Changes in Spasticity, Widespread Pressure Pain Sensitivity, and Baropodometry After the Application of Dry Needling in Patients Who Have Had a Stroke: A Randomized Controlled Trial

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Abstract

Objective: The purpose of this study was to determine the effects of deep dry needling (DDN) on spasticity, pressure sensitivity, and plantar pressure in patients who have had stroke.

Methods: A randomized controlled trial was conducted. Thirty-four patients who previously had a stroke were randomly assigned either an experimental group that received a single session of DDN over the gastrocnemius and tibialis anterior muscles on the spastic leg or a control group that received no intervention. Spasticity (evaluated with the Ashworth Scale); pressure pain thresholds over the deltoid muscle, second metacarpal, and tibialis anterior muscle; and plantar pressure (baropodometry) were collected by a blinded assessor before and 10 minutes after intervention.

Results: A greater number of individuals receiving DDN exhibited decreased spasticity after the intervention ($P < .001$). The analysis of covariance showed that pressure pain thresholds increased bilaterally in patients receiving DDN compared with those who did not receive the intervention ($P < .001$). The analysis of covariance also found that patients receiving DDN experienced bilateral increases of support surface in the forefoot, unilateral increase of the support surface in the rear foot of the treated (affected) side, and bilateral decreases in mean pressure (all, $P < .02$) as compared with those who did not receive DDN.

Conclusions: Our results suggest that a single session of DDN decreases spasticity and widespread pressure sensitivity in individuals with poststroke spasticity. Deep dry needling also induced changes in plantar pressure by increasing the support surface and decreasing the mean pressure. (J Manipulative Physiol Ther 2014;37:569-579)

Key Indexing Terms: *Stroke; Muscle Spasticity; Pain Threshold; Acupuncture*

Stroke is a leading cause of disability with an estimated annual incidence of 144 per 100 000 people in Iceland[1] and 118 per 100 000 in Spain.[2] A recent study found that the incidence of ischemic stroke in Sweden has decreased (3.7% per year) in older people (>65 years), slightly decreased (0.4% per year) in middle-aged people (45-65 years), but increased (1.3% per year) in young people (18-44 years) in the last 25 years.[3] Although stroke has dropped from being the third main leading cause of death to the fourth cause in the United States of America and Europe,[4] it still remains the leading cause of physical disability, particularly due to the presence of spasticity.

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Patients with spasticity exhibit lower motor activity performance than patients who do not have spasticity.[5] In fact, stroke patients with spasticity in the lower extremity exhibit several impairments associated with standing and walking resulting in high levels of disability.[6]

Spasticity usually develops slowly, peaking 1 to 4 months after the onset of stroke,[5] and is present in 38% of the patients 1 year after stroke.[7] It is defined as "a motor disorder characterized by velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as a component of upper motoneuron syndrome."[8] Although the primary lesion in subjects with spasticity is neural in origin, profound secondary changes occur in the muscle itself at the protein, single-fiber, and whole-muscle levels. For instance, electron microscopy found the presence of expanded connective tissue, decreased mitochondrial volume fraction, and appearance of intracellular amorphous material in spastic muscles.[9] It is widely accepted that muscle contractures occurring secondary to spasticity are due to a reduction in muscle fiber length and a decrease in the number of serial sarcomeres within muscle fibers.[10]

Intramuscular botulinum toxin A (BTX-A) injection is the most popular tool for the management of spasticity.[11] A recent meta-analysis found that application of BTX-A in patients who have experienced a stroke was associated with moderate improvement in upper extremity performance.[12] Recent case reports also support the use of BTX-A in combination with other manual therapy modalities, for example, neurodynamic interventions, for the management of spasticity.[13-15] Because some individuals exhibit allergic responses to BTX-A, it has been proposed that acupuncture can be also effective for treatment of poststroke spasticity. Several studies have investigated the effects of acupuncture on poststroke spasticity; however, the results are conflicting. Some studies have observed that acupuncture was effective for reducing spasticity,[16,17] but others did not find any significant effect.[18,19] Discrepancies between these studies may be related to the fact that these trials needled classical acupuncture points, which implies that the needle was not introduced into the spastic muscle. Therefore, it is possible deep dry needling (DDN) may be a viable alternative intervention for spastic musculature where the needle is inserted into the targeted muscle.[20]

Both mechanical and neurophysiological mechanisms are associated with DDN. It is purported that mechanical effects include disruption of contraction knots, localized stretch of the contractured cytoskeletal structures, and reduction of the overlap between actin and myosin filaments.[21,22] It has been demonstrated that contractured taut bands have greater stiffness than surrounding tissue[23] and that DDN is able to reduce muscle stiffness as assessed by ultrasound shear wave elastography.[24] Therefore, it is possible that DDN may decrease poststroke spasticity. In addition, it is also suggested that DDN can modulate the central nervous system through an antinociceptive effect.[25] Hence, DDN may also induce sensory changes in patients with stroke.

To our knowledge, no previous study has investigated the effects of DDN in patients with poststroke spasticity. The purpose of this randomized clinical trial was to determine the effects of a single session of DDN on spasticity, widespread pressure pain sensitivity, and plantar pressures (baropodometry) in individuals with chronic stroke. We hypothesized that patients receiving a single session of DDN would exhibit a greater reduction in spasticity and pressure pain sensitivity than those who did not receive DDN.

Methods

Design

A randomized controlled trial was performed (registered with ClinicalTrials.gov, NCT 01950338). The study protocol was approved by human research committee of the Universidad Rey Juan Carlos, Spain (URJC 52/2012), and all subjects signed an informed consent before participation in the study.

