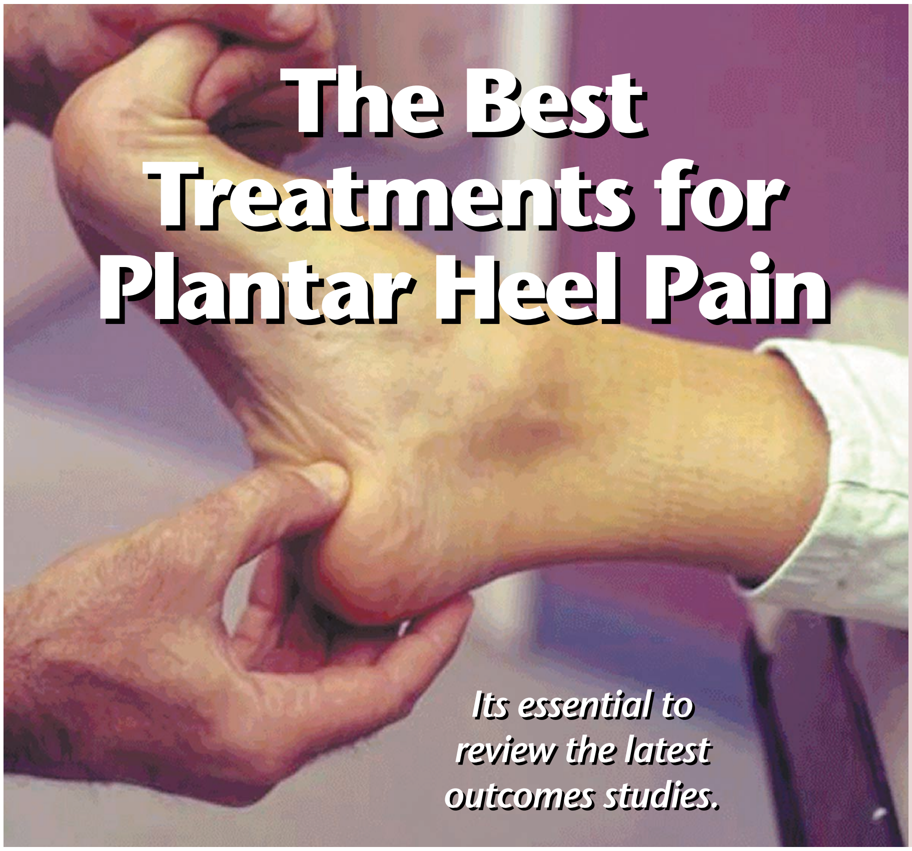
The Best Treatments for Plantar Heel Pain



Objectives

1) Understand the pathway to wound healing through the three phases of inflammatory, proliferative, and remodeling.

2) Introduce the reader to the concerns of bacterial burden, hydration, and nutrition in wound healing.

3) Better comprehend the inter-related biology of wounds and bacterial contamination.

4) Comprehend the relationship between wound physiology and healing potential.

5) Further the reader's understanding of comprehensive wound management.

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*Following this article, an answer sheet and full set of instructions are provided (p. 152).—**Editor***

Douglas H. Richie Jr. D.P.M.

Introduction

Few conditions affecting the human foot have stimulated more interest and controversy among health care professionals than plantar heel pain syndrome. Over the past decade, new surgical approaches have been popularized, approaches which have fueled considerable debate and critical analysis about the validity and success of all interventions for treatment of plantar heel pain—both operative and non-operative.[15,18]

Among all disciplines is near-universal agreement that the vast majority of patients with plantar heel pain will be successfully treated with non-operative strategies. However, there is no uniform agreement, either within or between these disciplines, as to which conservative interventions are most appropriate in achieving a successful outcome in treating plantar heel pain.

As managed care economics affected the medical profession during

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the 1990's, health care providers were pressured to develop evidence-based outcomes research to justify the costs and benefits of prevailing clinical treatment strategies. Standards for quality outcomes research have evolved for most health care disciplines, including podiatric medicine.[17] However, scrutiny of published research reporting outcomes of treatment of plantar heel pain in the podiatric and orthopedic literature reveals numerous shortcomings in terms of valid design, methodology, and interpretation of results.

The purpose of this article is to 1) Evaluate prevailing theories about the pathomechanics of plantar heel pain, 2) Present controversies that currently exist regarding etiology and treatment and, 3) Review outcomes reports of non-operative interventions used to treat large groups of patients with plantar heel pain syndrome.

Pathomechanics

In 1972 Snook and Chrisman, in reviewing the prevailing literature relevant to plantar heel pain, stated "It is reasonably certain that a condition which has so many theories about etiology and treatment does not have valid proof of any one cause."[23] Sadly, thirty years later, this statement is still true.

Patients presenting with pain in and around the plantar tubercules of the calcaneus have been theorized to have a wide array of possible injuries to various structures in and around the plantar heel area (Table 1). These conditions include plantar fasciitis, calcaneal periostitis, enthesopathy, calcaneal stress fracture, calcaneal spur, nerve entrapment, fat pad atrophy and subcalcaneal bursitis. Systemic inflammatory conditions are known to cause plantar heel pain, most notably the seronegative spondyloarthropaties. This article will focus on all nonsystemic etiologies. A summary of prevailing causes of plantar heel pain is presented in Table 2.

The four most popular theories of the pathomechanics of plantar heel pain include plantar fascial strain, heel impact shock, and nerve entrapment. The most compelling evidence supporting any of these theories is found in the category of plantar fascial overload and strain. Before discussing this area, the other two proposed mechanisms will be reviewed.

Nerve Entrapment Theories

The nerve entrapment theory of plantar heel pain has been popularized by Don Baxter M.D., who has co-authored several papers dealing with the anatomy, diagnosis and treatment of heel pain attributed to an entrapment of the first branch of the lateral plantar nerve.[1,27] This nerve has been thus named "Baxter's Nerve" even though it was first described as a cause of plantar heel pain by Tanz in 1963,[6] and later by Przylucki and Jones in 1981—ten years before Baxter's first paper on the subject.[2]

The first branch of the lateral plantar nerve (nerve to the abductor digiti quinti brevis) is thought to lie in the direct vicinity of the area where most patients complain of plantar heel pain. Contrary to original anatomic descriptions, Baxter showed in a cadaver series that the first

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Heel Pain Etiology

Calcaneal periostitis	Chang, 1934
Subcalcaneal Bursitis	Freeman, 1988
Strain and Degeneration of Plantar Fascia	Lapidus, 1965
Lateral plantar nerve entrapment	Tanz, 1963; Przylucki & Jones, 1981
Medial calcaneal neuritis	Lutter, 1986
Calcaneal stress fracture	Graham, 1983
Fat pad atrophy	Snook & Chrisman, 1972
Tarsal tunnel syndrome	Mann, 1986
Subcalcaneal spur	DuVries, 1957
Calcanealdynia	Leach, 1986

