2 stated, “it is reasonably certain that a condition which has so many different theories of etiology and treatment does not have valid proof of any one cause.” Since the symptoms are often similar, symptomatic therapy may often be successful.

The patient’s response to therapy may point to the etiology. In this case, the therapy is a provocative test. The goal of management is to decrease inflammation, to increase function, and to determine etiologic factors, which are usually mechanical. Most patients respond to nonoperative management.3,4,10,12

The concept of subcalcaneal pain (Fig 2) was introduced by Snook and Chrisman4 and then further explained by Bordelon.5 The inflammation may arise in the plantar fascia itself or it may be secondary to inflammation in that anatomic area. The inciting inflammation may be local or systemic. A complete list of these factors is given in Table 1.4,5,9,22,23 Subcalcaneal pain is best thought of as a syndrome that may involve the plantar fascia.5 Classically, the medical treatment of syndromes does not rely on the exact etiology; rather, it relies on the treatment of symptoms.

EPIDEMIOLOGY

In athletes, plantar fasciitis is more common in sports that involve running.10,12,24 The incidence in these sports is about 10%.10,18,25 Plantar fasciitis has been noted in the lowest level of running24 and in high mileage groups.18 Plantar fasciitis has also been frequently noted in dancers, tennis players, and basketball players.1,16,26,27

Plantar fasciitis also occurs in non-athletes in roughly the same percentage as athletes.28 Occupations requiring prolonged weight bearing have been associated with subcalcaneal pain.12

Plantar fasciitis has been said to predominate in males,5,12,18 but this may be more of a function of occupation (eg, laborers) and recreational activity (eg, long distance running). In
**Fig 1: Evaluation Based Rehabilitation.** Reprinted with permission from J.B. Lippincott Company.

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**Fig 2: Subcalcaneal pain syndrome.**

Furey's series of 103 patients without systemic disease, there was only a 55% male predominance. Two other large series also show about equal sex distribution.

Plantar fasciitis has been reported in patients from 7 to 85 years old. The majority of patients are over 40 years old and the occurrence may be related to atrophy of the fat pad.

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**Anatomy**

The plantar fascia is a tough, longitudinal fibrous connective tissue structure which originates on the medial tuberosity of the calcaneus. It has three parts: medial, lateral, and central. The largest is the central portion (Fig 3). The plantar fascia divides into five bands at the mid-foot level and attaches to the proximal phalanges. The muscles of the first layer (abductor hallucis, flexor digitorum brevis, and abductor digiti quinti) and the quadratus plantae all arise from the calcaneal tuberosity.

The medial calcaneal nerve supplies sensation to the medial heel. It then branches off to become the medial and lateral planter nerves. The tibial nerve or the medial calcaneal nerve may be compressed by the flexor retinaculum. The medial plantar nerve travels between the abductor hallucis and flexor digitorum brevis. The lateral plantar nerve passes between the quadratus plantae and flexor digitorum brevis.

The nerve to the abductor digiti minimi may also be compressed by the intrinsic muscles.

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**Pathomechanics**

The plantar fascia links the tarsal bones and ligaments of the foot and acts as a truss. It elongates with increasing loads to act as a shock absorber, but its ability to lengthen is limited.

The plantar fascia stiffens with increasing tension. When the metatarsophalangeal joints are extended passively, the plantar fascia is pulled...
distally, and the base of the truss gets shorter while the height of the arch increases. The process shortens the distance between the hind-foot and the forefoot and locks the tarsal joints into a flexed position. Plantar flexion of the toes relaxes the fascia and slightly lengthens the arch. Loss of the action of the plantar fascia results in a flatter foot.

The extrinsic muscles control the foot dynamically through the subtalar joint. The posterior tibialis is the most important muscle in control of the medial stability of the foot; loss results in collapse of the arch. The intrinsic muscles help provide dynamic stability of the foot during loading by the windlass mechanism. Planter flexor strength and flexibility deficits for dorsiflexion in the muscles of the posterior calf and foot have been documented. Tight posterior structures cause heel valgus at foot strike and weak plantar flexors increase load on the muscles and plantar fascia.

Pronation of the subtalar joint everts the calcaneus and lengthens the plantar fascia, increasing its tension. The pronated foot requires greater intrinsic muscle activity to stabilize the transverse tarsal and subtalar joints than does the normal foot.

