International Medical Case Reports Journal

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CASE REPORT

Effects of Dry Needling on Spasticity in Multiple Sclerosis Evaluated Through the Rate-Dependent Depression of the H Reflex: A Case Report

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Background: Spasticity is a common symptom of multiple sclerosis (MS) which affects mobility. Dry Needling (DN) has shown a reduction in spasticity in neuromuscular conditions such as stroke and spinal cord injury although the mechanism of action is still unclear. In spastic individuals, the Rate-Dependent Depression (RDD) of the H reflex is decreased as compared to controls and analyzing the effects of DN in the RDD may help to understand its mechanism of action.

Objective: To evaluate the effect of Dry Needling on spasticity measured by the Rate-dependent Depression (RDD) of the H reflex in an MS patient.

Methods: Three time points were evaluated: Pre-intervention (T1), Post-intervention assessments were carried out in the seventh week at two-time points: Before DN (T2) and After DN (T3). Main outcomes included the RDD and latency of the H reflex in the lower limbs at stimulation frequencies of 0.1, 1, 2, and 5 Hz in a five consecutive pulses protocol.

Results: An impairment of the RDD of the H reflex at frequencies ≥1 Hz was found. Statistically significant differences were found when comparing the mean RDD of the H reflex in Pre-intervention compared to Post-intervention at 1, 2, and 5 Hz stimulation frequencies. Mean latencies were statistically lower when comparing Pre- vs Post-intervention.

Conclusion: Results suggest a partial reduction in spasticity represented by decrease of the excitability of the neural elements involved in the RDD of the H reflex following DN. The RDD of the H reflex could be implemented as an objective tool to monitor changes in spasticity in larger DN trials.

Keywords: multiple sclerosis, dry needling, spasticity, H reflex, rate-dependent depression

Introduction

Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the central nervous system.[1] It is the most common debilitating neurological disease in young adults[2] affecting approximately 2.3 million people worldwide.[3] MS symptoms may include fatigue, poor balance, muscle weakness, pain, and spasticity[4] and it is estimated that associated costs range between 28,000 and 63,000 euros per patient,[3] with higher costs associated with an increase in severity.[5]

Spasticity is a feature of MS that affects around 34% of patients[6] and is manifested as increased muscle tone and reflex excitability, spasms, and contractures.[7] Treatment for spasticity includes a combination of pharmacological[8] and non-pharmacological[9] interventions. Pharmacological treatments used to reduce spasticity commonly produce undesired side effects such as drowsiness, somnolence, and weakness, among others. Moreover, patients may develop drug resistance.[8] In recent years, non-pharmacological approaches such as Dry Needling (DN) have been investigated for decreasing spasticity, which has shown to be effective to decrease spasticity in different neurological conditions such as

Received: 25 September 2022
Accepted: 20 December 2022
Published: 18 May 2023

stroke[10,11] spinal cord injury,[12] and more recently MS.[13] DN consists of the insertion of solid non-beveled needles at myofascial trigger points.[14] It has some similarities with western medical acupuncture, as both needling procedures involve penetration of the skin with solid filiform needles with therapeutic intent and are based on modern biomedical understandings of the human body. However, they have differences regarding the needling procedure (procedure duration, orientation of needle, etc) and the target structure (acupuncture points vs myofascial trigger points).[15]

The Hoffmann reflex (H reflex) evoked by electrical pulses applied on peripheral nerves and recorded in corresponding muscles is a tool to quantitatively evaluate spinal processing in normal and pathological conditions.[16] When the H reflex is produced by consecutive pulses at stimulation frequencies ≥1 Hz, a Rate-Dependent Depression (RDD) of the H reflex occurs. An impaired RDD has been observed in spastic patients after stroke,[17] spinal cord injury,[18–20] and MS.[21] Although several tests and scales are commonly used to evaluate spasticity, subject- and examiner-independent tests that objectively measure spasticity are highly desirable. The RDD of the H-Reflex could be a viable choice to objectively measure changes in spasticity. To our knowledge, there are no reports evaluating the effect of DN on spasticity in MS measured by the RDD of the H-reflex. In this case report as a primary outcome, we evaluate changes in spasticity measured by the RDD of the H reflex using DN. As a secondary outcome, we evaluate the effects of DN on two functional assessments: the 10-meter walking test (10 MWT) and the 6-min walk test (6 MWT).

