JAOA ORIGINAL CONTRIBUTION

Effect of Counterstrain on Stretch Reflexes, Hoffmann Reflexes, and Clinical Outcomes in Subjects With Plantar Fasciitis

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Context: Previous research indicates that osteopathic manipulative treatment based on counterstrain produces a decrease in the stretch reflex of the calf muscles in subjects with Achilles tendinitis.

Objectives: To study the effects of counterstrain on stretch reflex activity and clinical outcomes in subjects with plantar fasciitis.

Methods: In a single-blind, randomized controlled trial of crossover design, the effects of counterstrain were compared with those of placebo in adult subjects (N=20) with plantar fasciitis. The subjects were led to believe that both the counterstrain and placebo were therapeutic modalities whose effects were being compared. Ten subjects (50%) were assigned to receive 3 weeks of counterstrain treatment during phase 1 of the trial, while the other 10 subjects were given placebo capsules. After a 2- to 4-week washout period, phase 2 of the trial began with the interventions reversed. Clinical outcomes were assessed with daily questionnaires. Stretch reflex and H-reflex (Hoffmann reflex) in the calf muscles were assessed twice during each laboratory visit, before and after treatment in the counterstrain phase.

Results: No significant changes in the electrically recorded reflexes of the calf muscles were observed in response to treatment. However, changes in the mechanical characteristics of the twitches resulting from the electrical responses were observed. Peak force and time to reach peak force both increased ($P \leq .05$) in the posttreatment measurements, with the increase being significantly more pronounced in the counterstrain phase ($P < .05$). A comparison of pretreatment and posttreatment symptom severity demonstrated significant relief of symptoms that was most pronounced immediately following treatment and lasted for 48 hours.

Conclusions: Clinical improvement occurs in subjects with plantar fasciitis in response to counterstrain treatment. The clinical response is accompanied by mechanical, but not electrical, changes in the reflex responses of the calf muscles. The causative relation between the mechanical changes and the clinical responses remains to be explored.

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The results of a study previously published in abstract form[1] and published in full in this issue of *JAOA—The Journal of the American Osteopathic Association*[2] suggest that osteopathic manipulative treatment (OMT) based on counterstrain produces a decrease in the amplitude of the stretch reflex of the triceps surae (soleus plus the lateral and medial heads of the gastrocnemius muscles) in subjects with Achilles tendinitis. The purpose of the present study was to test the effect of counterstrain in subjects with plantar fasciitis by measuring: (1) changes in the reflexes of the triceps surae muscles, which insert directly on the Achilles tendon and indirectly, via the Achilles tendon and the calcaneus, on the plantar fascia, and (2) the clinical outcomes of the treatment in terms of pain relief and restoration of function.

Counterstrain has been defined by Yates and Glover[3] as an indirect myofascial technique focused on the neurologic component of the neuro-vascular-myofascial somatic dysfunction. The counterstrain technique was developed by Lawrence H. Jones, DO, during the 1950s[4] based on the proprioceptive theory of somatic dysfunction proposed by Irvin M. Korr, PhD.[5] Jones hypothesized that bringing a hypertonic muscle, whose spindles "report strain where there is none," into a shortened position could reverse the hyperactivity of the spindles, restoring normal stretch reflex gain and normal range of motion.[6]

There are two experimental methods that can be used to elicit the stretch reflex: (1) stretching the muscle, and (2) electrically stimulating the nerve to the muscle. The latter method is referred to as the Hoffmann reflex, or H-reflex. By measuring these two reflexes, one can determine whether an intervention, such as OMT, changes the reflex intensity by altering the sensitivity of the muscle spindles or by altering the transmission of the reflex signal within the spinal cord.

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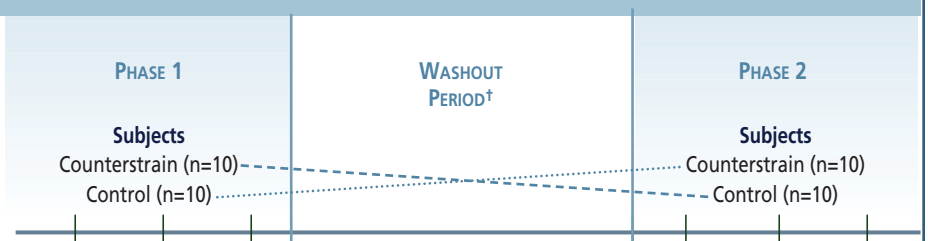
Counterstrain Study: Crossover Trial Design
Sequence for Each Subject Visit (N=20)*



Laboratory Visit	1	2	3		4	5	6
Procedures							
■ **Pretreatment**							
□ Questionnaire	X	X	X		X	X	X
□ Measurement	X	X	X		X	X	X
□ Diagnosis‡	X						
■ **Treatment§**	X	X	X		X	X	X
■ **Posttreatment**							
□ Questionnaire	X	X	X		X	X	X
□ Measurement	X	X	X		X	X	X

* X indicates that the procedure occurred during the visit. No entry indicates that the procedure did not occur.
† The washout period varied from 2 to 4 weeks among subjects, depending on subject availability.
‡ A diagnosis was made during the first visit only.
§ Treatment was administered to counterstrain subjects only.

