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How to Avoid Failure of Radial Pressure Waves and Focused Shock Waves Therapy in Plantar Heel Pain: Diagnostic, Technical, and Biomechanical Pitfalls

Osvaldo Valle Toledo[1]

Abstract

Mechanical wave-based therapies are a widely used and effective treatment for chronic plantar fasciopathy (PF); however, some patients do not achieve satisfactory clinical outcomes. Treatment failure is not necessarily related to the intrinsic efficacy of the method but is more frequently associated with modifiable clinical factors. This narrative review aims to analyze the most common causes of failure, focusing on diagnostic errors, inappropriate timing of treatment, inadequate technical application, and failure to address biomechanical abnormalities. Misdiagnosis is a major cause of therapeutic failure, as plantar heel pain may originate from multiple conditions other than PF, including tarsal tunnel syndrome, Baxter's nerve entrapment, heel fat pad syndrome, calcaneal stress fractures, bursitis, tumors, and systemic inflammatory diseases. Technical factors, such as inappropriate energy delivery, incorrect targeting, and the use of local anesthesia, may also negatively influence outcomes. Furthermore, uncorrected gastrocnemius contracture and altered foot biomechanics contribute to persistent overload of the plantar fascia. Finally, both radial pressure waves and focused shockwaves appear to be more effective in chronic than in acute PF. A better understanding of these factors may improve patient selection and optimize treatment outcomes.

Keywords: Heel pain, Radial pressure waves, Focused shock waves, Plantar fasciitis

Introduction

Heel pain is a common musculoskeletal complaint, with plantar fasciitis being the most common underlying cause [1,2,3]. Although its name refers to an inflammatory process, plantar fasciitis is fundamentally a degenerative process [3], so it is more correct to call it plantar fasciopathy (PF).

It affects approximately 10% of the general population, with 83% of cases occurring in economically active adults between 25 and 65 years of age [3]. PF has a substantial impact on sports medicine, accounting for approximately 8% of all running-related injuries [4].

Numerous therapeutic options have been described for the management of the symptoms of PF; however, the American College of Foot and Ankle Surgeons has developed a treatment algorithm that serves as a widely accepted reference framework [5]. The authors reached a consensus that mechanical wave-based therapies (MWT), including radial pressure waves and focused shockwaves, are a safe and effective treatment for PF [5].

Reported success rates for MWT vary substantially, likely reflecting differences in selection, treatment protocols, and underlying pathology [5,6,7,8,9]. Nevertheless, despite its established safety and effectiveness, approximately 10–20% of patients with heel pain experience an inadequate clinical response [5,6,7,8,9].

Treatment failure may result from several factors, including an incorrect diagnosis, suboptimal treatment application, inadequate assessment of biomechanical factors, and inappropriate timing of the intervention.

The aim of this review is to analyze the main causes of MWT failure and to propose a clinically oriented framework for improving therapeutic outcomes.

Misdiagnosis: The Leading Cause of MWT Failure

The leading cause of MWT failure is an incorrect diagnosis [10]. Not all cases of heel pain are attributable to PF; therefore, clinicians should maintain a broad differential diagnosis and consider other potential causes of this symptom. Applying MWT to an incorrect diagnosis is unlikely to produce satisfactory clinical outcomes.

Several conditions may mimic PF, including proximal or distal tarsal tunnel syndrome (TTS), heel fat pad syndrome (HFPS),

[1]Department of Orthopedic Surgery, Ankle and Foot Team, MEDS Clinic, Santiago de Chile, Chile.

Address of Correspondence
Dr. Osvaldo Valle Toledo,
Department of Orthopedic Surgery, Ankle and Foot Team, MEDS Clinic, Santiago de Chile, Chile.
E-mail: osvaldo.valle@meds.cl

Dr. Osvaldo Valle Toledo

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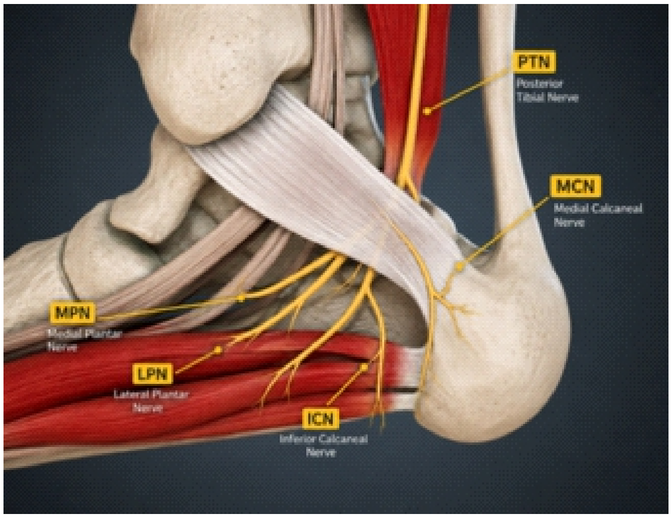

