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Case Report

Third-Party Focal Cutaneous Pressure Suppresses Movements in Coexisting Painful Legs and Moving Toes Syndrome and Electrophysiologically Supported Propriospinal Myoclonus: A Quantitative Case Report

Jo Van Vaerenbergh*

SPINDLE, Route du Rawil 27, 1950 Sion, Switzerland.

***Corresponding Author:** Jo Van Vaerenbergh, SPINDLE, Route du Rawil 27, 1950 Sion, Switzerland. Email: jo.vanvaerenbergh@spindle.rehab

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Abstract

Background: Painful legs and moving toes syndrome (PLMT) is a rare sensorimotor disorder characterized by lower-limb pain and involuntary toe or foot movements. Propriospinal myoclonus (PSM) is an uncommon axial movement disorder for which clinical characterization should be supported by multichannel electromyography and electroencephalographic back-averaging. Quantified modulation of coexisting PLMT and electrophysiologically supported PSM by focal somatosensory input is scarcely documented.

Case presentation: A 57-year-old man presented with painful continuous movements of the right toes and foot, superimposed jerks involving the lower limb, and a perceived abdominal onset with spread toward the limb. Surface polymyography showed electromyographic bursts longer than 100 ms with synchronous agonist–antagonist co-contraction; jerk-locked EEG back-averaging identified no time-locked cortical correlate, and video-polysomnography characterized sleep-onset-related movements. Brain and spinal magnetic resonance imaging, nerve-conduction studies, routine electromyography, and dopamine-transporter imaging did not identify an explanatory lesion or parkinsonian degeneration. Pramipexole produced no meaningful benefit. Several cutaneous sites were tested, but only direct manual pressure on the skin over the right T7–T8 region, when continuously applied by another person, markedly suppressed the movements. Self-administered pressure and transcutaneous electrical nerve stimulation did not.

Quantitative findings: Two repeated 10-second two-axis accelerometry recordings were analysed in each condition. Without pressure, a shared 5.2–5.5 Hz component was present in both axes. With pressure, vector root-mean-square acceleration decreased by 72.0% and 59.4% across the two trials, while 1–20 Hz acceleration power decreased by 92.2% and 83.5%. Amplitude at the unstimulated dominant frequency decreased by 91.6%–99.2% across axes. Results remained comparable after excluding 0.5 seconds at each recording boundary.

Conclusion: Externally applied focal pressure reproducibly disrupted a coherent oscillatory movement pattern in coexisting PLMT and electrophysiologically supported PSM. The site specificity and failure of self-applied pressure suggest an interaction between localized afferent input and internal prediction of self-generated sensory consequences. The intervention response does not by itself establish whether the modulating mechanism is spinal, supraspinal, functional, or mixed.

Keywords: *Painful Legs and Moving Toes; Propriospinal Myoclonus; Functional Movement Disorder; Sensory Modulation; Cutaneous Pressure; Accelerometry; Case Report*

Introduction

Painful legs and moving toes syndrome (PLMT), first delineated by Spillane and colleagues, combines persistent or recurrent lower-limb pain with continuous or semicontinuous involuntary movements of the toes or feet [1]. The movements may include flexion, extension, abduction, adduction, fanning, and torsional components. In the largest published clinical series, neuropathy, radiculopathy, previous trauma, and other peripheral or spinal disorders were frequent associations, although many cases remained cryptogenic and treatment responses were generally limited [2,3].

Propriospinal myoclonus (PSM) is a descriptive movement phenotype involving repetitive axial jerks, typically affecting the trunk, hips, and proximal limbs. The classical model proposes a spinal generator with slow propagation through long propriospinal pathways [4,5]. This model has been substantially revised: clinical examination alone does not reliably distinguish presumed spinal PSM from functional axial jerks, and contemporary assessment emphasizes multichannel polymyography together with jerk-locked electroencephalographic back-averaging when available [6,7].

A previous report described coexisting PLMT and PSM-like manifestations, suggesting possible shared sensorimotor mechanisms [8]. Separately, abdominal self-rubbing and transcutaneous electrical nerve stimulation (TENS) have been reported to suppress PSM [9]. The present case adds a different and objectively quantified observation: marked suppression of a painful moving-toes/axial-jerk phenotype by focal pressure applied by another person, despite a lack of benefit from self-administered pressure or TENS.

Case Presentation

Patient information and symptom history

A 57-year-old right-handed man reported an approximately five-year progressive history of pain and involuntary movement affecting the right toes, foot, lower limb, and thigh. The movements had become constant during the preceding 36 months. He perceived some larger jerks as beginning in an abdominal region and spreading toward the right lower limb, occasionally toward an upper limb. The jerks occurred in prolonged bursts against a background of near-continuous distal movement and were sometimes preceded by a premonitory sensation. Larger jerks were commonly accompanied by a brief, sharp exacerbation of pain.

