

Shock Wave Therapy for Chronic Proximal Plantar Fasciitis

John A. Ogden, MD[‡]; Richard Alvarez, MD**; Richard Levitt, MD[†];
G. Lee Cross, MD*; and Marie Marlow, RN[‡]*

Three hundred two patients with chronic heel pain caused by proximal plantar fasciitis were enrolled in a study to assess the treatment effects consequent to administration of electrohydraulic-generated extracorporeal shock waves. Symptoms had been present from 6 months to 18 years. Each treated patient satisfied numerous inclusion and exclusion criteria before he or she was accepted into this study, which was approved by the Food and Drug Administration as a randomized, double-blind evaluation of the efficacy of shock wave therapy for this disorder. Overall, at the predetermined evaluation period 3 months after one treatment, 56% more of the treated patients had a successful result by all four of the evaluation criteria when compared with the patients treated with a placebo. This difference was significant and corroborated the fact that this difference in the results was specifically attributable to the shock wave treatment, rather than any natural improvement caused by the natural history of the condition. The current study showed that the directed application of

electrohydraulic-generated shock waves to the insertion of the plantar fascia onto the calcaneus is a safe and effective nonsurgical method for treating chronic, recalcitrant heel pain syndrome that has been present for at least 6 months and has been refractory to other commonly used nonoperative therapies. This technology, when delivered using the OssaTron (High Medical Technology, Kreuzlingen, Switzerland), has been approved by the Food and Drug Administration specifically for the treatment of chronic proximal plantar fasciitis. The results suggest that this therapeutic modality should be considered before any surgical options, and even may be preferable to cortisone injection, which has a recognized risk of rupture of the plantar fascia and recurrence of symptoms.

Chronic heel pain syndrome is a significant problem of patients with musculoskeletal conditions.^{9,14,16,39,69} The specific pathologic features responsible for any patient's symptoms are not well understood. However, thickening of the proximal fascia, decreased vascularity, peritendinous inflammation, loss of normal elasticity (tensegrity), and alteration of nociceptor physiology all may play roles in the onset and persistence of the heel pain. This pain classically is present when the patient first stands on his or her feet after awakening. It may persist during the day and may be worsened by activities of daily living such as a job or recreational activity.

From *Atlanta Medical Center, Atlanta, GA; the **Memorial Hospital, Chattanooga, TN; [†]HealthSouth Doctor's Hospital, Coral Gables, FL; and [‡]HealthTronics, Marietta GA.

The use of the OssaTron® for the treatment of proximal plantar fasciitis was approved by the Food and Drug Administration October 12, 2000.

Reprint requests to John A. Ogden, MD, Skeletal Educational Association, Inc., 3435 Habersham Road, Northwest, Atlanta, GA 30305.

The clinical diagnosis of proximal plantar fasciitis is relatively easy. However, less frequent conditions such as seronegative arthropathies and nerve entrapment syndromes should be considered in the differential diagnosis.^{16,33,35,39,40,51,60,63,69} Radiographically, a heel spur on the inferior surface of the calcaneus frequently is evident but is not considered pathognomonic of the disorder.⁵²

Although the natural history may be associated with symptomatic improvement in the absence of any intervention, most patients have sufficient pain and incapacitation that they eventually seek medical evaluation and treatment. The initial treatment should be conservative (nonoperative) with modalities such as physical therapy (especially heel cord and fascial stretching), orthotics, night splints, shoe wear modification, and nonsteroidal anti-inflammatory drugs. Most patients improve to the point of satisfaction with one or more of these interventions. Steroid injections into the painful area also have been used. However, steroid injections are associated with a significant risk of subsequent rupture of the plantar fascia, and often are followed by a recurrence of symptoms.^{1,41,60,62}

Those patients do not respond to the aforementioned treatments often resort to open or endoscopic release of a portion of the plantar fascial insertion onto the calcaneus.^{3,5-8,12,27,30,35-37,52,64} Such surgery is not without significant risk and often is associated with prolonged healing and postoperative rehabilitation, and alteration of foot biomechanical integrity.^{12,13,18,28,31,43-45,47,65,69,71}

The current clinical study was directed prospectively at an evaluation of the potential for the electrohydraulic application of extracorporeal shock waves (orthotripsy) to the pathologically altered plantar tissue to bring about pain relief and modification of the plantar fascial tissue to lessen the risk of recurrent fasciitis. The shock waves used in the current study are comparable with those currently in widespread clinical use for the fragmentation of renal and ureteral stones.¹⁷ This technologic application has been used in Europe for several

years for the treatment of numerous musculoskeletal indications.^{10,11,19-21,23,24,32,53-59,61,66}

MATERIALS AND METHODS

The current study was designed as a randomized, placebo-controlled, double-blinded evaluation to determine the safety and the effectiveness of extracorporeal shock wave therapy for the treatment of chronic plantar fasciitis. This particular study was preceded by a Food and Drug Administration-approved feasibility study that suggested that extracorporeal shock wave therapy might be a reasonable and safe therapeutic adjunct. After the approval of the feasibility study, a concise protocol was devised and accepted by the Food and Drug Administration to conduct a multicenter study involving several hundred patients who would be randomized to either an active extracorporeal shock wave therapy treatment or a placebo treatment. Treated patients could receive a second treatment if they qualified, whereas the patients who received a placebo treatment, at a predetermined time, subsequently could choose to receive one or two treatments. An additional group of patients were not randomized to appropriately allow completion of training requirements for physician investigators participating in this study program. The institutional review board of each participating institution also approved the conduct of this study.

For the current study, chronic heel pain syndrome was defined as symptoms of moderate to severe heel pain in the involved foot at the origin of the proximal plantar fascia on the medial calcaneal tuberosity. The pain must have persisted for at least 6 months before the study enrollment, and the specific diagnosis must have been made by a licensed physician and corroborated by a second physician participating as an investigator in the study protocol, and responsible for specifically evaluating the inclusion and exclusion criteria.