Figure 1: Medial view of the ankle with the abductor hallucis partially removed depicting the PTN branches. After giving off a small MCN branch, the PTN bifurcates into the MPN and LPN plantar nerves within the tarsal tunnel. The first branch of the LPN is Baxter's nerve also known as the inferior calcaneal nerve. PTN: Posterior tibial nerve, MCN: Medial calcaneal nerve, MPN: Medial plantar nerve, LPN: Lateral plantar nerve.

calcaneal stress fractures, bursitis, primary bone tumors, and inflammatory arthropathies. Failure to recognize these alternative diagnoses may lead to inappropriate application of MWT and suboptimal clinical outcomes, as each condition has a distinct pathophysiology and requires a specific treatment strategy.

TTS

TTS, sometimes referred to as "tibial nerve dysfunction" or "posterior tibial nerve neuralgia," is an entrapment neuropathy arising from the compression of the tarsal tunnel structures, particularly the posterior tibial nerve [11, 12].

TTS can be classified according to the anatomical site of nerve entrapment into (Fig. 1):

a) Proximal (classic) TTS, when the posterior tibial nerve is entrapped as it passes through the tarsal tunnel (beneath the flexor retinaculum).

b) Distal TTS involves the terminal branches, most commonly the first branch of the lateral plantar nerve (Baxter's nerve or inferior calcaneal nerve).

Baxter's nerve (Fig. 2) is a mixed sensory-motor nerve: It provides motor innervation to the abductor digiti minimi, occasionally to the flexor digitorum brevis, and the lateral half of the quadratus plantae. It also provides sensory innervation to the periosteum of the medial aspect of the calcaneus, the long plantar ligament, and adjacent blood vessels [11].

Compression typically occurs between the abductor hallucis fascia and quadratus plantae or near the medial calcaneal tuberosity. It is a significant and underdiagnosed cause of heel pain [13, 14].

According to etiology, compression is categorized into three origins [13, 14]:

a) Intrinsic factors

Intrinsic causes arise within the tunnel itself and include benign tumors, such as ganglions, lipomas, and neurilemmomas; muscle hypertrophy or accessory muscles, such as accessory soleus; inflammation of the tendon sheaths; and vascular malformations. Varicose veins, including those of the Lazorthes venous network, may also act as intrinsic compressive factors within the tunnel.

b) Extrinsic factors

External causes that exert pressure on the tunnel, such as sequelae of trauma (severe sprains), bone deformities (osteophytes, malunions), excessively tight footwear, fluid retention (edema), or severe hindfoot valgus. A calcaneal spur contributes to heel pain only when it compresses Baxter's nerve against the posteromedial calcaneal tuberosity and the muscle belly of the flexor digitorum brevis [15,16,17]. In most cases, however, the spur itself – regardless of its size or morphology – is not the primary source of pain. Rather, symptom severity is determined by inflammation and irritation of the surrounding soft tissues, including Baxter's nerve, rather than by the morphological characteristics of the spur.

c) Idiopathic origin

In 20–40% of cases, the cause is idiopathic.

Patients typically present with burning pain, paresthesia, or shooting pain along the medial ankle and plantar aspect of the foot [13]. Symptoms may radiate proximally toward the calf and are often exacerbated by prolonged standing or walking.

Clinical suspicion should arise in the presence of fifth-toe abduction weakness (Fig. 3), a positive Tinel sign over the medial heel, or burning pain with neuropathic characteristics [13, 14].

Magnetic resonance imaging (MRI) is useful for diagnosis, demonstrating denervation changes in the abductor digiti minimi muscle, including edema in acute cases and fatty infiltration in chronic cases [15, 16,17].

