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Case Series

Clinical differentiation of spasticity and spastic dystonia after chronic stroke using surface electromyography: a case series

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ABSTRACT

Introduction: Spasticity affects a large number of stroke patients, limiting their mobility and autonomy. Surface electromyography (sEMG) is the most suitable option for quantifying spasticity and differentiating it from other phenomena. Despite evidence of dry needling's effect on spasticity, it has not been quantified using sEMG, nor have other patterns of muscle overactivity been explored.

Case presentation: Four cases of patients with chronic stroke and spasticity are presented, who received intensive therapy for 4 weeks, combined with a weekly session of ultrasound-guided dry needling in the medial gastrocnemius muscle. Muscle activity was evaluated using sEMG at rest and during stretching, the Modified Ashworth Scale (MAS), and walking tests at the start of the study (T0), after the first dry needling session (T1), and at the end of the treatment (T2). Different phenotypes were identified based on electromyographic activity: high activity during stretching, high variability, high activity at rest and during stretching, and a combination of the above.

Results: Electromyographic activity during stretching was reduced in cases with high response and variability to stretching without spastic dystonia, while resting activity was reduced in those cases that did present spastic dystonia. The MAS scale improved in all patients, and walking tests exceeded clinically significant thresholds. No adverse effects were reported.

Conclusion: A theoretical-practical framework is proposed for assessing muscle tone with sEMG, describing different clinical phenotypes in patients with chronic stroke and supporting the combination of dry needling and intensive therapy to improve both spasticity and gait.

Keywords: Dry needling; surface electromyography; spasticity; spastic dystonia; stroke; intensive care; neurorehabilitation.

1. Introduction

Stroke is a health problem in Spain, with an incidence of 120,000 cases and 25,000 deaths per year, along with an increase in incidence in recent years.⁽¹⁾ 25% of strokes occur in people aged 20 to 64, and the incidence increases significantly from the age of 65.⁽²⁾

Spasticity is a very common aftereffect in people who suffer from this disease, affecting 25% of patients⁽³⁾, causing disability, impairing mobility and functionality⁽⁴⁾, which can lead to comorbidity problems, which in turn hinder improvement over time.^(5–7)

The definition of spasticity has undergone changes since its initial formulation in the 1980s,⁽⁸⁾ and is currently defined as "a result of the hyperexcitability of the descending pathways from the brainstem, generating an exaggerated response to the stretch reflex that depends on the speed at which the stretch is performed and the muscle length at which the muscle is during the stretch."⁽⁷⁾

Studies using surface electromyography (sEMG) have been shown to be the most suitable option for quantifying spasticity compared to classic manual scales.^(9,10) In contrast to other instrumental measurements,⁽¹¹⁾ sEMG stands out as an inexpensive technique, quick to apply, and does not necessarily require additional external software.⁽⁹⁾

While it is true that spasticity is not the only sign affecting patients with central nervous system injuries and has traditionally been used as an umbrella term⁽¹⁰⁾, several authors have described spastic dystonia as 'an increase in resting tone with an inability to relax voluntarily.' This can be measured through electromyographic recordings and may or may not be accompanied by spasticity, which contributes to generating terminological confusion.^(12–15)

This spastic dystonia is very common in patients with upper motor neuron syndrome^(12–14), sharing the lesion territory within the central nervous system with spasticity.⁽¹⁶⁾ However, while spasticity is considered primarily an afferent phenomenon, spastic dystonia is regarded as a purely efferent phenomenon secondary to an abnormal supramedullary descending conduction pattern.^(7,16,17)

On the other hand, spastic dystonia seems more closely related to the stiffness and contracture problems patients experience in the long term, because sustained hypertonia leads to progressive immobilization, which causes structural changes in the soft tissues.^(15,16)

Within this context, a combination of spastic dystonia with spasticity has been described, which may or may not be modulated by stretching, with patients being found to have: 1) spasticity that is modulated by sustained stretching; 2) spasticity that is not modulated by sustained stretching; 3) spastic dystonia and spasticity that are modulated by sustained stretching; and 4) spastic dystonia and spasticity that are not modulated by sustained stretching.⁽¹⁴⁾

Despite evidence of the effect of dry needling on spasticity in people with stroke^(18–20), its effect has not yet been quantified using sEMG, nor has its effect on spastic dystonia been explored.

The main objective of this case series is to define a methodology for the objective measurement of spasticity through linear manual stretches and the identification of the presence of spastic dystonia, as well as to electromyographically evaluate the effects of dry needling on the medial gastrocnemius muscle, combined with intensive therapy, in different phenotypes of chronic-stage stroke patients who present with spasticity.

2. Description of case series

2.1 Patient information

The selected patients are part of the clinical trial ‘Effects of Dry Needling on Electromyographic Activity and Ultrasonographic Characteristics in Post-Stroke Spasticity’ with the registration code [clinicaltrials.gov](https://clinicaltrials.gov/ct2/show/study/NCT06017960) identifier NCT06017960, approved by the Ethics Committee of Aragón with reference C.I. PI23/333. All procedures were conducted in accordance with the Declaration of Helsinki, and patients provided informed consent before participating. For the article, the instructions and sections outlined in the CARE Guide were followed.