Figure 1. *Of the 20 subjects in the study population, 10 were assigned to receive osteopathic manipulative treatment (OMT) with the counterstrain procedure in phase 1 of the crossover trial, which lasted 3 weeks. The 10 control subjects were given placebo capsules during phase 1. After a 2- to 4-week washout period, phase 2 of the trial began, with the 10 OMT subjects reassigned to receive placebo and the 10 control subjects reassigned to receive OMT for 3 weeks. Subjects visited the testing laboratory once per week during each of the two 3-week trial phases for the following sequence of procedures: (1) pretreatment subject rating of symptom severity on a questionnaire; (2) pretreatment electromyogram measurements of stretch reflex and H-reflex; (3) diagnosis of plantar fasciitis by osteopathic physician (during first visit only); (4) treatment with counterstrain technique (OMT subjects only); (5) posttreatment subject rating of symptom severity on a second questionnaire; and (6) posttreatment electromyogram measurements of stretch reflex and H-reflex.*

Methods

All procedures were approved by Ohio University's institutional review board. Adult subjects with plantar fasciitis were recruited through public advertisements and referrals from physicians. Subjects were excluded from the study if they reported any of the following: (1) wearing a pacemaker; (2) having been diagnosed with cardiac arrhythmias; (3) possessing known conditions resulting in unstable blood pressure (eg, frequent fainting spells); (4) having been diagnosed with deep vein thrombosis; (5) having had serious trauma to the lower extremities within the previous year; (6) having known peripheral neuropathies affecting the lower extremities; (7) having known neoplasms affecting the lower extremities; (8) having been treated for plantar fasciitis during the previous month with any type of manual treatment or therapy (eg, OMT, physical therapy); (9) obesity; or (10) taking muscle relaxants, pain medication, or more than half the daily maximum dose of nonsteroidal anti-inflammatory drugs (NSAIDs). Twenty subjects (16 women, 4 men; age range, 20–66 y) were enrolled in the study. All subjects provided informed consent.

The present study compared the effects of counterstrain with those of placebo in a single-blind, randomized controlled trial of crossover design. The subjects were led to believe that both the counterstrain and the placebo were therapeutic modalities whose effects were being compared. The control intervention consisted of capsules, taken two times per day, con-

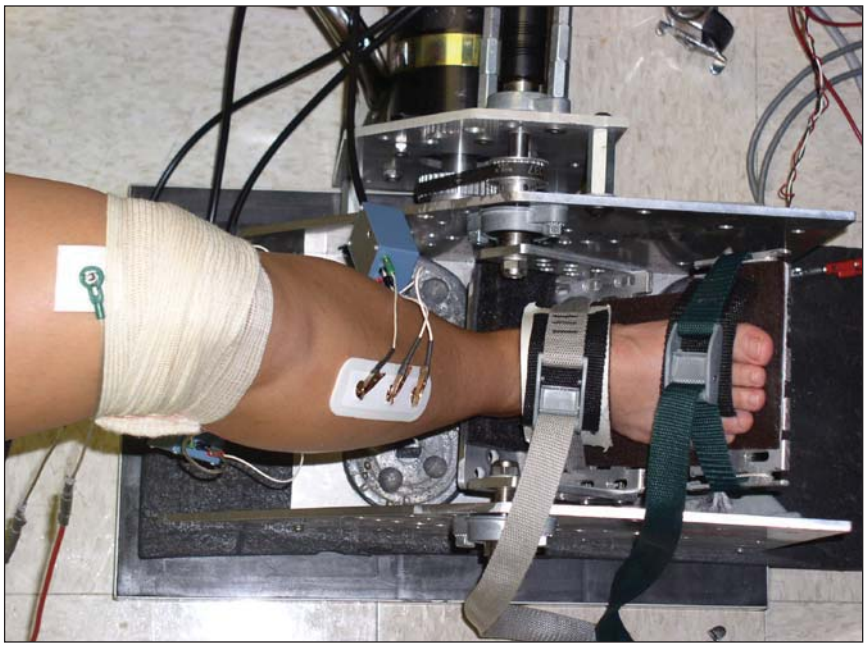


Figure 2. *The seated subject's leg is positioned in the stepper motor testing apparatus with the foot strapped to the foot plate and with a knee angle of 130 degrees and an ankle angle of 90 degrees. Electromyogram electrodes are visible on the subject's tibialis anterior. The electric motor (top) imposes a rapid dorsiflexion (5 degrees in 50 ms), which elicits the stretch reflex. The H-reflex is elicited by electrical stimulations of the tibial nerve.*

taining either cornstarch or, in the case of one subject who had hypersensitivity to corn, flour. Subjects were told the capsules contained an NSAID.

Ten (50%) subjects were assigned to receive counterstrain treatment during the 3 weeks of phase 1 of the trial, while the other 10 subjects were given placebo capsules to take during this period. Following a 2- to 4-week washout period (an interval that varied because of subject availability), phase 2 began, with the group interventions reversed (*Figure 1*). Subjects came to the testing laboratory one time per week during each of the two 3-week treatment periods for reflex testing and, when they were in the counterstrain phase of the study, for treatment. Subjects in the control phase were provided with a week's supply of placebo capsules at each visit.

At the beginning of each laboratory visit, subjects filled out "Pain and Dysfunction Questionnaire #1," on which they rated, on a scale of 0 (no symptoms) to 9 (extreme symptoms/pain), the severity of their plantar fasciitis symptoms according to their present pain, soreness to touch, stiffness, and how it affected their walking. Following electrode placement, both reflexes were measured. Subjects were examined by an osteopathic physician (J.M.B. or D.C.E.) to confirm the diagnosis of plantar fasciitis (during the first visit only). Subjects in the counterstrain phase were then treated with counterstrain. Prior to a second reflex measurement, subjects in both phases completed "Pain and Dysfunction Questionnaire #2."

At the conclusion of testing, subjects were given a packet of six "Take-Home Subject Questionnaires" to fill out at the same time each day between laboratory visits. On the forms, subjects rated, on the same scale of 0 to 9, the severity of their plantar fasciitis according to present pain, soreness to touch, stiffness, how it affected their walking, and how it affected their previous night's sleep. Subjects also reported on these forms if they had taken any NSAIDs that day.