The most common injury pattern to the plantar fascia is currently felt to be overload. As the speed of gait increases, the ground reaction force greatly increases while the period in stance phase greatly decreases predisposing to overload. Loading may increase to 200% body weight with jogging and running. Excessive pronation and overload appear to be common causes of overload in the general population. In runners, excessive pronation and training errors are common. A 15% incidence of plantar fascitis in 72 runners with pronation accounted for 58% in one study. Overload may cause traction spurs or stress fractures.

HISTOLOGY

Microtears and partial rupture in the plantar fascia near or at its insertion have been noted in patients with chronic plantar fascia. Biopsies showed collagen degeneration and necrosis, angiofiblastic hyperplasia, mucinoid degeneration, chondroid metaplasia, and matrix calcification. These findings are consistent with fatigue failure, inadequate healing and repair, and chronic inflammation.

COMMENTS

A. Pain at the Medial Aspect of the Calcaneus: Plantar fascitis is a clinically defined syndrome characterized by pain at the attachment of the plantar fascia to the medial tubercle of the os calcis. The pain may radiate to the medial side of the foot and medial malleolus and even distally on the sole of the foot.

B. Symptoms Worse with Rising in the Morning or Activity: A common complaint is pain in the morning. Fury found that 83.5% of his 116 patients had their worst pain on rising in the morning. The most severe pain in the
morning occurs with the first 50-100 steps and then decreases for ordinary walking.1 It is a distinctive symptom of the disorder.4,15,18,30 Occasionally, patients may have such severe pain that it may occur even when non-weight bearing.38

C. Confirm Fascial Origin of Pain: Typically, the patient complains of pain under the heel, worse on the medial side.1,4,5,7,12,13,36 The onset is usually gradual and there is usually no history of trauma.1,4,6,12 It decreases but later increases during the day.1,8 The condition is not usually disabling but may severely limit weight bearing activities, particularly high intensity ones such as running and jumping. Patients may have jobs or recreational activities that involve prolonged weight bearing. A limp may be present and athletes may notice a change in stride pattern or poor performance.1 The pain may be worse when the area is cold or contracted.7

The pain is usually unilateral; bilateral in 4% to 30%, 1,6,12,14,29

Bilateral heel pain in patients with plantar fasciitis has been reported in 4% to 30%.6,12,14,29

The pathognomonic feature is tenderness to palpation of the plantar fascia insertion onto the medial tubercle of the calcaneus.1,3,7,12,15,18

Other signs to note include other areas of tenderness, heel pad atrophy, crepitus, and pain associated with stretching the plantar fascia. Alignment and gait are observed with emphasis on foot and ankle alignment, leg length discrepancy, heel strike, and pushoff.1,9,10,12,13,18,36,43 Pes planus was diagnosed in 22%.6 A brief neuromuscular examination and inspection of the skin should be documented. Generalized burning pain may indicate nerve entrapment. One study of 30 patients with plantar fasciitis included seven patients (23%) with chronic low back pain and sciatica.14

Skin changes and bilaterality may alert the examiner to systemic disease. Other significant peripheral joint problems were diagnosed in 22.3%.6

Acute rupture of the plantar fascia44,45 presents somewhat differently and is uncommon. Patients complain of acute pain after intense running, jumping, or accelerating. There may be a history of multiple steroid injections.44,45 When localized swelling and acute tenderness subside, a defect in the plantar fascia may be palpated. This was replaced by a firm mass in two of four patients.44 Patients usually resume full activity after 3 to 4 weeks.45

Chronic plantar fasciitis with fibrotic nodules also has a slightly different presentation.13,15,44 The history is protracted and there may have been an acute episode of pain corresponding to rupture.44 There may be no history of steroid injections.15 Patients may have a history of high impact sports participation.13,44 The nodules are firm and tender.15 There may be palpable tenderness of the abductor hallucis.13 The incidence is not known. It is unassociated with systemic disease. With plantar fibromas, nodules are not painful.

D. Contributing Factors: We classify contributing factors of plantar fasciitis as structural (including overload states), pain syndromes (including systemic disease and nerve compression syndromes), or both.4 Structural abnormalities of the foot may predispose it to overload states. Overload may occur in the presence of a normal foot, particularly if the patient runs extremely high mileage or is training incorrectly.10 A minor structural abnormality may be magnified by high mileage, resulting in "accumulated impact loading."10 Pain syndromes may involve the plantar fascia secondarily. For example, a patient who initially has entrapment of the medial calcaneal nerve may develop inflammation of the plantar fascia.

