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Ischemic compression and joint mobilisation for the treatment of nonspecific myofascial foot pain: findings from two quasi-experimental before-and-after studies

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Objective: *The aim of this study was to evaluate the efficacy of myofascial therapy involving ischemic compression on trigger points in combination with mobilization therapy on patients with chronic nonspecific foot pain.*

Study design: *Two quasi-experimental before-and-after studies involving two different baseline states.*

Method*: Foot pain patients at a private clinic were divided into two separate cohorts: A, custom orthotic users; and B, non-users. In Study A, 31 users received 15 experimental treatments consisting of ischemic compressions on trigger points and mobilization of articulations through the foot immediately after study enrollment. In study B, ten non-users were prescribed a soft prefabricated insole and were monitored for five weeks before subsequently receiving 15 experimental treatments after the initial five-week delay.*

Outcome measures: *The Foot Function Index (FFI) and patients' perceived improvement score (PIS) on a scale from 0% to 100%.*

Results: *The Study A group (n=31) maintained a significant reduction in the FFI at all three follow-*

Objectif : *L'objectif de la présente étude est d'évaluer l'efficacité de la thérapie myofasciale impliquant une compression ischémique sur des points gâchettes combinée à une thérapie de mobilisation chez les patients souffrant de douleurs chroniques non spécifiques au pied.*

Plan d'étude : *Deux études quasi expérimentales avant/après sur deux états de référence différents.*

Méthodologie : *On a formé deux groupes avec les patients souffrant de douleurs au pied d'une clinique privée. A : les utilisateurs d'orthèses adaptées. B : ceux qui n'en utilisent pas. Dans l'étude A, 31 utilisateurs ont reçu 15 traitements expérimentaux impliquant une compression ischémique sur des points gâchettes et une mobilisation des articulations du pied immédiatement après l'inscription à l'étude. Dans l'étude B, 10 non-utilisateurs ont reçu une prescription de semelle souple préfabriquée et ont fait l'objet d'un suivi de 5 semaines. Après les 5 semaines de suivi, ils ont reçu 15 traitements expérimentaux.*

Mesures des résultats : *L'index de fonction du pied (IFP) et l'amélioration perçue par le patient (APP) sur une échelle de 0 % à 100 %.*

Résultats : *Le groupe de l'étude A (n=31) a montré une diminution importante de l'IFP aux trois évaluations*

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up evaluations. Mean improvement from baseline in FFI was 47%, 49% and 56% at 0, 1 and 6 months, respectively, post-treatment. Mean PIS was 58%, 57%, and 58%, again at 0, 1 and 6 months post-treatment. For the Study B group, mean improvement in FFI was only 19% after the monitoring period, and 64% after the experimental treatment period. Mean PIS was 31% after monitoring, and 78% after experimental treatment. In repeated measures analyses, experimental treatment was associated with a significant main effect in both of these before-and after studies (all P values<0.01).

Conclusion: *Combined treatment involving ischemic compression and joint mobilization for chronic foot pain is associated with significant improvements in functional and self-perceived improvement immediately and at up to six-months post-treatment. Further validation of this treatment approach within a randomized controlled trial is needed.*

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KEY WORDS: foot pain, myofascial trigger points, ischemic compression, joint mobilization, chiropractic

de suivi. L'amélioration moyenne selon la référence de l'IFP était de 47 %, 49 % et 56 % après 0, 1 et 6 mois respectivement. La moyenne d'APP était de 58 %, 57 % et de nouveau 58 % 0, 1 et 6 mois après le traitement. Le groupe de l'étude B a montré une amélioration moyenne de l'IFP de seulement 19 % après la période de suivi, et de 64 % après la période de traitements expérimentaux. La moyenne d'APP était de 31 % après le suivi et de 78 % après les traitements expérimentaux. Dans les analyses de mesures répétées, on associe le traitement expérimental à un effet principal important dans les deux études avant/après (toutes les valeurs P<0,01).

Conclusion : *Un traitement combiné impliquant une compression ischémique sur des points gâchettes et une mobilisation des articulations pour soulager des douleurs chroniques au pied est associé à des améliorations fonctionnelles importantes et une amélioration cernée par le patient sur une période allant d'immédiatement après le traitement jusqu'à six mois plus tard. Il est nécessaire d'effectuer un essai contrôlé aléatoire pour valider ce traitement.*

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MOTS CLÉS : douleur au pied, myofascial, points gâchettes, compression ischémique, mobilisation des articulations, chiropratique

Introduction

Foot pain is very common. In one population-based study of 4,060 subjects, 17.4% had foot pain, aching or stiffness in either foot.[1] Plantar fasciitis affects about 10% of the population at some time during their lives.[2] Regarding hallux valgus, one systematic review and meta-analysis of 76 pooled surveys (496,957 participants) estimated that the prevalence was 23% in adults aged 16-65 years and 35.7% in elderly people aged 65 years or older. It was 30% in females compared to 13% in males.[3] Foot problems are commonly encountered in chiropractic practice.[4,5] It is often assumed that biomechanics is the cause of almost any condition seen in the foot;[6] this is seemingly proved by the biomechanical abnormalities present in the pathological foot.[6] But while biomechanical abnormalities are common, it should not be assumed that they are necessarily causatively associated with foot symptoms. In this regard, it is important to rule out other medical or specific causes of foot pain before assuming that a biomechanical fault is at play.[6]

Specific foot pain conditions

For background purposes, we summarize a number of common specific conditions that should be ruled out prior to applying a working diagnosis of nonspecific biomechanical foot pain.