EMG Recording and Data Acquisition

Following skin preparation by shaving, if necessary, and rubbing with prep pads containing alcohol and pumice, self-adhesive bipolar silver/silver chloride (Ag/AgCl) electrodes (1 cm diameter, separated by 2 cm) were placed on the skin overlying the soleus, the lateral and medial heads of the gastrocnemius, and the tibialis anterior muscles. A cathodal-stimulating electrode was placed on the skin of the popliteal fossa, and an anodal-stimulating electrode was placed on the skin just above the patella (*Figure 2*). Trial stimuli were delivered to ensure correct placement of the stimulating electrode and to determine the range of stimulus intensities required to produce the H-reflex. The electromyogram (EMG) signals were amplified

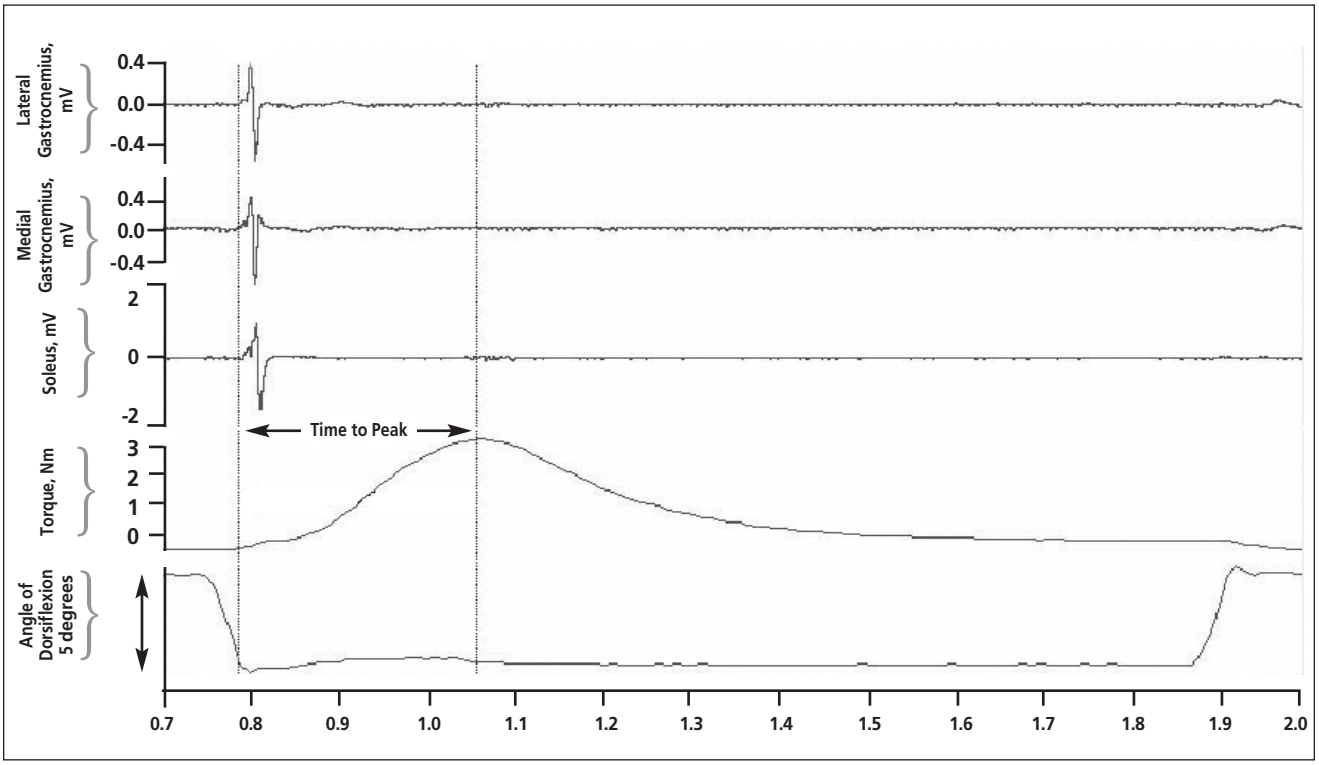


Figure 3. *Sample electromyogram (EMG) traces of torque and reflex action potentials for all three muscles of the triceps surae (soleus, medial gastrocnemius, lateral gastrocnemius) from a single subject in response to a 5-degree angle of dorsiflexion. Time to reach peak torque is represented by broken vertical lines. The mean of 30 such responses from each subject was used for pretreatment and post-treatment comparisons. Sampling frequency in EMG records is 2 kHz.*

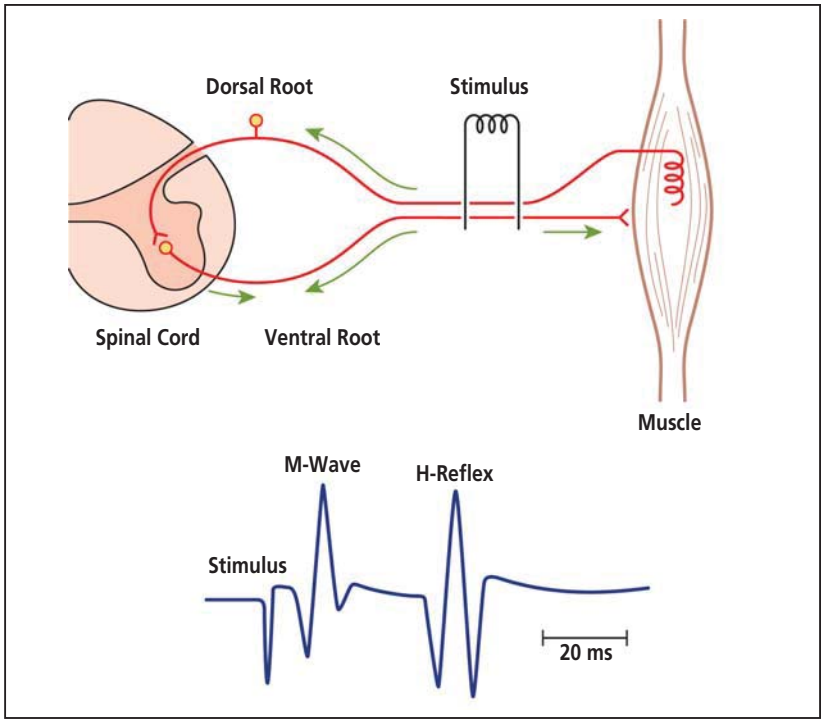


Figure 4. *Low intensity stimuli delivered to the tibial nerve activates only the largest fibers within the nerve, namely the Ia sensory fibers, generating a reflex electromyogram (EMG) response in the muscle—the H-reflex. Higher intensity stimuli also activate motor fibers. Action potential conduction within these fibers in the orthodromic direction generates a short latency EMG response in the muscle—the M-wave. Action potentials traveling in the opposite direction, antidromically, travel toward the spinal cord and collide with action potentials generated reflexly from sensory fiber stimulation. These collisions prevent the reflexly generated signal from traveling to the muscle. With increasing intensity of stimuli, more motor fibers are stimulated, causing these collisions in a greater proportion of the motor fibers. The intensity of the H-reflex signal decreases as the M-wave becomes larger, disappearing with stimuli greater than 20 mA.*