The study was conducted at the European Centre for Neurosciences in Madrid between 2023 and 2025. The inclusion criteria were: [1] being over 18 years old; [2] understanding and voluntarily signing the informed consent before undergoing the intervention; [3] having a medical diagnosis of chronic stage ischemic or haemorrhagic stroke (at least 6 months of progression);^(21,22) [4] having a score between 1 and 3 according to the modified Ashworth Scale (MAS) in the triceps surae; [5] being able to walk independently to perform the 10 Meter Walk Test (10MWT), 6-Minute Walk Test (6MWT), and Timed Up and Go Test (TUG). The exclusion criteria were: [1] patients with recurrent stroke, [2] who had received previous type A botulinum toxin treatments in the last 3 months, or [3] who had undergone dry needling in the past month, [4] severe cognitive deficits that, in the evaluator's judgment, would hinder administering the study assessments, [5] fear of needles, or [6] metal allergies.

The following is a description of the patients in this case series:

Case 1

A 51-year-old man with an ischemic stroke affecting cortico-subcortical areas of the left hemisphere, with 7 months of progression. The intervention consisted of 4 sessions of ultrasound-guided dry needling along with 20 hours of weekly therapy, distributed in 4 hours per day, 5 days a week, for 4 weeks, totalling 80 hours of therapy. (Table 1)

Case 2

A 63-year-old woman with a haemorrhagic stroke affecting the right frontoparietal area, with 9 years of evolution. The intervention consisted of 4 sessions of ultrasound-guided dry needling, along with 15 hours of weekly therapy, distributed as 3 hours per day, 5 days a week, for 4 weeks, for a total therapy time of 60 hours. (Table 1)

Case 3

A 42-year-old man with an ischemic stroke affecting the right frontoparietal area with 9 months of evolution. The intervention consisted of 4 sessions of ultrasound-guided dry needling, along with 25 hours of weekly therapy, distributed as 5 hours daily, 5 days a week, for 4 weeks, for a total therapy time of 100 hours. (Table 1)

Case 4

69-year-old woman with a haemorrhagic stroke affecting cortico-subcortical areas of the left hemisphere, with a course of 4 years and 4 months. The intervention consisted of 4 sessions of

ultrasound-guided dry needling along with 6 hours of therapy per week, distributed as 2 hours daily, 3 days a week, for 4 weeks, totalling 24 hours of therapy. (Table 1)

Table 1. Demographic characteristics of the case series.

	Age (years)	Gender (M/F)	Most affected side (L/R)	Stroke type (I/H)	Location of the lesion	Evolution time (months)	Weekly therapy hours
Case 1	51	M	L	I	Cortico-subcortical	7	20
Case 2	63	F	R	H	Frontoparietal	108	15
Case 3	42	M	R	I	Frontoparietal	9	25
Case 4	69	F	L	H	Cortico-subcortical	52	6

Type of stroke: Ischemic/Haemorrhagic.

2.2 Clinical variables and assessment tools

Assessment of muscle activity using surface electromyography

For the sEMG assessment, the mDurance device (mDurance Solutions SL, Granada, Spain) was used, with a sampling frequency of 1024 Hz and a bandwidth of 8.4 kHz. The EMG signal resolution was 24 bits, with overall amplification of 100-10,000 V/V. This signal was filtered using a fourth-order Butterworth filter with a cutoff frequency of 20-450 Hz. Subsequently, the rectified signal was processed using the root-mean-square method, with all values expressed in microvolts (µV).⁽²³⁾ The electrodes used were pre-gelled Ag/AgCl with a diameter of 30 mm.

The electrodes were placed 2 centimeters apart, parallel to the muscle fibers, distal to the knee, and 2 centimeters medial to the midline, following clinical guidelines, with the area to be evaluated cleaned prior to placement.⁽²⁴⁾ (Figure 1) All measurements were taken on a stretcher with the patient in a supine position in a quiet environment, with the lower limbs in a neutral position at the hips and knees; the feet were positioned in their natural stance, which for most people is at the mid-range of motion.^(24,25)

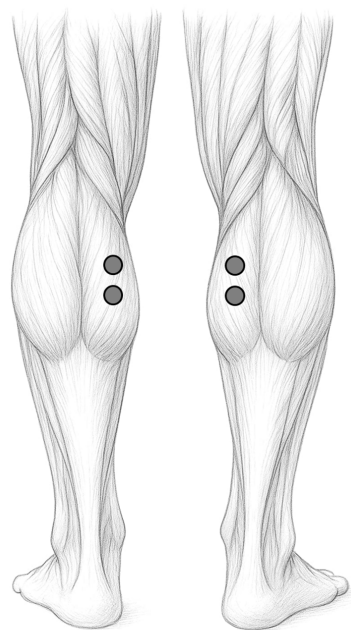


Figure 1. Placement of the electrodes, 2 centimeters apart from each other, parallel to the muscle fibers, distal to the knee, and 2 centimeters medial to the midline.