Plantar fasciitis

The plantar fascia is a thick band of longitudinally arranged fibers which run from the tuberosity of the calcaneus. The thick, central portion divides distally into five slips (one for each digit), and inserts into the sides of the

sheath of the flexor tendon of each toe.[7] The cause of plantar fasciitis is a repetitive microtrauma overload injury.[8] The point of injury is usually located at the attachment of the plantar fascia on the inferior aspect of the calcaneus. On digital palpation a point of hyperirritability or severe point tenderness is found at the insertion of the plantar fascia into the calcaneus. The most common symptom is "first-step pain," immediately after prolonged non-weight bearing, which quickly diminishes after the next few steps. In proximal plantar fasciitis, over-pronation is commonly associated, as are simple training errors.[8] Less commonly, tenderness over the distal and mid portion of the plantar fascia indicates the presence of distal plantar fasciitis.[8,11] Plantar spurs occur in 20% of normal patients without plantar fasciitis and in about 50% of patients with plantar fasciitis, so their presence is suggestive, but by no means diagnostic.[9,11]

The natural history of plantar fasciitis is unclear.[11] Bone scans are indicated only in problematic cases.[9] Commonly administered treatments include nonsteroidal anti-inflammatory drugs (NSAIDs), heel pads or cups, orthotics, steroid injections, ultrasound, deep friction massage, shock wave therapy, and active release.[2,4,7,12] Surgery is thought to be indicated after nine months of failed conservative treatment.[5,8,9,10] Although there is consensus that conservative treatments are effective most of the time, there is no agreement as to which specific modality is most effective.[11]

Hallux valgus, hallux rigidus, turf toe, and bunion

There are many interrelated painful conditions of the great toe. Some are considered to be complications of acute or chronic sprain/strain of the first metatarsophalangeal joint. This category accounts for the most common athletic disorders of the foot.[13] Each of these diagnoses is often based simply on the elicitation of exquisite tenderness during passive extension of the great toe, with or without simultaneous observation of a bunion.[14] The exact cause of hallux valgus is unknown.[15] The literature suggests that advanced imaging studies and lab studies are uninformative for hallux problems in general.[15] Commonly administered treatments for hallux problems include manipulation, transverse friction therapy, gradual axial elongation and progressive mobilization of the first metatarsophalangeal joint.[13,14,16,17] There is some evidence that mobilization of the first metatarsophalangeal joint is of value in the treatment of hallux rigidus.[13] Yet, in one Cochrane systematic review, there was no evidence of a difference in effectiveness between various conservative treatments and no treatment for hallux problems. Furthermore, that same review found no evidence from randomized controlled trials of any benefit (e.g., diminution of joint deformity or pain) from the use of orthotics or splints.[14] For hallux valgus in particular, treatment has often been aimed at reducing deformity, and improving pain, function, and patient satisfaction.[15,16] However, the genuine effectiveness of non-surgical interventions for hallux valgus remain questionable.

Metatarsalgia

The main symptom is a burning sensation located at the ball of the foot. When speaking of metatarsalgia, it is customary to exclude conditions affecting the first metatarsophalangeal joint, as those conditions tend to fall under the umbrella of hallux valgus, hallux rigidus and other related conditions of the big toe.[18]

Morton's neuroma

Morton's neuroma is a misnomer as the underlying pathology does not involve a proliferative state of the nerve tissue.[19] For this reason, the term "Morton's metatarsalgia" is preferred by some authors. In any event, this condition is a paroxysmal neuralgia affecting the web spaces in the toes, typically the third. The pain may be sharp and lancinating when walking. The diagnosis is typically based on a history of symptoms such as pain, numbness or pins and needles in the ball of the foot and/or toes, particularly during walking. On examination digital pressure applied to the third and/or fourth intermetatarsal area provokes pain. Neuromas are perhaps the most misdiagnosed of all podiatric complaints and, in reality, are not nearly as prevalent as once believed. Instead, arthritis and other conditions causing inflammation of the metatarsophalangeal joints, and pressure on the interdigital nerves can mimic a true neuroma.[6]

Many patients undergo surgery for this condition, yet there is little evidence on the effectiveness of surgical as well as non-surgical interventions for Morton's neuroma.[19] As surgical removal of a misdiagnosed neuroma may cause an exacerbation of the symptoms and even disability,[6] it is imperative to exhaust non-invasive approaches before contemplating surgery.

Longitudinal arch pain

Often, pain along the longitudinal arch occurs at the cuboid-metatarsal articulation. There are three mechanisms of injury associated with this condition: 1) acute injury from forceful plantar flexion and inversion of the foot and ankle, 2) compensatory weight bearing over the lateral border of the foot in response to antecedent medial plantar heel pain, and 3) biomechanical predisposition to a lateral forefoot sprain, such as in the presence of pes cavus deformity.[20] In treating this problem, mobilization, manipulation, long axis distraction, and orthotics have been suggested.[20,21]

Tendinopathy of the Achilles tendon

Posterior heel pain most commonly arises from the Achilles tendon. The clinical picture is pain and swelling immediately above the heel, with related impairment of physical function. The Achilles tendon is the conjoint tendon of the soleus and the two heads of the gastrocnemius muscles. Patients with insertional tendinopathy present with posterior heel pain, mostly in the middle region and insertion of the tendon. Overuse is the principal cause. Usual treatments include orthosis, stretching, anti-inflammatory medication and steroid injection, the latter of which should be performed only sparingly due to the potential risk of tendon rupture. Surgery is considered only when all nonoperative treatments have been tried.[22]

Nonspecific foot pain

In the absence of the above-mentioned specific causes of chronic foot pain, we suggest that a diagnosis of nonspecific foot pain is applicable. For nonspecific foot pain, we have utilized a treatment approach that appears to be promising from an anecdotal perspective, but has not yet been formally validated to date.

The proposed treatment assumes that nonspecific foot pain has, at least in part, a myofascial component which may be present either exclusively or in addition to the aforementioned specific causes of foot pain. Our use of ischemic compression and joint mobilization is therefore based on the theoretical rationale that myofascial trigger points (TrPs) may be located within muscles, ligaments, tendons, fascia, and articular capsules of the painful foot.[23,24] Previous studies (further detailed in the Discussion section) have shown that other manual myofascial therapy techniques such as friction massage, mobilization, and Graston Instrument Mobilization Technique have helped alleviate many foot problems.[9,13,14,16,21,25]

Study rationale

The objective of this study was to determine the efficacy of ischemic compression and joint mobilisation for the treatment of common nonspecific foot pain of presumed myofascial origin.