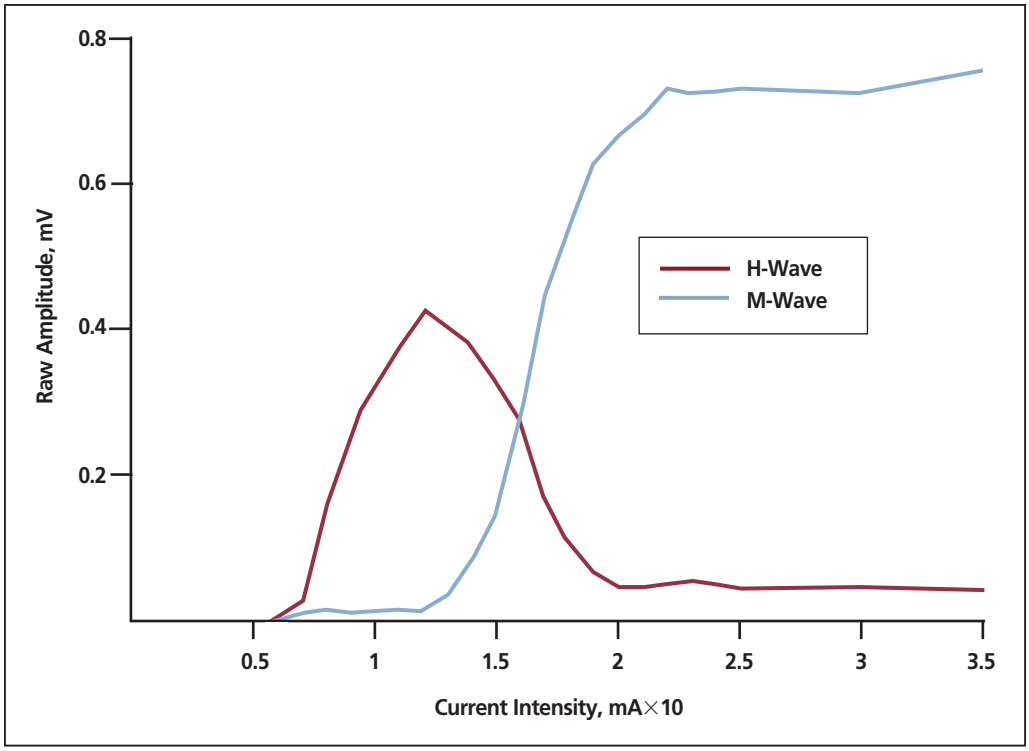


Figure 5. *Plot of the H-wave and M-wave as a function of intensity of the stimulus applied to the tibial nerve. The raw electromyogram amplitude is shown.*

(Z_1=100 GΩ; Intronix Technologies, Bolton, Ontario), filtered between 10 Hz and 1000 Hz, and sampled at 10 kHz with a Spike II data acquisition system (Model 1401; Cambridge Electronics Design, Cambridge, England). Muscle force output, detected with a strain gauge in the foot apparatus, was measured as torque around the ankle joint. During data acquisition, subjects were instructed to remain relaxed and to maintain their gaze with no head movement.

Measurement of the Stretch Reflex

Subjects were seated comfortably with one foot strapped firmly to a foot plate at a knee angle of 130 degrees and an ankle angle of 90 degrees (*Figure 2*). The stretch reflex was elicited by a quick movement (50 ms) applied to the foot plate by a computer-controlled stepper motor (Model M112; Superior Electronics, Bristol, Conn), causing a 5-degree dorsiflexion of the foot and a corresponding stretch of the triceps surae muscles. This process was repeated 10 times, approximately every 5 seconds, but at slightly irregular intervals to prevent the subject from anticipating the exact time of the stimulus. Each set of 10 stretch reflexes was repeated three times for a total of 30 stretch reflex measurements (*Figure 3*).

Measurement of the H-Reflex

The H-reflex was elicited by a series of electrical stimulations of the tibial nerve in the popliteal fossa (Model DS7; Digitimer Ltd, Welwyn Garden City, England) (*Figure 4*). Varying intensities of current were applied to the tibial nerve, beginning with the intensity required to produce a maximum M-wave and decreasing progressively. The amplitude of the H-reflex was recorded as the ratio of peak amplitudes of the H- and M-waves seen in the EMG recording, and was plotted as a function of stimulus intensity (*Figure 5*).

Osteopathic Manipulative Treatment

Counterstrain treatment was performed by physicians (J.M.B and D.C.E.) in the Osteopathic Manipulative Medicine section of the Department of Family Medicine at Ohio University College of Osteopathic Medicine in Athens. No time limit was enforced for OMT, which began with the physician performing an osteopathic structural examination focused on the foot, ankle, and lower leg to locate tender points. Each tender point was then treated individually using the counterstrain method.