Muscle activity at rest – Spastic dystonia

With the patient relaxed in the described position, spastic dystonia was assessed, defined as “the inability to voluntarily suppress muscle activity at rest”^(12,13,15) of the medial gastrocnemius muscles of the affected and less affected sides for 30 seconds.^(14,25)

To establish the tone threshold of spastic dystonia, the less affected muscle of each subject was used as a reference, calculating the mean (μ) and standard deviation (σ) of the contralateral side and considering it pathological when the mean RMS of the affected side exceeded $\mu + 2 \cdot \sigma$.^(26,27)

Linear manual stretching – Spasticity

Spasticity was also assessed using sEMG, defined as “the degree of reflex hyperexcitability, expressed by the maximum electromyographic activity reached by the muscle during controlled passive stretching” of the ankle through linear manual stretches synchronized with a metronome.

For this purpose, the protocol described and validated by Marinelli et al. (2013) was used, which demonstrated highly reproducible results across evaluators after a training period of less than 20 minutes.⁽²⁸⁾

The protocol consisted of 4 phases: [1] the evaluator adjusted the metronome frequency via headphones so that only he could hear the set rhythm of 60 BPM, during which the average angular velocity was $106 \pm 24^\circ/\text{s}$. [2] The passive ankle dorsiflexion movement was synchronized with the headphone rhythm, allowing a full range of motion to be completed in 1 beat. [3] Once the evaluator had synchronized the movement with the rhythm, the movement was stopped at maximum plantar flexion, and he continued to imagine the movement for at least 15 seconds during the first repetition, maintaining the metronome rhythm. [4] Finally, the evaluator performed the linear movement from maximum plantar flexion to maximum ankle dorsiflexion in 1 metronome beat (1 second) in a synchronized manner.

Following this same methodology, 10 manual stretches were performed with 10-second intervals between measurements, in accordance with recommendations from previous studies to minimize fatigue and ensure recovery of muscle fibers from the previous stretch.⁽²⁹⁾ The maximum activity of each repetition was taken as a reference to subsequently calculate the mean and standard deviation.

In this case, comparing the affected side with the contralateral side, as was done in the resting measurement, is neither appropriate nor feasible manually, since the stretch reflex in a healthy muscle is generally evoked at 300 to 350°/s.^(30,31) To compare several subjects in spasticity measurements, the electromyographic signal was normalized to the maximum voluntary isometric contraction (MVIC), with results expressed in absolute values (μV) and normalized values (%MVIC), as recommended by international consensus.⁽³²⁾

To perform the measurement, the ankle was placed in a mid-range position of the available joint range, and the patient was asked to perform a sustained isometric contraction for 5 seconds. This measurement was performed 3 times, with a 1-minute interval between trials. The maximum electromyographic activation values from each repetition were selected to obtain the average value.⁽³³⁾

Assessment of spasticity using clinical scales

Additionally, spasticity was assessed using the Modified Ashworth Scale (MAS), a scale widely used in central nervous system disorders⁽¹¹⁾, with moderate intra-rater reliability in stroke patients.⁽³⁴⁾

The scale assesses muscle tone from 0 to 4. A score of 0 means there is no resistance; 1 means there is resistance at the end of the movement; 1+ means a slight increase in muscle resistance followed by minimal resistance for the rest of the movement (less than half); 2 means muscle resistance throughout the range of motion, but easily movable; 3 means a considerable increase in muscle tone, with resistance throughout the range of motion and difficult to move; and 4 means the affected part remains rigid with no possibility of movement.

Gait assessments

Walking speed was assessed with the 10MWT, walking endurance with the 6MWT, and walking functionality with the TUG.^(35,36)

The TUG test involves measuring the seconds it takes for the subject to get up from a chair approximately 46 centimeters high, walk 3 meters, turn around, and sit down again. This scale has shown excellent intra-rater reliability in stroke patients.⁽³⁶⁾ The minimal detectable change (MDC) of the scale in people with chronic stroke is 2.9 seconds.⁽³⁷⁾ In this case, this parameter was selected because values for the minimal clinically important difference (MCID) in the chosen population are not established in the literature.

The 10MWT test measures the time it takes the subject to walk 10 meters in a straight line to obtain the comfortable and fast speed in meters per second (m/s), with excellent intra-rater reliability.⁽³⁸⁾ The MCID of this test is set at 0.14 m/s.⁽³⁹⁾

The 6MWT test evaluates the distance in meters that the subject covers over 6 minutes and has shown good intra-rater reliability in stroke patients.⁽⁴⁰⁾ The MCID for this assessment is set at 55m.⁽³⁹⁾

2.3 Measurement moments

Muscle activity was measured with sEMG at rest, during manual stretching, and while performing the MVIC at three time points: T0) before the start of the study; T1) after the first dry needling intervention; and T2) at the end of the fourth and last dry needling intervention. In addition, clinical assessments of spasticity were performed using the MAS scale at the same time points (T0, T1, and T2), and assessments of walking ability were conducted using the TUG, 10MWT, and 6MWT scales at the beginning and end of the intervention (T0 and T2). Figure 2 shows the timeline of the assessment and intervention protocol.