Our primary hypothesis was that private clinic patients with chronic foot pain who are treated with ischemic compression on trigger points, as pinpointed through palpation and patient corroboration, in combination with joint mobilization, would exhibit significant improvement in the severity of symptoms and functional status after 15 experimental treatments.

Methods

We conducted a controlled before-and-after study in which data was collected in two populations contemporaneously within a private clinic setting in Trois-Rivières, Québec. However, as both populations were exposed to an experimental treatment phase, each constituted a separate quasi-experimental study individually as well as a controlled before-and-after study collectively.

Each individual study involved a single group repeated measures design within a distinct cohort of foot pain patients. Study A was a before-and-after study of the effect of manual therapy in a cohort of custom orthotic users. Study B was a before-and-after study of the effect of delayed manual therapy in a cohort of non-users who were prescribed a soft prefabricated orthotic for five weeks before undergoing the experimental manual therapy approach over an additional five weeks. Therefore, during the first five weeks of data collection only the Study A cohort received experimental treatment while the Study B cohort was monitored contemporaneously without receiving experimental treatment.

All participants received experimental manual therapy from one of two experienced chiropractors (GH, AML). All data collection was performed by the principal investigator (GH) who was clearly aware of each participant's degree of progress throughout the study. A study statistician, on the other hand, was blinded to the types of treatments being administered and analyzed. The study was approved by the ethics committee of the Université du Québec à Trois-Rivières.

All subjects were recruited through local newspaper advertisements. In order to take part in one of the studies, participants had to be between 20 and 60 years of age and had to have suffered from daily foot pain for at least three months. Foot pain was defined as pain anywhere in the foot, either at rest or during movement. The intensity of the pain had to be rated at least a "6" on a 10 cm visual analogue scale. Participants agreed to receive, free of charge, 15 manual therapy treatments over five weeks (one month), at a frequency of three times per week.

The exclusion criteria for both studies included past surgery to the symptomatic foot, body weight greater than 200 pounds, a history of steroid or local anaesthetic injection to the foot within the past month, a current history of local tumour, infection, fracture, rheumatoid arthritis or any other active arthropathy.

As podiatry is taught at the Université du Québec à Trois-Rivières, most of the participants (readers of the local newspaper) were residents of the local community who had already seen a Doctor of Podiatric Medicine and were also already using custom orthotics. Arbitrarily, we recruited all participants who were already wearing a custom orthotic into Study A while participants not wearing a custom orthotic were recruited for Study B.

Written informed consent was obtained from each participant.

Experimental interventions

Ischemic compression treatment over trigger and/or tender points.

The diagnosis of a treatable TrP in this study involved the patient pinpointing the exact location of pain on the foot, and the attending chiropractor reproducing the patient's pain upon applying thumb-over-thumb pressure over the location in question. If the patient's pain was reproduced, ischemic compression treatment was administered during each visit, and consisted of a single application of 15-seconds of pressure (again using a thumb-over-thumb contact) over each treatable TrP identified in the foot. As the TrP could be very sensitive, the pressure was light during the first few seconds, and gradually increased to the point of the patient's maximum tolerance.

Patients were positioned in a standardized manner depending upon the area of the foot being treated:

1. Plantar fascia: The patient was in either a supine position with the legs extended or in a prone position with the knee flexed to 90 degrees.

2. Dorsal aspect of the foot: To target TrPs over the dorsal metatarsal or tarsal regions, ischemic compression was applied with the patient supine and the foot flat on the table. The pressure was applied perpendicularly to the dorsal surface of the foot. The therapist sometimes used his body weight to augment the application of pressure through the thumbs.

3. Lateral longitudinal arch: The patient would lie in a recumbent position with the affected side (foot) up, and the medial aspect of the affected foot resting on the table. In this position, the plantar surface of the cuboid bone was the most common site treated.

4. Achilles tendon: With the patient in a prone position, the tendon was palpated along its entire length. Pressure was applied perpendicularly to the skin surface over tender points anywhere along the length of the tendon. TrPs in the soleus and the gastrocnemius muscles were also treated.

5. Center of the fat pad of the heel: The patient was treated either in a prone or standing position with the dorsal surface of the forefoot facing down and resting flat on the table, and with the plantar surface of the heel exposed and facing up. Often, treatable TrPs were detected and therefore targeted over the entire plantar surface of the heel.

6. Anterior capsule of the foot: While the patient was supine, the therapist stabilized the foot by applying pressure to the ball of the foot (and therefore forceful ankle dorsiflexion) with his thigh. At the same time the ankle-mortise joint was probed deeply with the thumbs in order to test for the presence of treatable TrPs and/or movement restriction in the ankle. Particularly in this region of the foot, ischemic compression and joint mobilisation were often administered simultaneously.

Mobilization

Joint mobilisation consisted of applying forceful flexion or extension movement, within the patient's level of tolerance, through the hypomobile articulation for 15 seconds per administration. The following standardized approaches were used:

1. First metatarsophalangeal articulation (big toe): Depending on the direction of detected restriction, pressure for 15-seconds was applied in either extension, flexion, or both directions. Usually, treatment was given in the direction that was limited by pain. In symptomatic pa-

tients, pressure was applied very lightly during the first few seconds, then gradually increased to the patient's maximum tolerance. Thereafter, pressure was sustained without moving the joint any further. The same treatment was used for treating an underlying hallux rigidus. The diagnosis of a concomitant hallux rigidus was based on the presence of exquisite tenderness upon passive extension of the big toe, with or without bunion formation.[16]

2. Third and fourth metatarsophalangeal articulation (sometimes in the presence of concomitant Morton's neuralgia): Mobilisation was carried out with the patient in a supine position. Hyperirritability was tested for by hyperextending and hyperflexing the third and fourth metatarsophalangeal joints. If pain was reproduced, then the affected joint was treated by administering gradual, yet forceful, hyperextension or hyperflexion to tolerance, for 15 seconds per visit. Ischemic compression therapy was often concomitantly administered to treatable TrPs over the lateral aspects of metatarsophalangeal joints.

