# Stretch Reflex and Hoffmann Reflex Responses to Osteopathic Manipulative Treatment in Subjects With Achilles Tendinitis

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**Context:** Irvin M. Korr, PhD, hypothesized that sensitivity of the monosynaptic stretch reflex (ie, deep tendon reflex) plays a major role in the restriction-of-motion characteristic of somatic dysfunction, and that restoration of range of motion through osteopathic manipulative treatment (OMT) could be achieved by resetting of the stretch receptor gain.

**Objective:** To test Korr's hypothesis in the context of Achilles tendinitis, examining whether OMT applied to patients with Achilles tendinitis reduces the strength of the stretch reflex.

**Methods:** Subjects were recruited through public advertisements and referrals from healthcare professionals. There were no recruitment restrictions based on demographic factors. Amplitudes for stretch reflex and H-reflex (Hoffmann reflex) in the triceps surae muscles (the soleus together with the lateral and medial heads of the gastrocnemius) were measured in subjects with diagnosed Achilles tendonitis (n=16), both before and after OMT. These measurements were also made in asymptomatic control subjects (n=15) before and after sham manipulative treatment.

**Results:** As predicated on the concepts of the strain-counterstrain model developed by Lawrence H. Jones, DO, the use of OMT produced a 23.1% decrease in the amplitude of the stretch reflex of the soleus ($P<.05$) in subjects with Achilles tendinitis. Similarly significant responses were measured in the lateral and medial heads of the gastrocnemius in OMT subjects. The H-reflex was not significantly affected by OMT. In control subjects, neither reflex was significantly affected by sham manipulative treatment. By using a rating scale on questionnaires before treatment and daily for 7 days posttreatment, OMT subjects indicated significant clinical improvement in soreness, stiffness, and swelling.

**Conclusion:** The reduction of stretch reflex amplitude with OMT, together with no change in H-reflex amplitude, is consistent with Korr's proprioceptive hypothesis for somatic dysfunction and patient treatment. Because subjects' soreness ratings also declined immediately after treatment, decreased nociceptor activity may play an additional role in somatic dysfunction, perhaps by altering stretch reflex amplitude.

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In 1975, Irvin M. Korr, PhD, hypothesized that altered gain, or sensitivity, of the monosynaptic stretch reflex (ie, deep tendon reflex) plays a major role in the restriction-of-motion characteristic of somatic dysfunction, and that restoration of range of motion through osteopathic manipulative treatment (OMT) could be achieved by resetting of the stretch receptor gain.[1] He proposed that hyperactive stretch reflexes cause resistance to the stretch of dysfunctional muscles.[1] Lawrence H. Jones, DO, who introduced the osteopathic counterstrain procedure, appealed to Korr's hypothesis as the underlying mechanism of somatic dysfunction.[2] More recently, the focus of attention in somatic dysfunction theory has shifted away from the proprioceptors and toward the nociceptors.[3,4] Bailey and Dick[5] have suggested that both proprioceptive and nociceptive components may be at work in counterstrain. However, neither hypothesis—the one relating to proprioception or the other to nociception—has previously been tested experimentally. The purpose of the present study was to test the Korr hypothesis regarding proprioceptors[1] in the context of treatment for patients who have Achilles tendinitis.

The choice of Achilles tendinitis as an experimental example of somatic dysfunction was based, in part, on a conversation with the late Dr Jones in the fall of 1994, in which he indicated success in using counterstrain to treat patients for this condition. Another reason Achilles tendinitis was chosen for the present study was because reflexes from the muscles that insert onto the Achilles tendon (tendo calcaneus) are easy to measure.

In testing for reflex sensitivity of the triceps surae muscles (the soleus together with the lateral and medial heads of the

gastrocnemius), we chose to measure both the stretch reflex and the H-reflex (Hoffmann reflex). The H-reflex, which is elicited by electrical stimulation of the tibial nerve in the popliteal fossa, activates the Ia afferent nerve fibers from the muscle spindles of the triceps surae muscles to produce a reflex contraction.[6] The H-reflex is similar to the stretch reflex except for the fact that the H-reflex bypasses these muscle spindles, which serve to initiate the stretch reflex.[7] Because the H-reflex bypasses the spindles, it cannot be modulated by the gamma efferent system, which modulates the stretch reflex.[8]

If an experimental or clinical intervention alters the stretch reflex, but not the H-reflex, alteration of spindle sensitivity is suggested. If an intervention alters both reflexes, the mechanism is more likely to relate either to altered alpha motoneuron 2 excitability or to altered presynaptic inhibition at Ia afferent fiber endings on alpha motoneurons.

Early work on the Jendrassik maneuver, a clinical procedure in which a patient hooks his or her flexed fingers together and then pulls them apart as hard as possible to enhance weak stretch reflexes, indicated that the maneuver is ineffective in enhancing the H-reflex.[9] This suggested that the underlying mechanism of the maneuver is to increase spindle sensitivity to stretch by increasing gamma efferent fiber tone.