Participants

Consecutive subjects who had experienced a stroke were screened for eligibility criteria from January 2013 to October 2013. Participants were recruited from the local community and had a documented diagnosis of stroke from their neurologist. To be included, they must have met the following criteria: (1) first-ever unilateral stroke, (2) hemiplegia resulting from stroke, (3) unilateral equinovarus gait with independent walking, and (4) able to ambulate without supportive device. Participants were excluded if they exhibited any of the following: (1) recurrent stroke; (2) previous treatment with nerve blocks, motor point injections with neurolytic agents for spasticity at any time, or with BTX-A in the 6 months preceding the study; (3) not independent in the basic activities of daily living; (4) severe cognitive deficits; (5) progressive or severe neurologic diseases, for example, heart conditions, unstable hypertension, fracture, or implants in the lower extremity; (6) fear to needles; or (7) any contraindication for deep dry needling, for example, anticoagulants, infections, bleeding, or psychotic.

Spasticity: Modified Modified Ashworth Test

Spasticity in the affected ankle joint was evaluated with the Modified Modified Ashworth Scale (MMAS).[26] The examiner passively moved the ankle in a dorsiflexion direction, back and forth at least 5 times, and evaluated the degree of resistance to the movement on a scale from 0 to 4. The MMAS comes from a modification of the modified Ashworth Scale,[27] which is the most commonly used scale

for assessing spasticity,[28] where the grade "1+" is omitted and the grade "2" is redefined.

In the MMAS, spasticity is scored on an ordinal scale from 0 to 4 as follows: 0, no increase in muscle tone; 1, slight increase in muscle tone (minimal resistance at the end of the range of motion); 2, marked increase in muscle tone (resistance throughout the range of motion, but some sections are easily moved); 3, considerable increase in muscle tone (passive movement difficult throughout the full range of motion); or 4, affected part(s) rigid in flexion and/or extension. The MMAS has exhibited good intraexaminer ($\kappa = 0.85$)[29] and also interexaminer ($\kappa = 0.74$)[30] reliability for assessing ankle plantar flexor spasticity in patients who had experienced a stroke.

Mechanical Pain Sensitivity: Pressure Pain Thresholds

Pressure pain threshold (PPT), defined as the amount of pressure applied for the pressure sensation to first change to pain,[31] was assessed bilaterally with a mechanical pressure algometer (Pain Diagnosis and Treatment, Inc, New York, NY) over the deltoid muscle, the second metacarpal, and the tibialis anterior muscle to determine changes in widespread pressure sensitivity. Subjects were instructed to press a switch when the sensation first changed from pressure to pain. The mean of 3 trials was calculated, converted to kilopascal (SI unit) and used for analysis. A 30-second resting period was allowed between each trial. Several studies have documented high intraexaminer and interexaminer reliability (Intraclass correlation coefficient, 0.80-0.97) for PPT assessment in patients with pain.[32,33]

Baropodometry

A baropodometric study was performed with a Foot-Work force platform (V-PLATE, Norm EN 46003; Medicapteurs, Balma, France) with the following specific features: real capture, 40 × 40 cm; sensor size, 10 × 10 mm; sensor thickness, 4 mm; sensor number, 1600 (40 × 40); acquisition frequency, 100 MHz.[34] The system consists of a force platform placed on the floor. It was calibrated to the weight of each individual. Data collection was performed with the subjects standing barefoot in a comfortable bipedal position on the platform according to standardized procedures: heels of both feet were separated 2 cm with the forefoot creating a 30° angle (Fig 1). This assured the center of gravity was placed within a support triangle formed by the foot. A reference point was located in front of the patients, depending on their height, and they were asked to maintain their gaze fixed on the reference point and hold their position for 1 minute.

The following data were collected bilaterally from each patient: support surface (square centimeters), percentage of load (percentages), and force distribution (percentages) of both forefoot and rear foot. In addition, we also calculated mean and maximum pressures (grams per square centimeter) of each foot, affected and nonaffected side.

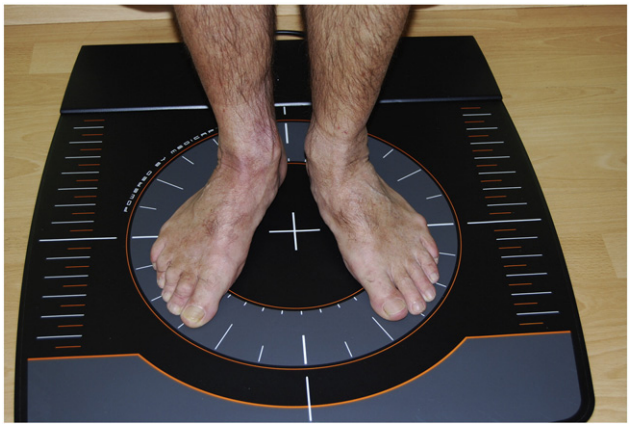

Fig 1. *Baropodometric data collection in a patient with stroke: standing barefoot in a comfortable bipedal position on the platform with both heels separated 2 cm with forefoot creating a 30° angle.*

Deep Dry Needling

Patients within the experimental group received a single session of DDN with disposable stainless steel needles (0.3 × 50 mm; Novasan, Madrid, Spain) that were inserted into the skin over taut bands of the gastrocnemius and tibialis anterior muscles. In this study, the fast-in and fast-out technique described by Hong[35] was applied. Once the most painful spot was located within a palpable spastic taut band with pincer palpation in the gastrocnemius muscle or with flat palpation in the tibialis anterior muscle, the overlying skin was cleaned with alcohol. The needle was then inserted, penetrating the skin approximately 15 to 20 mm, until the first local twitch response (LTR) was obtained. It is suggested that LTRs should be elicited during DDN for a proper technique.[35] Once the first LTR was obtained, the needle was moved up and down (4-5 mm vertical motions with no rotation) in the muscle at approximately 1 Hz for 25 to 30 seconds. Each patient received DDN over taut bands of the following muscles: the medial gastrocnemius (Fig 2A), the lateral gastrocnemius (Fig 2B), and tibialis anterior muscle (Fig 3).