3. Generalized metatarsophalangeal stiffness: Simultaneous treatment to multiple metatarsophalangeal articulations was carried out with the patient in a supine position. The attending chiropractor would grasp the last four toes of the patient's foot by applying a thenar and hypothenar contact against the plantar surfaces of the toes with one hand, and by reinforcing his grasp with the other hand. All four toes were then simultaneously hyper-extended to the limit of the patient's tolerance and held in that position for 15 seconds. In most case this treatment was performed in combination with ischemic compression therapy over treatable TrPs in the ball of the foot.

Most patients suffered from the presence of concomitant specific foot conditions. In the presence of heel pain, plantar fasciitis, arch pain, and fat pad pain, TrPs located in the calf muscles were also treated. In the foot itself, there were typically one or two TrPs for each condition.

Patients were encouraged to taper off any existing analgesic medications as soon as possible. During treatments, patients were monitored for even the slightest signs of distress and were repetitively asked if the administered pressure or mobilisations were bearable.

Treatments were repeated until either hyperirritability was completely gone or a maximum of 15 visits had been administered. The presence of hyperirritability (pain reproduction) was the basis for the diagnosis of treatable TrPs or joint hypomobility. If the problem was a TrP, the tender point pinpointed by the patient was easily confirmed through palpation over the area in question. If the location was at an articulation, hyperirritability, if any, was detected by forcing the articulation to the end of flexion and/or extension.

Additional Interventions (Study B)

In study B, patients were initially treated for five weeks with only a soft prefabricated insole (Holiday, Pedag International, Germany). After five weeks each participant was invited to receive, free of charge, a course of experimental therapy as described above.

Outcome measures

Foot Function Index (FFI) questionnaire

The FFI was used to measure the impact of pathology on function in terms of pain, disability, and activity restriction. It is a self-administered index consisting of 23 items making up three sub-scales. The test-retest reliability of total and sub-scale scores ranges from 0.87 to 0.69. Internal consistency ranges from 0.96 to 0.73.[26] The FFI is an easily administered clinical index which provides a practical method of measuring foot function in an outpatient setting.[26]

Perceived Improvement Score (PIS).

The PIS is ascertained by asking a patient to rate his/her perceived degree of improvement on a numerical scale from 0 to 100%, where 0 means no improvement and 100% means complete improvement

Timing of measurements

In the Study A, FFI measurements were ascertained at baseline. Subsequently, follow-up FFI and PIS measurements were ascertained at one month post-enrolment (immediately after 15 treatments), two months post-enrolment (one month post-treatment) and seven months post-enrolment (6 months post-treatment).

In Study B, measurements were again obtained at enrolment (baseline). Subsequently, both FFI and PIS were ascertained at one month post-enrolment (after one month of soft orthotic use), and then at two months post-enrolment (immediately after completion of 15 manual therapy treatments in combination with ongoing soft orthotic use).

Statistical Analysis

The percent change in FFI was calculated by subtracting the baseline FFI score from the follow-up measurements, and then dividing by the baseline score. Percent change in PIS was measured directly from the raw questionnaire score and therefore required no statistical transformation.

To test the effects of experimental treatment over time, a one-way repeated-measures ANOVA was performed for each study. Multiple paired t-tests were performed to compare each follow-up measurement to the baseline measurement.

Also, to exploit the controlled before-and-after component of the data, the immediate post-treatment percent changes in the FFI and the PIS from the Study A group were compared to the corresponding one-month post-monitoring period changes in the Study B group using a t-test for independent samples. In this latter analysis Study B participants who were issued a prefabricated orthotic and only monitored during the first month were used as an external control group for Study A participants who were both active custom orthotic users and had received experimental manual therapy during the first month. For all analyses, statistical significance was set at a conventional level of $P < 0.05$.

Results

Figure 1 depicts the flow of patients and timing of measurements for Studies A and B. A total of forty-one patients met initial inclusion criteria and agreed to participate. Thirty-one participants were active users of custom orthotics and were therefore enrolled into Study A. Ten participants were not active custom orthotic users and were therefore enrolled into Study B. There were no statistically significant differences between the two groups in terms of baseline characteristics except for sex, in which case, women accounted for a much higher proportion of participants in Study A (Table 1). The most common specific diagnoses and/or locations of pain among participants involved the heel or Achilles tendon, the plantar fascia, and longitudinal arch (Table 2). In this trial, only three patients in the Study A group were lost to follow-up at six months.