The counterstrain procedure involved application of brief mechanical pressure on each tender point with one fingertip in order to determine tenderness and tissue tension. The physician then moved the appropriate joint into various positions of ease until a position was achieved in which there was at least 70% to 80% relief of discomfort at the tender point when the same pressure was reintroduced. This position was then main-

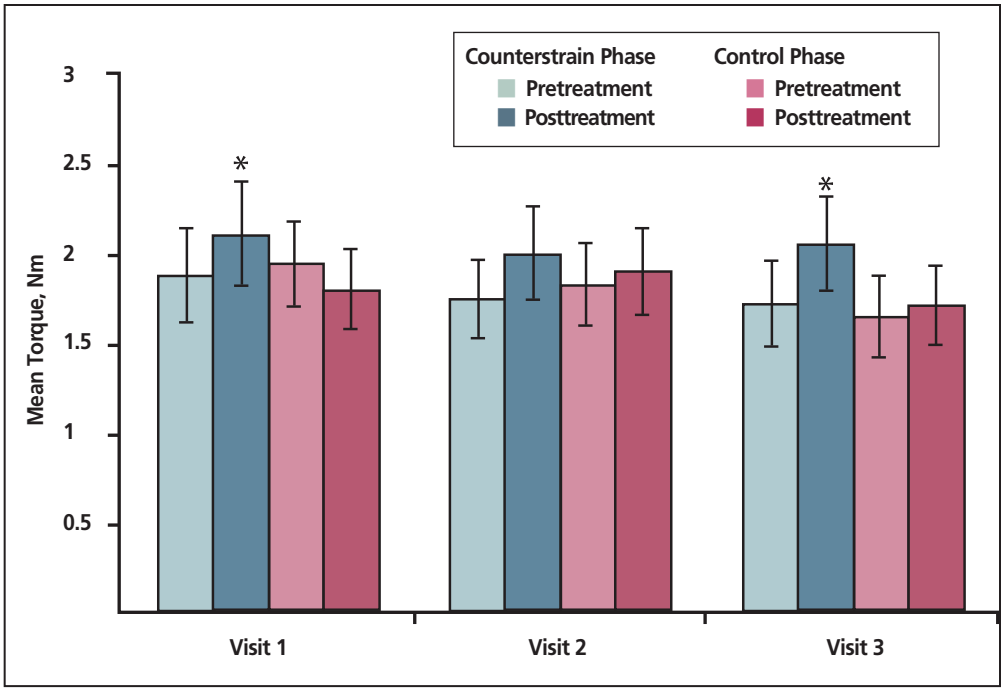


Figure 6. *Peak torque of the stretch reflex increased significantly (*) after subjects were treated with the counterstrain technique during laboratory visits 1 and 3 of the counterstrain phase (P<.05). No significant changes in peak torque were observed in any of the visits of the subjects in the control (placebo capsule) phase.*

tained for 90 seconds. Following the 90-second period, the physician slowly returned the appropriate joint to a neutral position and reexamined the area in which the tender point was located. Because no set OMT sequence exists in the literature for plantar fasciitis, the counterstrain tender points were treated by the physician according to functional anatomical considerations, overall strain throughout the lower extremity, and patient tolerance.

Data Processing and Analysis

Comparisons of the subjects' reflex responses before and after treatment were made as follows.

For the stretch reflex, the EMG records of the soleus, medial gastrocnemius, and lateral gastrocnemius were rectified and integrated; the areas under the curves of the evoked responses were expressed as a ratio of the stretch reflex amplitude to the maximum M-wave amplitude (S/M_{max}). Thirty stretch reflexes were averaged for both the pretreatment and posttreatment periods.

For the H-reflex, EMG records were similarly rectified and integrated, and the areas under the curves of the evoked responses were expressed as the ratio of the H-reflex amplitude to the maximum M-wave amplitude (H/M_{max}). To determine the H-reflex amplitude, the highest three points on the H recruitment curve were averaged for the pretreatment and posttreatment periods.

Paired *t* tests were used to analyze the reflex data. Analysis of variance (ANOVA) was used in the analysis of the clinical outcomes data, followed by the Tukey posthoc test to localize specific significant differences.

Results

Electromyogram measurements of both reflexes revealed no significant change in response of the triceps surae muscles to either OMT with counterstrain treatment or to placebo. However, significant mechanical changes in the twitches resulting from the electrical responses were observed. Peak torque of the stretch reflex (*Figure 6*) and the H-reflex (*Figure 7*) increased significantly in response to counterstrain treatment ($P<.05$). A trend toward increasing torque values was also seen in the H-reflex during the control phase but reached significance only in the second of the three visits. In addition, the time to reach peak torque of the stretch reflex increased significantly with treatment but not in the control phase (*Figure 8*). Significant increases in time to reach peak torque of the H-reflex were seen in both the treatment and control phases ($P<.05$) (*Figure 9*).

Osteopathic manipulative treatment resulted in a marked decrease in symptom severity in subjects with plantar fasciitis, as measured immediately following treatment (*Figure 10*). When symptom severity was analyzed by repeated-measures ANOVA (RMANOVA) over the 6 days following each laboratory visit, no statistical significance between the phases emerged (*Figure 11*). When the analysis was limited to the first 2 days posttreatment, a group interaction was observed, indicating a difference in the responses over that time between the counterstrain and control phases.

Comment

The research by Howell et al[2] published in the present issue of the *JAOA* demonstrates a decrease in stretch reflex amplitude