Control Group

Patients within the control group did not receive any intervention with the aim being to determine the natural course of the condition. Outcomes were assessed twice with 10 minutes between measurements. During this time, patients rested in a chair.

Allocation

After the baseline examination, patients were randomly assigned to receive DDN (experimental group) or no intervention (control group). Concealed allocation was performed using

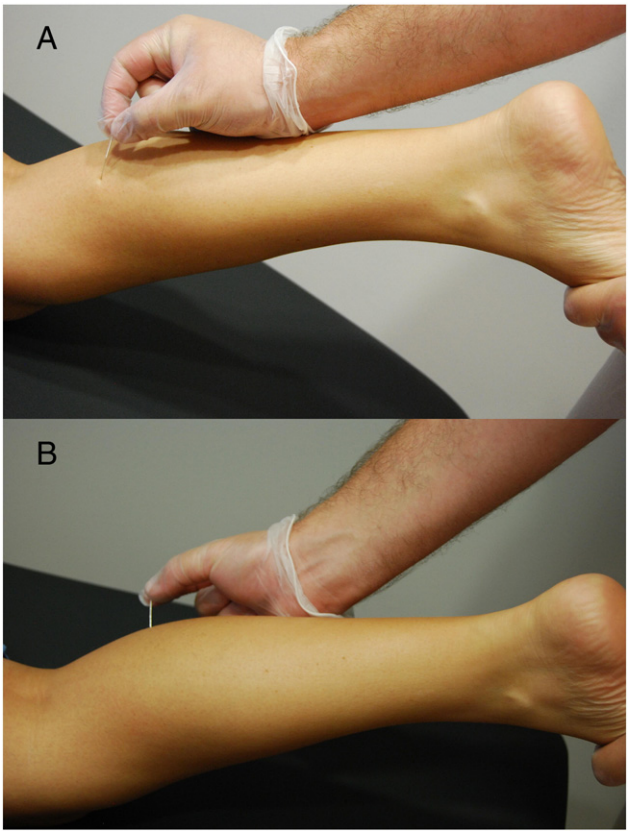

Fig 2. *Deep dry needling applied over taut bands within the medial (A) and lateral (B) gastrocnemius muscles.*

a computer-generated randomized table of numbers created before the start of data collection by a researcher not involved in the recruitment or treatment of patients. Individual and sequentially numbered index cards with the random assignment were prepared. The index cards were folded and placed in sealed opaque envelopes. A second therapist, blinded to baseline examination findings, opened the envelope and proceeded with treatment according to the group assignment. Outcome measures were taken before and 10 minutes after the intervention by an assessor blinded to group allocation.

Sample Size Calculation

The sample size was calculated with the ENE 3.0 software (GlaxoSmithKline; Universidad Autónoma, Barcelona, Spain). The calculations were based on detecting between-group differences of 20% on PPTs,[36] assuming an SD of 10, 2-tailed test, an α level of .05, and a desired power (β) of 80%. The estimated desired sample size was calculated to be at least 16 subjects per group.

Statistical Analysis

Data were analyzed with SPSS version 18.0. Mean, SD, or 95% confidence intervals (CIs) were calculated for each variable. The Kolmogorov-Smirnov test showed normal distribution of quantitative data. Baseline demographic and clinical variables were compared between both groups using independent Student *t* tests for continuous data and χ^2 tests of independence for categorical data. Because participants received a unilateral intervention on the affected leg, sides were classified as ipsilateral (affected) or contralateral (unaffected) to the treated side.

A mixed χ^2 test (McNemar-Bowker test) was applied to analyze changes in MMAS between groups at baseline and after the intervention. A 2 × 2 × 2 mixed model repeated-measure analysis of covariance (ANCOVA) with time (baseline, immediate after) and side (ipsilateral, contralateral to the treated side) as within-subject factors, group (DN, control) as the between-subject factor, and baseline scores as covariate was used to determine the effects of the intervention on PPTs. Separate ANCOVAs were conducted with each point as the dependent variable. Similarly, 2 × 2 × 2 mixed model ANCOVAs were also used to evaluate the differences in support surface, percentage of load, and force distribution of forefoot and rear foot, separately, and mean and maximum pressure of each foot with time (baseline, immediate after) and side (ipsilateral, contra lateral to the treated side) as the within-subject factors, group (DN, control) as the between-subject factor, and baseline scores as covariate. The hypothesis of interest was the group × time interaction. $P < .05$ was considered statistically significant.