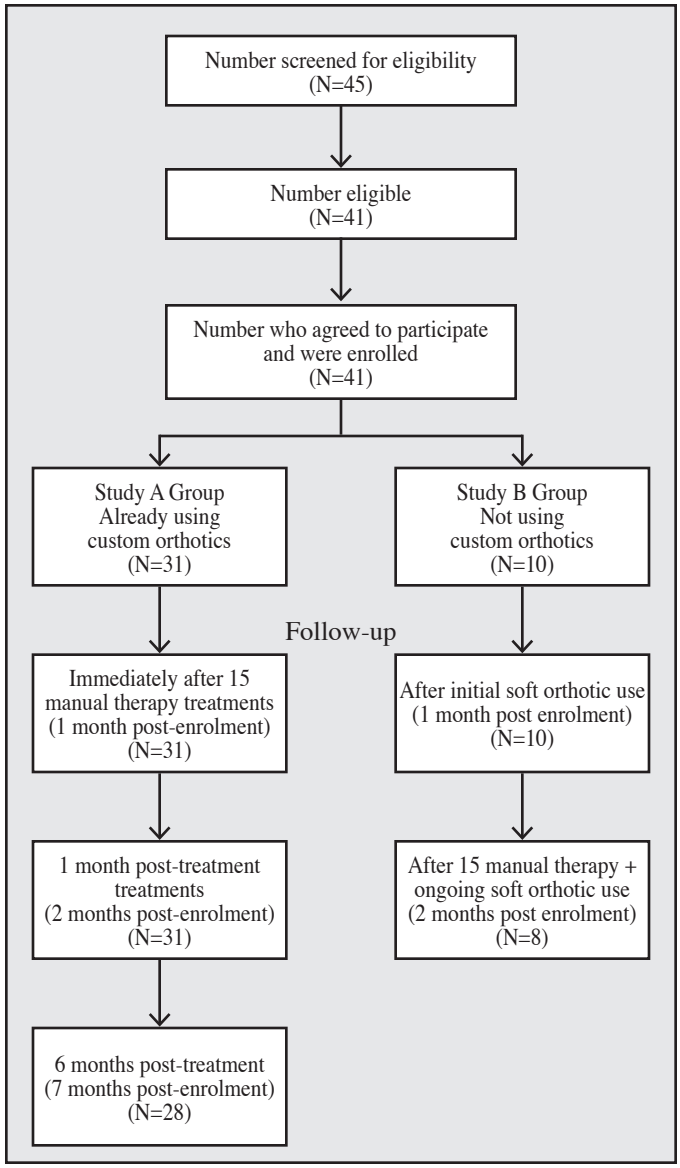

Figure 1:
Patient flow chart.

Foot Function Index (FFI)

In the Study A group, the mean FFI score (and standard deviation [SD]) was 89.5 (SD, 26.2) at baseline (see Table 1); 51.4 (SD, 33.7) after 15 treatments; 47.5 (SD, 32.5) at one month post-treatment, and 37.4 (SD, 28.2) at six months post-treatment. Table 3 shows the corresponding improvement in percent from baseline at each follow-up point. The Study A group maintained a significant reduction in the mean FFI score at all three follow-up evaluations (one-way ANOVA: $F(2, 18) = 30.664$, $p < 0.01$).

Table 1:
Baseline characteristics of participants.

	Study A Group	Study B Group
n	31	10
Women	22	4
Mean age	45	43
Mean duration of symptoms (years)	3.7	4
Baseline FFI questionnaire	89	94

Table 2:
Concomitant specific diagnoses and locations of otherwise nonspecific foot pain among participants.

Diagnosis or location	n (%)
Heel pain (Achilles tendon)	19 (46)
Plantar fasciitis	13 (31)
Painful arches (interior or exterior)	11 (27)
Metatarsalgia	10 (24)
Big toe pain	7 (17)
Pain in other toes	6 (15)
Ankle pain	3 (7)
Anterior tarsus pain	2 (5)
Painful fat pad	1 (2)
Morton's neuroma	1 (2)

*Some patients had multiple diagnoses, therefore, total of percentages exceeds 100.

For the Study B group, the repeated-measures ANOVA showed that a statistically significant improvement occurred only after the period of experimental manual therapy. The mean FFI score was 94.3 (SD, 40.7) at baseline (see Table 1); 77,7 (SD, 32.6) after five weeks (one month) of monitoring and soft orthotic use; and 30.7 (SD, 18.2) after 15 experimental manual therapy treatments (one-way ANOVA $F(1, 9) = 11.412$, $p < 0.01$) at 1 month post-treatment. Again, the corresponding percent improvement from baseline is shown in Table 3 for each follow-up time point.

Figure 2 depicts the mean changes in FFI in both groups over time. Mean FFI and corresponding SDs are presented in Table 3. Admittedly, the two study groups (i.e., active users and nonusers of custom orthotics) are likely systematically different in terms of unmeasured confounders. However, for exploratory purposes only, we tested for, and found, a significant difference ($t(39) = 2.678$, $p = 0.011$) between the two groups at one month post-enrollment. Again, this time point corresponds to the time at which Study A participants completed 15 experimental manual therapy treatments and Study B participants completed one month of monitoring and soft orthotic use without any manual therapy. Overall, the mean percentage of improvement (and standard deviation) was 47% (SD, 30.4) and 19% (SD, 19.5) for the Study A and Study B groups, respectively.

Perceived Improvement Scores (PIS)

The Study A group mean score (and SD) was 58% (SD,

Table 3:
Mean percent improvement in Foot Functional Index (FFI) and Perceived Improvement Scale (PIS) scores.

	Study A		Study B	
	FFI [a]	PIS [b]	FFI [a]	PIS [b]
1 month post-enrolment	47 (30) (after 15 trt's)	58 (26) (after 15 trt's)	19 (19) (after 1 mo. of soft orthotic use)	31 (23) (after 1 mo. of soft orthotic use)
2 months post-enrolment	49 (29) (1 mo. post-trt.)	57 (28) (1 mo. post-trt.)	64 (31) (after 15 trt's)	78 (16) (after 15 trt's)
7 months post-enrolment	56 (33) (6 mo. post-trt.)	58 (33) (6 mo. post-trt.)		

