

# Management of Heel Pain Syndrome with Acetic Acid Iontophoresis

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This study was undertaken to determine the effectiveness of acetic acid iontophoresis in the treatment of heel pain. Thirty-five patients with chronic heel pain were treated with acetic acid iontophoresis over a 4-year period. Ninety-four percent of patients had complete or substantial relief of heel pain after an average of 5.7 sessions of acetic acid iontophoresis over an average period of 2.8 weeks. Heel pain levels were rated from 0 to 10, with 10 representing the most severe pain. Heel pain prior to iontophoresis treatment received an average rating of 7.5; by the end of therapy, the average rating had decreased to 1.8. At an average follow-up time of 27 months, heel pain levels averaged 0.64, indicating continued reduction in heel pain. Ninety-four percent of participants said that they would recommend acetic acid iontophoresis to someone with similar heel pain. (J Am Podiatr Med Assoc 89(5): 251-257, 1999)

Acetic acid iontophoresis has been considered an effective treatment modality for the relief of heel pain<sup>1</sup> and has become a standard approach to the treatment of calcific bursitis and tendinitis of the shoulder.<sup>2</sup> Iontophoresis introduces ionizable substances into the body for therapeutic purposes by means of a direct current (Table 1).<sup>1</sup> In this case, the acetate radical is introduced under the cathode and deposited subcutaneously, permeating the area around the calcific deposit (Fig. 1). Potential areas of inflammation and subsequent calcium deposition leading to retrocalcaneal spurs are the tendo Achillis insertion on the calcaneus, retrocalcaneal bursae, and superficial

Achilles bursae (Fig. 2). Infracalcaneal spurs are formed in the same manner in areas of inflammation and subsequent calcium deposition at the insertion of the plantar fascia on the calcaneus (Fig. 2).

The acetate radical is more chemically active than the carbonate radical in the calcific area, replacing the carbonate radical and forming calcium acetate, which is blood-soluble. The presumed chemical reaction is as follows<sup>3</sup>:



Although acetic acid iontophoresis has been used to treat heel pain, no controlled study of this therapeutic modality has been reported in the literature. This report investigates acetic acid iontophoresis as an alternative conservative physical therapy modality for the management of heel pain.

## Iontophoresis Theory

Ion transfer, or iontophoresis, is the introduction of topically applied, physiologically active ions into the epidermis and mucous membranes of the body by

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**Table 1. Iontophoretic Substances: Source and Properties/Indications**

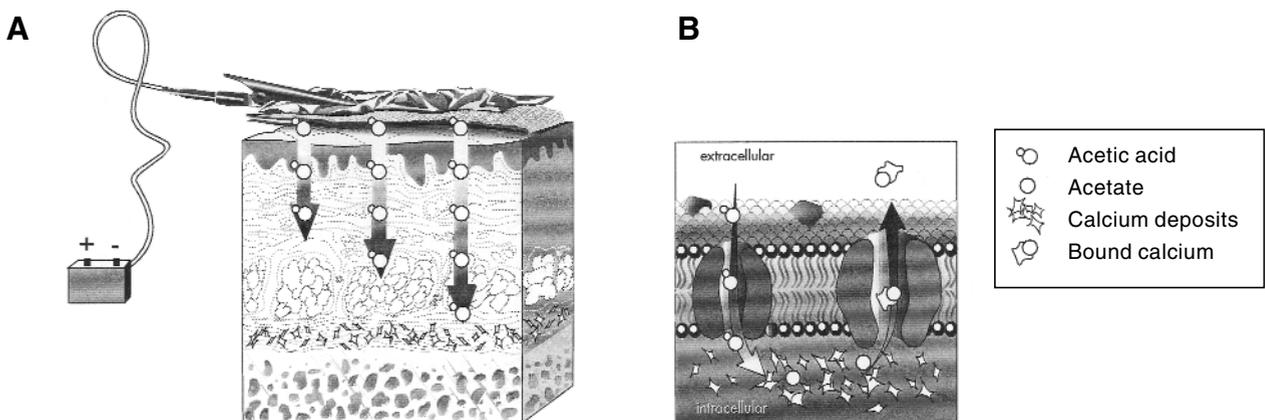
Substance	Source	Properties/Indications
Acetic acid	Stock acetic acid 10% diluted to 2% or 5%	Calcific deposits, myositis ossificans, frozen joints
Calcium	Calcium chloride 2% solution	Myospasm, frozen joints, trigger finger, mild tremors
Chlorine	Sodium chloride 2% solution	Sclerolytic agent; reduces keloids
Copper	Copper sulfate 2% solution	Antiseptic, antifungal, allergic rhinitis, tinea pedis
Iodine	Iodine 4.7%, methyl salicylate 4.8% ointment	Sclerolytic, antiseptic, analgesic
Hydrocortisone	Hydrocortisone 1% ointment	Anti-inflammatory
Salicylate	Salicylate 10% preparation	Analgesic
Zinc	Zinc oxide 20% ointment	Open skin lesions, dermatitis
Lithium	Lithium chloride 2% solution or lithium carbonate 2% solution	Gouty tophi
Magnesium	Magnesium sulfate 2% solution	Antispasmodic, analgesic, vasodilator
Lidocaine	Lidocaine 5% solution	Anesthesia, neuritis, bursitis, painful range of motion