Among the important study inclusion criteria were requirements such as failure to respond to at least three attempts at conservative treatment, including at least two prior courses of intervention with physical therapy (stretching exercises) and the use of orthotics (heel cup, molded shoe insert, night splints), and at least one prior course of pharmacologic treatment (nonsteroidal antiinflammatory drug or cortisone injection). If the patient had a cortisone injection, extracorporeal shock wave therapy could not be given until at least 4 weeks had

elapsed since the injection, an investigator assessment of pain in the proximal plantar fascia greater than or equal to 5 on a 10 cm visual analog scale, and subject self-assessment of pain after the first 5 minutes of walking in the morning greater than or equal to 5 on the 10 cm visual analog scale. Exclusion criteria were (1) history of previous plantar fascial surgery; (2) other pathophysiologies such as seronegative arthropathies, osteomyelitis, recent trauma, or documented foot and ankle fracture; (3) neurologic, vascular or metabolic diseases, including diabetes mellitus; and (4) history of or documentation of a spontaneous or steroid-induced rupture of the plantar fascia.

A minimum of two investigators (physicians) participated at each study site so that one investigator could serve as the treatment-blinded evaluator for baseline inclusion and posttreatment followups. The actual study procedure (whether extracorporeal shock wave therapy or placebo) was done by a second physician who was aware (non-blinded) of the treatment; however, this second physician did not play a role in evaluating the patient before or after treatment, and thus was blinded to the evaluation.

The study treatment protocol was as follows: Each patient received an ankle block anesthetic (patients who received treatment) or multiple subcutaneous wheal injections (patients treated with a placebo) before the treatment. The affected leg then was draped from the direct view of the patient. Ear protection devices were used by the subject and all involved personnel. The extracorporeal shock wave treatments were applied using the OssaTron® machine (High Medical Technology, Kreuzlingen, Switzerland). This device generated the shock wave by the electrohydraulic method, which has been shown to be superior to other generation methods (electromagnetic, piezoelectric) in comparison studies for lithotripsy.¹⁷ Each study subject assigned to active treatment underwent extracorporeal shock wave therapy for a total of 1500 shocks at an 18 kV power setting. The treating physician continually manipulated the heel and foot against the therapy head throughout the shock wave applications. For those subjects assigned to placebo treatment, a styrofoam block was placed against the coupling membrane (treatment head) to absorb the shock waves by the presence of the multiple air cavities. In addition, a fluid-filled intravenous bag was placed between the styrofoam block and the subject's heel to mimic the feel of the therapy head. Subjects who received

placebo treatment did not have any coupling gel (ultrasound gel) applied to the heel, as did the patients who receive active treatment. Patients who received the placebo treatment also had 1500 shocks delivered at 18 kV, effectively duplicating the duration and noise of active treatment. Patients who received the placebo treatment and patients who received the active treatment were kept apart in the recovery room to negate any discussions about concepts of what occurred in the treatment room.

All subjects then were followed up with a detailed evaluation (assessment questionnaires and physical examinations, including a dolorimeter) at 4, 8, and 12 weeks after treatment. An initial success or fail status was assigned based on the subjective and objective results 12 weeks after treatment. The 12-week interval was selected because it was expected that the healing process would likely be evident within this interval (although not necessarily complete). Additionally, because of the well-described natural history of proximal plantar fasciitis, it was expected that symptoms of chronic heel pain still could resolve with time in some of the subjects during the continued course of study participation, even in this selected treatment patient population in whom multiple previous nonoperative treatments had failed. It was perceived that status assignment at the 12-week interval could help differentiate improvement because of spontaneous healing from actual treatment effect.

At the 12-week followup study subjects were assigned a success or failure status according to the following four criteria: (1) Investigator assessment of pain: Minimum 50% improvement over the baseline, with a visual analog scale score of 4.0 or greater. This criterion was subjective, based on the use of a dolorimeter that was applied to the same pressure reading after treatment as had been recorded during the pretreatment evaluation; (2) Study subject's self-assessment of pain on first walking in the morning: Minimum of 50% improvement over pretreatment baseline and a visual analog scale of 4.0 or greater. According to these 1 and 2 scores, a patient with a baseline visual analog scale greater than 8.0 who achieved a 50% improvement would not be considered to have a successful result in a grading category, because it would be greater than 4.0; (3) Subject's self-assessment of activity. This measured the distance and time the subject was able to walk without heel pain. The study subject had to show improvement of one point or more on a five-point scale, or main-

tain a 0/1 baseline level (no pain or minimal pain); and (4) Use of pain medications: No prescription analgesics were given after treatment. If the patient self-treated with over-the-counter analgesic medications, this was documented with a medication log returned at each monthly evaluation. To be assigned a successful result, the patient could not have taken any such medication for heel pain in the treated heel between 10 and 12 weeks after treatment.

The aforementioned success criteria were applied rigidly. Subjective symptomatic improvement of any kind did not necessarily constitute success. Each study subject had to meet all four success criteria to attain an overall status assignment of success at the stipulated 12-week evaluation.

To determine the durability (longevity) of any successful outcome, all subjects assigned a success status at 12 weeks after treatment were required to continue followup at 6, 9, and 12 months (long-term followup data still are incomplete). Any study subjects who were assigned a 12 week status of fail were informed that they could withdraw from the study to pursue alternative treatment modalities except extracorporeal shock wave therapy or continue in the study by trying additional treatments as allowed by the study protocol. If the study subject chose to continue study participation, he or she was informed that a possible additional treatment would be active extracorporeal shock wave therapy. Thus, a patient who received an actual treatment, but did not attain all four success criteria, could undergo a second treatment. Study subject who received the placebo, and who did not achieve the required four success criteria then could enter the treatment arm to receive at least one, and in certain cases, two treatments with detailed evaluation under the same criteria as those study subjects receiving the extracorporeal shock wave therapy primarily. All evaluations of retreatment or placebo-to-treatment were done by the same protocol as the primary treatment and placebo study arm.

Seven investigational sites participated in the study including Memorial Hospital, Chattanooga, TN; Atlanta Medical Center, Atlanta, GA; Doctor's Hospital, Coral Gables, FL; American Sports Medicine Institute, Birmingham, AL; Washington University School of Medicine, St Louis, MO; University of Rochester School of Medicine, Rochester, NY; and Baylor College of Medicine, Houston, TX. At all institutions the study was conducted through

the Department of Orthopaedics and had local institutional review board approval.