The patient described the baseline pain as dull, throbbing, and associated with hypersensitivity. Its intensity varied with physical activity. He did not report an urge to move the limb, relief through movement, or a clear circadian pattern. The movements increased slightly in the supine position and partly subsided when he lay on his left side. Attention or distraction could modestly reduce their amplitude. He also reported substantial fatigue.

Restless legs syndrome had previously been considered. Pramipexole, initiated at 0.088 mg once daily approximately three hours before bedtime and increased gradually to 0.54 mg, did not meaningfully improve the abnormal movements. At assessment, he was taking paracetamol 500 mg three times daily and tramadol 50 mg in the late afternoon.

Clinical findings

The patient was alert and oriented, with no evident cognitive or thought disorder during clinical interaction. Cranial-nerve examination was unremarkable. Upper-limb bulk, tone, strength, sensation, and arm-holding tests were normal. In the right foot and toes, involuntary movements varied in amplitude and apparent frequency. No focal lower-limb weakness was identified. Achilles tendon reflexes were absent bilaterally. Sensory examination demonstrated bilateral hypaesthesia over the dorsum of the feet, absent vibration perception at the malleoli (0/8), reduced thermal and pinprick sensation, and impaired graphesthesia over the foot dorsum. Romberg testing, stance, and gait were clinically normal. Body mass index was 37.7 kg/m². General cardiovascular, respiratory, and abdominal examination was unremarkable.

Diagnostic assessment

Dopamine-transporter imaging did not demonstrate a presynaptic dopaminergic deficit. Magnetic resonance imaging of the brain and spine, routine electroencephalography, nerve-conduction studies, and routine electromyography were reported as unremarkable. Multichannel surface polymyography showed electromyographic bursts longer than 100 ms with synchronous agonist–antagonist co-contraction. Jerk-locked EEG back-averaging found no time-locked cortical correlate, arguing against a demonstrated cortical generator. Video-polysomnography characterized movements related to sleep onset. Glucose dysmetabolism, vitamin deficiency, thyroid disease, renal or hepatic disease, and alcohol-related neuropathy were excluded as explanations for the distal sensory findings. There was no relevant family history. The combination of painful involuntary toe movements and distal sensory abnormalities supported PLMT. The axial phenomenology, longer-duration co-contraction bursts, absence of a time-locked cortical potential, and sleep-onset association supported a noncortical/propriospinal classification of the superimposed myoclonus [15,16]. Because the available summary does not specify a fixed rostrocaudal EMG propagation pattern or conduction velocity, the diagnosis is described as electrophysiologically supported PSM rather than proven solely by spinal propagation.

Clinical timeline

Time	Clinical event
≈5 years before assessment	Progressive onset of right lower-limb pain, toe/foot movements, and larger jerks.
≈36 months before assessment	Movements became constant.
Before specialist assessment	Restless legs syndrome and parkinsonism considered; pramipexole ineffective; dopamine-transporter imaging negative.
Diagnostic work-up	Brain and spinal MRI, routine EEG, nerve-conduction studies, and routine EMG were unremarkable. Polymyography showed >100-ms bursts with synchronous agonist–antagonist co-contraction; jerk-locked back-averaging found no cortical correlate; video-polysomnography characterized sleep-onset-related movements.
Experimental examination	Multiple cutaneous sites were tested. Only direct pressure on the skin over the right T7–T8 region, applied by a third party, suppressed movement; self-pressure and TENS did not.
Quantitative recording	A 10-second baseline/rest recording was followed by a 10-second stimulation recording during continuous third-party pressure; this paired sequence was repeated after a 10–30-second rest/washout interval.
Follow-up	No adverse effects were observed. No home intervention or longer-term follow-up was planned because the examination was intended to demonstrate immediate suppressibility.

Differential diagnosis

Alternative	Features considered	Interpretation in this case
Restless legs syndrome	Urge to move, worsening at rest/ evening, relief with movement.	No urge, relief, or clear circadian pattern; pramipexole ineffective.
Parkinsonian tremor	Rhythmic rest tremor with other parkinsonian signs.	No supporting examination findings; dopamine-transporter imaging negative.
Epilepsia partialis continua	Repetitive focal movement with epileptiform EEG and/or relevant cortical lesion.	EEG and brain MRI unremarkable; toe phenomenology not typical.
Akathisia	Generalized inner restlessness, often medication-associated.	No characteristic drug exposure or generalized motor restlessness.
Spinal segmental myoclonus	Jerks restricted to one or a few contiguous myotomes, often with a structural spinal lesion.	Perceived multisegmental spread and distal toe movements were not typical; spinal MRI unremarkable.
Functional movement disorder	Positive features may include variability, distractibility, entrainment, suppressibility, and altered attentional or agency effects.	Modest attentional modulation and the third-party-only pressure response make a functional contribution plausible. However, longer-duration co-contraction bursts and absence of a time-locked cortical potential supported a noncortical/propriospinal classification; these findings do not exclude mixed functional modulation.