Table 1

Causative Factors Of Heel Pain:

Obesity	O'Brien, Shikoff, Tanz
Pes Planus	Bordelon, 1993
Pes Cavus	Culter, 1986
Tight Heel Cord	Kibler, 1991
Pronation of STS	Baxter, 1984 Gould, 1959 Hicks, 1954 Davis, 1990
Shock	McKenzie, 1985
Windlass Dysfunction	Ellis, 1988

Table 2

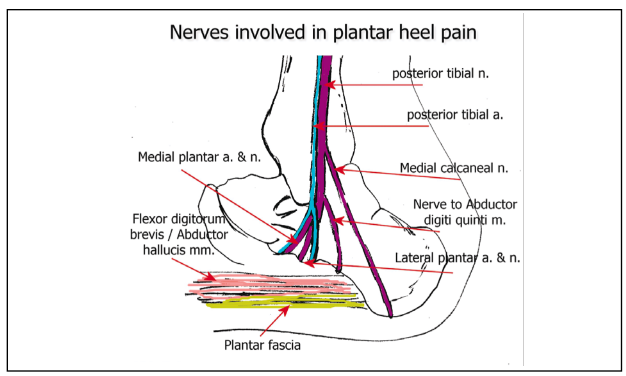

Figure 1

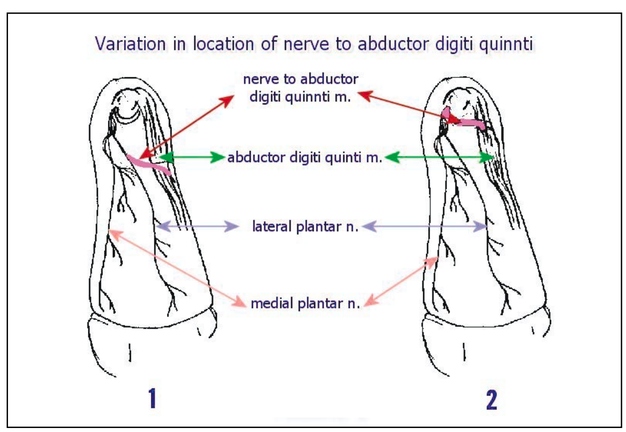

Figure 2

Figures 1 and 2 were adapted from illustrations that appeared originally in *Clinical Orthopaedics*. Baxter DE, Pfeffer GB: Treatment of Chronic Heel Pain by Surgical Release of the First Branch of the Lateral Plantar Nerve: Clin Orthop. 279:229-235, 1992.

branch of the lateral plantar nerve is more proximal, and penetrates a tight myofascial septum separating the abductor hallucis muscle from the quadratus plantae muscle, then courses plantarly, just anterior to the medial calcaneal turbercle (Figures 1 & 2). An entrapment is thought to occur at this fascial septum, or just under the calcaneal margin. How such an entrapment occurs, however, has not been proposed by any author. Surgical release of the entrapment along with neurolysis of the first branch of the lateral plantar nerve has shown success in 89% of patients with recalcitrant plantar heel pain.[27]

Other nerve entrapment theories of plantar heel pain include involvement of the medial calcaneal nerve[3] as well a tarsal tunnel nerve entrapment.[14] Hendrix et al demonstrated a 95% success rate with surgical decompression of the tarsal tunnel in his series of 51 patients with chronic heel pain.[14] The pathomechanics of this entrapment was speculated to be an inverted gait pattern, which was a compensation for a pre-existing painful heel.

Heel impact shock is commonly quoted as a causative factor in the development of plantar heel pain syndrome.[23] Further scrutiny of these reports shows no valid objective data to justify such a conclusion. Certainly, a group of patients have been identified with plantar fat pad atrophy that is subject to periostitis and bursitis due to lack of intrinsic cushioning.[12] However, this anatomic characteristic is not found amongst the majority of patients treated for plantar heel pain in this country.

The calcaneal stress fracture theory is primarily based upon a traction force applied to the calcaneus by the plantar fascia, rather than an impact shock mechanism.[23] Calcaneal stress fractures have been reported in high-mileage runners who are subject to repetitive impact, heel cord and plantar fascial loads. These patients make up a small percentage of patients treated for plantar heel pain syndrome in this country.

Later, the use of cushioning modalities will be discussed in the treatment of heel pain syndrome. The results of cushioning strategies are, for the most part, only marginally effective, which indirectly invalidates impact shock as a primary etiology of plantar heel pain syndrome.

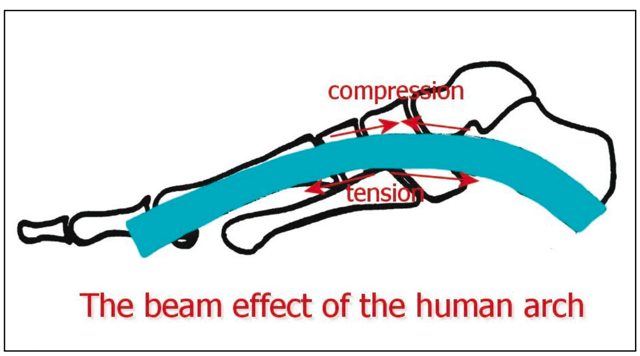

Figure 3

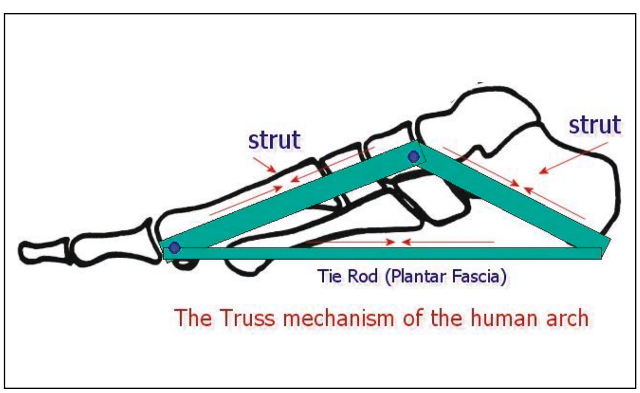

Figure 4

Figures 3 and 4 were adapted from illustrations that appeared originally in *Foot and Ankle International*. Sarrafian SK: Functional Characteristics of the Foot and Plantar Aponeurosis under Tibiotalar Loading. Foot Ankle. 8:4-17, 1987.

Plantar Fascial Strain

The most widely-accepted theory of the etiology of plantar heel pain syndrome is plantar fascial strain. Biopsies of plantar fascia samples taken from patients with chronic heel pain have consistently demonstrated histologic findings compatible with mechanical tear and inflammatory response.[8,12] The location of this mechanical injury is most often at the plantar-medial margin of the calcaneus.[7]

A widely-accepted consequence of chronic plantar fascial strain is the development of a plantar-calcaneal spur.[13,23] In fact, many clinicians commonly label all patients with plantar heel pain as having "heel spur syndrome."[26]

Myths About Heel Spurs

In a series of elegant anatomic studies using cryomicrotomy, McCarthy and Gorecki clearly showed that the common plantar calcaneal spur is not invested by the plantar fascia.[19] Rather, the spur is invested by the abductor hallucis, quadratus plantae and flexor digitorum brevis muscle origins and is clearly found superior to the origin of the plantar aponeurosis. In light of these findings, the direct link between plantar fascial overload and the formation of calcaneal spurs must be questioned.