Methods

Case Description

The patient is a 48-year-old male (66 kg, 1.73 m), with a 25-year diagnosis of MS, with his right side being the most affected. A clinical history of disc hernia and hyperthyroidism and a familiar background of cerebral stroke, fibromyalgia, cancer, and hyperthyroidism was reported. The subject also reports no physical activity, no smoking, or alcohol consumption. A wheelchair or a wheeled walker and bilateral ankle-foot orthosis are used by the patient to provide support during gait. According to the Penn scale, the patient has spontaneous spasms occurring more than ten times per hour, which prevents function and causes moderate discomfort. He also presents bilateral spontaneous clonus due to light mechanical stimulation. The patient receives three physical therapy sessions per week, consisting of pain management, mobility, and gait training. Management for spasticity included oral baclofen (Mylinax, Teva; 60 mg per day) and the use of Taopatches®.

Intervention

The protocol consisted of seven DN sessions, once per week. DN was performed bilaterally in the *vastus lateralis, vastus medialis, biceps femoris*, and *semitendinosus* muscles (Figure 1). The site of DN was determined by palpation to identify myofascial trigger points (MTrPs). The patient reported having burning pain in his lower legs bilaterally due to vascular problems, so DN was not performed in gastrocnemius muscles. DN was done by a certified physiotherapist in the DNHS® (Dry Needling for Hypertonia and Spasticity) method, using the following diagnostic criteria used for MTrPs to define the area of needle insertion: 1) Within the ensemble of taut bands, the one that displays the highest degree of tension (in muscles that are accessible); 2) The nodular zone within the band or the more sensitive area, if this exists; 3) Assessment of the movement and function of the patient; 4) Restriction of the range of movement, an increase in the resistance to passive movement or the triggering of a myotatic reflex, or other reflexes. The DN technique consisted of needling the diagnosed Myofascial Trigger Points with rapid in-and-out motions performed with solid, non-beveled 1.0 inch (0.32 mm x 25 mm) disposable needles (Wabbo®, Silverstar) at about 1 Hz (1 entry per second). Local twitch responses (LTRs) were achieved for every muscle treated in order to ensure that the MTrP was targeted. The application followed the criteria described for patients with a central nervous system lesion: 1) Place the muscle to be treated in a position of submaximal stretch; 2) Perform explorations of MTrPs using the needle, while controlling the stability of the segment until a significant cessation in the excessive muscular activity occurs; 3) Maintain the position during a brief period of time until the neural release appears or contraction ceases; 4) Remove the needle to the skin layer and then move the treated muscle into a position of sub-maximal stretch before proceeding with further needle explorations. The

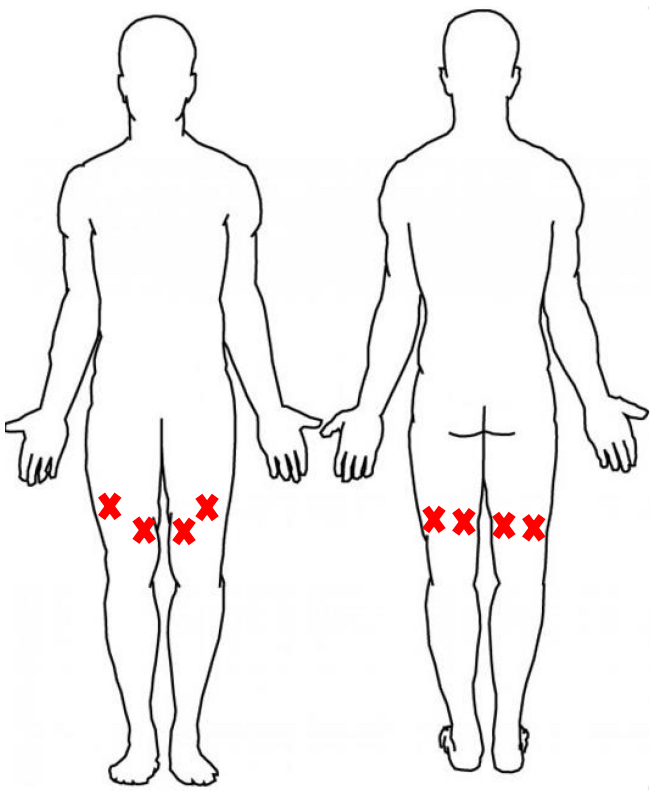

Figure 1 Sites of dry needling. Dry needling was done bilaterally in the *vastus lateralis, vastus medialis, biceps femoris*, and *semitendinosus* muscles.

intensity of the application was adjusted according to the patients' tolerance to interrupt the treatment. No adverse effects were reported during any of the DN sessions.