[a] Percent improvement in FFI = ((follow-up FFI – baseline FFI)/baseline FFI) x 100%; with standard deviation of calculated improvement scores in parentheses

[b] Percent PIS as directly reported on the Percent Improvement Scale; standard deviation of PIS scores in parentheses

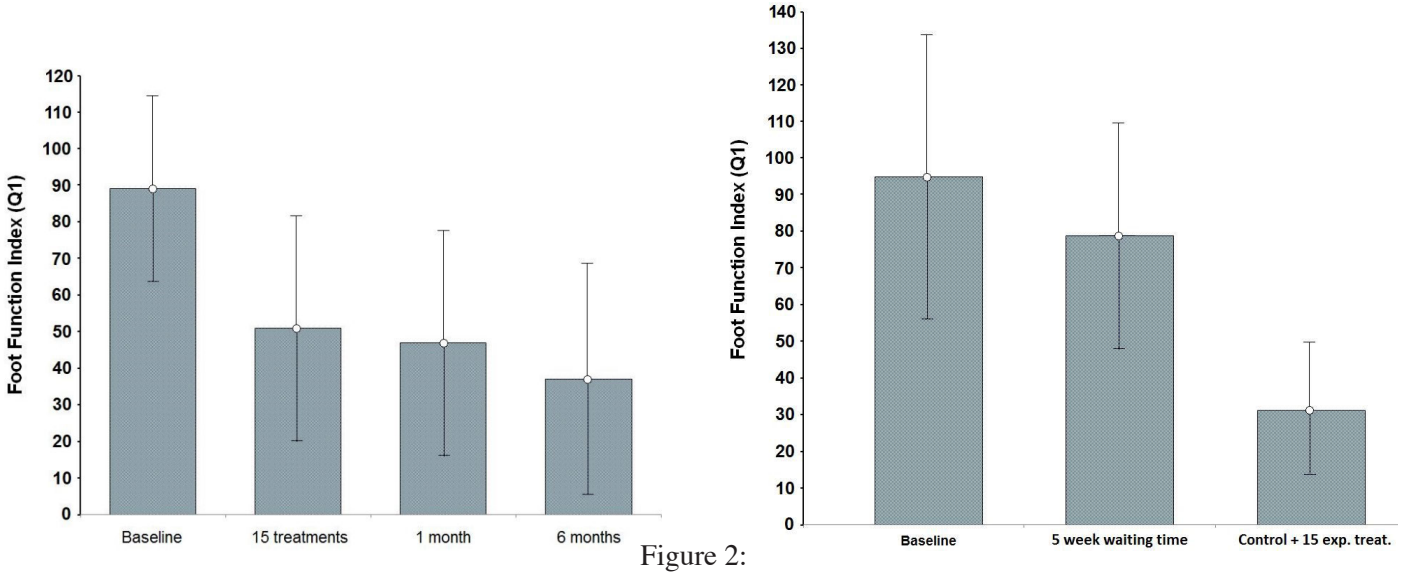

Figure 2:
Mean and standard deviation bars for foot function over time.

26.2) after 15 treatments; 57% (SD, 28.5) one month post-treatment, and 58% (SD, 31) at six months post-treatment (one-way ANOVA: $F(2, 60) = 0.007$, $p > 0.05$). There was no significant difference in the PIS between any of the three follow-up evaluations.

For the Study B group, the mean PIS (and SD) was 31% (SD, 22.9) after one month of prefabricated soft orthotic use without experimental manual therapy, and 78% (SD, 14) after 15 treatments with experimental manual therapy. The repeated-measures ANOVA yielded a significant increase in the PIS only after the experimental manual therapy period ($F(1, 9) = 67.314$, $p < 0.001$).

Between study cohorts, a significant difference was tested for, and detected between the groups A and B at one month post-enrolment, which corresponds to the time that the Study A group completed 15 manual treatments ($t(39) = 2.920$, $p = 0.006$),and the Study B group had completed one month of monitoring and soft orthotic use without any manual therapy. Mean PIS and (and SDs) at each individual time are presented in Table 3. Across all time points overall, the mean PIS was 58% (SD, 26.2) and 31% (SD, 22.9) for the Study A group and Study B groups, respectively.

In these studies, we did not observe any significant adverse effects from treatment. There were few reports (three) of increased sensitivity in the feet among some participants after the first few treatments, however this sensitivity invariably diminished subsequently.

The sum of the above frequency counts exceed 100% as many patients had more than one foot problem.

Discussion

The main findings of the two studies are that significant improvements in outcomes were detected after immediate as well as delayed experimental treatment. In Study A, FFI scores improved significantly from baseline following 15 sessions (i.e., one month) of experimental manual therapy. Similarly, in Study B, FFI scores were seen to be significantly greater only after implementation of delayed experimental manual therapy.

A comparison between the two study cohorts also suggested a benefit in favor of experimental manual therapy. In this regard, mean improvement in foot pain related disability (as assessed by the FFI) was more improved in the Study A group (47%) than the Study B group (19%). A similar benefit in the Study A cohort was observed in terms of self-perceived improvement (58% in the Study A cohort versus 31% in the Study B cohort). On the one hand, these between-cohort comparisons should be interpreted cautiously as they are potentially confounded. Theoretically, for example, orthotic users in Study A may have been better off socioeconomically and there-

fore more able to afford orthotics. Similarly, they might have been more securely employed and therefore better covered by extended health insurance in comparison to nonusers of custom orthotics in Study B. Either circumstance could have been associated with a superior initial outcome in the Study A cohort independent of any effect of immediately-administered experimental therapy.

On the other hand, the results of the between-cohort comparisons are consistent with those of our within-cohort findings. Our findings are clinically important and particularly compelling within Study B, which included a five-week monitoring phase prior to the administration of experimental treatment. In Study B, the greatest improvements occurred only after the experimental treatment phase (from 19% pre-treatment to 64% post-treatment improvement in FFI, and from 31% pre-treatment to 78% post-treatment for PIS).

We partially attribute our observed outcomes to the targeted treatment of myofascial trigger points. By definition, a trigger point is a hypersensitive zone of harder than normal consistency, which triggers pain when stimulated.[27,28,29,30] The most pathognomonic symptom of myofascial pain syndrome is the presence of pressure sensitive palpable nodules that replicate the chief complaint of the patient.[30] Trigger points are thought to develop after trauma, overuse or prolonged spasm of muscle.[29,31] Ischemic compression has been demonstrated as being effective in the treatment of TrPs.[27,32,33] When the treatment is given carefully, by paying attention to the patient's slightest reaction and asking if the pain is bearable, it is likely to be well tolerated.