In 1963, Rubin showed that only 10% of patients with radiographic evidence of heel spurs were actually symptomatic.[5] Since then, many authors have demonstrated that the majority of plantar heel spurs found on foot x-rays are asymptomatic.[4,10]

Thus the role of a heel spur in the pathomechanics of plantar heel pain syndrome is still poorly understood. Yet, clinicians commonly use the term heel–spur syndrome and many treatment strategies employ methods to theoretically off-load a plantar calcaneal spur.

Theories of Plantar Fascia Overload

Plantar fascia strain has been speculated to result from every imaginable foot type and biomechanical etiology. Both the podiatric and orthopaedic literature are replete with unsubstantiated explanations of plantar fascial strain resulting from cavus foot types, flat foot types, pronated feet and supinated feet.[13,21,23] In almost every case, these pathologies have been speculated to cause a lowering of the medial arch of the foot, resulting in fascial strain.

A valid and well-substantiated arch-lowering force on the human foot is a tight heel cord.[12] Tightening of the heel cord in cadaver models not only lowers the

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arch, it causes significant rotational movements of the forefoot upon the rearfoot, including midtarsal joint pronation and dorsiflexion and inversion of the first ray segment.[29]

Interestingly, the presence of a tight heel cord has not been consistently found in groups of patients with plantar heel pain syndrome. In her study of 91 patients with heel pain, Barbara Warren found that the heel cord was actually tighter in a control group than in a group of patients with plantar heel pain.[20] Conversely, Kibler found that the heel cord was tighter on the symptomatic side of patients with heel pain, but could not rule out a cause vs. effect relationship.[11] Amis found that 75% of his patients with heel pain had a tight heel cord.[10] However, there is no universal agreement as to the "normal" range of ankle joint dorsiflexion necessary for humans, so studies of tight heel cords are open to considerable subjective interpretation. The essential factor in evaluating this possible link between a tight heel cord and plantar heel pain is the role of calf and Achilles stretching in non-operative treatment programs. Indeed, although stretching is integral in most recommended treatments, the success of such intervention is questionable. This will be discussed later in this article.

Artificially supporting the arch does not necessarily reduce strain in the fascia.

The plantar fascia is the most important arch-supporting mechanism of the human foot. In his study of cadaver models subjected to axial load, Thornardsen found that the plantar fascia had a two fold greater contribution to arch stability than the posterior tibial tendon.[24] In his series on the role of the plantar fascia and arch support, Sharkey found a significant elongation and deformation of the arch with complete fasciotomy.[29,30]

The arch of the human foot has been described as both a beam and a truss.[34] Recent experimental evidence has validated the truss mechanism as the primary explanation of stability. A beam relies on the interlocking relationship of the building blocks (bones) and the soft tissue connections on the concave surface (ligaments)(Figure 3). The truss is described as two struts connected by a tie rod (plantar fascia)(Figure 4).

Cadaveric studies have shown that, without intact ligaments, the bone architecture of the human foot is incapable of maintaining an arch configuration when axial load is applied.[22,24] When the entire central band of the plantar fascia is severed, the human arch integrity is severely compromised, with documented

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shift of alignment of the tarsal bones in all three cardinal body planes. In addition, Sharkey showed, in cadaver models void of an intact central plantar fascia, that greater loads were transmitted to the central metatarsals, due to loss of plantar stabilization of the proximal phalanx on the metatarsal head.[30] Bending and strain of the metatarsals can possibly lead to stress fractures in patients who have undergone complete plantar fasciotomy.

In static, resting stance, the muscles of the leg and foot are inactive. Maintenance of arch integrity is entirely dependent on the osseous locking of the tarsus and the truss mechanism of the plantar fascia. Without the aid of the extrinsics to maintain the arch, static stance may be the most stressful situation for the plantar fascia.

Anecdotally, clinicians have reported the most difficult challenges in managing plantar fasciitis in patients engaged in prolonged standing activities.[15,36] Comparisons between the dynamic foot condition and the resting, static foot position offer interesting challenges for treatment options for off-loading the plantar fascia.

Recent insights into the effects of off-loading the plantar fascia in a static foot model were offered in a series of studies conducted by Kogler et. al. In nine cadaver specimens, axially loaded, six degree medial wedges placed under the forefoot caused an increased strain in the plantar fascia while six degree lateral wedges caused a significant decreased strain. Rearfoot wedges, both medial and lateral, had no significant effect on plantar fascial strain.[32]

In another study published by Kogler, the effect of heel elevation on plantar fascia strain was determined.[33] Simple blocks of 2,4 and 6 cm thickness were placed under the heel of 12 cadaver specimens and plantar fascia strain was measured and compared to the heel flat condition. Surprisingly, there was no evidence of any reduced fascial strain with heel elevation. However, when the heel was elevated with shank contour platforms (simulating the effect of footwear with elevated heels) there was a significant decrease in strain of the plantar fascia with increased elevation of the platform.

The results of Kogler's research validates the experience of many patients who obtain relief of plantar heel pain syndrome by wearing shoes with elevated heels. The mechanical off-loading of the plantar fascia cannot, however, be explained by heel elevation alone. In fact, with a true truss mechanism, elevating the proximal strut (calcaneus) can be expected to actually increase strain in the tie rod (plantar fascia)(figure 5). The reduction in plantar fascia strain occurring with contoured shank platforms, according to Kogler, may be the re-

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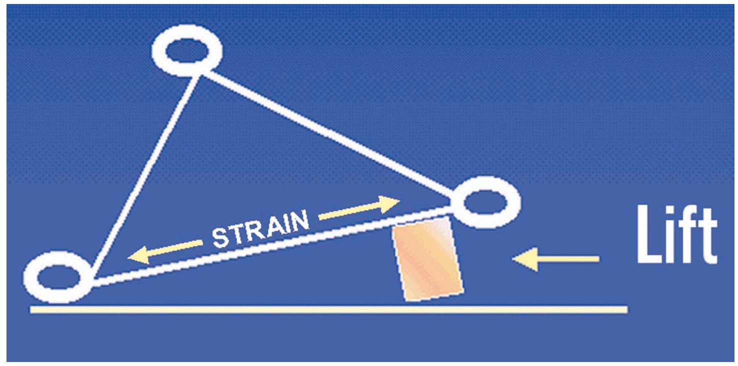