Assessment

The protocol consisted of seven DN sessions performed weekly. Baseline data of the RDD of the H reflex, 10 MWT, and the 6MWT were evaluated in the first session before DN (Pre-intervention -T1-). Post-intervention assessments were carried out in the seventh week at two-time points: Before DN (T2) and After DN (T3). The latter allows us to evaluate any possible chronic effect and acute effects of DN on spasticity. An outline of the protocol is shown in Figure 2. The 10 MWT and the 6MWT were done with the aid of the patient´s walker, but excluding the use of his ankle-foot orthosis. To compare the RDD of the H reflex with a control, a 51 y/o healthy subject was enrolled (73 kg, 1.66 m). The patient and the healthy participant signed an informed consent form authorizing the use of his information for this case report. Institutional approval was not required to publish the case details. The case report (CARE) guidelines were followed.[22]

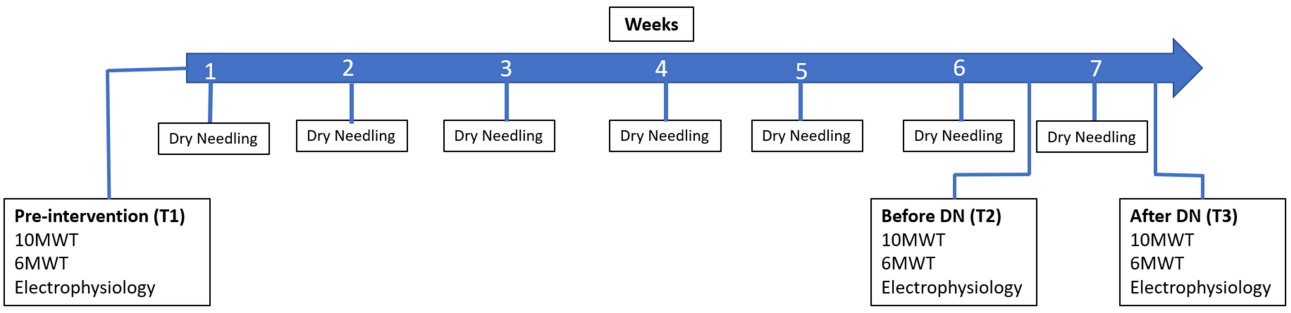

Figure 2 Outline of the protocol. Assessments were performed during Pre-intervention (1st week, T1) and Post-intervention, before (T2) and after (T3) the last DN session (7th week).

H-Reflex Assessment

The H reflex was evoked in the right leg with the subject lying in a prone position. Briefly, 1 ms, monophasic square pulses were applied (Digitimer D8SR) at the popliteal fossa with a bar electrode (MFI Medical). Two disposable surface electrodes (2.5 cm diameter, 3M) were placed over the midline at the belly of the gastrocnemius muscles, while a third electrode was placed over the Achilles tendon. Recordings were sampled at 10 kHz, amplified (PowerLab, ADInstruments), and stored for offline analysis. Stimulation currents producing around 50% of the maximal amplitude of the H reflex were used to evaluate the RDD of the H reflex.[23] Peak-to-peak amplitudes and latencies of the H reflex were measured using Clampfit v.10.7 (Molecular Devices). As a control, the H reflex was evoked at 0.1 Hz. At this stimulation frequency, there is no RDD as described previously.[17,19,21] Then, three trains of five consecutive pulses were applied at 1, 2, and 5 Hz with thirty seconds intervals between trains. This approach diminishes discomfort produced by several pulses applied at high-frequency stimulation. To evaluate the RDD of the H reflex, the amplitude of the H wave from the second to the fifth pulses (Hn) was divided by the amplitude of the first pulse (H1). A closer ratio Hn/H1 to 1 at stimulation frequencies of 1, 2, and 5 Hz, means an impaired RDD of the H reflex. Graphs of Hn/H1 vs the number of pulse were constructed for each stimulation frequency and then data were averaged to obtain the mean RDD for each stimulation frequency. To avoid any possible misreading of the H reflex acutely, the patient was asked to remove the Taopatches® and not to attend his physiotherapy session 48 hours before the Post-intervention assessment (week 7).