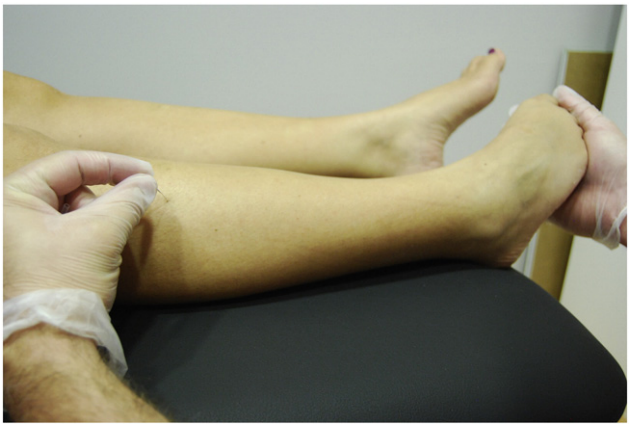

Fig 3. *Deep dry needling applied over taut bands within the tibialis anterior muscle.*

Results

Forty consecutive patients who had experienced a stroke were screened for eligibility criteria. Thirty-four (mean ± SD age, 50 ± 11 years; 53% female) satisfied the eligibility criteria, agreed to participate, and were randomized into the experimental (n = 17) or control (n = 17) group. The reasons for ineligibility are found in Figure 4, which provides a flow diagram of patient recruitment. Baseline features between

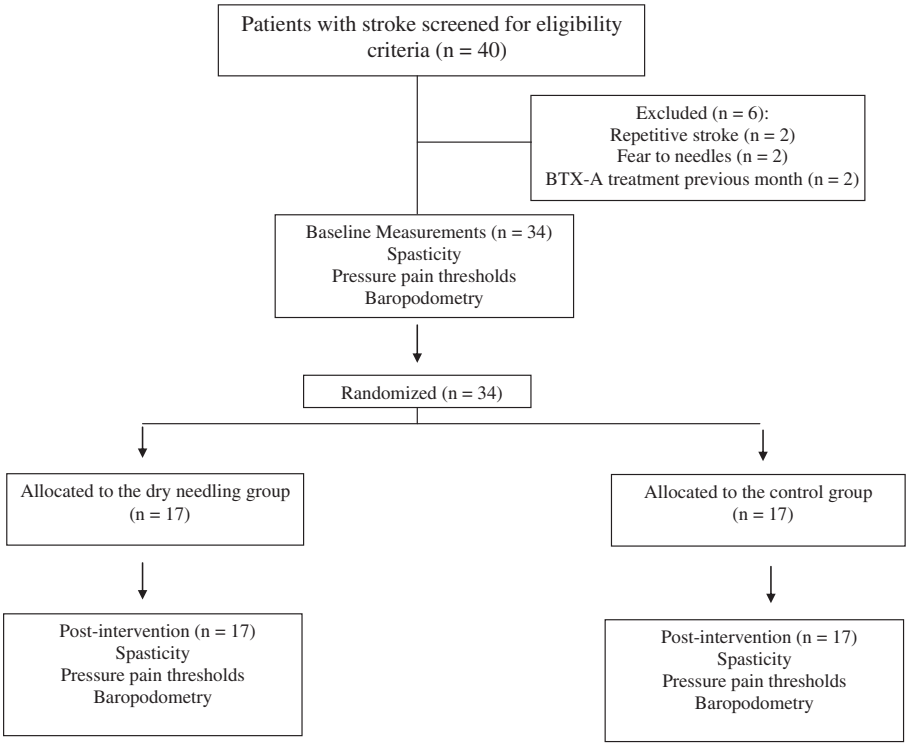

Fig 4. *Flow diagram of patients throughout the course of the study. BTX-A, intramuscular botulinum toxin A.*

Table 1. *Baseline Clinical, Demographics, and Sensory Outcomes for Both Groups*

	Experimental Group (n = 17)	Control Group (n = 17)	*P*
Clinical features			
Sex (male/female)	8/9	8/9	.999
Age (y)	49 ± 9	51 ± 11	.499
Affected side (right/left)	11/6	9/8	.486
Ashworth scale, n (%)			
Grade II	7 (41%)	10 (59%)	.494
Grade III	10 (59%)	7 (41%)	
Pressure pain thresholds (kPa)			
PPT deltoid muscle affected side	395.5 ± 78.8	415.4 ± 97.0	.586
PPT deltoid muscle nonaffected side	356.1 ± 77.5	396.0 ± 98.0	.310
PPT second metacarpal affected side	306.8 ± 98.0	356.0 ± 99.0	.172
PPT second metacarpal nonaffected side	326.4 ± 99.0	375.8 ± 98.0	.165
PPT tibialis anterior muscle affected side	454.9 ± 79.0	445.1 ± 79.0	.701
PPT tibialis anterior muscle nonaffected side	395.2 ± 59.3	445.5 ± 89.2	.190

Values are expressed as mean ± SD except for sex, affected side, and Ashworth scale.
PPT, pressure pain threshold.

both groups were similar for all sensory (Table 1) and baropodometric (Table 2) variables.

Changes in Spasticity

The nonparametric McNemar-Bowker test revealed a significant reduction in MMAS (χ^2 = 19.071; $P < .001$) after the intervention in the experimental group: a greater number of patients receiving DDN exhibited a lower degree of spasticity (Table 3).

Changes in Pressure Pain Sensitivity

The 2 × 2 × 2 ANCOVA revealed significant group × time interactions for PPT at all locations: deltoid muscle (F = 59.022; $P < .001$), second metacarpal (F = 70.573; $P < .001$), and tibialis anterior muscle (F = 18.452; $P < .001$). No significant group × time × side interactions were observed at any location: deltoid muscle (F = 1.829; $P = .181$), second metacarpal (F = 0.083; $P = .774$), and tibialis anterior muscle (F = 2.495; $P = .119$). The inclusion of baseline scores as a covariate did not influence any PPT (all, $P > .347$). Pressure pain threshold increased significantly bilaterally in subjects who had experienced stroke receiving a DDN session compared with those who did not receive any intervention. Table 4 summarizes before and after intervention scores as well as within-group and between-group differences with their associated 95% CI for PPT data.