The idea that the stretch reflex and H-reflex are identical, except for the participation of the spindles, has been in retreat in recent years.[10,11] Although both reflexes are probably dominated by monosynaptic activation of alpha motoneurons by Ia afferent fibers, it now seems clear that oligosynaptic connections from both Ia afferents and other afferent fibers also play a role and that the different kinds of afferent fibers are activated differently in the two reflexes.[11] The stretch reflex activates both Ia afferent fibers and group II afferent fibers from the spindles. Electrical stimulation of the tibial nerve activates not only Ia afferents, which are the largest fibers, but also the Ib afferents, which overlap in size with Ia afferents. The Ib afferents originate from the Golgi tendon organs and have different effects than the Ia afferents have within the spinal cord.[8,11] Furthermore, amplitudes of the stretch reflex and H-reflex are often quite different under experimental conditions, raising questions as to whether one is truly looking at the same reflex process in these two methods of reflex elicitation.[12] In light of these findings, caution is required in interpreting similarities and differences between the stretch reflex and the H-reflex.

The present study was designed to test the Korr hypothesis in the context of OMT for subjects with Achilles tendinitis and to determine whether reflex changes, if present, were reflected in measures of amplitude for each reflex response. If stretch reflexes failed to decrease in response to treatment, it
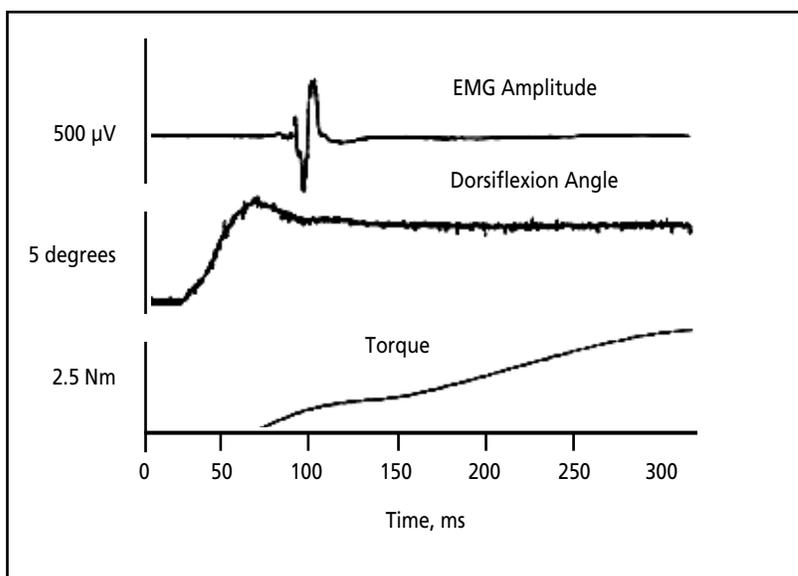


**Figure 1.** *Sample electromyogram (EMG) (top) and torque (bottom) responses of a subject's soleus muscle to a single rapidly imposed dorsiflexion of 5 degrees (middle). The initial elevation of torque results from passive stretch. The subsequent rise represents the muscle contraction resulting from the action potential seen in the EMG response.*

would cast doubt on Korr's proprioceptive theory,[1] at least in the context examined in the present study.

## Methods

All procedures used in the present study were approved by Ohio University's institutional review board in Athens. Subjects were recruited through public advertisements and referrals from healthcare professionals. No demographic restrictions (ie, age, sex, or race) were used in subject recruitment. Exclusion criteria included the presence of a neoplastic process, a history of surgical intervention in the lower limbs, and a history of manual treatment of the foot/ankle within the preceding 4 months. Sixteen patients (7 women, 9 men; average age, 38.5 y; age range, 15–70 y) were selected to receive OMT (counterstrain) for their Achilles tendinitis, and 15 asymptomatic control subjects (7 women, 8 men; average age, 34.6 y; age range, 15–61 y) were selected to receive sham manipulative treatment.

Upon arrival at the testing laboratory, all subjects were provided with a verbal description of the study procedures and were shown the testing apparatus. Each subject was then asked to read and sign a consent form, which reiterated the description of the study and the exclusion criteria.

After an initial clinical assessment of the study participants to confirm the presence of Achilles tendinitis in the OMT subjects, measurements of the stretch reflex and H-reflex were performed with the subjects seated. The subjects then

moved to a manipulation table, where either counterstrain treatment or sham manipulative treatment was provided to them as determined by group assignment. A second set of reflex measurements was performed after treatment, again with the subjects seated.

Prior to treatment, the OMT subjects were asked to rate their clinical symptoms of soreness, stiffness, and swelling on a questionnaire, with each of these symptoms rated separately on a scale of 0 to 9. After treatment and reflex testing, these subjects were given a similar questionnaire on which to rate their clinical symptoms. The subjects were directed to record their ratings 6 hours after treatment and then once daily for 1 week.

### Measurement of the Stretch Reflex
Each subject was prepared for measurement of the stretch reflex by applying electrodes to the subject's skin. The subject then sat in a chair with the foot of the leg to be treated strapped to a computer-controlled foot plate, which was equipped with a strain gauge for force measurement. The subject's knee and ankle angles were set to 130 degrees and 90 degrees, respectively, by adjusting the positions of the chair and foot plate.