means of a continuous direct current. Discovered by LeDuc<sup>4</sup> in 1903, iontophoresis is based on the principle that an electrically charged electrode will repel a similarly charged ion. Therefore, ions with a positive charge can be introduced into the tissues from the positive electrode, and negatively charged ions can be introduced by the negative pole.

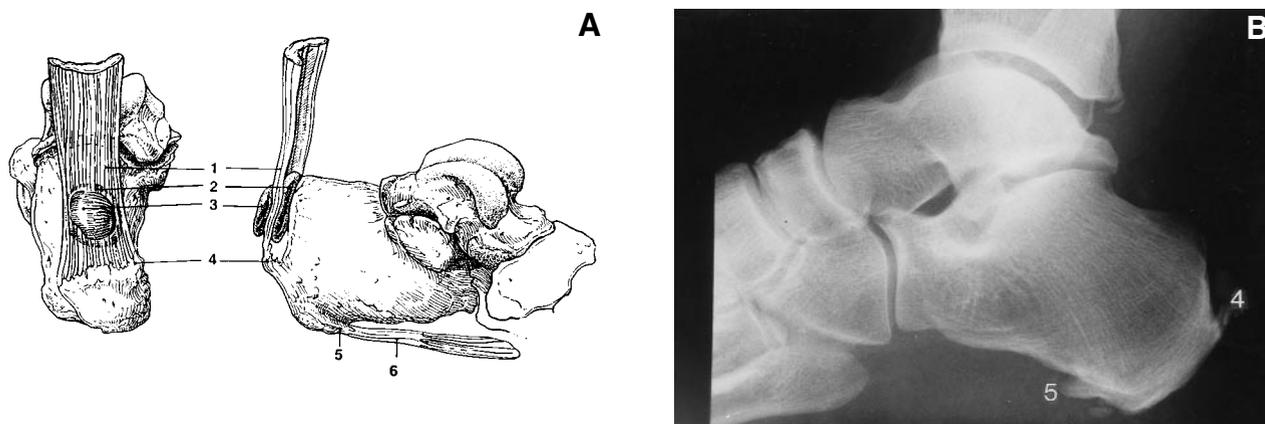
The force acting to move an ion through the surface of the body depends on the strength of the electric field and the resistance of the tissues of the body to current flow. The medical specialist may compensate for skin resistance by altering the applied voltage as necessary to achieve the desired current density. It is the current density at the electrode-skin interface that is responsible for the velocity of the

charged ions as they pass into the body. The current density may be increased either by decreasing the size of the electrode or by increasing the amount of current. Because body tissues, especially the skin and mucous membranes, have a limited tolerance for the passage of an electric current, the principal guide to a safe current density is the comfort of the patient.