Changes in Baropodometry

The 2 × 2 × 2 mixed model ANCOVA found a significant group × time interaction for the support surface (F = 7.496;

Table 2. *Baseline Scores (Mean ± SD) of Baropodometry for Both Groups*

	Experimental Group (n = 17)	Control Group (n = 17)	*P*
Affected side			
Support surface forefoot (cm^2)	58.2 ± 15.5	51.1 ± 12.0	.148
Force distribution forefoot (%)	27.9 ± 8.5	24.8 ± 7.2	.268
Percentage of load forefoot (%)	56.5 ± 16.6	65.3 ± 23.2	.211
Support surface rear foot (cm^2)	39.8 ± 14.4	31.1 ± 19.9	.154
Force distribution rear foot (%)	20.7± 10.3	16.2 ± 12.3	.260
Percentage of load rear foot (%)	43.1 ± 16.8	34.7 ± 23.2	.239
Mean pressure (g/cm^2)	353.1 ± 39.5	378.5 ± 74.2	.222
Maximum pressure (g/cm^2)	818.3 ± 120.1	857.6 ± 169.1	.440
Nonaffected side			
Support surface forefoot (cm^2)	43.4 ± 12.2	49.0 ± 11.4	.177
Force distribution forefoot (%)	19.4 ± 6.2	23.6 ± 7.1	.077
Percentage of load forefoot (%)	37.9 ± 11.5	40.5 ± 11.5	.518
Support surface rear foot (cm^2)	49.4 ± 15.6	49.3 ± 8.6	.989
Force distribution rear foot (%)	32.2 ± 8.3	35.3 ± 12.1	.387
Percentage of load rear foot (%)	61.6 ± 11.4	59.2 ± 11.6	.545
Mean pressure (g/cm^2)	397.5 ± 64.9	419.7 ± 89.2	.413
Maximum pressure (g/cm^2)	972.3 ± 347.4	987.3 ± 320.2	.362

Table 3. *Changes in Ashworth Scale Before and After the Intervention in Both Groups*

	Experimental (n = 17)	Control (n = 17)	*P*
Ashworth preintervention, n (%)			.001
Grade II	7 (41%)	10 (59%)	
Grade III	10 (59%)	7 (41%)	
Ashworth postintervention, n (%)			
Grade I	12 (70%)		
Grade II	4 (24%)	10 (59%)	
Grade III	1 (6%)	7 (41%)	

P = .008) but not for the force distribution (F = 1.728; *P* = .193) or percentage of load (F = 1.165; *P* = .285) of the forefoot: patients receiving DDN experienced a bilateral increase of support surface in the forefoot as compared with those who did not receive an intervention (Fig 5). A significant group × time × side interaction was observed for the percentage of load (F = 0.903; *P* = .045) but not for support surface (F = 0.229; *P* = .634) or force distribution (F = 0.009; *P* = .924) of the forefoot: patients with stroke who received DDN experienced an unilateral increase of the percentage of load in the forefoot of the contralateral side (unaffected) as compared with those who did not receive the intervention. Table 5 provides details of before and after scores as well as within-group and between-group differences with their associated 95% CI for baropodometric outcomes of the forefoot.

The 2 × 2 × 2 mixed model ANCOVA did not reveal any significant group × time interaction for the support surface (F = 1.714; *P* = .195), force distribution (F = 0.094; *P* = .760), or percentage of load (F = 0.019; *P* = .891) of the rear foot. A significant group × time × side interaction was observed for the support surface (F = 7.476; *P* = .006) but not for percentage of load (F = 1.175; *P* = .283) or force distribution (F = 2.006; *P* = .185) of the rear foot: patients with stroke who receiving DDN experienced an unilateral increase of the support surface in the rear foot of the treated side (affected) as compared with those who did not receive treatment (Fig 6). Table 6 provides details of before and after intervention scores as well as within-group and between-group differences with their associated 95% CI for baropodometric outcomes of the rear foot.

Finally, a significant group × time interaction was observed for mean pressure (F = 6.335; *P* = .014) but not for maximum pressure (F = 1.438; *P* = .235). No significant group × time × side interaction was found (mean pressure: F = 0.376; *P* = 0.542; maximum pressure: F = 0.653; *P* = .422). Subjects receiving DDN experienced a bilateral decrease in mean pressure compared with those not receiving the intervention (Table 7). The inclusion of baseline scores as a covariate did not influence any baropodometric outcome (all, *P* > .445).

Discussion

The current randomized controlled trial demonstrated that the application of a single session of DDN decreased spasticity and widespread pressure pain sensitivity in subjects who had experienced a stroke. In addition, DDN induced changes in plantar pressures by increasing the support surface and decreasing mean pressure bilaterally.