The stretch reflex was elicited by the imposition of a 5-degree angle of dorsiflexion imposed over a period of 40 milliseconds by a computer-controlled stepper motor system (Model M112; Superior Electronics, Bristol, Conn). This reflex was elicited 10 times, with intervening intervals of 9 seconds. Three trials of 10 stretches were obtained, and the resultant traces of electromyogram (EMG) and torque responses were averaged for both the pretreatment phase and posttreatment phase. *Figure 1* illustrates a sample EMG record of a subject's soleus muscle response to a single rapidly imposed dorsiflexion, along with the torque response of the muscle.

### Measurement of the H-Reflex
The H-reflex was initiated by stimulation of the tibial nerve in the popliteal fossa. The electrical stimulator's anode and cathode, which were both silver/silver chloride (Ag/AgCl) electrodes (1 cm diameter), were placed on the subject's skin just proximal to the patella and centrally within the popliteal fossa, respectively. The electrodes were wrapped in an elastic bandage to help maintain proper skin contact. Stimulus pulses of 0.5-millisecond duration were delivered from the electrical stimulator (Model DS7; Digitimer Ltd, Welwyn Garden City, England) while the subject was seated with the foot strapped to the force plate. Varying intensities of current were applied to stimulate the tibial nerve, from the H-response threshold to the maximum evoked EMG response—the maximum M-wave ($M_{max}$) (*Figure 2*). The H-wave and M-wave recruitment curves were obtained pretreatment and posttreatment using the same range of current intensities for stimulation.

### EMG Recording and Data Acquisition
For EMG recording, the subject's skin was prepared by shaving, if necessary, and rubbing with prep pads containing alcohol and pumice. Bipolar Ag/AgCl electrodes (1 cm diameter, separated by 2 cm) were placed on the subject's skin overlying the soleus, the lateral and medial heads of the gastrocnemius, and the tibialis anterior muscles. The electrodes were treated with electrode gel before application to the skin. The EMG signals were fed into amplifiers ($Z_i$=100 GΩ; Intronix Technologies Corp, Bolton, Ontario), filtered between 20 Hz and 500 Hz, and sampled at 10 kHz, using a Spike II data acquisition system (Model 1401; Cambridge Electronics Design, Cambridge, England). The amplifiers were calibrated with external test signals to verify that the gains of the amplifiers were correct and uniform.

### Osteopathic Manipulative Treatment
Treatment was provided by physicians (A.G.C. 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. The OMT subjects lay prone on a standard manipulation table during the assessment and treatment phases of the experiment, each of which took approximately 10 minutes. The affected leg of each subject was pas-
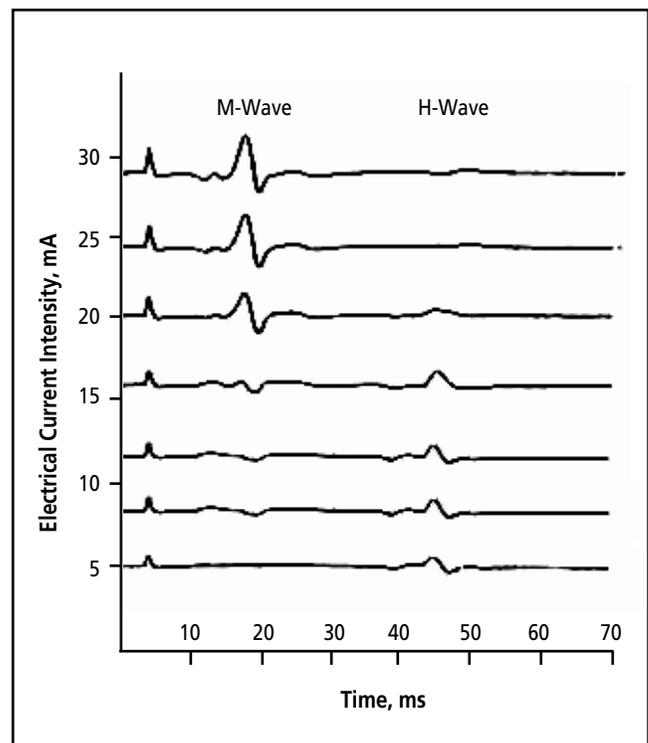


**Figure 2.** *Sample electromyogram (EMG) responses of a subject's soleus muscle to tibial nerve stimulation of varying intensities. Low-level current intensities were sufficient to stimulate only the largest fibers within the tibial nerve, producing an EMG response with a latency of approximately 40 milliseconds—the H-wave. Higher-level intensities also activated the smaller motor nerve axons, producing an EMG response with a latency of about 10 milliseconds—the M-wave.*

sively bent to approximately 90 degrees at the knee and ankle, and the subject was instructed to relax the leg muscles. This position was considered the neutral position for the purposes of the present study. During the initial assessment phase, before the first set of reflex measurements were made, the physician checked the following areas for tender points related to Achilles tendinitis: the Achilles tendon, the triceps surae above the Achilles tendon, the insertion of the Achilles tendon on the calcaneus, the lateral and medial malleoli, the navicular and cuboid bones, the subtalar joint, and the tarsometatarsal joints. The tender points comprise the system originally described by Jones[2] in strain/counterstrain treatment. The number and position of each point and the degree of subject discomfort at each point were recorded. Each subject was then moved to the reflex testing chair for the first set of reflex measurements.