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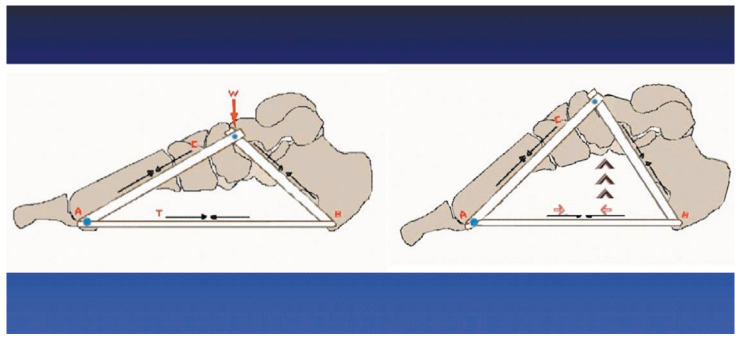

Figure 6

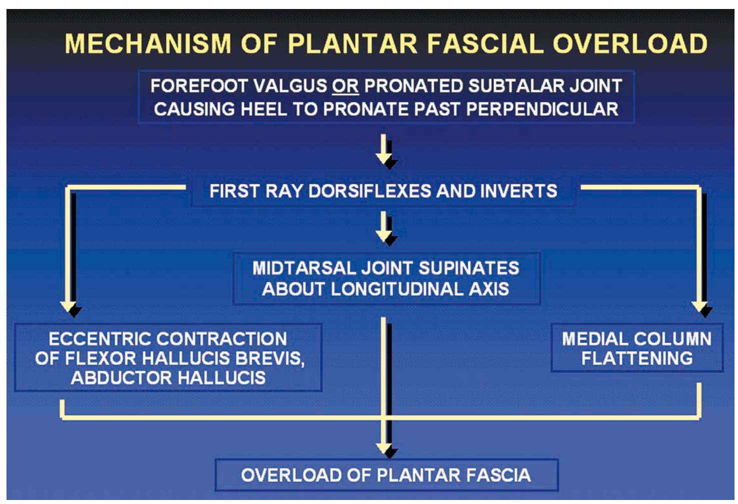

Figure 7

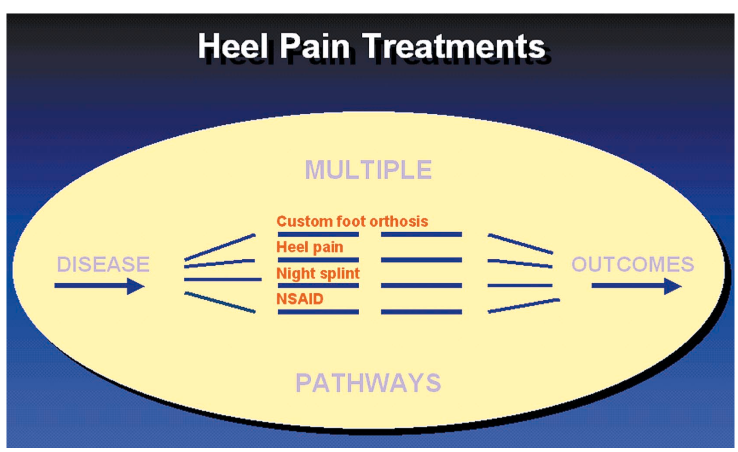

Figure 8

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sult of lateral arch elevation which secondarily raises the medial arch and thus decreases strain on the medial fascial structures (Figure 6).

The problem with most of the cadaver studies on plantar fascia biomechanics is the fact that the investigators did not evaluate or report on the foot-type of the specimens. The presence of a forefoot varus or valgus could have significant effect on the response to medial and lateral wedging of the forefoot, as well as the lateral arch-raising effect of a shank contoured elevation platform.

Another issue to consider is the phase in the gait cycle that is simulated in cadaver studies of lower extremity function. Kogler, Thornardsen and Kitaoka all evaluated their cadaver specimens in a foot-flat, static stance position. Sharkey evaluated plantar fascia mechanics in terminal stance (propulsive phase) when maximal strain on the plantar fascia is thought to occur. This assumption is somewhat debatable. Although ground-reaction forces peak at heel rise and extrinsic muscle activity also reaches maximal tension, the windlass mechanism allows transmission of tension into kinetic movement of pedal and leg skeletal segments. Therefore, energy or strain in the plantar fascia is transferred to other parts of the foot during terminal stance. Without a functioning windlass, occurring with hallux limitus or during static stance, strain develops in the plantar fascia and cannot be dissipated or transferred into joint movement.

Utilizing Off-loading Principles

Podiatric physicians have long employed biomechanical principles in the treatment of patients with plantar heel pain. However, there is no consensus in the podiatric literature in terms of a uniform treatment approach utilizing functional foot orthoses and proper footwear prescription.

Many practitioners seek to control pronation of the subtalar joint through the use of medially-posted foot orthoses, both in the rearfoot and forefoot. As Kogler's work has shown, and from a simple understanding of the truss mechanism of the plantar fascia, the application of a medial post under the forefoot will actually increase strain in the plantar fascia for most foot types. One possible exception is the true forefoot varus, which is uncommon, and in the author's experience, is rarely associated with plantar heel pain syndrome.

> ***Many patients have formed opinions that their pain will never be totally cured.***

Insight into the prevalence of plantar heel pain in certain foot types was provided by Scherer and the Biomechanics Graduate Research Group at the California College of Podiatric Medicine.[26] In a prospective study of 88 patients with 133 painful heels, 115 had a structural deformity that would result in a compensation with supination of the forefoot on the rearfoot, presumably through a longitudinal axis of rotation. Of these, 63 had a forefoot valgus, 20 had a plantarflexed first ray, and 32 had an everted heel in stance. All three groups, therefore, would have a compensation mechanism which would invert (supinate) the forefoot on the rearfoot. Scherer and co-workers theorized that this movement would increase strain on the medial portion of the central band of the plantar fascia. Indeed, the later work of Kogler, using lateral wedges to reduce forefoot inversion, validated Scherer's theory of foot mechanics and plantar fascia strain.[32] A proposed mechanism of plantar fascia overload appears in Figure 7.

Many practitioners simply rely on over-the-counter arch supports or on custom foot orthoses designed primarily for arch support as a remedy for plantar heel pain syndrome.[21]

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The notion that support of the medial longitudinal arch will decrease strain on the medial portion of the central band of the plantar fascia has not yet been substantiated. Although it has been well-proven that the plantar fascia is the most important soft tissue support mechanism of the human arch, artificially supporting the arch does not necessarily reduce strain in the fascia.