Continuous direct (galvanic) current is the preferred mode of current, as it ensures maximum ion transfer per unit of applied current. Other current forms, such as conventional high-voltage galvanic, sine wave, and interferential currents, are not effective for iontophoresis.<sup>5</sup> Normal, intact skin will not tolerate a current density greater than 1 mA/cm when the applied current is continuous direct current. Skin



**Figure 1.** Diagram showing mechanism of acetic acid iontophoresis. A, Acetic acid introduced under the cathode changes to acetate (which has a negative charge), which is deposited subcutaneously, permeating the area of inflammation where the calcium deposit is found. B, The acetate ion then binds to calcium, forming calcium acetate, which is removed from the area of inflammation.



**Figure 2.** A, Diagram of the rearfoot showing potential areas of inflammation and subsequent calcium deposition. 1 = Tendo Achillis; 2 = retrocalcaneal bursa; 3 = superficial Achilles bursa; 4 = retrocalcaneal spur; 5 = infracalcaneal spur; 6 = plantar fascia. B, Lateral radiograph depicting both retrocalcaneal (4) and infracalcaneal (5) spurs.

resistance is even lower in areas where the skin is abraded or lacerated, in areas of scarring, and in individuals with fair skin.

The alkaline reaction occurring under the cathode is much more caustic to the skin than is the acidic reaction occurring at the anode. To minimize the possibility of tissue destruction under the cathode while still maintaining an effective treatment, the therapist should selectively decrease the current density under the cathode. This can be accomplished by increasing the surface area of the cathode. As the current density under the cathode decreases, the magnitude of the caustic alkaline reaction in the body tissue, which occurs as the continuous direct current passes through the cathode, also decreases. The cathode should be twice as large as the anode at all times.<sup>5</sup>

An additional concern is the anesthetic effect of continuous direct current.<sup>5</sup> Therefore, close monitoring of the skin underlying the electrodes is recommended during treatment. In addition, as a normal response to current flow, mild-to-moderate hyperemia (capillary dilation) is to be expected under both electrodes.

The effectiveness of a specific ion depends on the number of ions transferred, the depth of penetration, whether the ions combine chemically with other substances in the skin and precipitate, and whether the ions enter the capillaries and are carried away from the site of application by the blood.

The number of ions transferred into the body through iontophoresis is related to the current density at the active electrode, the duration of the current flow, and the concentration of the ions in the solution. The quantity of ions introduced across the body sur-

face is directly proportional to the current density. The amount of time that the current is allowed to flow also influences the number of ions transferred, as it is the direct current passing through the electrolytic solution that causes the ions to migrate according to their charge. The number of ions transferred is proportional to the cube root of the product of the current density and the duration of its application. Therefore, the longer a current is applied, the greater the number of ions that will be transferred. However, as the duration of the treatment increases, the resistance decreases and the chance of an electrical burn increases.

Resistance to current flow and to ion transfer is primarily a function of the skin. However, certain dermal structures such as hair follicles and sweat glands represent areas of decreased skin resistance and provide gaps in the skin through which an electric current and ions may readily flow. Moreover, the overall resistance of the skin to current flow will fall somewhat during the treatment as the skin becomes more saturated with the electrolyte and as the physiologic reaction to the current flow results in increased vasodilation to the area under the electrodes. Another factor inhibiting the transfer of ions is the tendency of some ions to form insoluble precipitates as they pass into the tissue. Precipitates are formed with the transfer of heavy-metal ions, such as iron, silver, copper, and zinc.

## Materials and Methods

From 1994 to 1998, 35 patients with chronic heel pain were treated with acetic acid iontophoresis by one of the authors (L.G.) at St. Albans Veterans Affairs Ex-

tended Care Center in St. Albans, New York. Patients were recruited by referral to the Department of Physical Medicine and Rehabilitation from the podiatry clinics and primary care clinics located at the Veterans Affairs Medical Centers in St. Albans, Brooklyn, Manhattan, and Staten Island, New York.