Following the first set of measurements, each subject returned to the manipulation table. Based on the subject's rating of discomfort at each tender point, treatment was applied to the most painful point first, followed by the next most painful point, until all the tender points had been treated. The treatment at each point consisted of the following steps:

1. Mechanical pressure was applied to the tender point with one fingertip to determine the degree of tenderness and tissue tension.

2. The physician moved the ankle joint through the various ranges of motion (compression, dorsiflexion, eversion, inversion, lateral, medial, plantarflexion, traction, and translation [anterior and posterior]), asking the subject to indicate which position reduced the discomfort at the tender point when pressure was applied.

3. By continually monitoring tissue tension with the fingertip placed on the tender point, the physician was able to refine the subject's position of comfort until the subject reported that the pain at that point had significantly decreased. The physician kept the subject in this position for a period of 90 to 120 seconds.

4. Following the treatment of each tender point, the physician slowly returned the subject's leg to the neutral position and reexamined the originally defined area of dysfunction.

5. After treatment, the subject returned to the testing chair for a second set of reflex measurements, which were completed within 30 minutes of treatment.

### Sham Manipulative Treatment

Like the OMT subjects, the asymptomatic control subjects lay prone on the manipulation table after the first set of reflex measurements, with one leg passively bent to approximately 90 degrees at the knee and ankle. The physician then held the ankle and foot in that position for a period of 10 minutes, which was comparable with the time required for treating OMT subjects.

### Data Processing and Analysis

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

For the stretch reflex, the soleus, medial gastrocnemius, and lateral gastrocnemius, EMG records were rectified and integrated. The areas under the curves of the evoked responses for each subject were normalized by expressing them as the ratio of the measured 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 of the evoked responses were normalized by expressing them as the ratio of the measured H-reflex amplitude to the maximum M-wave amplitude ($H/M_{max}$). To determine the value of H, the highest three points on the H-wave recruitment curve (*Figure 2*) 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. The Tukey posthoc test was performed to localize specific significant differences between the subject groups.

### Results

Normalized amplitudes of stretch reflexes (expressed as $S/M_{max}$) for the triceps surae muscle group before and after treatment, averaged for the 16 OMT subjects, are listed in *Table 1* and illustrated in *Figure 3*. A 23.1% decrease was seen in the soleus muscle stretch reflex after treatment of the identified tender points of the lower leg and ankle (paired *t* test, *P*=.002). Likewise, stretch reflexes in the medial and lateral heads of the gastrocnemius decreased significantly, by approximately 18.3% (*P*=.025) and 25.7% (*P*=.003), respectively. No significant changes were observed in the amplitudes of the H-reflexes (expressed as $H/M_{max}$) after treatment of OMT subjects, as listed in *Table 1* and illustrated in *Figure 4*.

In the control subjects, no significant differences were noted in either the stretch reflex (*Figure 5*) or H-reflex (*Figure 6*) after sham manipulative treatment. The normalized values of these reflexes before and after sham manipulative treatment are listed in *Table 2*.

The absolute values of peak-to-peak voltage of the stretch reflexes for the soleus (20–300 µV), medial gastrocnemius (10–150 µV), and lateral gastrocnemius (10–100 µV) fell. For the H-reflexes of the three muscles, these values were 3 mV to 13 mV, 2 mV to 10 mV, and 2 mV to 11 mV, respectively.

To detect any changes in motor neuron excitability, which might account for altered reflex amplitudes, background EMG levels were obtained before and after treatment. Background EMG levels during the 300-millisecond intervals prior to stimulation for each of the stretch reflexes was averaged for the 30 repetitions. The background EMG values for the soleus, medial gastrocnemius, and lateral gas-

### Table 1
### Normalized Stretch Reflex and H-Reflex Amplitudes for Triceps Surae Muscles in Subjects With Achilles Tendinitis Before and After Osteopathic Manipulative Treatment (n=16)

| Muscle | Stretch Reflex* | | | H-Reflex† | | |
|---|---|---|---|---|---|---|
| | Pretreatment | Posttreatment | Difference, %‡ | Pretreatment | Posttreatment | Difference, % |
| Soleus | 0.078 | 0.062 | −23.081 | 0.500 | 0.485 | −3.173 |
| Medial gastrocnemius | 0.043 | 0.036 | −18.303 | 0.278 | 0.261 | 6.426 |
| Lateral gastrocnemius | 0.025 | 0.019 | −25.733 | 0.185 | 0.168 | −9.305 |