In his series of cadaver studies of plantar fascial strain, Kogler used five different types of custom and non-custom

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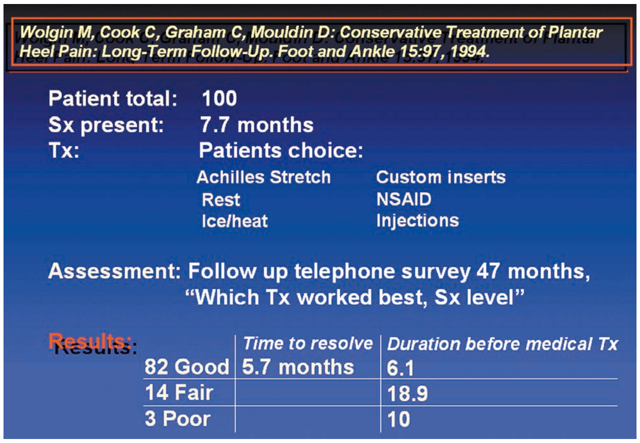

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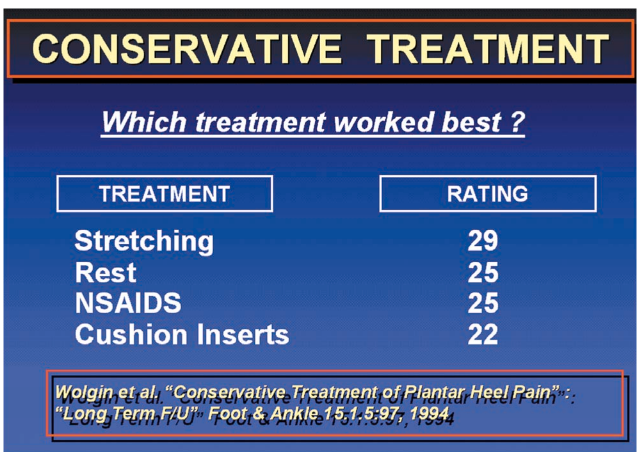

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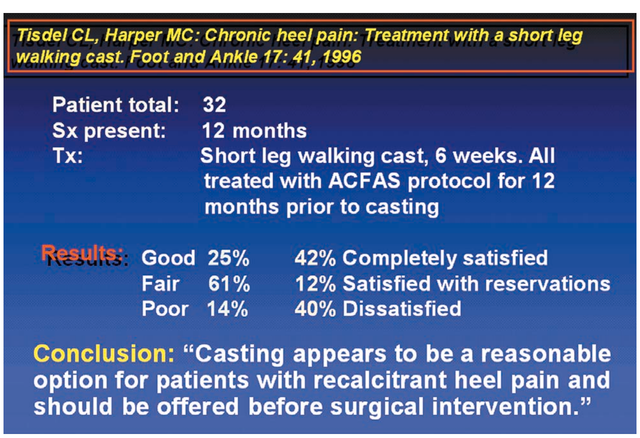

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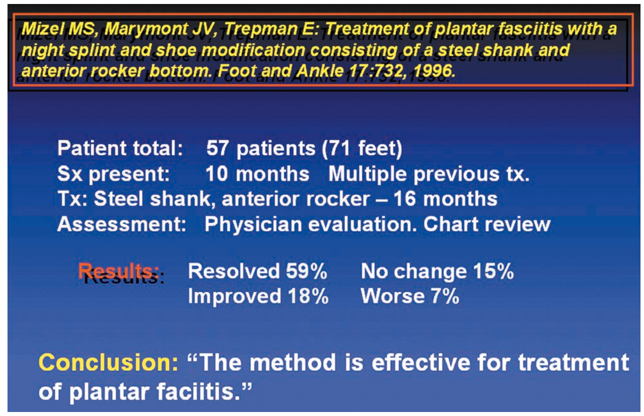

Figure 12

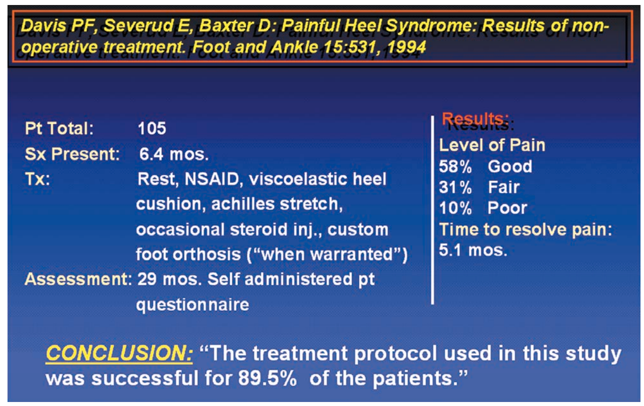

Figure 13

Figure 14

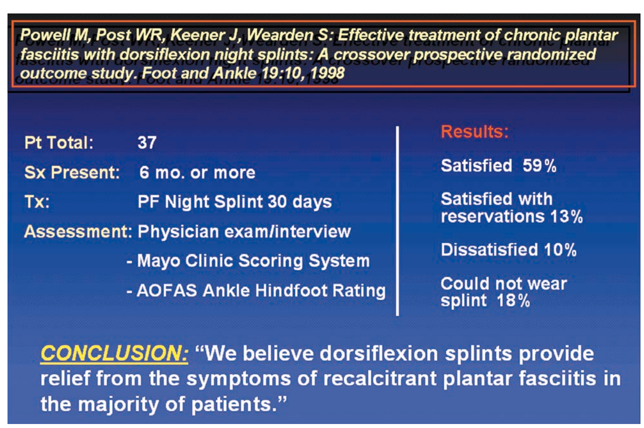

Figure 15

foot orthoses to determine effects on plantar fascial strain in seven cadaveric lower limbs.[31] These test orthoses included: a prefabricated stock arch insole, a custom "soft" accommodative design orthosis composed of viscoelastic material, a "semi-rigid" accommodative design orthosis composed of co-polymer, a "rigid" custom orthosis with a Root Functional design, and a UCBL design rigid orthosis. The prefabricated device and the Root rigid device actually increased strain in the plantar fascia while the other three custom devices decreased strain compared to the barefoot condition.

The devices that most effectively reduced strain were those with higher apical arch height and increased slope of arch shape in the central region of the medial arch. Although all five devices could be considered arch supports, the shape of the device and the conformity to certain key areas of the medial arch appeared critical in determining effectiveness to offload the plantar fascia. Thus, non-conforming arch supports have the potential to actually increase strain in the plantar fascia.

Evaluating Treatment Outcome Reports

A total of 10 published papers reporting outcomes of various treatments of plantar heel pain will be reviewed. These papers have all been published within the past decade and combine similar modalities commonly employed in this country for treatment of plantar heel pain. Although the treatment strategies are similar, the results and conclusions vary significantly amongst the investigators (Figure 8).