Data Analysis

Latencies and amplitude (P-P) of the H reflex were analyzed with Clampfit v.10.7 (Molecular Devices) and are reported as mean ± SD. To compare data at the three-time points in the MS subject, one-way repeated measures ANOVA was performed. The normality of data was assessed by the Shapiro–Wilk test ($p > 0.05$). Pairwise multiple comparisons were done by the Holm-Sidak method. If data were not normally distributed, ANOVA on Ranks and Tukey's test pairwise multiple comparisons were performed. Statistically significant differences were established if $p < 0.05$. SigmaPlot v.14 was used for statistics and graphical representation.

Results

RDD of the H-Reflex

Examples of recordings of the H reflex are shown in Figure 3 for the MS (Figure 3A) and healthy (Figure 3C) subjects. In the case of the MS subject, data were obtained at T1. For simplicity, a single train of five pulses is shown. The first pulse in each plot (Figure 3A and C) is represented by the black line. Note that in the subject with spasticity and in the healthy volunteer there was no RDD differences at 0.1 Hz as expected (ie the amplitude of the H reflex is similar). However, loss of RDD was observed at 1, 2, and 5 Hz in the MS subject (Figures 3A) compared to the healthy subject (Figures 3C). The ratio Hn/H1 of the recordings on the left at each stimulation frequency is also shown for the MS (Figure 3A) and the healthy subjects (Figure 3C). In Figure 3B, note that the mean RDD did not decrease under 0.5 in the MS subject (red dotted line, left panel) while in the healthy subject, the mean RDD decreased below this value (Figure 3D, red dotted line, left panel), indicating an impaired RDD secondary to spasticity in the MS subject. Mean latencies of the H reflex at each stimulation frequency are shown in Figure 3B (MS Subject, right panel) and Figure 3D (Healthy subject, right panel).

Mean RDD and latency of the H reflex were determined for the Pre-intervention at week 1 (T1, gray line) and compared to the Post-intervention (week 7) at two time-points: before (T2, black line) and after (T3, red line) DN in the MS subject (Figure 4A). RDD values are shown in Table 1. At 0.1 Hz, RDD values were similar at T1, T2 and T3, with no significant differences between time points ($p = 0.42$). At 1 Hz, mean RDD at T1 and T2 were similar ($p = 0.91$); however, T3 was higher compared to T1 ($p < 0.001$) and T2 ($p < 0.001$). At 2 Hz, statistical differences were found when comparing T1 vs T2 ($p = 0.04$) and T3 ($p = 0.03$) and T1 vs T2 ($p < 0.001$). At 5 Hz, statistical differences were found when comparing T1 vs T2 ($p = 0.01$) and T3 ($p < 0.001$) as well as when comparing T2 vs T3 ($p = 0.01$).

Mean latencies are shown in Figure 4B and Table 2. At 0.1 Hz, mean latencies of the H reflex measured at T1 showed significantly lower values than T2 ($p < 0.001$) and T3 ($p < <0.001$). Mean latency at T2 was also significantly lower