Intervention and Quantitative Methods

Focal stimulation protocol

Motivated by a previous report in which abdominal self-rubbing suppressed PSM [9], multiple body locations and sensory inputs were explored during the examination. TENS did not visibly alter the movement. Pressure at the other tested locations was also ineffective. Only gentle manual pinching or pressure applied directly to the skin over the right T7–T8 region produced an immediate and marked reduction. Movement returned when pressure was removed, and the site-specific response could be reproduced. Notably, self-administered pressure at the same region was ineffective; suppression occurred only when another person applied the manoeuvre. The patient was aware of stimulus onset and offset, and no sham site or concealed-timing condition was used. During both analysed stimulation recordings, third-party pressure was maintained continuously for the complete 10-second interval. Pressure force and contact area were not instrumented. No adverse effects were observed.

Accelerometry and calibration

A two-axis TP-ACC20 accelerometer (TiePie Engineering, Sneek, the Netherlands; nominal range ±2 g) was secured to the proximal phalanx of the right hallux to capture the composite mechanical output of toe, foot, and lower-limb movement. Signals were acquired with a Handyscope HS4 system. Static calibration used the sensor response at −1 g, 0 g, and +1 g. The resulting sensitivities were 409.45 mV/g for the X axis and 410.80 mV/g for the Y axis; voltage values were converted to m/s² using 1 g = 9.81 m/s².

Signal processing and outcomes

Four raw calibrated recordings were analysed. Each paired sequence comprised a 10-second baseline/rest recording without stimulation followed by delivery of the focal pressure stimulus with simultaneous 10-second acquisition; the paired sequence was then repeated. A 10–30-second rest/washout interval separated trials. Third-party pressure was maintained throughout each stimulation recording. Each file contained 97,656 samples, corresponding to an effective sampling frequency of 9,765.625 Hz calculated from the recorded time vector. The X- and Y-axis signals were filtered using a fourth-order zero-phase Butterworth band-pass filter from 1 to 20 Hz.

Orientation-independent vector root-mean-square acceleration was calculated as $a_{RMS} = \sqrt{[(1/N)\ \Sigma(a_{x,i}^2 + a_{y,i}^2)]}$. Band-limited acceleration power was calculated as the summed mean-square filtered acceleration across both axes. Full-record discrete Fourier transforms provided a frequency resolution of approximately 0.1 Hz. The dominant frequency was defined as the largest spectral component between 1 and 20 Hz. Amplitude during pressure was also evaluated at the dominant frequency observed in the corresponding unstimulated trial.

Because these recordings represent repeated observations in one patient rather than independent experimental units, results were summarized descriptively without inferential p values. Robustness was assessed by repeating the RMS and power calculations after excluding the first and final 0.5 seconds of each signal and by examining non-overlapping one-second windows.

Results

Without pressure, both axes contained a pronounced shared quasi-periodic component: 5.2 Hz in trial 1 and 5.5 Hz in trial 2. Harmonic structure was visible, particularly near 10–11 Hz and, in trial 1, near 15–16 Hz. During pressure, the common dominant component was markedly attenuated, and the remaining acceleration was lower in amplitude and less spectrally concentrated (Figures 1 and 2).

Table 1. Quantitative movement suppression during continuously applied third-party focal pressure. RMS, root mean square. Power denotes summed two-axis band-limited mean-square acceleration.

Trial	Dominant frequency without pressure	Vector RMS without pressure	Vector RMS with pressure	RMS reduction	1–20 Hz power reduction
1	5.2 Hz	11.04 m/s²	3.09 m/s²	72.0%	92.2%
2	5.5 Hz	9.17 m/s²	3.73 m/s²	59.4%	83.5%

At the dominant frequency identified without pressure, acceleration amplitude decreased by 99.2% on the X axis and 92.1% on the Y axis in trial 1. Corresponding reductions in trial 2 were 91.6% and 94.2%. The effect remained stable after removal of the first and final 0.5 seconds: vector RMS decreased by 71.4% and 59.2%, and band-limited power by 91.8% and 83.4%, in trials 1 and 2, respectively. In the one-second-window analysis, every window recorded during pressure had lower vector RMS acceleration than every window recorded without pressure.

These results demonstrate substantial but incomplete suppression. Short residual bursts remained during pressure; the data therefore do not support the stronger formulation of complete movement cessation. Pressure did not directly affect the patient's pain, although he reported feeling more relaxed during the manoeuvre. Pain intensity was not measured objectively or quantified concurrently; the relaxation report should therefore be interpreted as a subjective observation rather than evidence of analgesic efficacy.