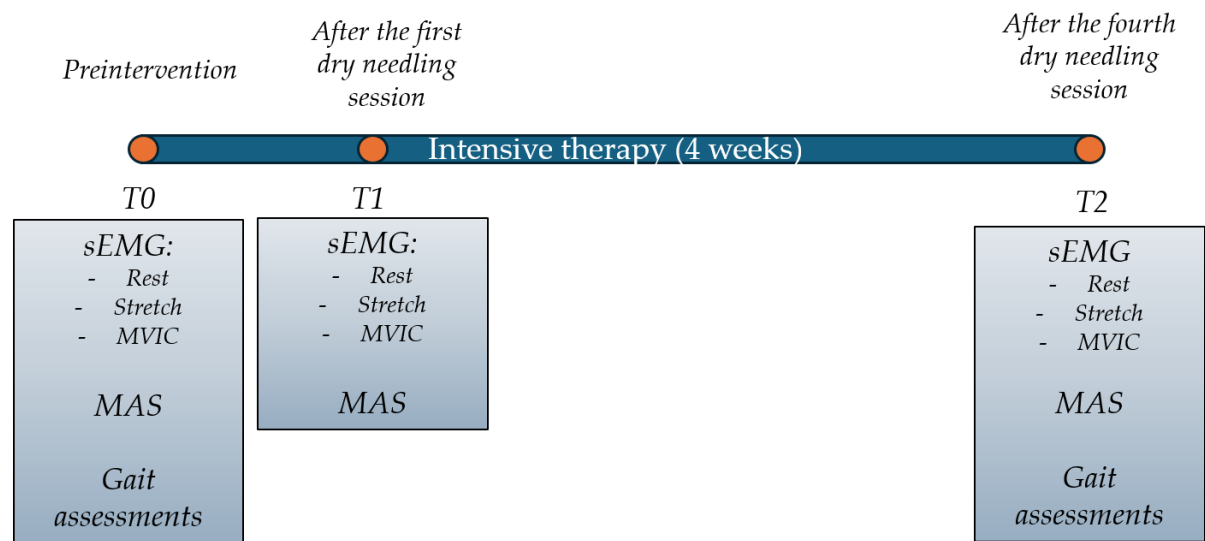


Figure 2. Timeline of the intervention and the assessments performed. sEMG evaluations were conducted at rest, during stretching, and during maximum voluntary isometric contraction (MVIC), along with the MAS scale and gait assessments, at the three time points of the study: before (T0), after the 4-week intensive therapy (T1), and at the end of follow-up (T2).

2.4 Therapeutic intervention

Ultrasound-guided dry needling

The subjects received a weekly ultrasound-guided dry needling session for 4 weeks in the medial gastrocnemius muscle using disposable stainless-steel needles (0.30mm x 0.40mm, Agupunt, Barcelona, Spain). The ultrasound-guided procedure was chosen to ensure a safe approach, guarantee accurate targeting of the structure, and observe local spasm responses in real time.⁽⁴¹⁾

To determine the location of the points to be treated, the diagnostic criteria of the DNHS® technique developed by Herrero et al. (2017) were applied, adapted to the characteristics of the study and the procedure for its application. To locate the specific point where the technique should be performed, two essential diagnostic criteria were followed: [1] select the tense band within the set of tense bands that presents the greatest tension; [2] select the nodular area within the tense band, if one exists.⁽⁴²⁾

For the performance of the technique, a variation of the recommended procedure was carried out to adapt it to the ultrasound-guided intervention:⁽⁴²⁾ [1] insert the needle above the selected point in the subcutaneous tissue; [2] place the ultrasound device next to it to guide the intervention; [3] continue inserting the needle until a significant reduction in muscle activity is achieved (usually after a local or global spasm response that can be confirmed by ultrasound during the procedure), known as neural release, since it involves structures such as the neuromuscular junction. To standardize the intervention, dry needling was performed at 1 Hz for 30 seconds, as tolerated by the patient.^(43–45) Possible adverse events related to the intervention were recorded.

Intensive therapy

In addition to ultrasound-guided dry needling interventions, all patients received physical therapy as intensively as possible, focused on gait recovery. The weekly therapy hours are shown in Table 1.

The intensive treatment consisted of an intensive therapy aimed at improving gait, following Clinical Practice Guidelines, with specific objectives agreed upon with the patient, and using high technology to allow the patient to practice the challenging task. This included body-weight-supported treadmill and overground training, gait training with step variability, the incorporation of virtual reality tools, functional electrical stimulation, and strength training.^(46–48)

3. Results

Four cases of people with chronic stroke and spasticity (MAS score between 1 and 3) were selected, who received the intervention of ultrasound-guided dry needling combined with intensive therapy to improve gait. No adverse effects related to the dry needling intervention or the intensive treatment were found.