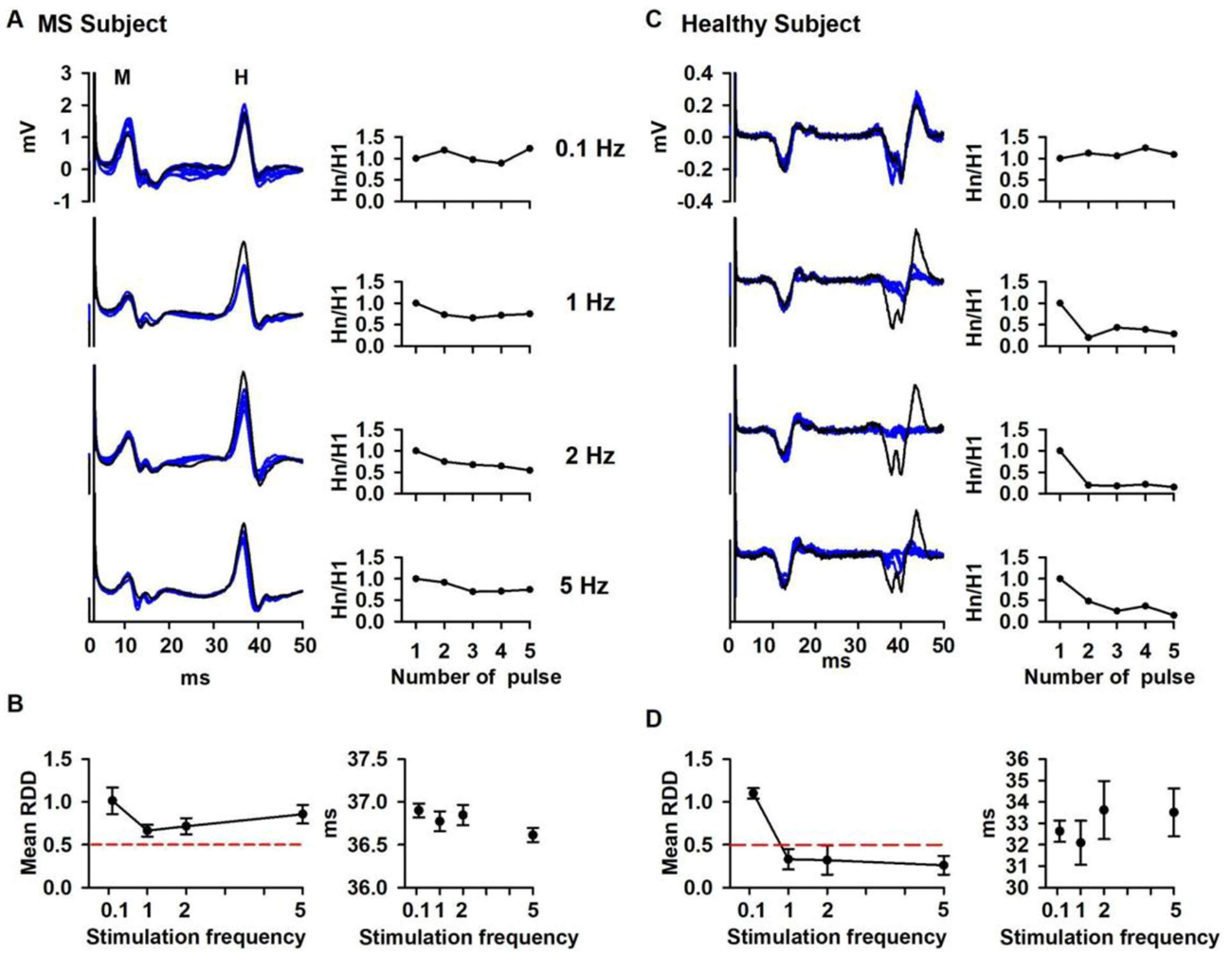

Figure 3 Loss of RDD of the H reflex in the MS patient. (**A**) MS subject. Left panel, superimposed traces of the H reflex evoked at 0.1, 1, 2, and 5 Hz. Black traces represent the first pulse while the blue ones represent the following pulses (2–4). Right panel, corresponding graphs of the RDD expressed as Hn/H1. (**B**) Mean RDD (left panel) and latency of the H reflex (right panel). (**C**) Corresponding traces and graphs as shown for the MS subject are presented in a healthy subject for comparison. (**D**) Mean RDD (left panel) and latency of the H reflex (right panel) in the healthy subject. Red dotted lines in B and D point out 0.5 as a reference value for comparative purposes.

compared to T3 ($p < 0.001$). At 1 Hz, T1 was significantly lower than T2 ($p < 0.001$) and T3 ($p > 0.001$). Mean latency at T2 was also significantly lower compared to T3 ($p < 0.001$). Similar results were observed at 2 Hz, where T1 was significantly lower compared to T2 ($p = 0.01$) and T3 ($p < 0.001$). Mean latency at T2 was also significantly lower compared to T3 ($p = 0.01$). At 5 Hz, T1 was significantly lower compared to T3 ($p < 0.001$), but not to T2 ($p = 0.11$). At this same stimulation frequency, T2 was significantly lower compared to T3 ($p < 0.01$).