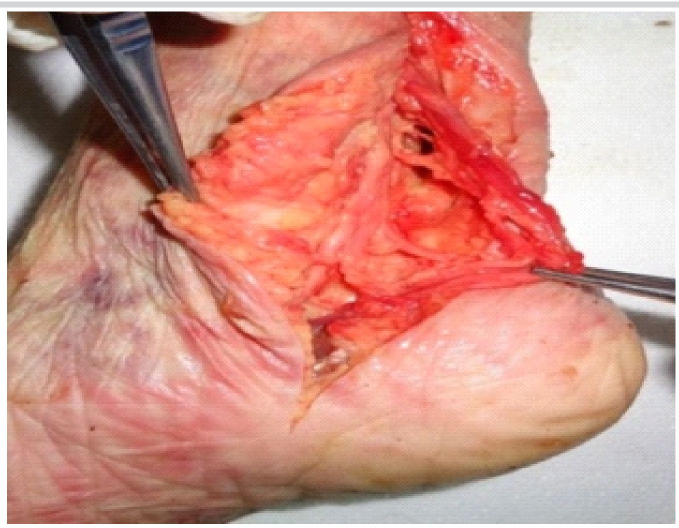

Figure 2: Anatomical variants of the posterior tibial nerve and its terminal branches: A study on cadaveric donors – a preliminary report. Admission paper for the Chilean Society of Orthopedics and Traumatology (Osvaldo Valle Toledo, MD).

Figure 3: Photograph by the author demonstrating, on the left side, an abduction deficit of the fifth toe resulting from Baxter's compressive neuropathy.

HFPS

HFPS is an underrecognized cause of plantar heel pain. It is characterized by degeneration or thinning of the fibro-adipose heel cushion; however, a variety of other pathological conditions may also affect this structure [18].

One prospective population-based cohort study – the Feet First Study – has evaluated the epidemiology of HFPS [19, 20]. The study identified HFPS as a common foot disorder among community-dwelling older adults in the United States, with a prevalence of 4.2%, making it the second most prevalent cause of heel pain after PF, which had a prevalence of 6.9% [19]. HFPS may coexist with PF [21].

Patients typically report central heel pain that worsens during barefoot walking or prolonged standing, in contrast to the "first-step pain" commonly seen in PF [20].

Ultrasound is useful for diagnosis, with a heel fat pad thickness of <3 mm supporting the condition [20, 21]. MRI may show fat pad degeneration or thinning [20].

Etiological factors include aging, repetitive overload, obesity, diabetes, rheumatologic disease, and corticosteroid injections [20].

Because ESWT does not restore fat pad structure, treatment failure is expected when HFPS is the primary diagnosis.

Heel bursitis

Bursitis around the heel can be another cause of pain [22]. These conditions are often related to mechanical overload, footwear issues, or inflammatory processes. Heel bursitis comprises three main entities:

a) Retrocalcaneal bursitis (deep posterior bursitis): The most common form, located between the Achilles tendon and the posterior aspect of the calcaneus. It is frequently associated with repetitive high-impact activities, jumping sports, insertional Achilles tendinopathy, or poorly fitting footwear.

b) Inferior calcaneal bursitis: Located beneath the calcaneus at the plantar aspect of the heel, it is typically associated with repetitive overload of the plantar heel and may coexist with PF.

c) Subcutaneous calcaneal bursitis (superficial calcaneal bursitis): Located between the skin and the Achilles tendon, it is commonly caused by repetitive friction or direct pressure, particularly from footwear.

Although heel bursitis may mimic PF, pain is typically localized to the posterior or central plantar heel rather than the medial calcaneal tuberosity, and tenderness is usually elicited directly over the affected bursa.

Calcaneal stress fracture

Calcaneal stress fractures are an important differential diagnosis of plantar heel pain and may be associated with significant bone marrow edema [22,23]. Pain is typically activity-related and worsens with weight-bearing.

Primary tumors

Although rare, primary bone tumors of the calcaneus should be considered in atypical or refractory cases. Imaging is essential for early detection [24].

Systemic diseases

Up to 15% of plantar heel pain cases may be associated with systemic inflammatory diseases [3] such as spondyloarthropathies, particularly in younger patients or those with bilateral symptoms [25]. Heel pain was the initial presenting symptom in 15.7% of a cohort of 275 patients with spondyloarthritis [25].

Timing of Treatment: Acute Versus Chronic PF

Treatment timing is another key determinant of MWT success. Clinical evidence suggests that MWT is more effective in chronic PF (symptoms persisting longer than 3 months) than in acute presentations [26,27]. This difference likely reflects their underlying biological mechanism, which primarily promotes tissue remodeling, neovascularization, and regeneration rather than acute anti-inflammatory effects [28].

In acute plantar fascia injuries or early inflammatory stages, conventional conservative management may be more appropriate as first-line therapy [27].