We also partially attribute our observed outcomes to the mobilization component of our management approach. Joint mobilization involves manual techniques without thrusting or sudden movement. Repetitive passive movement of a skeletal joint is performed with the aim of achieving a therapeutic effect.[34] In the present studies, the need for joint mobilization was determined by detection of a loss of joint play and/or provocation of pain during forceful flexion, extension, or rotation of the articulation of the foot. Subsequently, treatment consisted of forcing passive movement through the painful articulation, to the patient's limit of tolerance, and then maintaining that forceful contact for a duration of 15 seconds. This technique was repeated at each visit until the pain was completely eliminated or until the end of the 15 treatments.

We have found few clinical trials using mobilization, ischemic compressions, or massage to counter foot pain. Existing studies were conducted on patients with plantar fasciitis and hallux problems. Brantingham conducted a randomized clinical trial of the treatment of hallux abductovalgus (bunions).[17] Sixty subjects were randomized to two groups. The experimental group received mobilization of the first metatarsophalangeal articulation, in conjunction with cryotherapy and adjustment of all other putative fixations found in the foot and ankle. The placebo group received de-tuned microcurrent therapy. Six treatments were given over a two-week period. Outcomes were measured weekly throughout the treatment period and then at only one week post-treatment. In terms of objective findings, the experimental group showed a statistically significant improvement post-treatment, which was not the case in the control group. In terms of patients' subjective response to treatment, both groups experienced a statistically significant decrease in pain perception; however, the experimental group achieved a statistically significant improvement at one week whereas the placebo group did so only at three weeks. Furthermore, a statistically significant improvement in foot pain and related disability (as measured using the Foot Function Index) was observed in the experimental group only.[17]

Another study by Brantingham, this time on plantar fasciitis, involved a retrospective review of files of 29 patients who were treated with manipulations, physiotherapy, orthotics, and soft tissue massage of the plantar fascia.[35] Twenty-two of the 29 patients demonstrated excellent results (defined as greater than 75% pain reduction) following an average of eight treatments.

Sweeting et al. conducted a systematic review of the effectiveness of stretching in the treatment of plantar heel pain.[36] Six studies involving 365 symptomatic patients were included in the analysis. They reported that most patients with plantar heel pain who stretch tend to improve over time in terms of both pain and function, but when stretching is compared to other interventions, including sham treatment, no statistically significant benefit was observed.

Study limitations

Our studies had several limitations. We did not assess the reliability of our diagnostic protocol for detecting trigger points and foot dysfunction in advance. However, our

studies were intended to be preliminary investigations of the effectiveness of a manual therapy protocol on trigger points and joint hypomobility, as customarily diagnosed in everyday clinical practice.

We acknowledge that our outcomes were collected by the principal investigator who was aware of both the study questions and the phase of treatment for each participant at each follow-up visit. Having utilized only two treating doctors, our interventionists were not necessarily representative of other chiropractors in the general community. Furthermore, while we detected statistically significant differences, our sample sizes were small and therefore our point estimates are likely unstable and will require further corroboration in larger studies.

Also, our findings are potentially vulnerable to various threats to validity that are specifically associated with quasi-experimental studies.[37] One important threat to the validity of findings from a before-and-after study is the potential effect of history, meaning that some events occurring externally to the study, but concurrently with treatment, may affect outcomes independently of the experimental treatment. However, to our knowledge, our study participants were not systematically exposed to any events in the community (e.g., a public health initiative for foot disorders) that might have coincidentally caused the effects that we observed in our studies. A more likely threat to validity in our studies is maturation, or the effect of naturally occurring changes in outcomes (i.e., foot pain and related disability) over time, which could easily be confused with the effect of experimental treatment. However, this phenomenon is unlikely to account for the fact that the timing of clinical improvement among both of our study cohorts coincided precisely with the timing of two differently timed intervention periods (early treatment in Study A, and delayed treatment in Study B). Finally, regression to the mean is yet another potential threat to the validity of our observed effects. Admittedly, our study participants may have been seeking treatment (and were therefore enrolled) at a time when they were particularly disabled by their symptoms. In such patients, a natural regression from higher to lower disability status over the study period might have occurred independently of treatment, however regression to the mean would also not explain the observed consistency in the timing of observed improvements immediately after each of two differently timed experimental treatment periods. Moreover, the Study B cohort was exposed to an initial five-week monitoring period, at the end of which little to no regression of baseline outcomes was observed.

In Study B, our findings were potentially susceptible to the effect of experimental mortality in that two subjects declined experimental treatment after the initial monitoring phase and were therefore not included in the analysis. Finally, participants were not blatantly aware of the researchers' expectations but may have had personal expectations for improvements in outcomes following experimental manual therapy exclusively.

Conclusion

Our findings constitute preliminary evidence that myofascial therapy consisting of ischemic compressions and joint mobilizations may reduce the symptoms of patients suffering from chronic non-specific foot pain. The study intervention was heterogeneous and tailored to each individual patient, however it was applied pragmatically and consistent with the way in which it would be administered customarily in everyday clinical practice. A treatment period of five weeks was associated with improved self-reported outcomes. We hope these preliminary findings provide a foundation for other researchers to further evaluate the effectiveness of our approach within randomized controlled trials.