Functional Assessment

A complete summary of the results is presented in Table 3. The 10 MWT showed a decrease at T2 (90 s) compared to T1 (108); however, at T3 the total time was similar to T1. The 6 MWT showed an improvement from 32.4 m at T1 to 34.75 m at T2 and 37.75 m at T3.

Discussion

This case report aimed to evaluate the effect of DN on spasticity in a MS subject measured by the RDD of the H reflex. The RDD of the H reflex is commonly impaired in MS patients as compared to healthy subjects.[21] In this study, we confirmed this observation (Figure 3) and extended previous data by implementing a protocol based on three trains of five pulses at 1, 2, and 5 Hz stimulation frequencies to evaluate the RDD (Figures 3 and 4). As shown in Figure 3B, Mean RDD at 1, 2 and

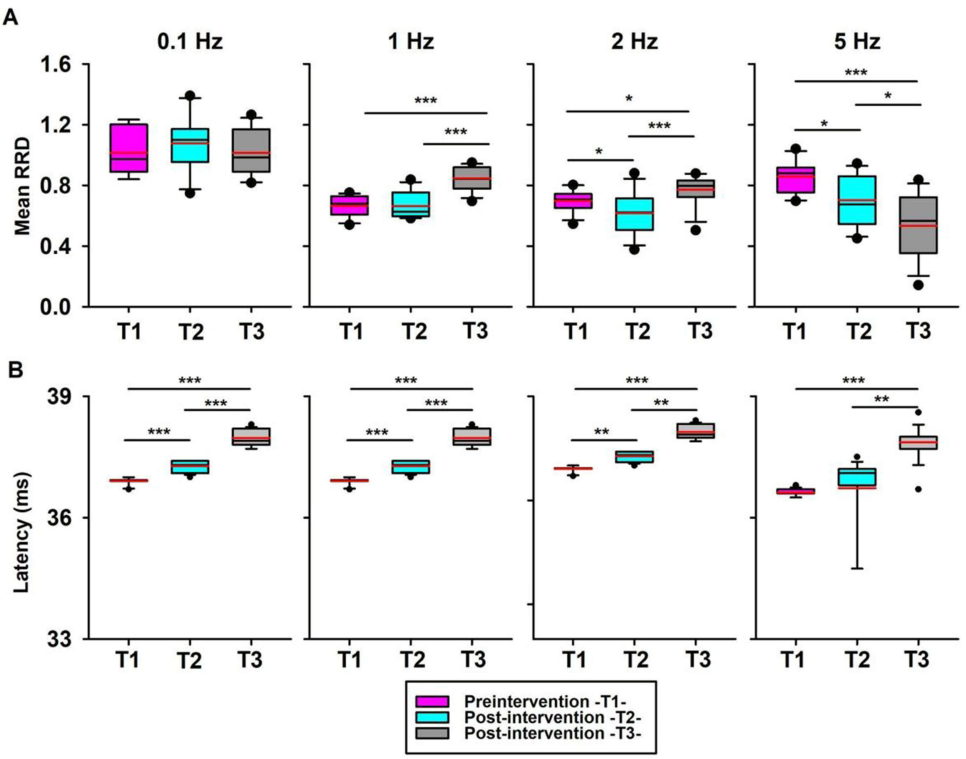

Figure 4 (**A**) Mean RDD at Pre-intervention -T1-, and Post-intervention at week 7 before DN -T2-, and after DN -T3-. (**B**). Latency of the H reflex at Pre-intervention -T1-, Post-intervention before DN -T2- and after DN -T3-. Data shown correspond to 0.1, 1, 2 and 5 Hz stimulation frequency. Color codes: T1, magenta; T2, cyan, and T3, gray. Black lines inside the boxes represent the median, while the red ones are the mean. *Statistically significant ($p < 0.05$), **Statistically significant ($p < 0.01$), ***Statistically significant ($p < 0.001$).