All patients enrolled in the study had a previous diagnosis of recalcitrant heel pain. There were 31 men and 4 women with an average age of 58.6 years (range, 33 to 78 years). The right heel was involved in 17 cases and the left heel in 12 cases. Six patients had bilateral heel pain and subsequent bilateral iontophoresis therapy. Of the 35 patients completing the treatment, 33 patients were available for follow-up evaluation.

Comorbid conditions contributing to heel pain were noted. Patients were interviewed before each treatment by a licensed occupational therapist. Individuals were asked about their current pain level, ability to walk without pain, and pain with the first step in the morning and during the day. The body mass index, defined as the weight in kilograms divided by the square of the height in meters, was calculated for each patient. Patients with a body mass index of 27.8 or greater were considered obese.<sup>6,8</sup>

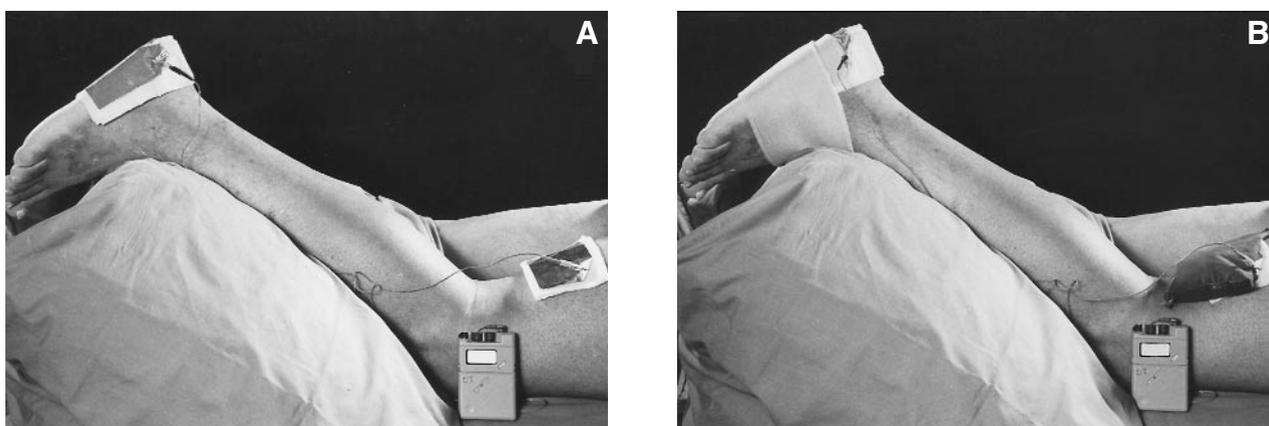
Heel pain was measured using a numeric pain-intensity scale ranging from 0 to 10, with 0 representing no pain and 10 representing the worst possible pain.<sup>9</sup> First-step pain was used as a measure of heel pain for the purposes of this study. Pain levels before and after completing treatment were compared using the *t*-ratio. The conservative treatment of patients before the beginning of the study varied. However, during the treatment regimen patients received no therapy other than acetic acid iontophoresis.

## Iontophoresis Technique

A solution of acetic acid diluted to 5% in distilled water was used. Two Crosstex (Cross County Paper Products Co, Hauppauge, New York) uncoated paper towels measuring approximately 5 × 7 inches were soaked in this solution and placed over the heel (Fig. 3). A pad of household heavy-duty aluminum foil folded to several thicknesses and rolled smooth was superimposed on the towels and connected to the negative pole lead of a continuous direct current generator (Dupel, Empi, Inc, St Paul, Minnesota) with an "alligator" clip (Fig. 4). The reference (positive) electrode (one 5 × 7-inch Crosstex paper towel, folded to measure 3 × 5 inches, soaked in plain tap water) was placed homolaterally above the popliteal fossa to reduce resistance in the circuit. The electrodes were held in position by lightweight sandbags or Nylatex (Chattanooga Group, Chattanooga, Tennessee) wraps depending on the anatomy involved. Two or three milliamperes of continuous direct current was passed through the circuits for approximately 15 to 20 minutes. Treatment was administered two or three times weekly. Patients were checked after each treatment for hyperemia. Patients had x-rays taken both before and after the procedure.