The results obtained are shown in Table 2, and the electromyographic values are graphically represented in Figure 3.

Table 2. Evolution of cases prior to the start of the study (T0), after the first dry needling intervention (T1), at the end of the fourth and final dry needling intervention (T2), and the difference between the beginning and the end of the study ($\Delta T2-T0$).

Assessment		T0	T1	T2	ΔT2-T0	
Case 1	sEMG	Resting activity affected side (μV)	9,71±0,31	10,4±0,26	8,87±0,19	-0,84
		Resting activity less affected side (μV)	11,9±0,23			
		Stretching (%MVIC)	130±29,4	65,9±18,4	70±12,2	-60
	MAS	2	2	1+	-1	
	Gait Variables	TUG	13,66		7,58	-6,08*
		10MWT (comfortable)	0,56		1,09	0,53*
		10MWT (fast)	0,77		1,59	0,82*
		6MWT	333		503	170*
Case 2	sEMG	Resting activity affected side (μV)	8,02±0,43	8,3±0,47	6±0,2	-2,02
		Resting activity less affected side (μV)	8,27±0,2			
		Stretching (%MVIC)	504±131	333±111	130±75,8	-374
	MAS	1+	1+	1	-1	
	Gait Variables	TUG	16,67		13,07	-3,6*
		10MWT (comfortable)	0,81		0,84	0,03
		10MWT (fast)	0,94		1,05	0,11
		6MWT	283,3		333	49,7
Case 3	sEMG	Resting activity affected side (μV)	47,1±0,98	55,6±1,1	21,3±0,63	-25,8
		Resting activity less affected side (μV)	22,7±0,5			
		Stretching (%MVIC)	202±78,9	103±10,6	104±14,1	-98
	MAS	3	3	2	-1	
	Gait Variables	TUG	21,7		17,23	-4,47*
		10MWT (comfortable)	0,59		0,8	0,21*
		10MWT (fast)	0,82		1,18	0,36*
		6MWT	189,5		264,1	74,6*

Assessment		T0	T1	T2	ΔT2-T0	
Case 4	sEMG	Resting activity affected side (μV)	85,3±2,8	71,5±1,7	6,79±2,2	-78,51
		Resting activity less affected side (μV)	25,2±0,57			
		Stretching (%MVIC)	276±462	105±14,6	288±152	-59
	MAS	3	2	2	-1	
	Gait Variables	TUG	37,07		29,66	-7,41*
		10MWT (comfortable)	0,26		0,37	0,11
		10MWT (fast)	0,28		0,43	0,15*
		6MWT	98,9		109,8	10,9

Data: Mean $\pm$ standard deviation (SD). *MCID or MDC

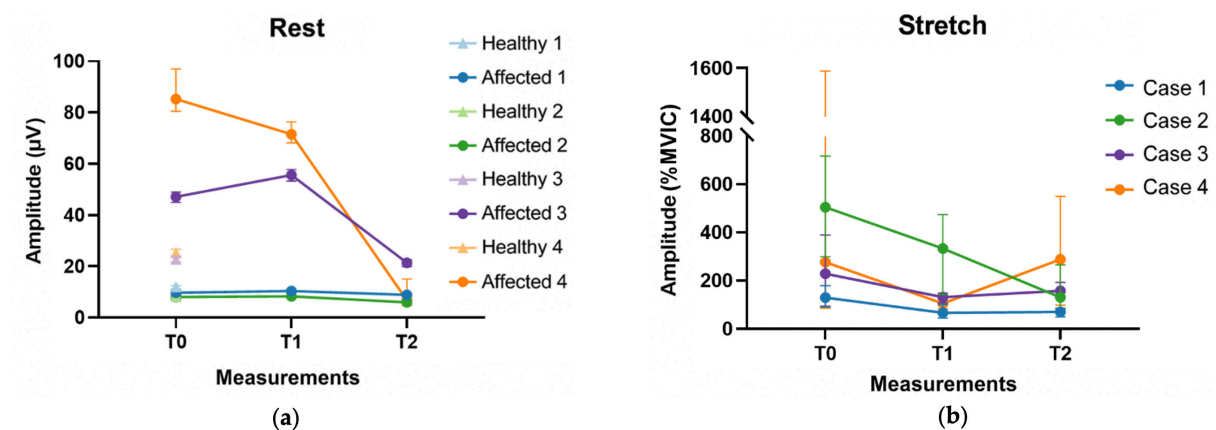


Figure 3. (a) Evolution of resting electromyographic activity in the cases throughout the study. (b) Evolution of electromyographic activity during manual stretches in the cases throughout the study.

Case 1: Phenotype with high electromyographic activity during stretching

This case shows a phenotype with spasticity (Table 2) and high electromyographic activity during manual stretches. The average electromyographic activity of the 10 manual stretches decreased from 130 $\pm$ 29.4% MVIC to 70 $\pm$ 12.2% MVIC at the end of the study (Figure 2). Similarly, the MAS score decreased from 2 to 1+ by the end of the study.