5 Hz were above 0.5 in the MS subject in contrast with the matched control subject, where mean RDD values decreased under 0.5, indicating that in spasticity there is a loss of the RDD (ie, loss of spinal inhibition). Significant differences were found in the mean RDD at 1, 2, and 5 Hz stimulation frequencies across time points (Figure 4A) except between T1 and T2 for 1 Hz. This could be due to this frequency not being sensitive enough to account for changes in RDD. At 1 and 2 Hz, mean RDD at T3 was significantly higher compared to T2 and T1; although we expected lower mean RDD compared to T1 (basal data). Interestingly, at 5 Hz, a partial recovery of the RDD was observed both at T3 and T2 compared to T1 (Figure 4A). For instance, in Figure 3B, mean RDD at 5 Hz in the MS subject at T1 had a clear loss of the RDD (0.85 ± 0.10). On the other hand, the mean RDD at this stimulation frequency at T3 (0.53 ± 0.20) was close to an arbitrary cutting value of 0.5 representing a "normal RDD" as presented in Figure 3D for a healthy subject. Although our results should be taken cautiously, differences in the RDD of the H reflex suggest changes in the spinal processing attributable to DN. Moreover, these results stress the importance of evaluating stimulation frequencies > 2 Hz. Moreover, it is important to

Table 1 Summary of Pre-Intervention (Week 1) vs Post-Intervention (Week 7) Mean RDD Values and Absolute Differences Between Time Points

Stimulation Frequency (Hz)	T1	T2	T3	Dif T2-1	Dif T3-T2	Dif T3-T1
0.1 Hz	1.01 ± 0.15	1.07 ± 0.18	1.01 ± 0.14	0.06	0.06	0
1 Hz	0.66 ± 0.06	0.66 ± 0.08	0.84 ± 0.07	0	0.18 ***	0.18***
2Hz	0.69 ± 07	0.61 ± 0.10	0.77 ± 0.10	0.08*	0.16***	0.08*
5 Hz	0.85 ± 0.10	0.70 ± 0.17	0.53 ± 0.20	0.15*	0.23*	0.32***

Notes: *Statistically significant ($p<0.05$). ***Statistically significant ($p < 0.001$).
Abbreviation: Dif, difference.

Table 2 Summary of Pre-Intervention (Week 1) vs Post-Intervention (Week 7) Mean Latencies and Absolute Differences Between Time Points

Stimulation Frequency (Hz)	T1	T2	T3	Dif T2-1	Dif T3-T2	Dif T3-T1
0.1 Hz	36.90 ± 0.08	37.27 ± 0.13	37.96 ± 0.18	0.37***	0.69***	1.06***
1 Hz	36.77 ± 0.11	37.32 ± 0.15	38.17 ± 0.13	0.55***	0.85***	1.4***
2Hz	36.84 ± 0.11	37.26 ± 0.10	38.30 ± 0.32	0.42**	1.04**	1.46***
5 Hz	36.61 ± 0.08	36.71 ± 1.22	37.86 ± 0.39	0.1	1.15**	1.25***

Notes: Results are expressed in milliseconds (ms). **Statistically significant ($p<0.01$). ***Statistically significant ($p < 0.001$).
Abbreviation: Dif, difference.

Table 3 Summary of Pre-Intervention (Week 1) vs Post-Intervention (Week 7) Functional Assessment

Test	Pre-Intervention T1 (Week 1)	Post-Intervention (Week 7)	
		Before DN -T2-	After DN -T3-
10MWT (min)	1.8	1.5	1.8
6MWT (m)	32.5	34.75	37.75

Abbreviations: 10 MWT, 10 meters walking test; 6 MWT, 6-minutes walk test.

mention that due to pain experienced by the patient, DN was not performed in the gastrocnemius muscles, where the H-reflex was recorded. However, significant statistical changes in the RDD and latency were observed. In this context, Taki et al[24] described changes in fluctuations of amplitudes of the H reflex in synergistic muscles attributed to a common source of noise, meaning that spinal circuits and particularly those involved in the H reflex could be modified across several segments (ie the lumbar enlargement). If DN has an impact not only in the intervened muscle but also in other segmentally related muscles has to be clarified in future experiments.