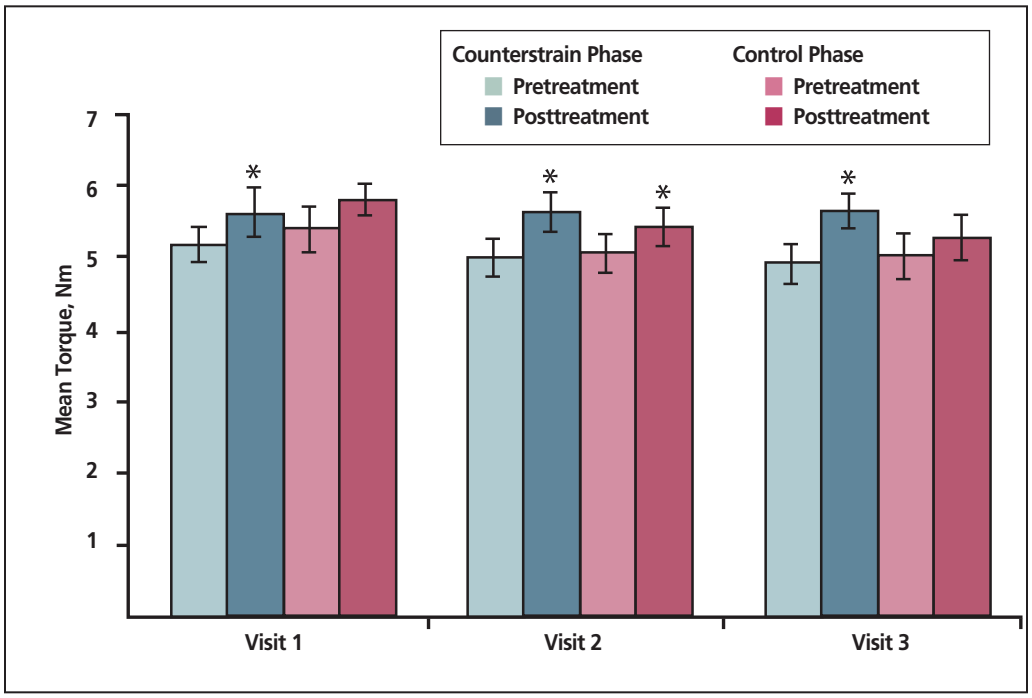


Figure 7. *Peak torque of the H-reflex increased significantly (*) after subjects were treated with the counterstrain technique during all three laboratory visits of the counterstrain phase (P<.05). A significant increase in peak torque was also observed for subjects in visit 2 of the control (placebo capsule) phase (P<.05).*

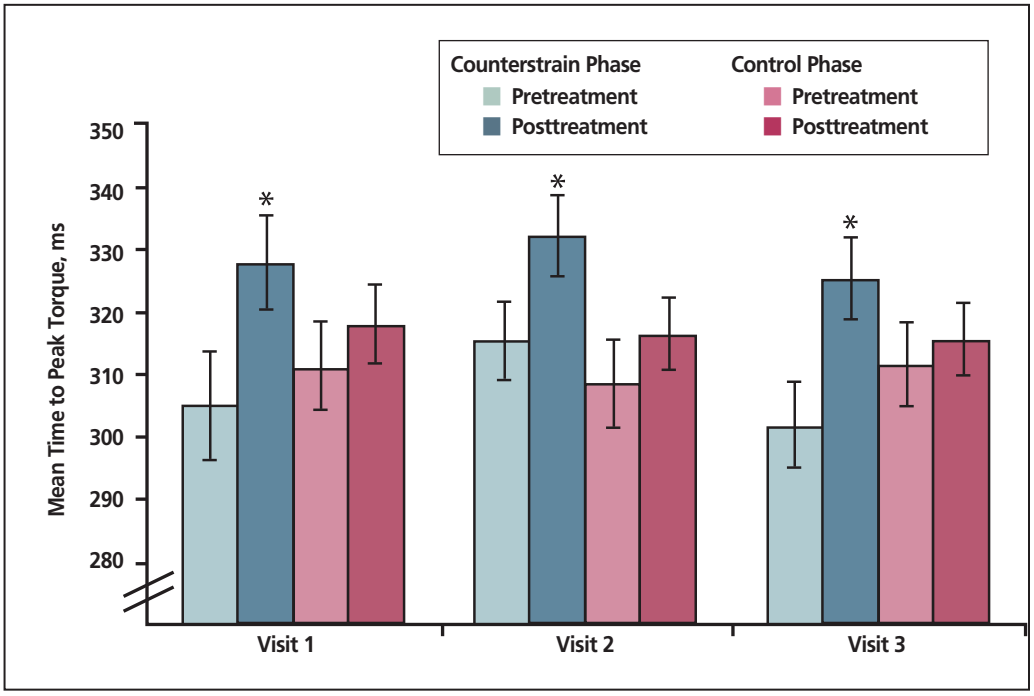


Figure 8. *Time to reach peak torque of the stretch reflex increased significantly (*) after subjects were treated with the counterstrain technique during all three laboratory visits of the counterstrain phase (P<.05). No significant changes in time to reach peak torque were observed in any of the visits of the subjects in the control (placebo capsule) phase.*

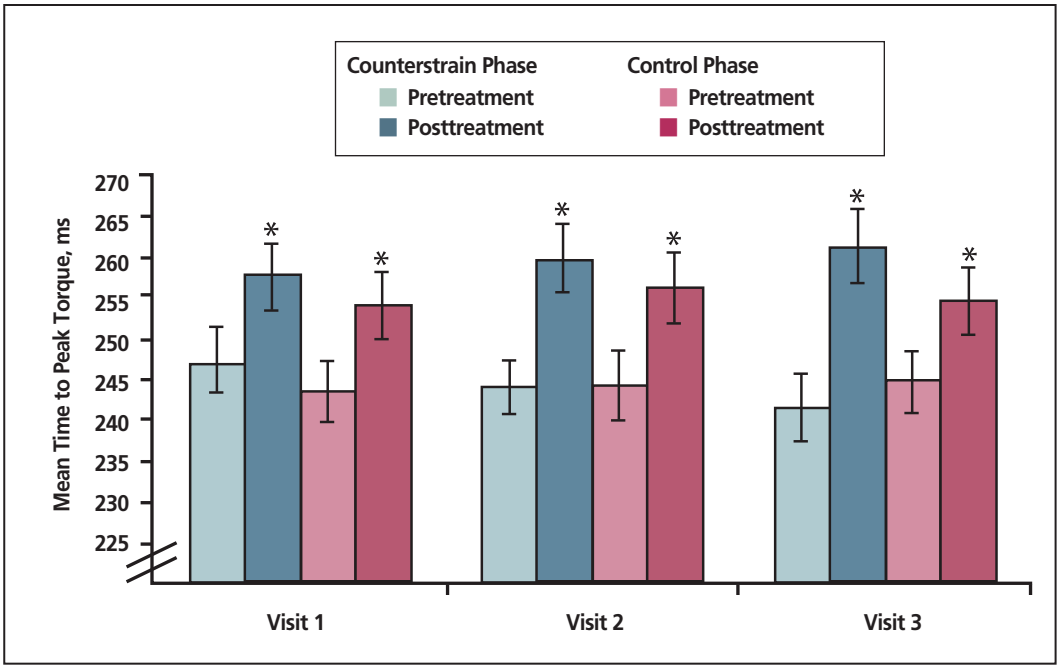


Figure 9. *Time to reach peak torque of the H-reflex increased significantly (*) after subjects were treated with the counterstrain technique during all three laboratory visits of the counterstrain phase (P<.05). Time to reach peak torque of the H-reflex also increased significantly for subjects in all three visits of the control (placebo capsule) phase (P<.05).*

with counterstrain treatment in subjects with Achilles tendinitis—a result that is consistent with Korr's hypothesis that OMT works by altering the gain of the stretch reflex.[5] Despite the mechanical continuity of the triceps surae and the plantar fascia around the calcaneus, no such reflex change in the triceps surae was seen in the present study with counterstrain treatment of subjects with plantar fasciitis. Nor were there any trends in the data suggesting that a larger sample size might reveal such changes. It is possible that such an effect might be seen in EMG recordings from the intrinsic muscles of the foot and/or the long digital flexors, but no recordings from these muscles were made in this study.