Factors responsible for fatigue failure encompass anatomic, biomechanical, and environmental factors.7 Cyclic loading (long distance running) is the most common mode of failure, such as in long distance running. Failure may occur due to high demand on normal tissues or normal demand on abnormal tissues. Some factors that increase demand include structural abnormalities in the foot, such as pronation, pes planus, pes valgus, pes cavus, increased body weight, tight Achilles tendon or limited dorsiflexion, and leg length inequality.3,6,10,12,17,29,30 Cavus feet are relatively rigid and put more stress on the loaded plantar fascia. The pronated foot also increases the demand on the intrinsic muscles of the foot.37 Kwong et al9 have shown an increase in the tensile stress on plantar fascia insertion in the pronated foot. This is in part due to eversion of the calcaneus in the pronated foot.37 Local or systemic disease can cause inflammation of the plantar fascia which may weaken the fascia.

Of 43 competitive or recreational athletes with unilateral symptomatic plantar fasciitis, 38 lower extremities had deficits in ROM.7 With the knee in extension, the most common motion abnormality was lack of dorsiflexion 5° beyond neutral on the affected side or lack of 10° dorsiflexion compared to the nonaffected side.7 The only significant factor in one study was decreased plantar flexion ROM.11 The plantar fasciitis group had more ROM in plantar flexion. A tight gastrosoleus may cause increased compensatory pronation.42

Several studies have noted the association
between being overweight and painful heels. One study examined 77 consecutive patients with planter heel pain. Using sex and age specific tables, 28 of 36 men (77.8%) and 36 of 44 women (81.8%) had weights above the 50th percentile. Twelve patients (15.6%) had bilateral involvement, and 10 of these had weights above the age-specific 50th percentile. No other specific etiologic factor could be identified. Of the 11 patients who failed non-operative management and required plantar fascial release and/or spur resection, 10 had an age-specific weight more than the 50th percentile. Other studies show a somewhat lower proportion of overweight, ranging from 28% to 60%.

Prediction of runners predisposed to plantar fasciitis based on running history, anatomy, running analysis, and leg length discrepancy has not been very successful. Leg length inequality of 5 mm to 25 mm was not a predisposing factor.

In the older population, plantar fasciitis is felt to be more common due to the atrophy of the heel fat pad, which degenerates with age. The above factors are additive. Gradual progression from a normal plantar fascia to a symptomatically abnormal anatomical, physiological, and biomechanical configuration results in symptomatic plantar fasciitis.

E. Radiographs and Diagnostic Imaging: A weight bearing radiograph of the foot in the anteroposterior and lateral projections are obtained to rule out fracture, periosteal elevation, spurs, and degenerative joint disease. Oblique views (particularly a 45° medial oblique view) and calcaneal views are helpful on an individual basis and should be considered if history points to a stress fracture. Bone spurs are associated with plantar fasciitis but are not felt to cause it. Many studies now show no clear association between spurs and plantar fasciitis. Of the studies on patients with plantar fasciitis, 10% to 70% of the patients have an associated ipsilateral calcaneal spur. However, most of these studies showed spurs on the contralateral asymptomatic foot. One epidemiologic study found an 11% incidence of heel spurs in adults. Heel spurs may be symptomatic when associated with atrophy of the heel pad. A higher incidence of spurs has been noted among the obese patients, especially in the 50th or greater percentile. In the only prospective study investigating heel pain, 70% of patients (21 of 30) had a spur. Of the 13 patients with bilateral spurs, six had bilateral symptoms. Bone scans are indicated in patients who present with a history suggestive of stress fractures (bilateral pain of acute onset after a fall or marked increase in activity) or metabolic bone disease. For those patients who fail to respond to therapy after 6 months, a bone scan should be ordered. Stated that about 5% to 10% of patients will require bone scans. In the absence of calcaneal stress or other fractures, the bone scan exhibits minimally to moderately increased activity on the early blood pool images.

F. Rule Out Systemic Disease or Nerve Entrapment Syndromes: Patients with idiopathic heel pain should be evaluated for inflammatory disease associated with plantar fasciitis, including seronegative and seropositive arthritides and sarcoidosis. The foot is second to the knee as the site of initial presentation in rheumatoid arthritis. In addition, signs of rheumatoid arthritis on radiographs may precede those in the hand. Retrocalcaneal bursitis is the most common lesion of rheumatoid arthritis at the heel and may present with ankylosing spondylitis and gout. Men under 40 years old presenting with bilateral heel pain should be evaluated for Reiter's syndrome and ankylosing spondylitis. Gout can also cause inflammation of the plantar fascia. In a study of 116 patients, 16.4% had an inflammatory arthritis (10 with rheumatoid arthritis, 4 with ankylosing spondylitis, and 2 with gout). Rule out these disorders if the signs and symptoms present initially or if the patient fails management after 6 to 12 months (Table 2).