* Stretch reflex amplitudes are expressed as the ratio of measured stretch reflex amplitude to maximum M-wave amplitude in the same subject.
† H-reflex (Hoffmann reflex) amplitudes are expressed as the ratio of measured H-reflex amplitude to maximum M-wave amplitude in the same subject.
‡ Significant differences between pretreatment and posttreatment stretch reflexes were noted for all three muscles ($P<.05$). No significant differences were noted between pretreatment and posttreatment H-reflexes.
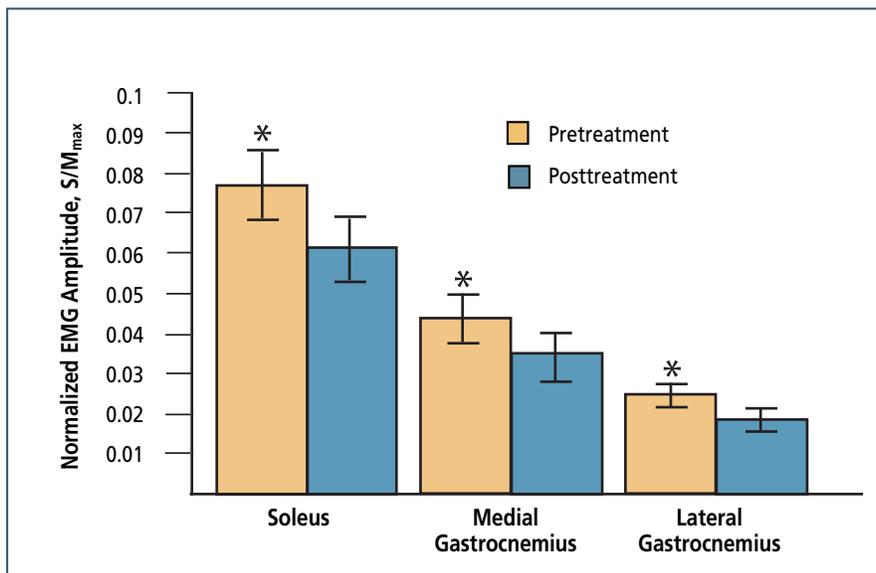


**Figure 3.** Normalized electromyogram (EMG) amplitudes of stretch reflexes for the three triceps surae muscles of subjects with Achilles tendinitis (n=16) before and after osteopathic manipulative treatment. Amplitudes are expressed as the ratio of measured stretch reflex amplitude (S) to maximum M-wave amplitude ($M_{max}$) in the same subject. The authors found statistically significant differences (*) between pre- and posttreatment reflexes for all three muscles ($P<.05$).
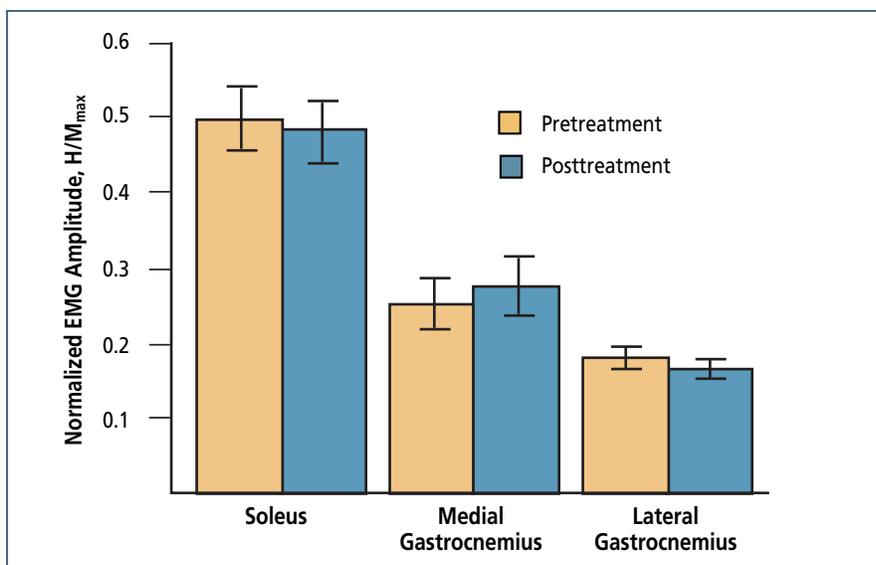