References

1. Hill CL, Gill TQ, Menz HB, Taylor AW. Prevalence and correlates of foot pain in a population-based study. J Am Med Assoc. 1987;77:308-311.
2. DeMaio M, Paine R, Mangine RE et al. Plantar fasciitis. Orthopedics.1993; 16:1153-63.
3. Nix S, Smith M, Vicenzino B. Prevalence of hallux valgus in the general population: a systematic review and meta-analysis. J Foot Ankle Res. 2010;3:21.
4. Polkinghorn BS. Posterior calcaneal subluxation: an important consideration in chiropractic treatment of plantar fasciitis (heel spur syndrome). Chirop Sport Med. 1995; 9(2):44-51.
5. Stuber K, Kristmanson K. Conservative therapy for plantar fasciitis: a narrative review of randomized controlled trials. J Can Chirop Assoc. 2006; 50(2):118-33.
6. Pack LG, Julien PH. Differential diagnosis of lesser metatarsalgia. Clin Podiat Med Surg. 1990; 7(4):573-7.
7. Kasmahl EM, Kosmahl EK. Painful plantar heel, plantar fasciitis, and calcaneal spurs: etiology and treatment. J Orthop Sport Ther. 1987;9(1):17-24.
8. Davison WT, Hyde TE, Reicher DL, Werner JS. Grand

Rounds: Heel pain. J Neuromusculoskeletal System. 1997; 5(1):39-46.
9. Shapiro SL. Heel pain management starts with correct differential diagnosis. Biomechanics. 1997; 4(9):25-27.
10. Dimou ES, Brantingham JW, Wood T. A randomized controlled trial of chiropractic manipulation and achilles stretching vs. orthotics for the treatment of plantar fasciitis. J Am Chirop Assoc. 2004; 41(9):32-42.
11. Gill LH. Plantar fasciitis: diagnosis and conservative management. J Am Acad Orthop Surg. 1997; 5(2):109-117.
12. Speed C. A systematic review of shockwave therapies in soft tissue conditions: focussing on the evidence. Br J Sports Med. 2014; 48(21):1538-1542.
13. Brantingham JW, Chang MN, Gendreau DF, Price JL. The effect of chiropractic adjusting, exercises and modalities on a 32-year-old professional male golfer with hallux rigidus. Clinical Chiropractic. 2007; 10(2):91-96.
14. GlascoW, Glasco G. Conservative evaluation and intervention of a sport-related injury: turf toe. Sport Chirop Rehab. 1998; 12:2.
15. Ferrari J, Higgins JPT, Prior TD. Intervention for treating Hallux Valgus and Bunions (Review).The Cochrane Library 2007; Issue 4.
16. Manral DB. Hallux rigidus: a case report of successful chiropractic management and review of the literature. J Chirop Med. 2004; 3:1.
17. Brantingham JW, Guiry S, Kretzmann HH et al. A pilot study of the efficacy of a conservative chiropractic protocol using graded mobilization, manipulation and ice in the treatment of symptomatic hallux abductovalgus bunion. Clinical Chiropractic. 2005; 8:117-133.
18. Quirk R. Metatarsalgia. Austr Fam Phys. 1996; 25(6):863-69.
19. Thomson CE, Gibson JNA, Martin D. Interventions for the treatment of Morton's Neuroma (Review). The Cochrane Library 2007, Issue 4.
20. Subonick S. Peroneal Cuboid Syndrome: an often overlooked cause of lateral column foot pain. Chiropractic Technique. 1998; 10(4):156-162.
21. Watt LH. Conservative chiropractic management of recalcitrant foot pain after fasciotomy: a retrospective case review. J Manip Physiol Thera. 2006; 29(5):398-402.
22. Solan M, Davies M. Management of insertional tendinopathy of the achilles tendon. Foot Ankle Clin Am. 2007; 12:597-615.
23. Travel JG, Simons DG. Myofascial Pain and Dysfunction; the Trigger Point Manual, Vol.1. Philadelphia: Williams and Wilkins;1983. p.19.
24. Graff-Radford SB. Myofascial pain: diagnosis and management. Current Pain and Headache Reports. 2004; 8:463-467.
25. Looney B, Srokose T, Fernandez-de la Penas C, Cleland JA. Graston instrument soft tissue mobilization and home stretching for the management of plantar heel pain: a case series. J Manip Physiol Thera. 2010; 32(2):138-141.
26. Budiman-Mak E, Conrad KJ, Roach KE. The foot function index: a measure of foot pain and disability. J Clin Epidemiol. 1991; 44(6):561-570.
27. Gerwin RD, Dommerholt J, Shah JP. An expansion of Simons' integrated hypothesis of trigger point formation. Current Pain and Headache Report. 2004; 8:468-475.
28. Alvarez DJ, Rockwell PG. Trigger points: diagnosis and management. Am Fam Phys. 2002; 65(4):653-660.
29. Schneider MJ. Tender points/fibromyalgia vs. trigger points/myofascial pain syndrome: a need for clarity in terminology and differential diagnosis. J Manip Physiol Thera. 1995; 18(6):398-406.
30. Borg-Stein J, Stein J. Trigger points and tender points. Rheumatic Dis N Am. 1996; 22(2):305-323.
31. Fischer AA. New approaches in the treatment of myofascial pain. Phys Med Rehab Clin Am. 1997; 8(1):153-169.
32. Fernandez-de-la-Penas C, Alonso-Blanco C, Fernandez-Carnero J et al. The immediate effect of ischemic compression technique and transverse friction massage on tenderness of active and latent myofascial trigger points: a pilot study. J Bodywork and Mov Thera. 2006; 10:3-9.
33. Hou CR, Tsai LC, Cheng KF et al. Immediate effects of various physical therapeutic modalities on cervical myofascial pain and trigger points sensibility. Arch Phys Med Rehabil. 2002; 83:1406-1414.
34. Wikipedia/Joint-mobilization 2012.
35. Brantingham JW, Snyder WR, Dishman RW et al. Plantar fasciitis. Chiropractic Technique. 1992;4:3.
36. Sweeting D, Parish B, Hooper L et al. The effectiveness of manual stretching in the treatment of plantar heel pain: a systematic review. J Foot Ankle Res. 2011; 4:19.
37. Harris AD, McGregor DE, Perencevich EN et al. The use and interpretation of quasi-experimental studies in medical informatics. J Am Med Informatics Assoc. 2006; 13(1):16-23.

Note: Dr. Guy Hains DC passed away on October 13, 2014. He published a number of clinical studies over the last decade and made significant contributions to chiropractic research.