The initial resting activity on the affected side (9.71 $\pm$ 0.31 μV) was similar to that of the less affected side (11.9 $\pm$ 0.23 μV), with no spastic dystonia present. The activity on the affected side at the end of the intervention remained low (8.87 $\pm$ 0.19 μV) compared to the resting values of the same muscle in healthy subjects (3.14 $\pm$ 1.61 μV to 13.4 $\pm$ 6.3 μV).^(49,50) Improvements were found in all gait variables, with a reduction in the TUG scale from 13.66 seconds to 7.58 seconds, exceeding the MDC.

The walking speed measured with the 10MWT improved from 0.56 m/s to 1.09 m/s for comfortable speed and from 0.77 m/s to 1.59 m/s for fast speed, exceeding the MCID.

The distance covered in the 6MWT increased from 333 m to 503 m, exceeding the MCID.

Case 2: Phenotype with high stretch-induced activity and high variability

In this case, a phenotype is shown with spasticity and high variability, as defined by expert criteria as a standard deviation of $\pm 100\%$ MVIC relative to the mean during the 10 manual stretches. (Table 2) The average electromyographic activity of the 10 stretches decreased from $504 \pm 131\%$ MVIC to a value of $130 \pm 75.8\%$ MVIC at the end of the study (Figure 2), and the score on the MAS scale decreased from 1+ to 1 at the end of the study.

The initial resting activity on the affected side ($8.02 \pm 0.43 \mu\text{V}$) was similar to that of the less affected side ($8.27 \pm 0.2 \mu\text{V}$), with no spastic dystonia present. The activity on the affected side at the end of the intervention remained low ($6 \pm 0.2 \mu\text{V}$).

Improvements were found in all gait variables, with a reduction in the TUG scale from 16.67 seconds to 13.07 seconds, exceeding the MDC.

Walking speed measured with the 10MWT improved from 0.81 m/s to 0.84 m/s for comfortable speed, and from 0.94 m/s to 1.05 m/s for fast speed. The distance covered in the 6MWT increased from 283.3 m to 333 m.

Case 3: Phenotype with high resting activity and high stretch-induced activity

In this case, a phenotype is shown with the presence of spasticity accompanied by spastic dystonia (Table 2), with high average electromyographic activity during the 10 manual stretches, which decreased from $202 \pm 78.9\%$ MVIC to $104 \pm 14.1\%$ MVIC by the end of the study, and a score on the MAS scale that decreased from 3 to 2 at the conclusion of the study. The initial resting activity on the affected side ($47.1 \pm 0.98 \mu\text{V}$) was very different from that of the less affected side ($22.7 \pm 0.5 \mu\text{V}$), which, being higher than $\mu + 2 \cdot \sigma$ of the contralateral side, could confirm the presence of spastic dystonia at the start of the study (Figure 4). This resting hyperactivity decreased to $21.3 \pm 0.63 \mu\text{V}$ by the end of the intervention. Improvements were observed in all gait variables, with a reduction in the TUG scale from 21.7 seconds to 17.23 seconds, exceeding the MDC.

The walking speed measured with the 10MWT improved from 0.59 m/s to 0.8 m/s for comfortable speed and from 0.82 m/s to 1.18 m/s for fast speed, exceeding the MCID. The distance covered in the 6MWT test increased from 189.5 m to 264.1 m, exceeding the MCID.

Case 4: Phenotype with high resting activity, high stretch-induced activity and high variability

In this case, a phenotype is shown with the presence of spasticity accompanied by high variability and spastic dystonia (Table 2), with a high mean EMG activity during the 10 manual stretches, which increased from $276 \pm 462\%$ MVIC to $288 \pm 152\%$ MVIC at the end of the study (Figure 5), and a MAS scale score that decreased from 3 to 2 by the end of the study.

The initial resting activity of the affected side ($85.3 \pm 2.8 \mu\text{V}$) was very different from that of the less affected side ($25.2 \pm 0.57 \mu\text{V}$), and the presence of spastic dystonia at the beginning of the study could be affirmed. (Figure 5) This resting hyperactivity decreased to $6.79 \pm 2.2 \mu\text{V}$ by the end of the intervention.

Improvements were observed in all gait variables, with a reduction in the TUG score from 37.07 to 29.66 seconds, exceeding the MDC.

The walking speed measured with the 10MWT improved from 0.26 m/s to 0.37 m/s for comfortable speed and from 0.28 m/s to 0.43 m/s for fast speed, exceeding the MCID in fast speed.

The distance covered in the 6MWT test increased from 98.9m to 109.8m.