The observed increase in torque produced by both reflexes following counterstrain treatment cannot be explained by altered muscle excitation because no change in evoked EMG amplitude was observed. Torque was measured as pressure from the ball of the foot on the foot plate. It is likely that counterstrain treatment produced alterations in the mechanical properties of the foot, including in the plantar fascia, which could have altered the apparent mechanical output of the triceps surae during the reflex. It is also possible that the treatment, which included attention to tender points in the lower leg as well as the foot, somehow altered the mechanical output of the triceps surae muscles themselves, despite the constancy of the evoked action potentials recorded from them.

Even if one assumes that counterstrain treatment altered the subjects' foot mechanics, it is not clear why twitch tension increased. The effects of treatment are not likely to result from passive stretching of the plantar fascia or triceps surae, because counterstrain typically brings the involved tissues into positions of less strain.

In addition to the significant increase in peak torque and time to reach peak torque of both the stretch and H-reflex following OMT, the second measurement in the control group, corresponding to the posttreatment measurement in the counterstrain group, showed some increase as well, in some cases reaching significance (*Figure 7*). However, the greater torque increases for the counterstrain group suggest that the effect from OMT was greater than any effect from the repeated measurements themselves.

Counterstrain seemed to have a greater effect on the stretch reflex torque (10% increase) than on the H-reflex torque (8% increase). This difference may be related to the fact that the twitch resulting from the stretch reflex is a smaller twitch, approximately 40% of the size of that from H-reflex.[2]

Clinical outcomes of the present study, assessed as subject ratings of symptom severity on a 10-point scale, indicated that subjects experienced significant relief of symptoms immediately following counterstrain treatment (*Figure 10*). This effect was greatest following the first of three treatments in the counterstrain phase. Our use of an oral placebo did not serve as a sham treatment and, therefore, did not allow us to dis-

Editor's message: In the original print publication, there was a layout error in *Figure 10*. The values on the y-axis appeared beneath their interval marks (tick marks) instead of centered on them. The error has been corrected here.

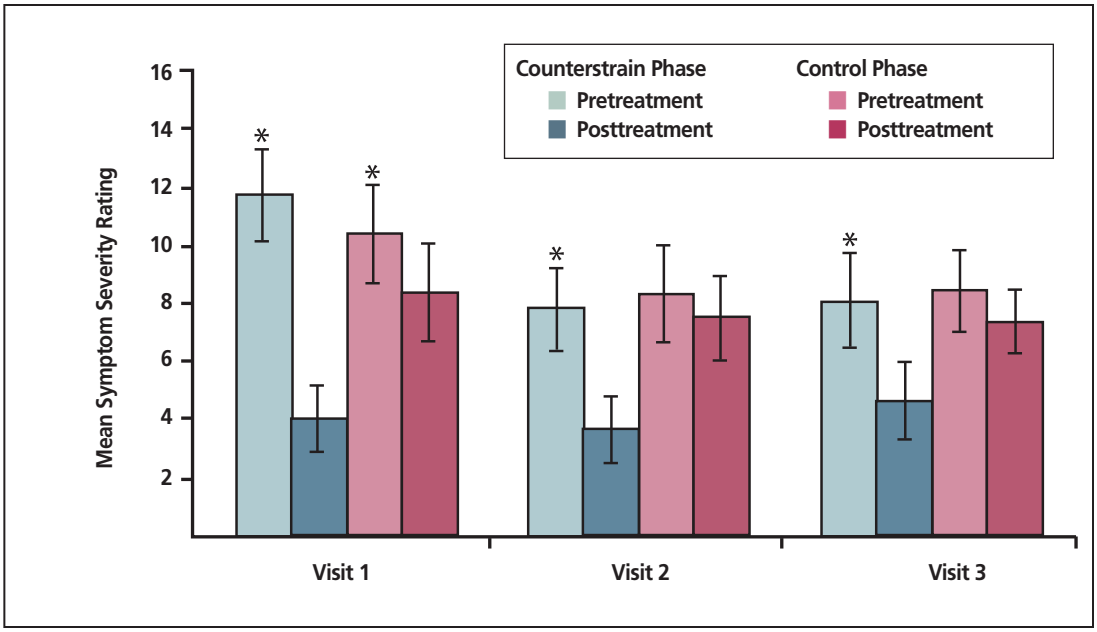


Figure 10. *Subjects' ratings of symptom severity before and after treatment in the three laboratory visits of the counterstrain phase and the three visits of the control (placebo capsule) phase. The data from one subject who failed to return one of the take-home questionnaires was discarded, leaving a sample size of 19. The ratings were recorded as the sum of each subject's rating of four factors: pain, soreness, stiffness, and mobility. Each rating was made on a scale of 0 (no symptoms) to 9 (extreme symptoms/pain), and the summed values for all subjects were then averaged. Thus, the minimum possible rating is 0 and the maximum possible rating is 36. The symptom severity ratings markedly decreased (*) immediately after treatment with counterstrain in all three visits of the counterstrain phase (P<.05). A significant, though slight, improvement in symptom severity was reported by subjects in the control phase only during the first visit (P<.05).*

tinguish between the mechanical and psychosocial effects of counterstrain. As shown in *Figure 10*, the mean rating of symptom severity immediately after the first and second laboratory visits for counterstrain treatment was reduced to 4, compared with mean pretreatment ratings of at least 8. Some subjects, however, did report complete relief of symptoms after counterstrain treatment.