Wolgin surveyed 100 patients who were treated with a variety of common non-operative interventions for plantar heel pain syndrome.[16] Average time for follow up survey was 47 months. Good results were obtained in 82 out of 100 patients, 14 achieved fair results and 3 had poor results (Figures 9 & 10). The patients rated Achilles stretching as the most effective treatment followed by rest and NSAID's, both of which were

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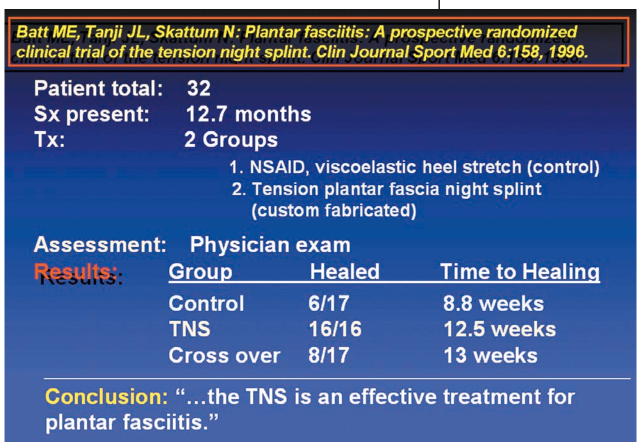

Figure 16

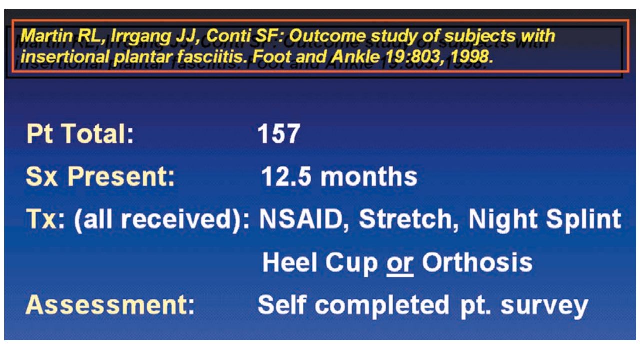

Figure 17

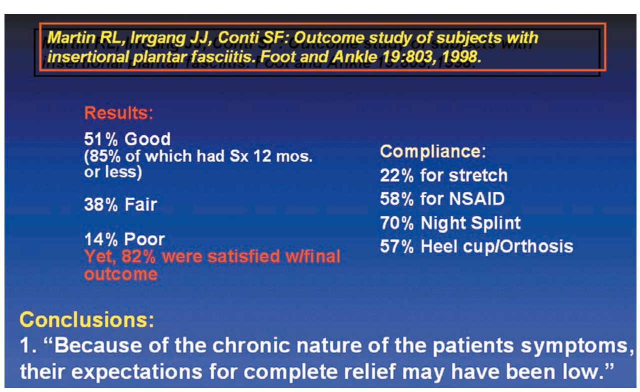

Figure 18

higher rated than "custom inserts." Patients who did poorly were overweight, had bilateral symptoms and had symptoms for a prolonged period of time (more than 10 months) before seeking medical attention.

Tisdel and Harper utilized a short-leg walking cast on 32 patients who had failed 12 months of non-operative treatment for plantar heel pain.[39] After six weeks of cast immobilization, the patients were then followed and interviewed at an average of 15 months post treatment (Figure 11). Despite the fact that good results were obtained in only 25% of the patients, and over 40% of the patients were dissatisfied, the authors concluded that "Casting appears to be a reasonable option for patients with recalcitrant heel pain and should be offered before surgical intervention."

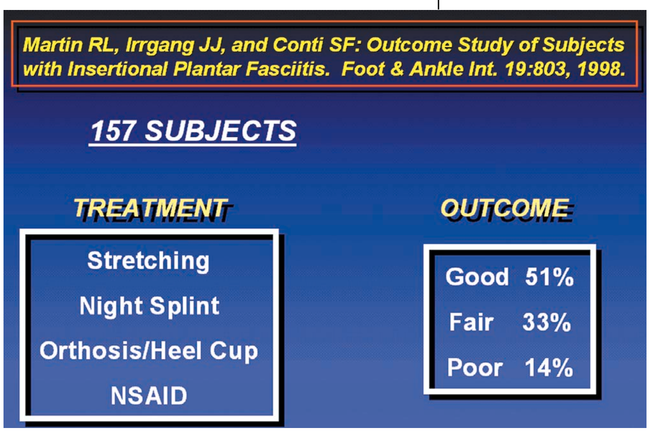

Figure 19

Mizel utilized a shoe modification with steel shank and anterior rocker for patients who had failed a 10-month course of previous treatment for plantar heel pain syndrome.[41] After 16 months of this treatment, 59% of the patients reported symptoms resolved, 18% were improved, 15% reported no change, and 7% were worse (Figure 12). After two years of treatment, with 59% of the patients resolved, the authors concluded that "The method is effective for treatment of plantar fasciitis."

Davis, Severud and Baxter reported on the results of non operative treatment of 105 patients with heel pain syndrome (38). A self-administered patient questionnaire was completed an average of 29 months after initiating treatment. Treatment included Rest, NSAID, viscoelastic heel pad, Achilles stretch, occasional steroid injection, and custom foot orthosis "when warranted." In rating their level of pain resolution, 58% of the patients reported good results, 31% fair, and 10% poor with an average time to resolution of 5.1 months (Figure 13). Somehow, the authors concluded that "The treatment protocol used in this study was successful for 89.5% of the patients."

Powell and co-workers used a plantar fascia night splint (Figure 14) on 37 patients who had 6 months of heel pain symptoms.[37]

Continued on page 146

The splint was used for 30 days, and the patients followed up at six months with physician interview. A survey revealed that 59% of the patients were satisfied, 13% satisfied with reservation, and 10% dissatisfied (Figure 15). Despite the fact that 18% of the patients could not tolerate the splint, and that only 59% of the patients were satisfied, the authors conclusion was "We believe dorsiflexion splints provide relief from the symptoms of recalcitrant plantar fasciitis in the majority of patients."

A more impressive result with night splinting was reported by Batt et. al., who used a custom-fabricated tension plantar fascia night splint on 32 patients and used a controlled, cross-over study design to compare splinting to NSAID, heel stretch and viscoelastic heel cushions.[40] All 16 out of 16 patients using the night splint were healed after 12 weeks, while only 6 of 17 patients were healed in the control group. In the cross-over group, 8 of 17 were healed once night splinting was utilized(Figure 16).

Martin studied results of treatment of 157 patients with an average of 12 months of heel pain prior to seeking care.[36] Treatment consisted of stretching, NSAID, night splint, and either a heel cup or a foot orthosis. Results were good in only 51% of the patients, 88% of whom had had symptoms for 12 months or less. Fair results were obtained in 38% of the patients, while 14% reported poor results. In evaluating patient compliance with treatment, only 22% were compliant with stretching, 57% with heel cup/orthosis, 58% with NSAID, and 70% with night splint (Figures 17,18 & 19). The authors concluded that early, aggressive non-surgical treatment within 12 months of onset of symptoms offers the best chance of a favorable outcome.

Gill and Kiebzak reported less effective outcomes of non-operative interventions described in the previous reports.[35] In a large patient population (246 female and 165 male) a treatment ratings survey showed that most interventions showed disappointing results. In terms of effectiveness, cast immobilization led to improvement in 65% of patients, steroid injection improved 45%, NSAID 25%, and heel pad 27%(Figures 20 & 21). However, the overall improvement with any treatment was rated poor or mild. The authors concluded that "The ineffectiveness of non surgical treatments noted in this study is at variance with most published clinical studies." Furthermore, these authors stated that "Physicians may be inappropriately attributing many of their success to their treatments, when, in fact, these treatments make very little difference in the actual outcome."