RESULTS

Three hundred two patients were involved in the current study and data analysis including 260 randomized study subjects and 42 nonrandomized subjects enrolled to accomplish investigator training requirements. Five of the 302 subjects (1.7%) withdrew from the study or were lost to followup before the 12-week determination visit. One of the five was a nonrandomized subject, although the remaining four were randomized. Of these four patients, one withdrew because of acute symptoms of a herniated disc that required treatment with medications that would affect one of the success criteria. The remaining three patients were lost to followup. Thus, 256 randomized and 41 nonrandomized subjects were assigned a success or fail status based on the 12-week followup findings.

The patient population was predominantly female (65.9%). The age of the study subjects at the time of enrollment was 20 to 79 years (mean, 49.6 years). Subjects were predominantly Caucasian, although Black and Asian individuals did participate.

Duration of Symptoms

The mean duration of symptoms for randomized subjects having active treatment was 968 days (2.65 years), with a range of 6 months to 13 years. The mean duration for the randomized subjects who received placebo treatment was 1078 days (2.95 years), with a range of 6 months to 18 years. The mean symptom duration for the nonrandomized subjects was 943 days (2.58 years), with a range of 6 months to 10 years. This latter group of patients was treated in the presence of physicians who subsequently would be treating patients in the randomized study so that they would be familiar with the treatment methods.

Radiographic Assessments

Eighty-seven of the patients who received active treatment and 88 of the patients who re-

ceived placebo treatment had heel spurs evident on at least one of three standard views (anteroposterior, lateral 45° medial oblique). Thirty-two nonrandomized subjects had similar findings. No study subject had any change in the appearance of the heel spur at 12 weeks. No other radiographic changes were evident. One patient who withdrew from the study because of continued and changed pain did not have pain at the treatment site (physician dolorimeter assessment), but had pain with subtalar motion. A magnetic resonance (MR) image revealed a fluid-filled cyst at the superior surface of the calcaneus, with a pathologic fracture and fluid extravasation into the subtalar joint. The patient has undergone curettage and grafting with relief of symptoms. Coincidentally, the patient has remained symptom-free.

Investigator Heel Pain Assessment

The blinded investigator used a pressure sensor to record and document the amount of pressure that when applied to the site of maximum sensitivity, elicited the baseline visual analog scale response. At each subsequent evaluation after treatment, the same baseline pressure was applied and the study subject was asked to evaluate the pain by the visual analog scale response, thus ensuring consistency of objective pressure evaluation while allowing for subjective evaluation of changes in pain perception after treatment. For the 130 subjects randomized to active treatment who had reported a pain score of 5.0 or greater on a 10 cm visual analog scale, no matter what the dolorimeter measurement (which served as control by repetition), the mean baseline visual analog scale score was 7.76. The mean visual analog scale score for patients randomized to placebo treatment was 8.02, and the mean baseline score for the nonrandomized subjects was 8.08. The randomized subjects who received active treatment had an improvement from a mean baseline of visual analog scale 7.68 to 3.13 at 12 weeks. The median visual analog scale was 1.90. Seventy-four (62.2%) of the patients met this success criteria. The randomized subjects who received placebo treatment had an im-

provement in the baseline score from 7.87 to 4.37 at 12 weeks, with a median of 4.70. Fifty-one (43%) of the patients who received placebo treatment met the success criteria. The nonrandomized subjects had an improvement from 8.05 to 2.01, with a median visual analog scale score of 0.80. Thirty-two (80%) subjects met this success criterion.

Subject Self-Assessment of Pain

For the subjects randomized to active treatment, the mean baseline visual analog scale score was 8.07. The mean baseline score of the subjects who received placebo treatment was 8.20. The mean baseline for the nonrandomized subjects was 6.84. Patients who received active treatment had an improvement in the visual analog scale from 8.02 to 3.48 (median visual analog scale, 2.60). Seventy-one (59.7%) subjects had a successful result. Subjects who received placebo treatment had an improvement in the visual analog scale from 8.14 to 4.20 (median visual analog scale, 4.05). Fifty-six (48.2%) patients met the success criterion. The nonrandomized subjects had an improvement in the visual analog scale from 6.86 to 2.10 (median visual analog scale, 1.50). Twenty-nine (70.7%) patients met the success criterion.

Subject Self-Assessment of Activity

Subjects receiving active treatment had an improvement in baseline score from 3.49 to 1.72 (median score, 1.00). The patients who received placebo treatment had an improvement from 3.53 to 1.88 (median score, 2.00). The nonrandomized subjects had an improvement from 2.63 to 0.85 (median, 0.00).

Use of Pain Medications

At baseline, 89.07% of the patients who received active treatment routinely were taking medication for pain, which decreased to 30.25% at 12 weeks. Thus, 69.74% of the subjects who received active treatment met the success criteria for this parameter. In contrast, 34.65% of the patients who received placebo treatment met this success criterion. Of the randomized sub-

jects, 80.5% met this criterion. To meet this criterion, the study subject could not be taking any over-the-counter or prescribed analgesic or antiinflammatory drugs at 12 weeks.

Report Procedures

Twenty-nine (22.3%) of the subjects who received active treatment, based on the extent of improvement, chose to undergo a second extracorporeal shock wave treatment. Of the subjects who received placebo treatment, 56 (43%) subsequently elected to have an active treatment. Of this patient cohort, 14 (24%) had a second treatment. Of the nonrandomized subjects, 10 (23.8%) elected to have a second treatment procedure.

Of the subjects randomized to active extracorporeal shock wave therapy, 47.06% met all four rigid success criteria, compared with 30.17% of the subjects who received placebo treatment. The *p* value for primary treatment comparison (active treatment versus placebo) was $p = 0.008$ (statistically significant). The success rate in subjects who received active treatment at 12 weeks was 56% higher than the success rate for patients who received placebo treatment. Of the nonrandomized subjects, 58.54% met all four success criteria.

Of the patients randomized to placebo treatment, 69.83% did not meet the four success criteria. Of these patients, 70% subsequently elected to have an active treatment, with 63.5% of these achieving a successful result at 12 weeks.