Tarsal tunnel syndrome is more common in women. Entrapment neuropathies of the lower extremity are more commonly diagnosed, particularly in diabetics. Tenderness at the medial tuberosity of the calcaneus may be absent and decreased sensation may be noted. A work-up for diabetes should be considered in patients with tarsal tunnel.

G. Therapeutic Modalities: Modalities such as ice and EMS (electro muscle stimulation) are used to relieve pain and inflammation. If the patient complains of acute pain and has acute tenderness, initial treatment with cryotherapy is more successful than heat modalities. Iontophoresis may reduce pain and inflammation associated with plantar fasciitis. This technique uses direct current to drive heavy metal ions through the skin. Deep friction massage has been suggested to help reduce scar tissue formation.

H. Non-Steroidal Antiinflammatory Drugs: Non-steroidal antiinflammatory agents (NSAIDs) may be prescribed to control pain. No one NSAID is more effective than another. NSAIDs are prescribed for 2 to 3 weeks in acute cases.
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I. Orthotics and Shoe Modification: The use of orthotics in treatment of plantar fasciitis is associated with positive results.1,3,10,22 The primary goal of the orthotic device is to restore normal biomechanics during support. An orthotic may prevent excessive pronation32 or maintain the supinated foot in neutral at heel strike6 by reorientation of the heel. In addition, pronation begins later and ends sooner and is associated with delayed dorsiflexion in this study.32 Orthotics also support the foot, relieve pressure, cushion impact, and elevate the arch.4,5,23 Shoes may be modified depending on the injury and the foot alignment. However, the rigid cavus foot and its associated injuries are usually not corrected by orthotics.10,24 A heel wedge between the inner sole and midsole of the shoe for plantar fasciitis and Achilles tendinosis is indicated. The goal of the wedge is to increase cushioning and relax the plantar fascia and Achilles tendon at heel strike for the rigid cavus foot.24 An 8 mm heel was measured to decrease the period and amount of maximum pronation.32

The rigid orthotic is made of hard plastic. Some authors believe that rigid orthotics are more durable and provide increased foot control.23 This type of orthotic may be more beneficial for high mileage runners.10 A plastic heel cap is prescribed for the patient’s shoes.4 We have found that for the recreational runner, the soft or semi-rigid orthotic has been most effective. The disadvantage is the decreased life span of the softer orthotic material used. Orthotics may be discontinued after the acute phase of plantar fasciitis, but many runners prefer to continue utilizing the orthotic device (Table 3). Custom-fit rigid and soft devices are available for patients with severe malalignment. While low dye taping is another means to decrease tension on the plantar fascia,6,53,54 our concern is that tape loosens.

Advise runners to train on relatively soft surfaces, like grass, to avoid high impact loads across the healing plantar fascia.10 A training shoe should meet the following criteria: 1) the heel counter should be very firm to control the hindfoot and have a well molded Achilles pad; 2) a beveled and flared heel may help provide heel stability; 3) a soft cushion with the heel elevated some 12 mm to 15 mm higher than the sole gives additional shock absorption; 4) the sole of the shoe under the forefoot should have a substantial midsole cushion, but should be flexible over the metatarsal heads.10,24 Newer models have improved shock absorption, rear foot control, and durability.24 There has also been a trend to provide support in midstance and flexibility at toe-off.24

J. Activity Modification: Total rest is necessary in some cases, usually with a duration of 16 weeks or longer.18 Reducing activity is usually required.1,10,22 A timetable should be outlined for each individual. James et al10 recommend a 6 week timetable starting with jogging a 7½ to 8 minute per mile pace 15 minutes a day the first week. For each week thereafter, 5 minutes are
Fig 4: Cylinder Roll: Place a strong cylinder (bottle, can, weight) under the foot. The patient slowly rolls the cylinder back and forth to stretch the arch.

Fig 5: Towel Curls: The patient sits with the feet on top of a towel. The patient curls his toes to gather the width of the towel under the arch.