**Figure 4.** Normalized electromyogram (EMG) amplitudes of H-reflexes for the three triceps surae muscles of subjects with Achilles tendinitis (n=16) before and after osteopathic manipulative treatment. Amplitudes are expressed as the ratio of measured H-reflex amplitude (H) to maximum M-wave amplitude ($M_{max}$) in the same subject. No significant differences between pre- and posttreatment reflexes were observed for any of the three muscles.
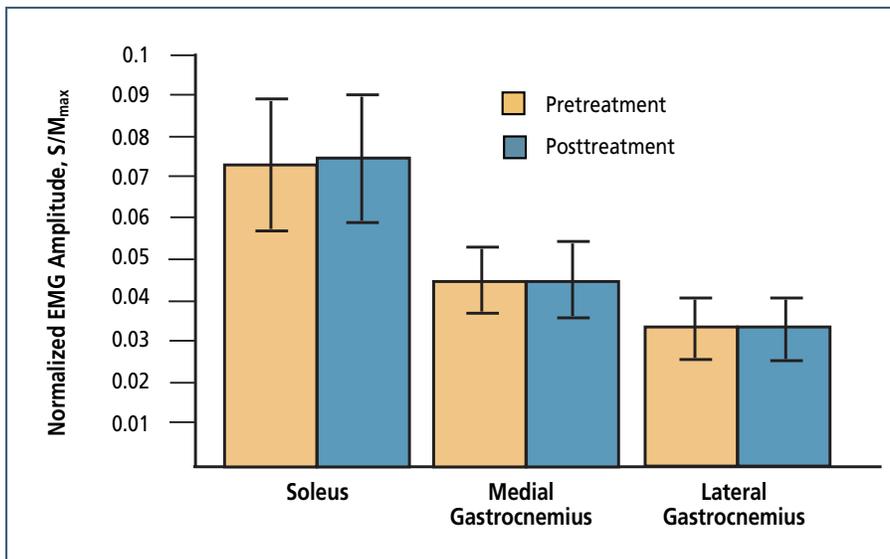
**Figure 5.** Normalized electromyogram (EMG) amplitudes of stretch reflexes for the three triceps surae muscles of asymptomatic control subjects (n=15) before and after sham manipulative treatment. Amplitudes are expressed as the ratio of measured stretch reflex amplitude (S) to maximum M-wave amplitude ($M_{max}$) in the same subject. No significant differences between pre- and posttreatment reflexes were observed for any of the three muscles.
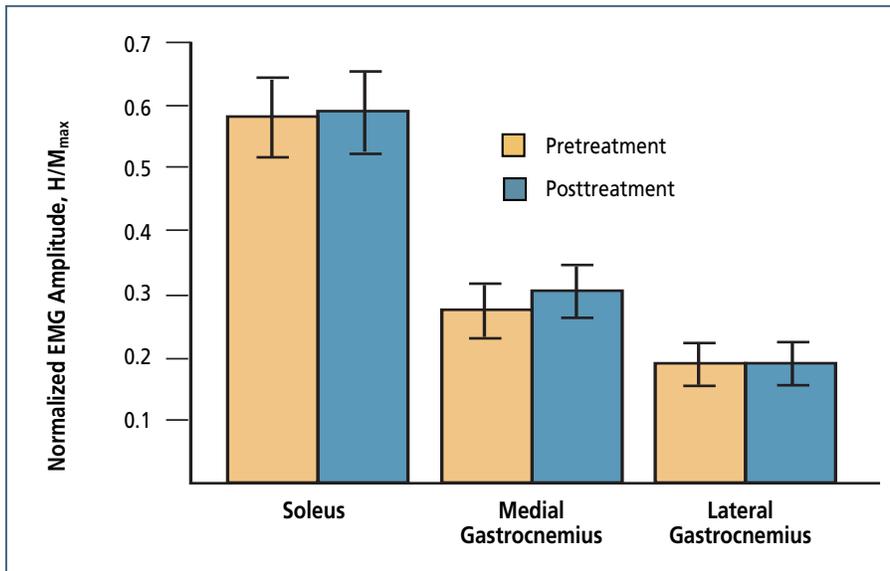


**Figure 6.** Normalized electromyogram (EMG) amplitudes of H-reflexes for the three triceps surae muscles of asymptomatic control subjects (n=15) before and after sham manipulative treatment. Amplitudes are expressed as the ratio of measured H-reflex amplitude (S) to maximum M-wave amplitude ($M_{max}$) in the same subject. No significant differences between pre- and posttreatment reflexes were observed for any of the three muscles.

**Table 2**
**Normalized Stretch Reflex and H-Reflex Amplitudes for Triceps Surae Muscles in Control Subjects**
**Before and After Sham Manipulative Treatment (n=15)**

| Muscle | Stretch Reflex[†] | | | H-Reflex[‡] | | |
|---|---|---|---|---|---|---|
| | Pretreatment | Posttreatment | Difference, % | Pretreatment | Posttreatment | Difference, % |
| Soleus | 0.075 | 0.076 | 1.326 | 0.586 | 0.596 | 1.636 |
| Medial gastrocnemius | 0.046 | 0.047 | 1.240 | 0.278 | 0.308 | 10.105 |
| Lateral gastrocnemius | 0.034 | 0.034 | −1.002[§] | 0.194 | 0.192 | −0.884 |

* No significant differences between pretreatment and posttreatment reflexes were observed for any of the three muscles.
† Stretch reflex amplitudes are expressed as the ratio of measured stretch reflex amplitude to maximum M-wave amplitude in the same subject.
‡ H-reflex (Hoffmann reflex) amplitudes are expressed as the ratio of measured H-reflex amplitude to maximum M-wave amplitude in the same subject.
§ Before rounding, the pretreatment amplitude was 0.0341 and the posttreatment amplitude was 0.0338.

trocnemius are shown in *Table 3*. No significant differences in these values between the pretreatment and posttreatment periods were detected.

### Clinical Outcomes of Counterstrain Treatment

Subjects who received OMT reported various ratings of soreness, stiffness, and swelling (*Table 4*) during the 1-week period following counterstrain treatment. Mean ratings of soreness and stiffness were found to be significantly reduced between pre- and posttreatment levels, as recorded at 6 hours posttreatment and daily for 7 days posttreatment (*P*<.05). Subjects reported that swelling was significantly reduced on posttreatment days 2 through 6 (*P*<.05).