We observed an immediate decrease in spasticity of the ankle muscles after the application of a single session of DDN over taut bands in subjects who had experienced a stroke. These results are similar to those previously reported after the application of acupuncture,[16,17] but contrary to others.[18,19] The main difference between our study and previous trials is that we inserted the needle directly into the spastic muscle (DDN), whereas previous studies used

Table 4. *Baseline, Final Treatment Session, and Change Scores for Pressure Pain Thresholds*

Outcome Group	Baseline	End of Treatment	Within-Group Change Scores	Between-Group Difference in Change Scores
PPT deltoid muscle affected side (kPa)				
Experimental	395.5 ± 78.8	454.9 ± 59.1	59.4 (29.7; 89.1)	59.4 (29.7; 89.0) [a]
Control	415.4 ± 97.0	415.4 ± 89.1	0.0 (−3.0; 4.0)	
PPT deltoid muscle nonaffected side (kPa)				
Experimental	356.1 ± 77.5	435.1 ± 69.7	79.0 (59.4; 100.2)	79.5 (59.4; 109.0) [a]
Control	396.0 ± 98.0	395.5 ± 98.5	0.5 (−9.0; 9.5)	
PPT second metacarpal affected side (kPa)				
Experimental	306.8 ± 98.0	425.3 ± 69.2	118.5 (69.3; 140.1)	88.8 (69.3; 128.6) [a]
Control	356.0 ± 99.0	385.7 ± 99.0	29.7 (0.0; 39.7)	
PPT second metacarpal nonaffected side (kPa)				
Experimental	326.4 ± 99.0	396.0 ± 68.5	69.6 (59.3; 128.3)	99.4 (70.3; 148.5) [a]
Control	375.8 ± 98.0	346.0± 1.1	−29.8 (−39.8; 10.1)	
PPT tibialis anterior muscle affected side (kPa)				
Experimental	454.9 ± 79.0	495.1 ± 10.1	40.1 (29.0; 59.4)	39.7 (10.1; 59.4) [a]
Control	445.1 ± 79.0	445.6 ± 89.0	0.5 (−10.1; 10.4)	
PPT tibialis anterior muscle nonaffected side (kPa)				
Experimental	395.2 ± 59.3	464.8 ± 58.5	69.6 (30.1; 99.0)	69.2 (29.9; 99.0) [a]
Control	445.5 ± 89.2	445.1 ± 79.1	0.4 (−10.0; 10.1)	

Values are expressed as mean ± SD for baseline and final mean values and as mean (95% CI) for within- and between-group change scores.

[a] Statistical significant differences (ANCOVA, group × time, $P < .001$).

PPT, pressure pain threshold.

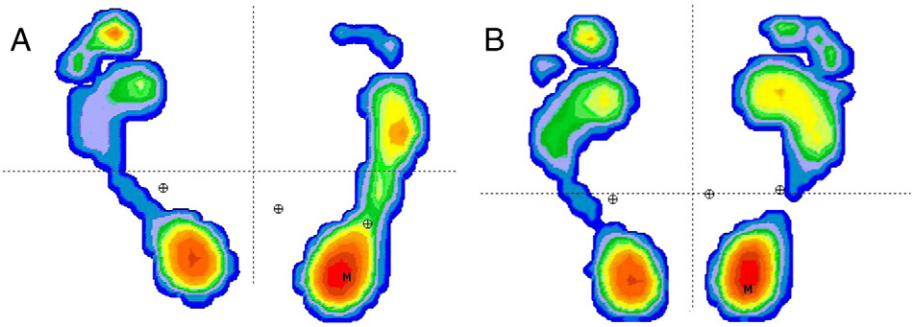

Fig 5. *Changes in support surface of the forefoot in patients with stroke before (A) and after (B) receiving DDN in the right (affected leg). B, The image shows bilateral increases of support surface in the forefoot after the intervention.*

acupuncture points. Changes in spasticity after DDN can be related to intrinsic modification induced to spastic muscles after its application. Because muscle contracture seems to be secondary to spasticity due to a reduction in the length of muscle fibers,[10] it is possible that DDN induce a localized stretch of the contractured cytoskeletal structures and reduction of the overlap between actin and myosin filaments. [21,22] This hypothesis is supported by the fact that DDN can reduce stiffness[24] of taut bands.[23] Therefore, because an increased resistance to passive ankle dorsal flexion in spastic muscles appears to be related to the inability of the muscle fascicles to elongate,[37] the restoration of sarcomere length and the decrease in stiffness of spastic taut bands may, at least in part, explain the decrease of poststroke spasticity observed in those patients receiving DDN.

In addition, increased motoneuron excitability induced by increased excitatory synaptic input, reduced interneuron inhibition, or alteration in intrinsic neuron properties is considered some of the main reasons behind spasticity.[38] It is also possible that DDN may modulate motoneuron activity and/or modify synaptic transmission from muscle afferents to spinal motoneuron by different reflex mechanisms. In fact, the local twitch response is an involuntary spinal reflex resulting from mechanical stimulation of a taut band with a needle and thought to occur in response to the presence of altered sensory spinal processing.[39] Chen et al[40] found that DDN had an inhibitory effect on spontaneous electrical activity of muscle motoneuron when local twitch responses were elicited during the technique. Therefore, changes in motoneuron excitability after the application of DDN require further investigation.

An interesting finding of the current study was that a single application of DDN was effective in harmonizing plantar pressures and support surface in both the affected and nonaffected feet. In fact, a decrease in spasticity of the gastrocnemius muscles would explain the bilateral increases of support surface in the forefoot and the unilateral

Table 5. *Baseline, Final Treatment Session, and Change Scores for Baropodometric Scores of the Forefoot*