When subjects' ratings of symptom severity were analyzed by RMANOVA over the 6 days following each visit, no statistically significant differences between the counterstrain and control phases emerged (*Figure 11*). Therefore, we cannot claim that there was a clinical effect of counterstrain compared with placebo in subjects with plantar fasciitis over this entire period of time. When the analysis was limited to the first 2 days posttreatment, however, a group interaction was observed, indicating a difference in subjects' responses over that time between the counterstrain and control placebo phases. *Figure 11* reveals that the symptom severity ratings over the first 48 hours either decreased or remained relatively low for subjects in the counterstrain phase while increasing for subjects in the control phase.

Expectations of the placebo effect would have shown up most clearly as a decrease in symptom severity ratings when subjects entered the control phase. Had the capsules contained an actual NSAID, the ratings would have decreased, as they did in the counterstrain phase. Many subjects admitted that they doubted that the capsules they were taking during the control phase contained an effective NSAID, based on the failure of the medication in the capsules to relieve headaches or menstrual cramps occurring during that time. Thus, the capsules probably served as a more effective placebo in the earliest part of the control phase than in the later part.

Effectiveness of counterstrain was greater immediately following treatment than over the successive days. Mean symptom severity ratings fell to approximately 4 immediately after treatment (*Figure 10*) but rose to approximately 8 by 24 hours afterward (*Figure 11*). These results indicate that the residual effect of counterstrain treatment between 24 and 48 hours after the treatment, though persistent, was considerably less than the immediate effect.

It should also be noted that counterstrain was administered to subjects in the present study without the benefit of adjunctive therapies, such as taping of the feet or prescription of specific exercises, to reinforce the treatment response.

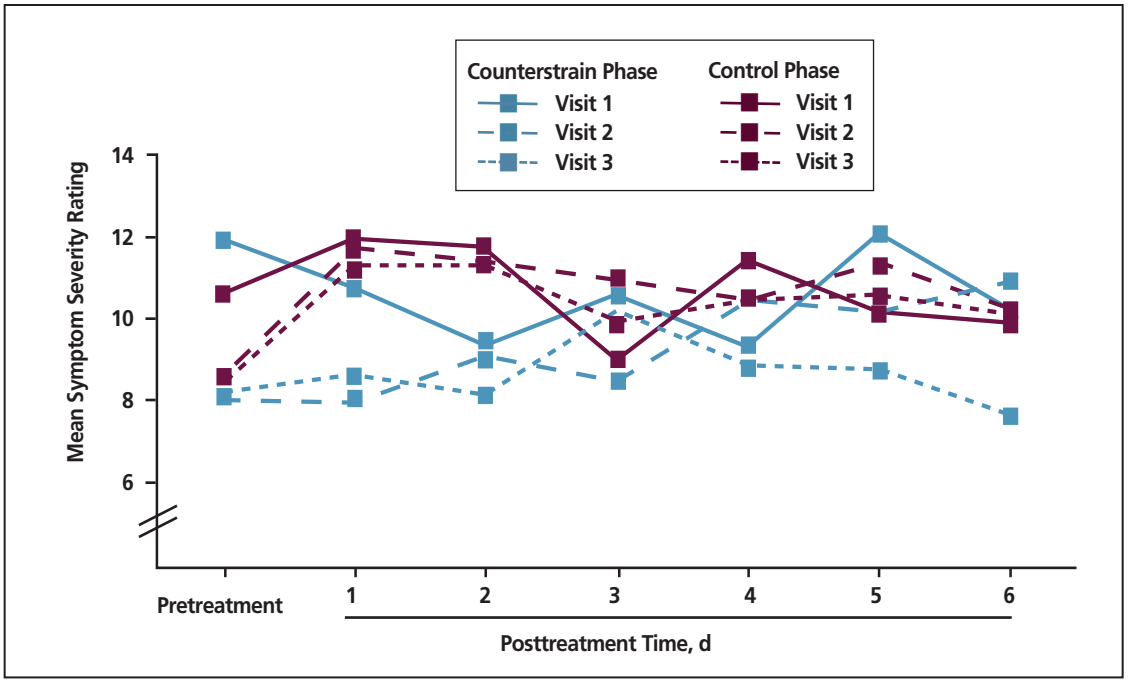


Figure 11. *Repeated-measures analysis of variance (RMANOVA) of subjects' ratings of symptom severity in the three laboratory visits of the counterstrain phase and the three visits of the control (placebo capsule) phase. The ratings were recorded as the sum of each subject's (N=19) rating of five factors: pain, soreness, stiffness, mobility, and effect on sleep. Each rating was made on a scale of 0 (no symptoms) to 9 (extreme symptoms/pain), and the summed values were then averaged. Thus the minimum possible rating is 0 and the maximum possible rating is 45. Statistical analysis using RMANOVA revealed no significant differences between the counterstrain and control phases in the ratings over the 6 days following each laboratory visit. However, a group interaction was observed during the first 2 days posttreatment, indicating a difference in responses over that time between the counterstrain and control phases.*

Furthermore, OMT was limited to the subjects' lower extremities, even in cases in which the plantar fasciitis might have arisen because of altered shock-absorber capacity of the leg resulting from dysfunction in the lower back, sacrum, and/or pelvis. Treatment designed more optimally for the individual subjects might have had more lasting effects. Greater uniformity in the activity levels of the subject population might also have improved the results. For example, one subject was a tennis teacher whose continued activity level chronically aggravated her condition.

Conclusions

Despite the limitations of the present study, the results provide evidence for the clinical effectiveness of counterstrain treatment for subjects with plantar fasciitis. Counterstrain treatment of subjects with this condition had no detectable effect on the stretch reflex or H-reflex of the triceps surae muscles as measured by EMG. The treatment did, however, alter the pattern of mechanical force output associated with these reflexes. Significant reductions in symptom severity were reported by subjects with plantar fasciitis immediately after counterstrain treatment. A smaller, but still significant, reduction in symptom severity persisted for more than 48 hours posttreatment.

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