### Comment

The present study tested Korr's hypothesis that OMT reduces the gain of the stretch reflex.[1] Our data indicate that, at least in the case of Achilles tendinitis, this does appear to be a mechanism. Two observations, in particular, indicate that the effect seen on the stretch reflex amplitude in these experiments was not simply an artifact of the measuring conditions (ie, a result of the second reflex measurement always being less than the first). First, no changes were observed in response to sham manipulative treatment in asymptomatic subjects. Second, the reduction in gain was seen in the stretch reflex but not in the H-reflex.

Our data clearly indicate a decreased stretch reflex amplitude following OMT in subjects with Achilles tendinitis. However, in the absence of randomized assignment of symptomatic subjects to a sham manipulative treatment group, we cannot exclude the possibility that the altered reflex amplitude observed in asymptomatic controls resulted from the nonspecific treatment effects of the sham protocol. It is also possible that asymptomatic control subjects in the present study failed to respond to sham manipulative treatment because their reflex gain was already at some minimum level that could not be further reduced. A sham manipulative treatment like the one given to these asymptomatic subjects might conceivably have caused a reflex change in the subjects with Achilles tendinitis.

The subjective clinical improvements recorded during posttreatment days 1 through 7 could, in principle, have resulted from any aspect of a subject's experience in the testing laboratory, including the treatment, the reflex measurements, and the psychological impacts of the measurements. However, the decrease in soreness and stiffness that was assessed immediately before and after treatment, with no intervening reflex measurements, suggests that the clinical improvement resulted from the treatment itself. Nevertheless, without evaluating sham manipulative treatment of subjects with Achilles tendinitis, it is not possible to know if either the clinical improvement or the reflex change resulted from the biomechanical or the psychological aspects of treatment.

A previous investigation by Newham and Lederman[13] of the effects of manual treatment on the amplitude of the quadri-

ceps stretch reflex in normal, asymptomatic subjects found no effects of static manipulation (massage and repeated isometric contractions). Decreases in stretch reflex amplitudes did occur in response to dynamic interventions (passive flexions/extensions of the knee joint and active extensions of the leg), but these effects lasted less than 1 minute. Neurophysiologic studies in feline and human subjects have documented depressions in H-reflexes with repeated nerve stimuli, an effect that may be related to presynaptic inhibition or other synaptic processes within the spinal cord.[14] However, as with the stretch reflex in the Newham and Lederman study,[13] these H-reflex depressions were also transient, lasting seconds or even less.[14] In the present study, the course of the reduction in stretch reflex amplitude was not followed carefully over an extended period of time, but the effect was noted to last at least several minutes. Five to 10 minutes were required following treatment to position our subjects back in the test apparatus and reconnect them for EMG recording.

It is tempting to conclude that the effects of treatment on stretch reflex, in the absence of any effect on either the H-reflex or the background EMG level, proves that the observed reduction in reflex amplitude was caused by decreased tone of gamma efferent fibers, resulting in decreased spindle sensitivity. Judgment on this possibility, however, must be suspended. If one assumes that the two reflexes are identical, except that the H-reflex bypasses the stretch receptors in the muscle, our results do suggest that OMT decreases the muscle sensitivity to stretch, possibly by decreasing gamma efferent activity. This was the explanation originally offered for the differential sensitivity of the stretch reflex and H-reflex to the Jendrassik maneuver.[7,9] The explanation has, however, fallen from favor with the demonstration that the H-reflex, when elicited with low-level stimuli to produce a reflex response comparable in amplitude to the stretch reflex, is enhanced by the Jendrassik maneuver.[12]

In addition, afferent nerve signals coming from the muscle spindles of the triceps surae muscles in response to stretch have been reported to be unaffected by the Jendrassik maneuver.[15] This observation, however, has recently been challenged.[16] Gregory et al[17] demonstrated that the Jendrassik maneuver fails to cause slack to be taken up in muscle spindles that are passively shortened following an isometric contraction, indicating that, under these conditions, the Jendrassik maneuver fails to activate the gamma efferent system. This finding seems to rule out modulation of the gamma efferent system as a mechanism of the Jendrassik maneuver. Nevertheless, still other recent reports[14,16] have demonstrated increased muscle spindle sensitivity in relaxed muscles in response to mental arithmetic tasks and the Jendrassik maneuver.

The idea that the Jendrassik maneuver alters the excitability level of motor neurons has been ruled out by the observation that, while it enhances the H-reflex, the Jendrassik maneuver does not alter the background EMG levels.[17,18] Some

**Table 3**
**Mean Background Electromyogram Levels for Triceps Surae Muscles in OMT and Control Subjects (N=31)**

| Muscle | Amplitude, Mean μV ± SE* | |
|---|---|---|
| | Pretreatment | Posttreatment |
| Soleus | 4.9 ±1.6 | 5.1 ±1.6 |
| Medial gastrocnemius | 12.8 ±6.4 | 15.7 ±13.1 |
| Lateral gastrocnemius | 12.7 ±5.3 | 16.2 ±10.2 |

\* No significant differences between pretreatment and posttreatment background electromyogram levels were observed for any of the three muscles.
**Abbreviation:** OMT, osteopathic manipulative treatment.

reflex is slow enough for oligosynaptic connections to contribute to it.[11,19] These oligosynaptic contributions, which may be excitatory or inhibitory, may cause the stretch reflex to respond differently than the H-reflex to synaptic inputs that arise from the Jendrassik maneuver or intervention with OMT.[20] Whatever the mechanism of Jendrassik enhancement of the stretch reflex may be, the mechanism involved in OMT (as seen in the present study) is not necessarily the same.