Thirty-eight complications occurred in the 302 patients who were treated. Eighteen of the 38 complications occurred in patients who received active treatment. Eight complications were related to the procedure, whereas 10 were unrelated to the procedure. Thirteen of the subjects who received placebo treatment experienced five related and eight unrelated events. An additional two complications (one related, one unrelated) occurred in the subset of patients who received placebo treatment who subsequently elected an actual treatment. Five adverse events occurred in the nonrandomized cohort: four related and one unre-

lated to the extracorporeal shock wave therapy. The most frequent procedure related complications in all three groups were pain after treatment and mild neurologic symptoms (numbness, tingling). One patient who received active treatment sustained a plantar fascial tear in the course of a vigorous activity 4 weeks after extracorporeal shock wave therapy; she had undergone multiple cortisone injections, with the most recent being 5 weeks before extracorporeal shock wave therapy.

Although only the randomized subjects were used for the statistics submitted to the Food and Drug Administration, the results may be assessed in another, more practical matter of clinical relevance. The first two categories (physician assessment and subject self-assessment of morning pain) could be a failure if the patient had a visual analog scale score greater than 8, but still had a 50% improvement. Such an improvement would be greater than 4, and thus was considered a failure by the data collection standards. Additionally, many patients were happy with the results of active treatment, as reflected by the smaller percentage of active patients requesting a second treatment compared with the patients treated with a placebo who requested an active treatment. Also, nonrandomized subjects who received active treatment were not included in the statistics. Accordingly, all patients who were treated were grouped together and then rated as having excellent results (four of four success criteria), good results (two of four or three of four success criteria), fair results (one of four success criteria), and poor results (zero of four success criteria). Using this grading system, 15% of patients had excellent results and 25% had good results, for a combined total of 76% of patients satisfied with the outcome, even when pain relief was not complete. Ten percent of patients had fair results and 14% had poor results, for a total of 24% of patients who were not satisfied with the outcome. The success rates were better in the second ½ of the study period, which probably reflected a relative learning curve on the part of the physician administering the treat-

ments, particularly relative to manipulation of the foot against the rubber membrane while the shock waves were delivered actively.

DISCUSSION

The plantar fascia is a dense band of fibers originating from the medial portion of the calcaneus and subsequently inserting and comingling with the various tissues of the forefoot.^{4,46} The fascia divides into medial, central, and lateral components.⁴² The central portion is the usual site of pathologic disorders.^{4,42} Histologically, the extracellular matrix within the plantar fascia is comprised of collagenous and elastic fibers. The elastic fibers are present in longitudinal strands and in wavy, bundled networks.⁴ These elastic fibers may alter orientation from wavy to straight under increasing amount of acute and chronic loading, leading to stiffening of the fascia. Interestingly, relative to the treatment method covered in the current study, heel strike while walking or running is associated with naturally produced shock waves.¹⁵ The heel pad and plantar fascia are integral to the dampening of these natural shock waves.⁵¹

Several authors promulgate the concept of the plantar fascia as a significant component of longitudinal arch integrity throughout the normal range of activity of the foot and ankle.^{4,29} This is in accord with the biologic and mechanical concept of tensegrity, the integration of tensile and compressive forces within a given structure during static (standing), and dynamic (running) loading.⁴⁶ Mechanistically, Hicks²⁶ appears to be the first to describe the windlass mechanism by which passive dorsiflexion causes the medial longitudinal arch to rise, the hind foot to supinate, the leg to externally rotate, and the plantar fascia to become more tense than when the foot and toes are in neutral.

Several investigators have showed a loss of this windlass mechanism after a complete plantar fascial release (resection), which led to a consistent decrease in height of the longitudinal arch, an increase in length of the longitudinal arch, and increased forefoot abduc-

tion.^{31,65} They implied that foot instability (and presumably chronic pain) would ensue after complete plantar fascial release. Certainly these same biomechanical changes would be expected to follow after rupture of the plantar fascia, whether spontaneous or consequent to cortisone injection.^{1,34} Unfortunately, no comparable studies have been reported that assess whether such changes (mechanically or symptomatically) occur after the partial plantar fascial release the currently is advocated, either in feet from cadavers or in patients who have had such surgery. There is a need for dynamic, functional studies to complement the theoretical studies that principally have relied on in vitro methodology.^{4,48}

Dysfunction in the plantar fascia attributable to any number of primary or secondary causes may lead to acute or chronic heel pain. Patients with undiagnosed heel pain represent as many as 20% of patients presenting to a physician for the diagnosis and treatment of foot problems. Although proximal plantar fasciitis undoubtedly is the most frequent diagnosis (cause) of inferomedial heel pain, other etiologies may be causal.⁶⁹ Hendrix et al²⁵ thought that the etiology of intractable heel pain after conservative treatment often was attributable to tarsal tunnel syndrome. Accordingly, it is imperative to undertake a detailed history, physical examination, and relevant diagnostic studies to ensure an accurate diagnosis. The exclusion criteria in this prospective study were directed at ascertaining the presence of any of the aforementioned potential causes of heel pain, because the authors thought they obviously might adversely affect outcome. Although many etiologies are described, it must be remembered that 90% of heel pain is attributable to proximal plantar fasciitis, and only 10% is attributable to the other etiologies. Two patient cohorts seem to have a particularly high incidence of plantar fasciitis, obese, middle-aged women, and young, male runners. The current inclusion and exclusion criteria were extremely strict compared with many previous studies of patients with chronic heel pain.^{3,5,8,13,27,28,30,35,41,60,67} Potential study subjects were assessed carefully to exclude those

with symptoms or physical findings consistent with any other heel pain diagnosis.