Inappropriate Technical Application of MWT

Inadequate technical application is a relevant and potentially preventable cause of both radial pressure waves and focused shockwave failure in plantar heel pain. Even in correctly diagnosed cases, suboptimal treatment parameters or incorrect targeting may significantly reduce clinical efficacy.

Treatment parameters

A wide variety of treatment protocols for PF have been described in the literature. For focused shock waves, the reported energy flux density ranges from 0.08 to 0.59 mJ/mm2, although low- to medium-energy protocols are most commonly used [29,30]. Energy settings for radial pressure wave therapy are even more heterogeneous. Some authors use fixed energy levels [31], whereas others recommend the highest energy tolerated by the patient [32] or progressively increase the energy level over successive treatment sessions [33].

Typically, 2,000–3,000 pulses are delivered per session for both methods, with treatment protocols consisting of three to five sessions performed at 7–10-day intervals. Treatment is generally administered at frequencies ranging from 3 to 5 Hz [29, 30, 31, 32, 33].

Targeting and vascular considerations

Both radial pressure waves and focused shock waves are generally applied over the area of maximal pain and associated myofascial trigger points. However, exclusive reliance on pain localization may not always reflect the true pathological substrate.

An alternative conceptual approach considers the vascular anatomy of the plantar fascia. The plantar fascia receives its blood supply primarily from branches of the posterior tibial artery, through the medial and lateral plantar arteries, with additional contributions from perforating branches of the dorsalis pedis artery [34].

Table 1: Mechanisms by which local anesthesia can affect the effect of mechanical waves

Mechanism	Effect
Inhibition of nociceptive signaling	Reduced neurogenic vasodilation and impaired local biological response
Loss of pain feedback	Reduced accuracy in identifying the pathological target area
Altered mechanotransduction	Potential reduction in tissue-level response to shock wave energy

The enthesis of the plantar fascia is relatively hypovascular, while surrounding perifascial tissues may demonstrate reactive hypervascularity in chronic PF. This vascular distribution may influence both disease pathophysiology and the biological response to MWT.

From this perspective, an alternative targeting strategy may involve directing mechanical pulses along vascular pathways rather than exclusively concentrating on the point of maximal pain. This hypothesis suggests that stimulation of arterial inflow may enhance the angiogenic response induced by mechanical stimulation. However, this concept remains theoretical and requires further clinical validation.

A proximal-to-distal or distal-to-proximal sweeping technique, aligned with vascular anatomy, may theoretically optimize biological stimulation, although current evidence is limited.

Local anesthesia

The use of local anesthesia during MWT has been associated with reduced clinical efficacy in PF [35]. This effect may be explained by interference with the biological mechanisms underlying mechanotransduction. Local anesthesia may negatively affect outcomes through several mechanisms (Table 1).

In addition, pain perception during treatment may serve as a useful guide for accurate targeting of pathological tissue.

Corticosteroid injections

Previous repeated corticosteroid infiltrations may negatively influence the clinical response to MWT. In particular, more than three prior injections have been identified as a negative prognostic factor for treatment success [9,36]. This may be related to corticosteroid-induced tissue degeneration and altered healing capacity.

Failure to Address Biomechanical Factors

Failure to consider underlying biomechanical dysfunction is another important cause of treatment failure in plantar heel pain.

A significant reduction in MWT success rates has been demonstrated in patients with gastrocnemius contracture or reduced ankle dorsiflexion [37,38,39]. Limited extensibility of the triceps surae increases mechanical load on the plantar fascia via the windlass mechanism and contributes to persistent symptomatology.

Accordingly, MWT should not be considered in isolation but rather as part of a comprehensive treatment strategy.

Combining MWT with eccentric strengthening and stretching of the triceps surae complex appears to improve clinical outcomes compared with MWT alone [40].

Adjunctive measures may include:

- Gastrocnemius-soleus stretching programs
- Eccentric strengthening of the triceps surae
- Plantar fascia-specific stretching exercises
- Footwear modification
- Orthotic support when indicated
- Load management strategies.

These interventions aim to reduce repetitive mechanical overload and improve long-term outcomes.

Conclusion

Successful treatment of plantar heel pain with MWT depends primarily on correct patient selection rather than the device itself.

Most cases of MWT failure can be attributed to four modifiable factors:

1. Incorrect diagnosis
2. Inappropriate timing of treatment
3. Inadequate technical application
4. Failure to address biomechanical abnormalities.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given the consent for his/ her images and other clinical information to be reported in the journal. The patient understands that his/ her names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflict of interest: Nil **Source of support:** None

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