## Results

The average heel pain rating decreased significantly, from 7.5 before treatment to 1.8 after the end of treatment ( $P < .01$ ;  $t = 28.9$ ). Obese patients experienced significantly higher levels of heel pain (mean rating, 8.04) than nonobese patients (mean rating,



**Figure 3.** A, Application of iontophoresis electrodes with unit for treatment of heel spur; B, electrodes secured at foot and thigh.



**Figure 4.** Materials needed for iontophoresis: heavy-duty aluminum foil, acetic acid solution, dual-channel iontophoresis system, paper towels, roller device.

6.92) before treatment ( $P < .01$ ;  $t = 2.32$ ). Patients required an average of 2.8 weeks of treatment with acetic acid iontophoresis to alleviate first-step pain; this is significantly less than the average of 7.8 weeks reported in other studies of conservative treatment modalities for heel pain ( $P < .01$ ;  $t = 10.4$ ) (Table 2). Ninety-four percent of patients reported total or substantial improvement of heel pain after completion of acetic acid iontophoresis therapy. Ninety-four percent said that they would recommend acetic acid iontophoresis therapy to someone with similar heel pain. No gross changes were noted on x-rays of calcaneal spurs after completion of treatment.

## Discussion

In this first long-term, prospective study of iontophoresis in the treatment of heel pain, 94% of patients treated with acetic acid iontophoresis reported relief of heel pain that had been present for a number of years. Patients obtained lasting relief with just 3 weeks of noninvasive, relatively inexpensive therapy. Studies of the effectiveness of various conservative modalities in the treatment of heel pain tend to show good results, with between 65% and 95% of patients obtaining relief from a variety of conservative therapies often used in combination (Table 2).

It is interesting that in the present study, obese patients did not respond as well to therapy as nonobese patients. Whereas obese patients obtained short-term relief after 3 weeks of therapy, when questioned during the follow-up period these patients reported that the heel pain had returned. Obesity is known to be a predisposing factor in heel pain,<sup>15</sup> and other studies have found that obese patients respond less well to conservative treatment for heel pain than nonobese patients.<sup>12</sup>

The final question to be explored is the mechanism by which acetic acid iontophoresis reduces heel pain. Acetic acid iontophoresis shifts calcium away from the site of inflammation. Calcium has been shown to be a component of the inflammatory response in the standard arachidonic acid cascade model.<sup>21</sup> Monocytes are theorized to raise calcium levels by inducing phospholipase A<sub>2</sub>, leading to cleavage of arachidonic acid from membrane glycolipid.

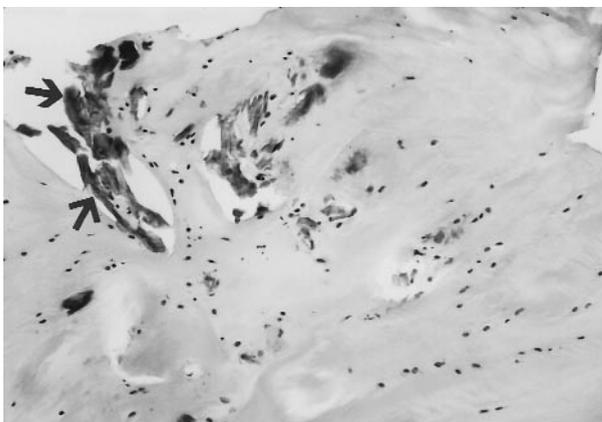
**Table 2. Effectiveness of Conservative Treatment Modalities in Various Studies**