An MRI scan of the normal plantar fascia, which may be observed from studies done for other pathologic conditions of the foot, shows a thin, well-delineated structure with a low signal intensity. The central cord has a consistent low signal throughout, whereas the medial and lateral bands have a more variable appearance. In plantar fasciitis, there is marked thickening of the proximal segment of the central cord.⁶⁸⁻⁷¹ Areas of high signal intensity on T2-weighted images within the fascia, edema at the fascia-muscle and fascia-fat interfaces and edema in the adjacent subcutaneous fat all are manifestations of the presumed inflammatory process that is characteristic of plantar fasciitis. Maffuli and coworkers³⁸ strongly suggested the term tendinopathy, which is more inclusive of changes within the tendon (or fascia) and external to the structure, with the deletion of the suffix *itis*, which implies only inflammation. Currently, certain drugs (nonsteroidal antiinflammatory drugs) may temporarily alleviate the inflammatory component of plantar fasciitis. But without correction or modification of the internal fascial changes manifested as marked thickening on the MRI scan, the inflammation can readily recur. The shock waves are directed at controlled internal fascial tissue microdisruption that initiates a more appropriate healing response within the fascia and a better long-term capacity to adapt to biologic and biomechanical demands.

Gibbon and Long¹⁹ assessed the plantar fascia with ultrasound. They found significant thickening in patients with unilateral and bilateral plantar fasciitis. In patients with unilateral plantar fasciitis the contralateral side did not show thickening. Patients with fasciitis had abnormal plantar aponeurosis echogenicity in 78% and bone spurs in 24%. Peritendinous edema was present in 5% of all symptomatic heels and intratendinous calcification was present in 3%. All patients who had calcifications had previous cortisone injections.

After an appropriate evaluation of a patient to determine that he or she has heel pain as a

consequence of proximal plantar fasciitis, there is no argument that the initial treatment program should be conservative (nonsurgical), an approach that may be successful in as many as 90% of patients presenting with acute symptoms by bringing significant, if not complete, relief of such symptoms.^{49,50,67} However, there is no consensus or overwhelming support for one specific treatment protocol compared with any other treatment protocol. Additionally, there are variable inconsistencies in the application and duration of any given therapy by an individual practitioner. Some practitioners apply therapies one at a time in a layered approach, whereas others apply multiple therapies at the same time. The usual initial treatment methods include nonsteroidal antiinflammatory drugs, physical therapy directed at stretching the Achilles tendon and plantar fascia or deep plantar muscles, heel cups, shoe modifications, custom fabricated orthoses, night splints, serial casting, and cortisone (by direct injection or passively by iontophoresis). When such methods fail, surgery usually is recommended. The latter may be an open or endoscopic procedure. The current study supports the use of extracorporeal shock wave therapy as a reasonable noninvasive treatment before any decisions about surgical intervention are made.

Unfortunately, studies of nonoperative methods have been difficult to assess statistically. Many studies investigators multiple and variable nonsurgical regimens within the same study.^{12,30,33,39,49} Other investigators have treated patients with heel pain without specifying any limitation to patients who only have documented insertional (proximal) plantar fasciitis.^{16,25,63} Martin et al³⁹ reviewed numerous nonsurgical studies for plantar fasciitis and showed a wide variation in acceptable outcome ranging from 44% to 82% (average, 60.3%) for complete relief of heel pain. However, all of these cited studies had multiple therapeutic methods. None of the nonoperative treatments have been analyzed with double blind studies.

The study reported by Martin et al³⁹ analyzed 237 patients treated with a regimen that

included noninflammatory nonsteroidal drugs, an exercise program with emphasis on heel cord stretching, daily use of night splints, and a heel cup or other type of orthosis when ambulatory. If the patient did not have significant improvement after 4 months, a steroid injection was offered. At 6 months, if there still was no acceptable improvement, surgery was offered. When these patients were analyzed at 6 months, 51% did not have pain, 34% had intermittent pain, and 14% had continued, constant pain. Regular use of night splints was statistically significant with a good outcome, whereas stretching exercises and use of a heel cup were not. The authors thought that their results (51% of the patients attained complete relief of their heel pain) were different from other reported studies because the subject population had more chronic disease and, thus, a poorer prognosis than the patients with more acute symptoms. In the study of Martin et al,³⁹ 29.7% of the patients had symptoms for more than 12 months and 53.6% had symptoms for more than 6 months. The duration of symptoms in their patients and the percentage of patients with asymptomatic relief were comparable with the results of the current shock wave therapy study. Interestingly, in the study of Martin et al,³⁹ although only 51% of patients were completely asymptomatic, 81.8% were satisfied with the final outcome relative to the amount of residual pain. Only a small number of the patients who still had pain agreed to additional therapy after 6 months. The authors found similar outcome perceptions by the patients who were treated with extracorporeal shock wave therapy.

An alternative to continued noninvasive therapy that many physicians advocate is the use of cortisone. The medication usually is given by injection into the painful region, but also may be delivered by iontophoresis. The use of such medication, as with nonsteroidal antiinflammatory drugs, is based on the premise that plantar fasciitis is an inflammatory disorder. Recent studies with MRI corroborate an inflammatory component to plantar fasciitis and changes within the fascia.⁶⁹⁻⁷¹ However,

injections are not without complications. Acevedo and Beskin¹ evaluated 39 patients who had rupture of the plantar fascia after injection. The majority of these patients had significant pain relief after rupture, not unlike the expected relief after surgical release. However, these patients, when followed up for a sufficient period after fascial rupture, subsequently had longitudinal arch strain, lateral and dorsal midfoot strain, lateral plantar nerve dysfunction, stress fracture, hammertoe deformity, swelling, and antalgic gait.

Most authors agree that subjects with insertional plantar fasciitis have a self-limited disease and that most will attain good results without significant intervention (injections or surgery). The current study was designed specifically to incorporate such a potential effect on the outcome. Even though the study subjects had to have failed at least three therapeutic interventions, there still was a relatively high placebo effect. However, when the outcome of treated patients was compared with the outcome of patients treated by placebo, the difference was statistically significant ($p = 0.008$), with 56% more of the treated patients attaining all success criteria compared with the patients treated by placebo.