4. Discussion

Through the series of cases presented in this publication, it has been possible to show how sEMG enables clear identification of different patient phenotypes, objectively differentiating dystonia from spasticity, an aspect that cannot be achieved with clinical scales commonly used in practice, such as the MAS. Although the results of this study show an improvement in the MAS score in all patients throughout treatment, this improvement did not correspond with electromyographic improvement during manual stretching, as a reduction in the MAS score was not always accompanied by a decrease in electromyographic activity, and vice versa. These results are in line with the study by Puce et al., where they also found no correlation between the clinical spasticity scale and the instrumental one.⁽¹⁴⁾ This suggests that both tests are not measuring the same thing; electromyography provides quantitative data on muscle activity, while the MAS scale assesses at what point in the range of motion the stretch reflex appears and how easily it can be overcome by the examiner. Therefore, we consider that both tests are not mutually exclusive but complementary, adding value to the evaluation of muscle tone.

Regarding surface electromyography measurements, absolute values were used to assess spastic dystonia (sEMG activity at rest), whereas for spasticity (sEMG activity during manual stretching), normalized data relative to MVIC were used. Current scientific evidence recommends quantifying resting activity in absolute values (μV), so that it can be identified whether there is some EMG activity at rest beyond the 'noise floor'. On the other hand, in the case of measuring dynamic muscle contraction, both passive and active, it is recommended to express the data as a percentage of MVIC.^(51,52) It is also important to consider that using the absolute resting value provides speed, simplicity, and ease of interpretation, as it is generally displayed directly on the EMG device without the need for post-analysis normalization. Conversely, for measurements during manual stretching, it is recommended to normalize the results because the healthy side cannot be used for comparison, as the myotatic reflex cannot be manually elicited there. Additionally, normalizing the data with MVIC helps reduce evaluator influence and facilitates comparison between subjects.

Another clinically relevant aspect is the possibility of determining, through the use of sEMG, whether a patient has spastic dystonia. In previous studies, a high prevalence of spastic dystonia was found in patients with upper motor neuron injury, ranging from 26% to 75% of patients included^(13,14), with increased resting muscle activity from 4.25 μV to 33 μV , depending on the muscle examined^(13,14,25). In our case series, we found much higher resting activity levels, even in the muscle on the less-affected side. If the criterion originally proposed by the Marinelli group had been followed⁽¹³⁻¹⁵⁾, our 4 cases would have been classified as having spastic dystonia. For this reason, we believe that the criterion for determining whether spastic dystonia

exists should not be the presence of zero basal muscle activity, but rather a cutoff point should be established, which currently does not exist, where values above it indicate the presence of spastic dystonia, or that there is a difference between the healthy and affected side of at least $\mu + 2 \cdot \sigma$,^(26,28) provided that both sides are not affected.

Based on this proposal for determining the presence of spastic dystonia, only cases 3 and 4 would indicate it. In these two cases, dry needling appears to improve the abnormal resting tone of this phenotype, although part of this improvement may be explained by the higher baseline muscle activity, a phenomenon known as regression to the mean, which is characteristic of many interventions. However, regarding spasticity itself, it was observed that electromyographic variability during manual stretching, as measured by the standard deviation across repetitions, could influence the magnitude of the improvement. In this sense, case 4, with high variability between repetitions, did not show an improvement in spasticity, whereas patient 3, with lower variability, did. A possible explanation for the immediate improvement in case 4 following dry needling, which later shows increased spasticity over the course of 4 sessions, is the presence of non-neural stiffness, which is often seen due to the sustained contraction produced by spastic dystonia. This hypothesis is based on the fact that dry needling achieves immediate improvements by directly reducing muscle activity through denervation of motor endplates and associated neuromodulation. However, the fact that a patient receives intensive treatment could explain the improvement in structural stiffness, which in turn may allow a myotatic reflex to be triggered that was previously not possible due to the non-neural limitation, leading to this seemingly abnormal or increased pattern at 4 weeks of treatment. Another hypothesis is that the older age of patient 4 and the lower dose of therapy received compared to the other cases could explain a smaller reduction in initial non-neural stiffness and, consequently, have influenced the recurrence of spasticity.

On the other hand, despite the final improvement in case 3 regarding their spastic dystonia, it is noteworthy that there was no immediate improvement after the first dry needling session, unlike in the other cases. This cannot be explained by the mechanisms of action of dry needling, which produce a mechanical denervation of the motor endplate⁽⁵³⁾, so it is necessary to analyze more cases with this phenotype to draw conclusions. However, the results obtained in this patient after 4 weeks showed improvement in tone, approaching that of the less affected side, indicating a recommendation to perform at least 4 weekly dry needling sessions, especially in patients who exhibit a spastic dystonia phenotype with little variability upon stretching.

Regarding the effect on muscles with spastic dystonia, interventions such as botulinum toxin injections, which have a chemical effect rather than the mechanical effect of dry needling, showed beneficial effects at 4 weeks after administration, although not as pronounced as in our study.⁽²⁵⁾ Possible hypotheses could be that our initial values were higher and allowed for a greater reduction in activity after the intervention; that dry needling produces immediate and cumulative effects compared to the gradual action of botulinum toxin;⁽¹⁸⁾ or that although botulinum toxin induces changes at the central level,⁽⁵⁴⁾ it may not reach the broad supraspinal modulation that dry needling provides,⁽⁵⁵⁾ which could help to curb the abnormal efferent signal that sustains the contractions of spastic dystonia.