Outcome Group	Baseline	End of Treatment	Within-Group Change Scores	Between-Group Difference in Change Scores
Support surface forefoot affected (treated) side (cm^2)				
Experimental	58.2 ± 15.5	62.1 ± 16.1	3.9 (0.8; 6.9)	3.5 (0.1; 6.9) [a]
Control	51.1 ± 12.0	51.5 ± 13.4	0.4 (−1.4; 2.2)	
Support surface forefoot unaffected (nontreated) side (cm^2)				
Experimental	43.4 ± 12.2	50.6 ± 13.4	7.2 (3.0; 11.4)	5.0 (0.6; 10.3) [a]
Control	49.0 ± 11.4	51.2 ± 11.5	2.2 (1.2; 5.7)	
Force distribution forefoot affected (treated) side (%)				
Experimental	27.9 ± 8.5	28.9 ± 8.5	1.0 (−1.0; 3.0)	1.1 (−1.3; 3.5)
Control	24.8 ± 7.2	24.7 ± 8.6	−0.1 (−1.5; 1.3)	
Force distribution forefoot unaffected (nontreated) side (%)				
Experimental	19.4 ± 6.2	21.1 ± 6.6	1.7 (0.5; 3.8)	1.4 (−1.6; 4.2)
Control	23.6 ± 7.1	23.9 ± 6.9	0.3 (−1.7; 2.4)	
Percentage of load forefoot affected (treated) side (%)				
Experimental	56.5 ± 16.6	55.3 ± 15.5	−1.2 (−6.8; 4.6)	−0.2 (−6.4; 5.9)
Control	65.3 ± 23.2	63.9 ± 25.2	−1.4 (−4.5; 1.4)	
Percentage of load forefoot unaffected (nontreated) side (%)				
Experimental	37.9 ± 11.5	42.3 ± 13.1	4.4 (0.8; 7.9)	3.7 (0.5; 7.9) [a]
Control	40.5 ± 11.5	41.2 ± 12.1	0.7 (−1.9; 3.2)	

Values are expressed as mean ± SD for baseline and final mean values and as mean (95% CI) for within- and between-group change scores.

[a] Statistical significant differences (ANCOVA, group × time, $P < .05$).

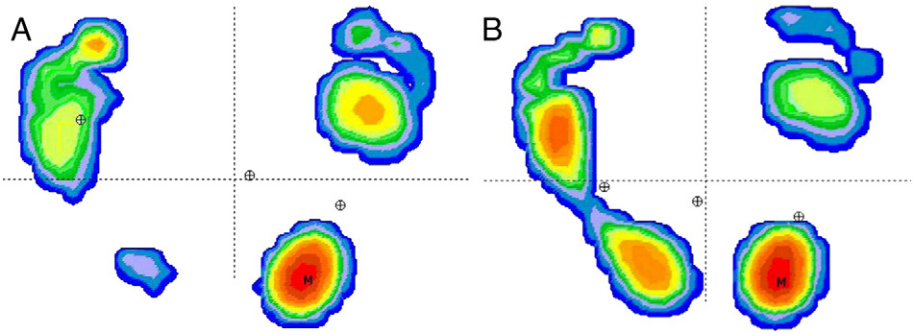

Fig 6. *Changes in support surface of the rear foot in patients with stroke before (A) and after (B) receiving DDN in the left (affected leg). B, The image shows a unilateral increase of support surface in the rear foot after the intervention.*

increase of the support surface in the rear foot of the treated side (affected) seen after DDN. These increases in the support surface of the forefoot may normalize distribution of the plantar pressures resulting in an increase of the percentage of load in the forefoot of the contralateral side. An increase in support surface will probably decrease mean plantar pressures of the foot. The harmonization of plantar pressures could be a first step for improving gait performance in patients who have had a stroke. A recent study found that resistive training with closed kinetic chain exercises was effective for improving normal gait patterns in patients who had experienced a stroke by increasing the contact area of the foot.[41] A single application of DDN was also able to increase the contact area of the affected foot; therefore, it would be interesting to examine if application of DDN within a multimodal rehabilitation program can help to improve standing and gait performance in patients who have had a stroke.

We also found that a single application of DDN reduced widespread pressure pain sensitivity in subjects who had experience a stroke because the experimental group experienced increases in PPT at both segmental (tibialis anterior) and nonsegmental distant (second metacarpal and deltoid muscle) points. Patients exhibited PPT increases ranging from 20% to 40% from baseline, suggesting potential real changes in pressure sensitivity.[36] Significant decreases in widespread pressure pain sensitivity support an antinociceptive effect of DDN.[25] The physiological mechanism for this effect remains unknown, and there currently exists much speculation surrounding the mechanisms of DDN, which potentially includes both segmental and central processes.[21,22,42,43] The fact that we found widespread changes in pressure sensitivity suggests that DDN is able to activate central antinociception. Roosink et al[44] observed that individuals with stroke, particularly those who develop pain, exhibit central sensitization. It is possible that successive applications of DDN can modulate sensitization mechanisms in this patient population and prevent the development of poststroke pain. This hypothesis requires further research.

Table 6. *Baseline, Final Treatment Session, and Change Scores for Baropodometric Scores of the Rear Foot*