Surgical release of the plantar fascia generally is reserved for those patients who have not responded to other conservative endeavors. Such surgery may be open, generally with a partial transection of the central portion, or may be accomplished endoscopically. Nerve decompression also may be done during such a procedure. Davies et al¹³ reviewed 43 patients (47 heels) who underwent open surgery. Overall, only 20 of 41 patients were totally satisfied with the outcome.¹³ They thought that patients needed to know that the average recovery time after surgical release could be prolonged. In most studies recovery is prolonged, as much as 8 months after surgery.¹³

Surgery also may be associated with complications. Patients have had stress fractures develop after endoscopic surgical release.^{28,60} Barrett et al,⁵⁻⁷ stressed a high level of complications in those patients who eventually had

surgery. In one study, only nine of 20 patients had complete pain relief, with no description of how long it took to attain such relief.⁴⁷

In a study of asymptomatic patients who previously had undergone open or endoscopic plantar fascial release, the MRI scan obtained after surgery revealed significant findings.⁷⁰ The average thickness of the plantar fascia remained two to three times the normal size. Additionally, although there was increased thickness, changes in morphologic features (persistent surgical defect), and changes in signal intensity within the fascia within the surgical site, similar changes were more prominent at the enthesis. In contrast, there was complete resolution of edema in and around the plantar fascia postoperatively. It seems that after surgery the plantar fascia does not revert to its normal appearance despite resolution of the inflammatory component. The fascia remains thickened and has indistinct superficial (fat) and deep (muscle) tissue margins compatible with perifascial fibrosis.

Because of the chronic nature of the subject's symptoms, the expectations for complete pain relief may be low. In many studies patients with intermittent discomfort after any therapeutic intervention may perceive their symptoms as mild or minimally interfering with their activities of daily living. Most patients with chronic heel pain would rather tolerate occasional discomfort than undergo invasive procedures such as surgery. The authors think this was a factor in many of the subjects who were studied.

Rupture of the plantar fascia is a significant complication in patients who were previously symptomatic or asymptomatic. The process may occur spontaneously and usually is associated with running and jumping sports.^{2,33,46,52} Rupture may occur after partial surgical release. Rupture also may occur as a relatively frequent complication after corticosteroid injection.^{1,40,60} The one patient in the current study who had a fascial rupture 4 weeks after extracorporeal shock wave therapy (confirmed by MRI) had three cortisone injections with the last one being given 5 weeks before extracor-

poreal shock wave therapy. Because this was the only patient to have such a rupture and the complication has not been reported after more than 1000 extracorporeal shock wave therapy cases in Europe and Asia, it seems probable that the cortisone injection was the most likely cause of the complication.^{10,11,32,55-59,61}

Because of the recognized risks and delayed healing often associated with surgery, alternative nonoperative therapeutic methods have been assessed. Particularly in Europe, since 1992, the potential use of shock waves for various musculoskeletal conditions has been assessed and has become widespread as nonoperative, noninvasive treatment for chronic conditions such as calcific tendinitis of the shoulder, lateral epicondylitis (tennis elbow), plantar fasciitis, and fracture nonunions.^{10,11,19,20,22,23,31,53-57,59} The technology of applying extracorporeal shock waves is similar to that used for lithotripsy.¹⁷ Several devices have been designed specifically for the treatment of musculoskeletal conditions. These devices generate the shock waves by one of three basic methods: electrohydraulic, electromagnetic, and piezoelectric. Comparison studies with lithotripters describe the electrohydraulic technology as the gold standard.¹⁷ The machine used for this musculoskeletal study used electrohydraulic shock wave generation. Comparison studies of machines used for orthotripsy have not been done, so there is no information concerning the efficacy of one type of shock wave generation over any other for musculoskeletal tissue applications. However, electrohydraulic treatment is based on one treatment, whereas other electromagnetic and piezoelectric devices routinely require multiple treatments. Electrohydraulic shock waves often are described as high energy waves, whereas electromagnetic and piezoelectric machines generate low energy waves.

Several authors have applied shock waves to the treatment of plantar fasciitis.^{31,53-57} These studies generated the shock waves electromagnetically. The protocols varied. Some included controls in which the control subject received a limited number of shocks (10 to 100), as opposed to patients who received

treatment with 600 to 1000 shocks. Treatments were given three times, with a week interval between each set of shocks. All ratings before and after treatment were subjective, and put a greater emphasis on improvement, rather than complete relief. The outcome criteria in each of these studies were much less restrictive than in the current study. In one study, 84% of the group of patients treated with 600 shocks (three doses) had an improvement whereas 64% of the patients in the control group treated with 100 shocks (three doses) had an improvement.³¹ In the other four studies, the control subjects received 10 to 30 shocks, whereas the patients who received active treatment had 1000 shocks at each of the three doses. The control subjects knew which study arm they were in, thus potentially biasing their results. Improvement rates in the patients who received active treatment varied from 40% to 84%.

Hammer et al²⁰ treated 44 patients with chronic plantar fasciitis with piezoelectric shock wave generation. There were no control subjects. In 24 patients (56%), the visual analog scale improved by 61% to 100%. Only 13 patients (29%) rated the outcome as excellent, which would be comparable with four of four success criteria in the current study.

Treatment with high energy (electrohydraulic) shock waves requires an anesthetic. In the current study, patients arbitrarily received an ankle block. Some patients only received local injection. Patients receiving treatment with low energy machines usually do not require anesthetic agents; however, one study stated the low energy treatment was "considered to be unpleasant by all patients."⁵⁷

In the current study, retreatment (a second treatment between 14 and 16 weeks after the first treatment) appeared to offer an improved outcome in subjects who initially did not meet all four success criteria. Of the 21 patients receiving a second treatment, 52% met all four success criteria and 81% met the objective (dolorimeter) success criteria for investigator assessment of pain. Of interest was the lower than expected rate of retreatment in the active

treatment arm of the study. The study was designed on the assumption that the majority of subjects who did not respond to primary treatment would elect to have a second treatment. However, only 29 of the 63 subjects who did not meet all four success criteria chose retreatment (46%), compared with 56 of 81 patients who received the placebo treatment (70%) who chose retreatment. This observation suggests that some of the subjects assigned a final fail status may have been sufficiently satisfied with their outcome.