Although the effect of dry needling on spasticity is clearly studied, reducing spinal hyperreflexia by decreasing the H-reflex, improving the central nervous system's ability to modulate reflex responses,^(56,57) normalizing the spontaneous activity of dysfunctional motor units, and possibly increasing cortical excitability thereby generating secondary top-down mechanisms,^(55,58) this is the first study to evaluate the application of dry needling in subjects with spastic dystonia to date. The possible mechanism underlying the clinical improvement from dry needling in spastic dystonia could be the modulation of the abnormal supraspinal efferent conduction observed in these patients.

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The effects of dry needling on gait are clear when combined with motor training⁽⁶⁰⁾; however, dry needling alone has been reported to yield mixed results.^(61,62) According to a published meta-analysis, there is a clear recommendation to combine dry needling treatment with rehabilitative therapy in stroke patients.⁽²⁰⁾ Intensive therapy may be the perfect complement, both for managing muscle tone and improving walking ability, as it allows the patient to take full advantage of the window provided by dry needling to perform exercises based on motor learning principles (intense practice, goal-specific, and focused on error correction) that enhance functional recovery.⁽⁶³⁾

Our study could be an example of this, in which the subjects achieved improvements that comfortably exceeded the MCID in most walking tests. In this regard, the different international Stroke Clinical Practice Guidelines set a goal of 3 hours of direct daily therapy, 5 days a week, adjustable according to patient tolerance^(46,47,64), with minimum recommendations for specific gait improvement of 20 total hours of therapy over a period of 4 to 6 weeks.⁽⁴⁸⁾

Furthermore, the guidelines themselves consider that any patient who has suffered a stroke has the potential to benefit from rehabilitation, regardless of the duration since the event,⁽⁴⁷⁾ in line with what was observed in the study conducted by Ward et al. 2021, in which they propose that patient improvement is proportional to the intensity of therapy, beyond the time since the stroke, and that a greater amount and intensity of therapy could compensate for a delayed start of rehabilitation.⁽⁶⁵⁾ This aligns with our results, which show improvement in patients with chronic stroke of up to 9 years post-event, as long as the intensity, dose, and scheduling of therapy are properly adjusted.⁽⁶⁵⁻⁶⁷⁾

The study presents several limitations that hinder precise phenotypic characterization, including the absence of a control group and a limited sample size with only one case per phenotype, resulting in high heterogeneity not only in outcomes but also in demographic variables. Additionally, the variability in total therapy time, which ranged from 6 to 25 hours per week due to treatment customization for each subject, is another factor to consider. On the other hand, the decision to use absolute values to assess muscle tone at rest and compare it with the healthy limb, but to normalize the results of the stretch measurement (since comparison with the contralateral limb is not possible), is an aspect to consider for interpreting the results. Finally, the lack of follow-up after the intervention prevents establishing the duration of the observed effects.

All of these limitations constitute important factors to consider for future lines of research, which should include conducting randomized double-blind clinical trials, where the effect of

combining dry needling and intensive therapy is compared to intensive therapy alone, establishing cutoff points for categorizing individuals with spastic dystonia, as well as identifying predictors of good and poor responders according to phenotype, including variables not covered in this study, such as structural and textural muscle analysis through ultrasound. Finally, it would be advisable to explore other invasive physiotherapy techniques that have so far been underutilized in neurological patients, such as percutaneous electrical stimulation applied to the muscle or the nerve.

5. Conclusion

A theoretical-practical framework is proposed for assessing muscle tone with sEMG, describing different clinical phenotypes in patients with chronic stroke and supporting the combination of dry needling and intensive therapy to improve both spasticity and gait.

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Conceptualization, D.J.M. and P.H.; methodology, D.J.M. and P.H.; software, D.J.M.; validation, D.J.M. and J.S.A.; formal analysis, D.J.M.; investigation, D.J.M. and J.S.A.; resources, D.J.M.; data curation, D.J.M.; writing—original draft preparation, D.J.M. and P.H.; writing—review and editing, D.J.M. and P.H.; visualization, D.J.M.; supervision, P.H.; project administration, P.H. All authors have read and agreed to the published version of the manuscript.

Informed Consent of the Patient

The selected patients are part of the clinical trial 'Effects of Dry Needling on Electromyographic Activity and Ultrasonographic Characteristics in Post-Stroke Spasticity' with the registration code [clinicaltrials.gov](https://clinicaltrials.gov/ct2/show/study/NCT06017960) identifier NCT06017960, approved by the Aragón Ethics Committee with reference C.I. PI23/333. All procedures were carried out in accordance with the Declaration of Helsinki, and the patients signed an informed consent document before participating.

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Data Availability Statement

The raw data on which the study results are based may be obtained upon request to the corresponding author.

Conflicts of Interest

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