Outcome Group	Baseline	End of Treatment	Within-Group Change Scores	Between-Group Difference in Change Scores
Support surface rear foot affected (treated) side (cm^2)				
Experimental	39.8 ± 14.4	46.2 ± 15.6	6.4 (0.2; 12.5)	5.0 (1.4; 11.3) [a]
Control	31.1 ± 19.9	32.5 ± 21.3	1.4 (0.8; 3.6)	
Support surface rear foot unaffected (nontreated) side (cm^2)				
Experimental	49.4 ± 15.6	50.7 ± 13.3	1.3 (−3.0; 5.6)	0.0 (−4.4; 4.5)
Control	49.3 ± 8.6	50.6 ± 8.8	1.3 (−0.6; 3.1)	
Force distribution rear foot affected (treated) side (%)				
Experimental	20.7 ± 10.3	24.0 ± 11.6	3.3 (0.6; 7.2)	2.7 (1.2; 6.7)
Control	16.2 ± 12.3	16.8 ± 12.9	0.6 (−0.8; 1.9)	
Force distribution rear foot unaffected (nontreated) side (%)				
Experimental	32.2 ± 8.3	30.5 ± 9.2	−1.7 (−4.9; 1.5)	−1.9 (−5.7; 1.8)
Control	35.3 ± 12.1	35.5 ± 12.5	0.2 (−2.1; 2.5)	
Percentage of load rear foot affected (treated) side (%)				
Experimental	43.1 ± 16.8	46.8 ± 18.4	3.7 (1.2; 9.4)	2.3 (−3.7; 8.4)
Control	34.7 ± 23.2	36.1 ± 25.2	1.4 (−1.4; 4.1)	
Percentage of load rear foot unaffected (nontreated) side (%)				
Experimental	61.6 ± 11.4	58.2 ± 13.6	−3.4 (−7.1; −0.4)	1.8 (−3.1; 6.9)
Control	59.2 ± 11.6	57.6 ± 13.4	−1.6 (−5.1; 2.0)	

Values are expressed as mean ± SD for baseline and final mean values and as mean (95% CI) for within- and between-group change scores.
[a] Statistical significant differences (ANCOVA, group × time, $P < .01$).

Table 7. *Baseline, Final Treatment Session, and Change Scores for Mean and Maximum Pressure*

Outcome Group	Baseline	End of Treatment	Within-Group Change Scores	Between-Group Difference in Change Scores
Maximum pressure affected (treated) side (g/cm^2)				
Experimental	818.3 ± 120.1	789.1 ± 130.3	−29.2 (−67.5; −9.0)	−13.7 (−67.8; 40.3)
Control	857.6 ± 169.1	842.2 ±194.1	−15.4 (−56.8; 25.8)	
Maximum pressure unaffected (nontreated) side (g/cm^2)				
Experimental	972.3 ± 347.4	911.8 ± 180.5	−60.5 (−182.6; 61.5)	−70.5 (−119.0; 56.0)
Control	987.3 ± 320.2	997.3 ± 401.3	10.0 (−54.5; 74.6)	
Mean pressure affected (treated) side (g/cm^2)				
Experimental	353.1 ± 39.5	331.6 ± 32.8	−21.5 (−39.1; −3.7)	−15.6 (−36.9; −5.9) [a]
Control	378.5 ± 74.2	372.6 ± 80.5	−5.9 (−19.4; 7.6)	
Mean pressure unaffected (nontreated) side (g/cm^2)				
Experimental	397.5 ± 64.9	366.3 ± 58.6	−31.2 (−50.3; −12.2)	−25.5 (−50.9; −0.1) [a]
Control	419.7 ± 89.2	414.0 ± 107.5	−5.7 (−24.0; 12.6)	

Values are expressed as mean ± SD for baseline and final mean values and as mean (95% CI) for within- and between-group change scores.
[a] Statistical significant differences (ANCOVA, group × time, $P < .05$).

Limitations

Although the results of our randomized controlled trial are promising, potential limitations should be recognized. First, this study only collected short-term outcomes. We do know if the observed changes lasted for longer durations or if the changes would have been similar between groups at a later time point. Significant changes have been observed, which supports future research in this area. The same clinician treated all patients in our study, which decreases the overall generalizability. We only used 1 treatment session with DDN; hence, inferences regarding multiple treatment sessions cannot be made. Future studies should include multiple treatment sessions with a greater number of clinicians and longer follow-up periods. Fourth, the use of the MMAS for assessing spasticity is under debate because it is a subjective scale and there are issues concerning validity and reliability. Nevertheless, the MMAS is the most commonly used tool in clinical practice and research. Finally, the sample size was small, and larger sample sizes would be needed to confirm the current results; however, the fact that significant and clinically relevant results were observed suggests that a greater sample would not alter the direction of the results. Future studies with larger sample sizes and long-term follow-ups are now needed.

Conclusion

The results of this trial suggest that a single session of DDN decreases spasticity and widespread pressure pain sensitivity in subjects with poststroke spasticity. Deep dry needling also induced changes in plantar pressure by increasing the support surface and decreasing the mean pressure.

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No funding sources or conflicts of interest were reported for this study.

Contributorship Information

Concept development (provided idea for the research): C.F.P., J.S.M., Z.S.M., R.O.S., M.P.C., S.T.D.
Design (planned the methods to generate the results): C.F.P., J.S.M., Z.S.M., R.O.S., M.P.C., S.T.D.
Supervision (provided oversight, responsible for organization and implementation, writing of the manuscript): C.F.P., R.O.S.
Data collection/processing (responsible for experiments, patient management, organization, or reporting data): J.S.M., Z.S.M., M.P.C.
Analysis/interpretation (responsible for statistical analysis, evaluation, and presentation of the results): J.S.M., Z.S.M., M.P.C., C.F.P.
Literature search (performed the literature search): C.F.P., J.S.M., Z.S.M., R.O.S., M.P.C., S.T.D.
Writing (responsible for writing a substantive part of the manuscript): C.F.P., J.S.M., Z.S.M., R.O.S., M.P.C., S.T.D.

Practical Applications

- This study suggests that a single session of dry needling decreases spasticity and widespread pressure sensitivity in patients who had experienced a stroke.
- Deep dry needling induced changes in plantar pressure by increasing the support surface and by decreasing mean pressure in patients who had experienced a stroke.

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