References

1. Acevedo JI, Beskin JL: Complications of plantar fascia rupture associated with corticosteroid injection. *Foot Ankle* 19:91-97, 1998.
2. Ahstrom JP: Spontaneous rupture of the plantar fascia. *Am J Sports Med* 16:306-307, 1988.
3. Apsalet I, Myhre T, Finsen V: Operative treatment of plantar fasciitis. *Foot* 10:87-89, 2000.
4. Aquino A, Payne C: Function of the plantar fascia. *Foot* 9:73-78, 1999.
5. Barrett SL, Day SV: Endoscopic plantar fasciotomy for chronic plantar fasciitis/heel spur syndrome: Surgical technique—Early clinical results. *J Foot Ankle Surg* 30:568-573, 1991.
6. Barrett SL, Day SV: Endoscopic plantar fasciotomy: Two partial endoscopic surgical techniques: Clinical results of 65 procedures. *J Foot Ankle Surg* 32:248-255, 1993.
7. Barrett SL, Day SV, Ignetti T, et al: Endoscopic plantar fasciotomy: A multisurgeon prospective analysis of 652 cases. *J Foot Ankle Surg* 34:400-411, 1995.
8. Benton-Weil W, Borrelli AH, Weil Jr LS, et al: Percutaneous plantar fasciotomy: A minimally invasive procedure for recalcitrant plantar fasciitis. *J Foot Ankle Surg* 37:269-272, 1998.
9. Berkowitz JF, Kier R, Rudicel S: Plantar fasciitis: MR imaging. *Radiology* 179:665-667, 1991.
10. Buch M, Siebert W: Shock Wave Treatment for Heel Pain Syndrome: A Prospective Investigation. In Coombs R, Schaden W, Zhou SS (eds). *Musculoskeletal Shockwave Therapy*. London, Greenwich Medical Media 73-77, 2000.
11. Coombs R, Schaden W, Zhou SS (eds): *Musculoskeletal Shockwave Therapy*. London, Greenwich Medical Media 2000.
12. Daly PJ, Kitaoka HB, Chao EY: Plantar fasciotomy for intractable plantar fasciitis: Clinical results and biomechanical evaluation. *Foot Ankle* 13:188-195, 1992.
13. Davies MS, Weiss GA, Saxby TS: Plantar fasciitis: How successful is surgical intervention? *Foot Ankle* 20:803-807, 1999.
14. DeMaio M, Paine R, Mangine RE, et al: Plantar fasciitis. *Orthopedics* 16:1153-1163, 1993.
15. Dickinson JA, Cook DA, Leinhardt TM: The measurement of shock waves following heel strike while running. *J Biomech* 18:415-422, 1985.

16. Furey JG: Plantar fasciitis: The painful heel syndrome. *J Bone Joint Surg* 57A:672-673, 1975.
17. Fuselier HA, Prats L, Fontenot C, et al: Comparison of mobile lithotripters at one institution: HealthTronics LithoTron™, Dornier MFL-5000 and Dornier DoLi. *J Endourol* 13:539-542, 1999.
18. Gentile AT, Zizzo CJ, Dahukey A, et al: Traumatic pseudoaneurysm of the lateral plantar artery after endoscopic fasciotomy. *Foot Ankle* 18:821-823, 1997.
19. Gibbon WW, Long G: Ultrasound of the plantar aponeurosis (fascia). *Skeletal Radiol* 28:21-26, 1999.
20. Hammer DS, Rupp S, Ensslin S, et al: Extracorporeal shock wave therapy in patients with tennis elbow and painful heel. *Arch Orthop Trauma Surg* 120:304-307, 2000.
21. Haupt G, Haupt A, Ekkernkamp A, et al: Influence of shock waves on fracture healing. *Urology* 39:529-532, 1992.
22. Hedrick MR: The plantar aponeurosis. *Foot Ankle* 17:646-649, 1996.
23. Heilbig K, Schostok T, Brown M, et al: Correlations Between Pain and Success. In Coombs R, Schaden W, Zhou SS (eds). *Musculoskeletal Shockwave Therapy*. London, Greenwich Medical Media 43-48, 2000.
24. Heller KD, Niethard FU: Der Einsatz der extrakorporellen stoßwellentherapie in der orthopädie: ein metaanalyse. *Z Orthop Ihre Grenzgeb* 136:390-401, 1998.
25. Hendrix CL, Jolly GP, Garbalosa JC, et al: Entrapment neuropathy: The etiology of intractable chronic heel pain syndrome. *J Foot Ankle Surg* 37:273-279, 1998.
26. Hicks JH: The mechanics of the foot: II. The plantar aponeurosis and the arch. *J Anat* 88:25-31, 1954.
27. Hofmeister EP, Elliott MJ, Juliano PJ: Endoscopic plantar fascia release: An anatomical study. *Foot Ankle* 11:719-723, 1995.
28. Jerosch J: Endoscopic release of plantar fasciitis: A benign procedure? *Foot Ankle* 21:511-513, 2000.
29. Kim W, Voloshin AS: Role of plantar fascia in the load bearing capacity of the human foot. *J Biomech* 28:1025-1033, 1995.
30. Kinley S, Frascione S, Calderone D, et al: Endoscopic plantar fasciotomy versus traditional heel spur surgery: A prospective study. *J Foot Ankle Surg* 32:595-601, 1993.
31. Kitaoka HB, Luo ZP, An KN, et al: Mechanical behavior of the foot and ankle after plantar fascia release in the unstable foot. *Foot Ankle* 18:8-15, 1997.
32. Krischek O, Rompe JD, Herbsthofer B, et al: Symptomatische niedrigenergetische stoßwellentherapie bei fersenschmerzen und radiologisch nachweisbarem plantaren fersensporn. *Z Orthop Ihre Grenzgeb* 135:169-174, 1998.
33. Kwong PK, Kay D, Voner ET, et al: Plantar fasciitis: Mechanics and pathomechanics of treatment. *Clin Sports Med* 7:119-126, 1988.
34. Leach R, Jones R, Silva T: Rupture of the plantar fascia in athletes. *J Bone Joint Surg* 60A:537-539, 1978.
35. Leach RE, Seavey MS, Salter DK: Results of surgery in athletes with plantar fasciitis. *Foot Ankle* 7:156-161, 1986.
36. Lester DK, Buchanan JR: Surgical treatment of plantar fasciitis. *Clin Orthop* 186:202-204, 1984.
37. Lewis G, Gatti A, Barry LD, et al: The plantar approach to heel surgery: A retrospective study. *J Foot Surg* 30:542-546, 1991.
38. Mafulli N, Khan KM, Puddu G: Overuse tendon conditions: Time to change a confusing terminology. *Arthroscopy* 14:840-843, 1998.
39. Martin RL, Irrgang JJ, Conte SF: Outcome study of subjects with insertional plantar fasciitis. *Foot Ankle* 19:803-811, 1998.
40. McCarthy DJ, Gorecki GE: The anatomical basis of inferior calcaneal lesions: A cryomicrotomy study. *J Am Podiatr Med Assoc* 69:527-536, 1979.
41. Miller RA, Torres J, McGuire M: Efficacy of first-time steroid injection for painful heel syndrome. *Foot Ankle* 16:610-614, 1995.
42. Mitchell IR, Meyer C, Krueger WA: Deep fascia of the foot: Anatomical and clinical considerations. *J Am Podiatr Med Assoc* 81:373-378, 1991.
43. Murphy GA, Pneumaticos SG, Kamaric E, et al: Biomechanical consequences of sequential plantar fascia release. *Foot Ankle* 19:149-152, 1998.
44. Nierenberg G, Hoffman A, Engel A, et al: Pseudoaneurysm with an arteriovenous fistula of the tibial vessels after plantar fasciotomy: A case report. *Foot Ankle* 18:524-527, 1997.
45. O'Brien SP, Simoni EJ, Jain KM, et al: Arteriovenous fistula and pseudoaneurysm formation following heel endoscopy. *Eur J Endovasc Surg* 13:240-242, 1997.
46. Ogden JA: *The Foot*. In *Skeletal Injury in the Child*. Ed 3. New York, Springer Verlag 1091-1119, 2000.
47. O'Malley MJ, Page A, Cook R: Endoscopic plantar fasciotomy for chronic heel pain. *Foot Ankle* 21:505-510, 2000.
48. Pai VS: Rupture of the plantar fascia. *J Foot Ankle Surg* 35:39-40, 1996.
49. Pfeffer G, Bachetti P, DeLand J, et al: Comparison of custom and prefabricated orthoses in the initial treatment of proximal plantar fasciitis. *Foot Ankle* 20:214-219, 1999.
50. Powell M, Post WR, Keener J, et al: Effective treatment of chronic plantar fasciitis with dorsiflexion night splints: A crossover prospective randomized outcome study. *Foot Ankle* 19:10-18, 1998.
51. Prischasuk S: The heel pad in plantar heel pain. *J Bone Joint Surg* 76B:140-142, 1994.
52. Prischasuk S, Subhadrabandhu T: The relationship of pes planus and calcaneal spur to plantar heel pain. *Clin Orthop* 306:192-196, 1994.
53. Reeve F, Laughlin RT, Wright DG: Endoscopic plantar fascia release: A cross sectional anatomic study. *Foot Ankle* 18:398-401, 1997.
54. Rolf C, Guntner P, Ericssater J, et al: Plantar fascia rupture: Diagnosis and treatment. *J Foot Ankle Surg* 36:112-114, 1997.
55. Rompe JD: Stoßwellentherapie: Therapeutische wirkung bei spekulativem mechanismus. *Z Orthop Ihre Grenzgeb* 134:13-19, 1996.
56. Rompe JD, Hopf C, Nafe B, et al: Low energy extracorporeal shock wave therapy for painful heel: A prospective controlled single-blind study. *Arch Orthop Trauma Surg* 115:75-79, 1996.