An interesting classification of non-operative treatments for plantar heel pain is provided by Lynch and coworkers.[25] In their randomized, prospective study of 103 subjects, three types of conservative therapy were utilized: Anti-inflammatory (dexamethasone injection), Accommodative (viscoelastic heel cup), and Mechanical (low-dye strapping and custom foot orthosis). After 12 weeks of treatment, good to excellent results were obtained in 70% of the patients in the mechanical group, 33% in the

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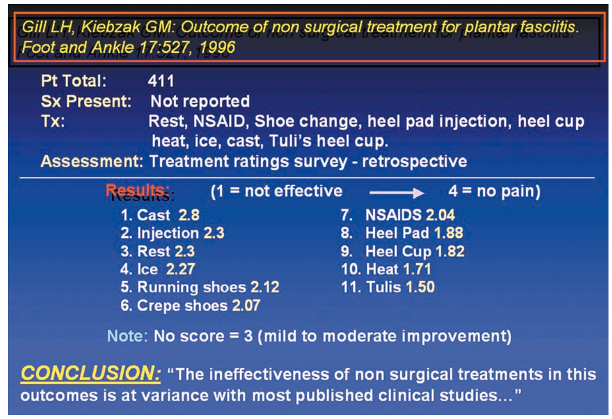

Figure 20

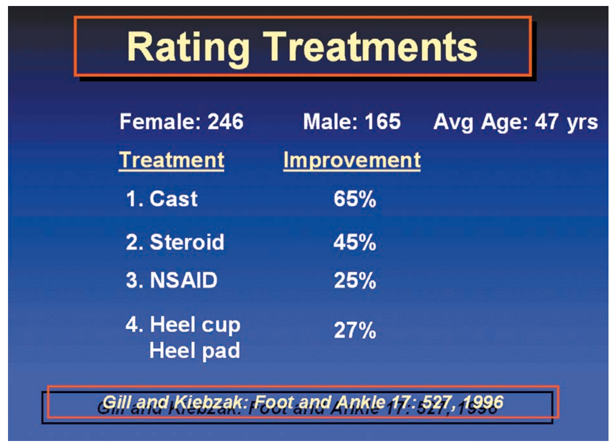

Figure 21

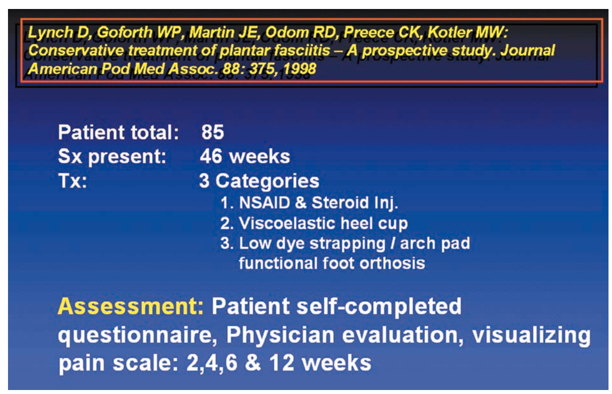

Figure 22

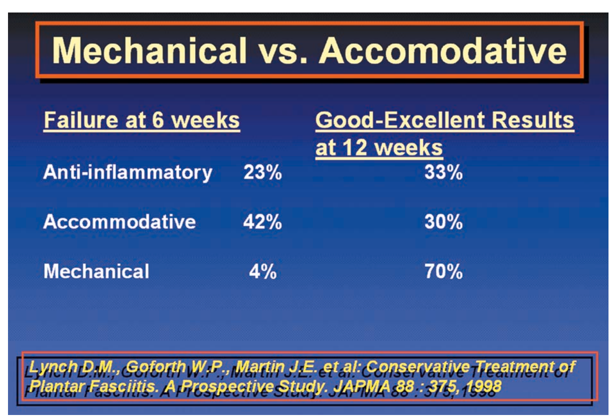

Figure 23

Heel Pain...

anti-inflammatory group and 30% in the accommodative group (Figures 22 & 23).

This same significant favorable outcome with a mechanical approach to off-loading the plantar fascia was obtained by Scherer and co-workers.[26] In their prospective study of 73 patients, a subgroup receiving low-dye strapping and custom functional foot orthoses only obtained good results in 81% of the patients, fair results in 15%, and poor results in 4%(Figure 24).

There are numerous difficulties in evaluating all of these outcome studies and drawing meaningful conclusions. Clearly, there were differences in assessment of success depending on whether the results were determined by confidential patient interview or obtained by interview from the treating practitioner. It is well known that patients will report a more favorable outcome to the treating doctor versus a more realistic assessment in a confidential survey, conducted by a neutral party.

Of interest is the disparity between a patient assessment of successful treatment outcome (Good, Fair, Poor) versus overall pain relief. In many studies, a majority of patients reported a good outcome, yet still had significant pain. As Martin states, in evaluating patients who have been treated for long term heel pain, "Because of the chronic nature of the patient's symptoms, their expectations for complete relief may have been low." Thus, many patients who present for treatment of plantar heel pain have already had their pain for an extended period and have formed opinions that their pain will never be totally cured.

Among almost all of these studies, there was near universal agreement that the longer the patient had experienced pain prior to treatment, the less likely would a successful treatment outcome occur. In general, those patients having pain for more than 12 months prior to treatment were most resistant to non-operative interventions.

In this regard, there appears a disparity among clinicians as to the amount of time necessary to expect significant pain relief with non-operative care. Several authors concluded that certain treatments were effective, even though the time of treatment needed to achieve measurable success exceeded two years. One has to question the overall efficacy of such interventions if, during the two years of treatment, the patient lost significant time from work or discontinued a potentially beneficial cardiovascular exercise program.

In the final analysis, the treatment strategy that yielded the best results in the shortest period of time was the combination of low-dye strapping[9] and prompt institution of custom functional foot orthotic therapy. Both Scherer and Lynch, utilizing these strategies, achieved

Continued on page 149

better results that the other groups in less than one-fourth the period of time (See Table 3).

Conclusions

Plantar heel pain syndrome continues to stimulate controversy regarding pathomechanics and treatment. Patients developing heel pain can expect to be offered a divergent approach, depending on the specialty they seek treatment from. Even within certain specialties, there is considerable variation of opinion regarding the pathomechanics and treatment of plantar heel pain syndrome.

Recent cadaveric studies have shed light on the role of the plantar fascia in supporting the arch as well as the effects of certain strategies to decrease strain in this structure. Some widely accepted notions about forefoot wedging and heel elevation have now been disputed. Relating the results of laboratory research to a practical clinical setting and to a variety of foot types remains a challenge to today's podiatric physician.