It is not possible to say whether the reduction of the stretch reflex gain seen in the present study is related to the clinical improvement reported by OMT

researchers have proposed that the Jendrassik maneuver might operate by reducing presynaptic inhibition.[18] However, the failure of the Jendrassik maneuver to add to the facilitation of the soleus H-reflex by femoral nerve stimulation casts doubt on this explanation because multiple sources of presynaptic inhibition characteristically exhibit summation.[17] Thus, the mechanism of even the most well-studied modifier of stretch reflex gain remains unclear.

The stretch reflex and H-reflex differ in ways other than the involvement (or lack of involvement) of the stretch receptors of the muscle spindles. Although both reflexes involve oligosynaptic and monosynaptic connections, the oligosynaptic connections differ in the two reflexes. For example, the rise time of the excitatory postsynaptic potential for the stretch

subjects as pain relief. However, if the reduced stretch reflex gain and the reported clinical improvement are related, it is not clear which of these factors is cause and which is effect. Korr[1] and Jones[2] hypothesized that the reduction in reflex gain would help unload the muscle, leading to the reduction of what is now known as overuse and, subsequently, to pain. Studies on feline subjects have shown that the electrical stimulation of group II and group III afferent fibers—but not group I afferent fibers—has excitatory effects on gamma motoneuron excitability.[21] Group III afferents include fibers that respond both to nociceptive and nonnociceptive inputs.[22]

Research with human subjects also suggests that nociceptive inputs in relaxed leg muscles acutely activate fusimotor neurons.[23,24] However, the stretch of elbow flexors that had been made sore by prior bouts of eccentric exercise does not generate reflex activity in the stretched muscles.[25]

Chronic activation of muscle nociceptors, as in experimentally induced myositis in feline subjects, actually decreases gamma motoneuron activity to the inflamed muscle.[26] This mechanism would serve to minimize the activity of chronically sore muscles. It seems possible that chronic nociception might decrease reflex excitability of the muscle generating the pain signals while increasing the reflex excitability of synergistic muscles, thereby encouraging them to take over for the injured muscle, as in muscle guarding.

In subjects with Achilles tendinitis, it is not clear whether the reflex activity in the triceps surae is increased or decreased compared with normal subjects. Although the present study suggests that stretch reflexes decrease with OMT, the neural mechanism responsible

**Table 4**
**Pretreatment and Posttreatment Ratings of Perceived Symptom Severity by Subjects With Achilles Tendinitis Who Received Osteopathic Manipulative Treatment (n=16)**

| Time | Symptom Rating, Mean±SE* | | |
|---|---|---|---|
| | Soreness† | Stiffness† | Swelling‡ |
| ■ Pretreatment | 5.69±0.64 | 5.38±0.52 | 2.44±0.43 |
| ■ Posttreatment | | | |
| □ 6 h | 4.25±0.69 | 3.31±0.49 | 1.88±0.22 |
| □ 1 d | 5.00±0.65 | 4.25±0.53 | 1.81±0.23 |
| □ 2 d | 4.38±0.69 | 3.81±0.59 | 1.75±0.28 |
| □ 3 d | 4.31±0.69 | 3.56±0.57 | 1.88±0.27 |
| □ 4 d | 3.84±0.65 | 3.56±0.61 | 1.81±0.28 |
| □ 5 d | 3.69±0.63 | 3.31±0.62 | 1.75±0.28 |
| □ 6 d | 3.88±0.70 | 3.06±0.62 | 1.75±0.28 |
| □ 7 d | 4.31±0.72 | 3.31±0.64 | 1.88±0.34 |

\* Subjects rated the severity of their symptoms on a scale of 0 to 9.
† All posttreatment ratings of soreness and stiffness were significantly lower than pretreatment ratings ($P<.05$).
‡ Posttreatment ratings of swelling on days 2 through 6 were significantly lower than pretreatment ratings ($P<.05$).

for this decrease remains unknown. One possibility is that the primary effect of OMT is to reduce pain, and decreased nociceptive input, in turn, decreases stretch reflex gain. This possibility is consistent with the proposals of Van Buskirk[3] and Bailey and Dick[5] that nociception plays a central role in the body's response to OMT. Nociception may play this role, at least in part, by altering proprioceptive gain. If OMT works via inhibition of pain afferent fibers, this inhibitory mechanism may account for the difference between our results and those of Newham and Lederman,[13] who reported no effect of manual treatment in control subjects.

## Conclusion

The results of the present study indicate that the amplitude of the stretch reflex decreases after OMT—compared with pretreatment reflex amplitudes—in subjects with Achilles tendinitis. This response is consistent with Korr's hypothesis[1] that OMT for somatic dysfunction may act by reducing the gain of the stretch reflex. Because OMT reduced subjects' pain in our study, the observed response is also consistent with the postulate that modulation of nociceptive inputs contributes to the body's response to manual therapy.

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