Authors	Treatment Modalities	Treatment Time (months)	Patients Receiving Relief	
			N	%
Current study	Acetic acid iontophoresis	0.25–8.2	33/35	94
Campbell and Inman <sup>10</sup>	Orthoses	1–3	31/33	94
Chang and Miltner <sup>11</sup>	Shoe modification, rest, cast, hot pack	3–9.6	17/25	68
Furey <sup>12</sup>	Shoe modification, injection, nonsteroidal anti-inflammatory drugs	2–5	114/116	98
Amis et al <sup>13</sup>	Orthoses, shoe modification	6	31/43	72
Kenzora <sup>14</sup>	Shoe modification, injection, nonsteroidal anti-inflammatory drugs	0.25	Not published	90
Lapidus and Guidotti <sup>15</sup>	Injection, nonsteroidal anti-inflammatory drugs	0.25	364/364	100
O'Brien and Martin <sup>16</sup>	Orthoses, injection	Not published	41/58	71
Shikoff et al <sup>17</sup>	Orthoses, injection, nonsteroidal anti-inflammatory drugs	5	127/195	65
Snook and Chrisman <sup>18</sup>	Rest, injection, nonsteroidal anti-inflammatory drugs	0.25	22/27	81
Lutter <sup>19</sup>	Physiotherapy	1–9	172/182	95
McBryde <sup>20</sup>	Shoe modification, nonsteroidal anti-inflammatory drugs, physiotherapy	2	82/100	82

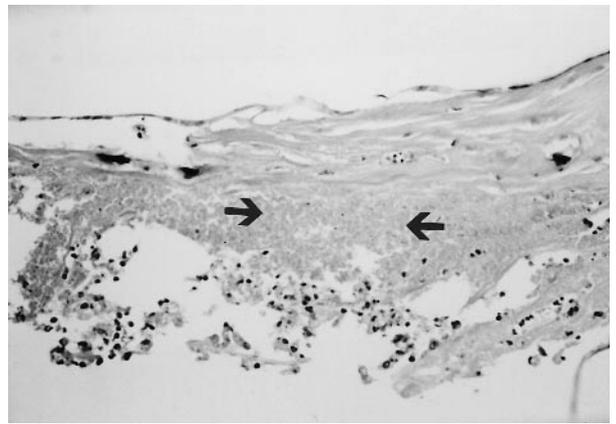
Histopathologic changes of calcaneal tendons and ligaments of patients who have heel pain include an initial low-grade chronic periosteal inflammation, edema, fibroblastic proliferation, and inflammatory cell proliferation. Dystrophic calcification will then occur, leading to acute pain at the fascial and tendon attachments.<sup>22</sup> Dystrophic calcification refers to calcium deposits found in inflamed, dead, or dying tissue despite normal blood calcium levels and normal calcium metabolism (Fig. 5).<sup>23</sup> One theory proposes that denatured proteins from damaged cells unmask reactive groups that bind phosphate radicals. In turn, the phosphate radicals bind calcium ions. The calcium ions then open tendon collagen fiber bundles, which cause tissue swelling and fat saponification and ultimately tissue disruption. These calcium ions break protein crosslinkages with chondroitin sulfate or other polysaccharides, disrupting hydrogen bonds or protein-interchange linkages.<sup>24</sup> The chronicity of the inflammation and necrosis will progress to fibrocartilage formation and eventually to osseous spur formation (Fig. 6). In addition, tendon thickening and nodularity with tendon calcification will occur. Tears of the tendo Achillis have been noted through areas of calcification immediately proximal to its insertion.<sup>25</sup> The patient will remain symptomatic as long as the inflammatory phase of adventitial bursitis, periostitis, or plantar fasciitis is present.

## Conclusion

The results of this study indicate that acetic acid iontophoresis is an effective conservative treatment modality for acute or recalcitrant heel pain. Further studies should be conducted to confirm these results.



**Figure 5.** Photomicrograph showing dystrophic calcification (between arrows) of the tendo Achillis (H&E,  $\times 20$ ).



**Figure 6.** Photomicrograph showing necrosis (between arrows) of the tendo Achillis (H&E,  $\times 20$ ).

**Acknowledgment.** Anne Erickson, MA, CMI, and Mike Yu for the medical illustrations and photographs; William Glynn for word processing; Kevin Cusack, PhD, Bimal C. Ghosh, MD, and Joseph Kahn, PhD, PT, for guidance.

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