Fig 6: Pick-ups: Place some pencils, pens, or marbles on the floor. The patient picks up these objects and then drops them in a different spot.

added to the daily schedule until 40 minutes of painless nonstop running is achieved. If the individual is progressing satisfactorily, return to a training regimen is allowed. Other modifications include decreasing velocity, shortening stride length, and decreasing heel contact.22

K. Weight Reduction: A decrease in loads that the foot supports will decrease the tension developed in the plantar fascia. Percent of body fat measurements more accurately correlate with fitness level than body weight. Recommendations regarding diet and proper exercise in those subjects with a higher percent of body fat for their age and height can be outlined. Obese patients have been noted to respond poorly to nonoperative treatment.6,29

L. Achilles Stretching and Plantar Fascia Stretching: To restore normal foot mechanics, stretching of the Achilles tendon by progressive dorsiflexion exercises and stretching of the plantar fascia is indicated.1,22,29 The plantar fascia may become fibrotic in chronic cases of plantar fasciitis. We recommend stretching for up to 8 weeks before considering night splints or a short leg walking cast. Stretching should be focused on the hamstrings, gastrosoleus, and plantar fascia.10,22 Avoid ballistic stretching.10 The Achilles tendon may be stretched on a slant box. Several other stretching exercises are shown in Figures 4-6. We prescribe these for 5 to 10 minutes, 3 to 4 times a day for 6 to 12 weeks.16,22

One study reported the peak torque in a group of 43 competitive or recreational athletes with unilateral symptomatic plantar fasciitis and 45 age and sex matched asymptomatic controls. The contralateral foot of the symptomatic athletes was also tested. Of the 43 feet with plantar fasciitis, 41 were found to have peak torque deficits in plantar flexor muscles compared to both the contralateral foot and controls. For plantar flexion, 37 of the 43 had deficits at only 180°/sec, while dorsiflexion was normal at 60 and 180°/sec. The 6 feet that did not have a
deficit of plantar flexion had navicular drop. Stretching has been shown to increase the strength of the musculotendinous junction. M. Maintaining Fitness and Cardiovascular Conditioning: Any activity that does not impact load the lower extremity and is designed to maintain or increase aerobic condition is indicated. Swimming, running in the water, and pool aerobic exercise are particularly good for patients with severe symptoms or for those who are obese. Patients who can tolerate some weight bearing may use cross country ski machines, bicycles, or stair machines. When the patient has not yet started the running program, we recommend three to four 20 to 30 minute sessions per week to maintain cardiovascular fitness. As the patient runs or jogs more, we decrease the fitness training by a similar amount.

N. Steroid Injection: Cortisone injections with a long acting anesthetic can be a helpful diagnostic and therapeutic maneuver. On occasion, a patient with severe acute, localized tenderness may benefit from an injection on presentation. If the patient has not responded to the initial management of NSAIDs, orthotics, and limited activity after 2 to 4 weeks, consider an injection. A maximum of three injections given once weekly, over 2 to 4 weeks, may be helpful. More injections may cause necrosis and atrophy of the fat pad. The effect of numerous corticosteroid injections on the plantar fascia is probably detrimental. Of Leach’s report, six reported patients with rupture of the acute plantar fascia, two had multiple injections and three had a history of steroid injections. Of the histologic specimens of chronic plantar fasciitis after steroid injection, chronic inflammation and necrosis have been documented. As Leach et al point out, the physician must weigh the advantages of the short-term effectiveness of a steroid injection against the potentially detrimental long-term sequelae, such as fat pad atrophy and weakening of the plantar fascia.

Pain should be localized and the injection given there. Injection into the medial tuberosity of the calcaneus showed a decrease of the symptoms associated with plantar fasciitis. Other sites of point tenderness may be differentially injected.

O. Ice Massage: Ice massage should be used for 5 to 10 minutes prior to therapy sessions to decrease inflammation. While there is no experimental data to support the use of ice in the presence of chronic inflammation, it is used by virtue of its anesthetic effect. Also, it is an adjunct to therapeutic exercise to improve mobilization by decreasing muscle spasm. Interestingly, ice increases collagen stiffness. However, this does not seem to be a predominant effect in our patients with plantar fasciitis who report improved flexibility after ice massage. Others report good results.

P. Taper NSAID: Once the patient’s pain and inflammation are controlled and therapeutic exercises begin, we taper the NSAID. Our goal is to have the patient completely off NSAIDs before full return to activity.