Non-operative treatment strategies for plantar heel pain have been evaluated in outcomes studies by a number of investigators. The results of these studies are contradictory, and conclusions must be made cautiously. The reasons for such skepticism are the following:

1. True outcomes research has yet to be conducted in this area, following accepted methodology and utilizing ap-

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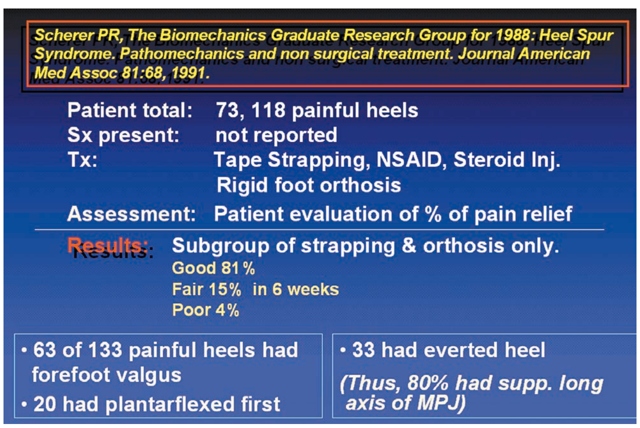

Figure 24

Author	Duration of Treatment (mos)	Outcome (%) G	F	P
Wolgin	5.7	82	15	3
Tisdel	12	28	61	14
Martin	12	51	33	14
Davis	5.1	58	31	11
Mizel	16	59	18	22
Scherer	1.2	82	28	8
Lynch	3	12	7	8

Table 3

Heel Pain...

propriate measurement techniques.

2. Assessment of a successful outcome of treatment will vary significantly depending on whether the patient versus the clinician provides the final analysis.

3. Patients with plantar heel pain have poor expectations for total, permanent pain relief when they present for treatment.

4. Clinicians can use treatments that require up to 24 months to achieve success, yet conclude such treatments are effective.

5. The longer a patient has symptoms prior to treatment, the less likely any non-operative treatment is going to be successful.

Although comparisons between studies are difficult to make, some findings appear worth noting. Specifically, those groups of patients with plantar heel pain, treated promptly with low-dye taping and custom functional foot orthosis therapy, had the most favorable outcome of treatment.

Until further insight into the pathomechanics of plantar heel pain is attained, there will continue to be controversy—and, unfortunately, continued significant numbers of patients suffering from this disorder. ■

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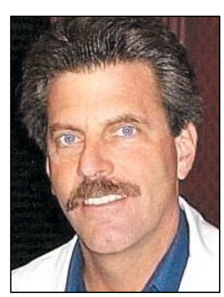

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EXAMINATION

See answer sheet on page 153.

1) Which of the following anatomic structures is NOT commonly implicated as a cause of plantar heel pain?
A) Plantar fascia
B) First branch of lateral plantar nerve
C) Calcaneal periosteum
D) Extensor digitorum brevis

2) The nerve most commonly implicated in plantar heel pain syndrome is:
A) Medial plantar
B) Medial calcaneal
C) Sural
D) First branch of lateral plantar

3) The primary structure(s) investing a heel spur are:
A) plantar aponeurosis
B) abductor hallucis and flexor digitorum brevis
C) peroneal tendon
D) posterior tibial tendon

4) The most important soft tissue support of the human arch is:
A) Plantar fascia
B) Posterior tibial tendon
C) Short plantar ligament
D) Spring ligament

5) Wearing elevated heel shoes decreases strain in the plantar fascia by:
A) Relieving the truss tie rod
B) Shifting weight to the toes
C) Providing a shank contour
D) Activating the intrinsic muscles

6) The plantar fascia appears strained when the midtarsal joint is:
A) Pronated
B) Supinated
C) Wedged
D) Rotated

7) Treating painful heels is most successful when patients seek treatment within:
A) Two years
B) Three years
C) 12 months
D) 14 months

8) Patients initially presenting for treatment with plantar heel pain usually have the following expectation level of achieving success with treatment:
A) High
B) Low
C) None
D) Surgical

9) Outcomes research on patients with plantar heel pain will have different results, depending on whether the results are determined by:
A) The treating physician
B) The patient
C) A neutral party
D) All of the above

10) The two most effective treatments reported in outcomes research in the treatment of plantar heel pain are:
A) NSAIDS and rest
B) Heel pad and stretching
C) Night splint and steroids
D) Low-dye taping and functional foot orthoses

11) At least one study of plantar fascia night splints, with cross-over design, has shown the following level of success:
A) Low
B) High
C) None
D) Variable

12) In static stance, the following muscles are active:
A) Posterior tibial
B) Anterior tibial
C) Abductor hallucis
D) None

13) A forefoot valgus causes the following compensation of the midtarsal joint:
A) Pronation
B) Supination
C) Dorsiflexion
D) Plantarflexion

14) In evaluating cadaver studies of plantar fascia strain, which variable NOT discussed would be most important:
A) Foot type of the specimen
B) Weight of the limb
C) Dorsiflexion
D) Pronation

15) Strain across the metatarsals has been observed after plantar fasciotomy because the following

Continued on page 152

Continuing Medical Education

EXAMINATION

(cont'd)

structure is de-stabilized:

A) Heel cord
B) Tibial nerve
C) Extensor tendon
D) Metatarsophalangeal joint

16) The following treatments for plantar heel pain have been reviewed, EXCEPT:

A) NSAID's
B) Heel cushion
C) Acupuncture
D) Orthotic

17) The windlass mechanism is compromised when the following clinical situation is present:

A) Functional hallux limitus
B) Nerve entrapment
C) Heel pad atrophy
D) Periostitis

18) Plantar fasciotomy can be expected to lead to the following change in the medial arch:

A) Shortening
B) Lengthening
C) Elevation
D) Adduction

19) Which of the following is NOT considered a mechanical approach to relieving plantar heel pain:

A) Custom foot orthotic
B) Low dye taping
C) Viscoelastic heel cup
D) Stretching heel cord

20) Impingement of the First Branch of the Lateral Plantar Nerve can involve the following structures EXCEPT:

A) Abductor Hallucis muscle
B) Quadratus Plantae muscle
C) Posterior calcaneal tubercle
D) Plantar medial edge of calcaneus

See answer sheet on page 153.

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EXAM #6/02
Plantar Heel Pain
(Richie)

Circle:

1.	A	B	C	D	11.	A	B	C	D
2.	A	B	C	D	12.	A	B	C	D
3.	A	B	C	D	13.	A	B	C	D
4.	A	B	C	D	14.	A	B	C	D
5.	A	B	C	D	15.	A	B	C	D
6.	A	B	C	D	16.	A	B	C	D
7.	A	B	C	D	17.	A	B	C	D
8.	A	B	C	D	18.	A	B	C	D
9.	A	B	C	D	19.	A	B	C	D
10.	A	B	C	D	20.	A	B	C	D

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