57. Rompe JD, Kirkpatrick CJ, Küllmer K, et al: Dose related effects of shock waves on rabbit tendo Achilles. A sonographic and histological study. *J Bone Joint Surg* 80B:546-552, 1998.
58. Rompe JD, Küllmer K, Eysel P, et al: Niedrigenergetische extrakorporale Stoßwellentherapie (ESWT) beim plantaren Fersensporn. *Orthop Prax* 32:271-275, 1996.
59. Rompe JD, Küllmer K, Riehle HM, et al: Effectiveness of low-energy extracorporeal shock waves for chronic plantar fasciitis. *Foot Ankle Surg* 2:215-221, 1996.
60. Schepsis AA, Leach RE, Gorzyca J: Plantar fasciitis: Etiology, treatment, surgical results, and review of the literature. *Clin Orthop* 266:185-196, 1991.
61. Schöllner C, Riedel C, Schwitalle M, et al: Shock-wave Treatment for Plantar Heel Pain. In Coombs R, Schaden W, Zhou SS (eds). *Musculoskeletal Shockwave Therapy*. London, Greenwich Medical Media 53-59, 2000.
62. Sellman JR: Plantar fascia rupture associated with corticosteroid injection. *Foot Ankle* 15:376-381, 1984.
63. Selth CA, Francis BE: Review of non-functional plantar heel pain. *Foot* 10:97-104, 2000.
64. Snider MP, Clancy WG, McBeath AA: Plantar fascia release for chronic plantar fasciitis in runners. *Am J Sports Med* 11:215-219, 1983.
65. Thordarson DB, Kumar PJ, Hedman TP, et al: Effect of partial versus complete fasciotomy on the windlass mechanism. *Foot Ankle* 18:16-20, 1997.
66. Tóth-Kischkat A: Principles of Shockwave Therapy. In Coombs R, Schaden W, Zhou SS (eds). *Musculoskeletal Shockwave Therapy*. London, Greenwich Medical Media 3-10, 2000.
67. Wapner KL, Sharkey PF: The use of night splints for treatment of recalcitrant plantar fasciitis. *Foot Ankle* 12:135-139, 1991.
68. Williams PL, Simbert JG, Cox R, et al: Imaging study of the painful heel syndrome. *Foot Ankle* 7:345-349, 1987.
69. Yu JS: Pathological and postoperative conditions of the plantar fascia: Review of MR imaging appearances. *Skeletal Radiol* 29:491-501, 2000.
70. Yu JS, Smith G, Ashman C, et al: The plantar fasciotomy: MR imaging findings in asymptomatic volunteers. *Skeletal Radiol* 28: 447-452, 1999.
71. Yu JS, Spigos D, Tomczak R: Foot pain after a plantar fasciotomy: An MR analysis to determine potential causes. *J Comput Assist Tomogr* 23:707-712, 1999.