Q. Maintenance Program: Once the patient’s symptoms have resolved, we prescribe a stretching program at least three times a week. The patient stretches both the plantar fascia and gastrosoleus tendons. This is particularly important between seasons to prevent recurrence. We do not have any data on the effectiveness of the stretching program in prevention of recurrence. However, our opinion is that it is empirically justified.

R. Gradual Return to Activity: Symptoms should guide the patient as he or she resumes impact activity.

S. Medical Workup: Careful consideration should be given to the diagnosis of inflammatory arthritis in a patient with idiopathic heel pain who does not respond to conservative management. This is particularly true for the patient who is compliant with rest and stretching but has persistent or worsening symptoms. The vast majority improve with nonoperative therapy regardless of etiology. Specific laboratory tests are directed toward the suspected etiology. The erythrocyte sedimentation rate is a good screening test. A bone scan may be helpful when a course of rehabilitation has failed to document increased uptake at the medial calcaneus and to rule out other pathology such as stress fracture, osteoarthritis, or inflammatory arthritis.

T. Night Splint Short Leg Walking Cast: A night splint with the foot in neutral or slight dorsiflexion is indicated when rehabilitative measures have failed. The splint maintains the plantar fascia in a lengthened position overnight. Minimizing or preventing contracture of the plantar fascia is a goal of treatment. Such splints are commercially available or may be made from Orthoplast or casting material. For patients with persistent and severe symptoms unresponsive to other therapy, a short leg walking cast with the ankle and foot in neutral or slight dorsiflexion is a treatment option. The cast should immobilize the foot with the plantar fascia in a lengthened position. The cast acts as a load sharing device and unloads the plantar fascia. The goal is to allow healing of the plantar fascia and to decrease the inflammation. Anecdotal reports warrant this treatment as an option before surgery.

U. Steroid Injection: If the patient has not yet
had an injection and continues to have symptoms, an injection may be considered. For patients who have had a prior injection, a repeat injection may be indicated. We inject for a total of three times. If there are signs of fat pad atrophy, our opinion is that repeat injection is contraindicated.

V. Consideration of Surgery: When all conservative therapies have failed, surgery is considered. Release of the plantar fascia is indicated in only a small percentage of patients. Lapidus and Guidotti treated 323 patients with 364 painful heels and operated on none. In one study of 182 patients with sport activity related subcalcaneal pain, only four required surgery. The consensus of opinion regarding surgery is as follows. Surgery is considered after 6 to 12 months of non-operative management. In an elite athlete, it may be considered sooner. Surgical management must be individualized and directed toward the structures causing pain. A patient with systemic disease should first have therapy directed toward this disease before fascial release is considered. We further recommend limited procedures (those that do not compromise the windlass mechanism) first. Diagnosis must be determined prior to surgery (Fig 7). Numerous procedures have been described including a limited release, a wide release (Steindler type), and a wide release combined with release of the nerve to the adductor digiti quinti and excision of the heel spur. A more extensive release includes release of the plantar...
fascia, a tarsal tunnel decompression, excision of the heel spur, and release of the nerve to the abductor digiti quinti. All procedures release part or all of the plantar fascia.

Limited release may be done under local block. It consists of releasing only the medial aspect of the plantar fascia and does not prevent a wider release at a later date. Incising the plantar fascia at its insertion alone can give good results. Snider et al. performed 11 releases in nine long distance runners whose symptoms had been present for an average of 20 months. Ten runners had excellent results and one had a good result. Eight of nine patients returned to full training at 4.5 months.

A Steinbender type release may be combined with excision of the heel spur and release of the nerve to the abductor digiti quinti. Leach et al. suggest that the medial calcaneal nerve should be identified, since it may be involved and resects all chronic granulomatous tissue along with a portion of the os calcis and bone spurs. Fourteen of 15 patients had complete success with return to previous level of activity. Most patients continued to improve for 6 months postoperatively. Stripping of the plantar fascia and the superficial plantar muscles from the calcaneus has been recommended. We do not recommend this routinely because it may destabilize the windlass mechanism particularly in the supple pronated foot. If the findings are consistent with nerve entrapment, that nerve should be released. EMGs may be helpful preoperatively. The medial calcaneal nerve may be resected or released. Release is preferred since resection results in numbness. The posterior tibial nerve, calcaneal nerve, and nerve to the abductor digiti quinti may also be released individually or together.

REFERENCES