Mean latencies at all stimulation frequencies were higher at T3 compared to T2 and T1 (Figure 4B). Except at 5 Hz, latencies at T2 were also significantly different compared to T1 (Figure 4B). This result is similar to the data reported at 3 weeks after DN in the wrist flexor by Babazadeh-Zavieh et al.[25] Although we did not measure latencies at 3 weeks, an increase in latencies was observed in our study at 7 weeks between T1 and T2-T3. Babazadeh-Zavieh et al suggested a decrease in alpha motor neuron excitability, although changes in the spinal circuitry should not be discarded as evidenced by our results in the mean RDD. In this context, changes in latency could be related to improvements in both functional assessments, 10 MWT and 6 MWT, although only changes in the 10 MWT achieved minimal clinical differences.[26,27] Overall, additional data is needed to establish a causal relationship. Interestingly, another study carried out by Jiménez -Sánchez et al[28] did not find differences in neurophysiological properties of the H reflex, including latencies and the H_{max}/M_{max} in non-injured subjects that received DN, suggesting that if any change occurs, this should be just evident in patients with neuromuscular disorders. Importantly, it has been discussed that the ratio H_{max}/M_{max} does not represent the motor pool excitability, as reported by Knikou.[23] In this context, the RDD could be a more representative and sensitive tool to compare the inhibitory spinal mechanisms between healthy and diseased individuals.

It is also important to mention that the patient wore Taopatches® during the study period except for 48 h previously to the last session where the Post-intervention effects of DN on spasticity were evaluated through the RDD of the H reflex. Amato et al implemented a home-based training protocol combined with the use of Taopatches® in seventeen MS patients and reported an increase in grip strength after the intervention period of ten weeks.[29] We assume that the use of Taopatches® did not affect the results of this study, as the patient manifested no perceived changes in spasticity for at least two months before the beginning of the protocol.

In this case report, DN was performed once per week for seven weeks. The use of DN once a week has shown improvements in pain and spasms[13] in patients with MS. In other populations such as stroke or SCI, similar results have been found. On the one hand, DN has shown to decrease muscle spasticity and to improve lower limb function and gait in the short term[30,31] in patients with stroke. On the other hand, DN has shown to have positive effects on spasticity, dynamic stability and walking velocity after 10 weeks in a patient with SCI.[12] Despite these positive clinical findings, the mechanism of DN to decrease spasticity is not well known and needs further research.[32] Current research supports the hypothesis that DN could have neuromodulation effects at different levels in the central nervous system,[33,34] with recent EEG studies highlighting the existence of variations of local parameters of the brain network in the delta, theta and alpha bands.[32] Moreover, there is no agreement neither on how many DN sessions per week and for how long this therapy would show the best results, although a period of one week between DN sessions is recommended considering tissue repair.[35]

Based on the above, the implementation of the RDD of the H reflex may represent an important tool to assess spasticity through the inhibitory spinal system; additionally, it could be a useful technique to compare upper vs lower limbs and right vs left side of the body. This technique could be also used to evaluate quantitative changes in spasticity due to pharmacological or non-pharmacological treatments, both acute and long-term effects.

To our knowledge, this is the first study evaluating the changes in spasticity measured by the RDD of the H reflex after DN. Additionally, we introduced a protocol evaluating 1, 2, and 5 Hz stimulation frequencies with trains of five pulses in a spastic subject due to MS, which allows to evaluate the RDD phenomenon and objectively quantify changes in spasticity (Figures 3 and 4). Limitations of this study must be considered, for example, measurements of the RDD of the H reflex were not obtained after DN in the first session (T1) and in intermediate time points; ie every one or two weeks. In future studies, recordings of the intervened muscle could be performed if there are no clinical constraints as well as a follow-up after DN to research carry-over effects.

Conclusions

The RDD of the H reflex could be used as a biomarker of spasticity. Concretely, stimulation frequencies >2 Hz in trains of 5 pulses should be considered when quantifying mean RDD to establish differences across time points. A larger sample is warranted for evaluating the RDD of the H reflex to study central and peripheral nervous mechanisms of DN on spasticity.

Acknowledgments

We thank the patient and his family for their kind support. We also thank the healthy volunteer for participating in this study.

Author Contributions

All authors made a significant contribution to the work reported whether that is in the conception, study design, execution, acquisition data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Funding

This study was supported by the Universidad Anáhuac México, Campus Norte.

Disclosure

Dr. Pablo Herrero is the creator of the DNHS technique and teaches courses about this technique. The authors report no other conflicts of interest in this work.

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