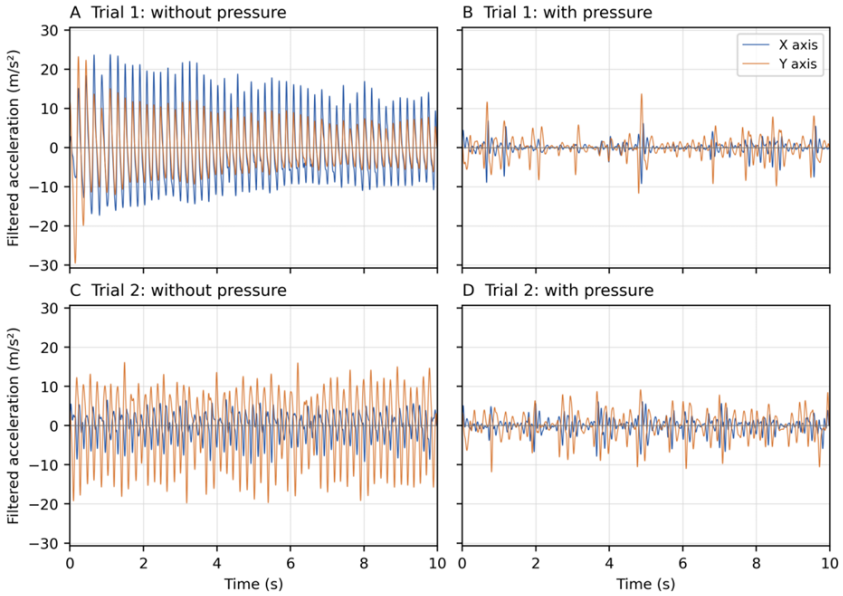

Figure 1. Band-pass-filtered two-axis acceleration during two repeated 10-second trials without focal pressure (A, C) and during continuous third-party focal pressure at the right T7–T8 region (B, D). All panels use the same vertical scale.

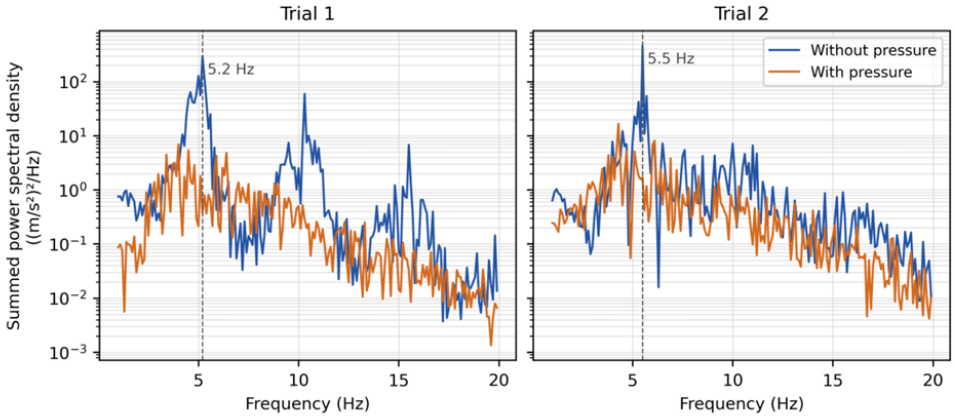

Figure 2. Summed X- and Y-axis power spectral density between 1 and 20 Hz. The unstimulated recordings showed shared dominant components at 5.2 Hz and 5.5 Hz with harmonic structure. Continuous focal pressure markedly attenuated these peaks and reduced band-limited power.

Discussion

This case documents marked, reproducible modulation of a rare combination of movement disorders. The painful, continuous multidirectional toe and foot movements fit the clinical spectrum of PLMT. The superimposed axial jerks were supported as noncortical/propriospinal myoclonus by their clinical distribution, surface-EMG bursts longer than 100 ms with synchronous agonist–antagonist co-contraction, absence of a time-locked cortical correlate on jerk-locked back-averaging, and association with sleep onset on video-polysomnography [15,16]. A previous report described coexisting PLMT and PSM manifestations [8], but the present observation adds quantitative two-axis accelerometry and identifies a condition-dependent response to externally delivered focal pressure.

The magnitude and spectral findings are mutually reinforcing. Third-party pressure reduced vector RMS acceleration by 59%–72% and band-limited power by 83%–92%. More importantly, the shared 5.2–5.5 Hz component present across both axes was largely abolished. The intervention therefore appeared to disrupt an organized oscillatory pattern rather than merely produce a proportional reduction in all movement. Replication across two trials, stability after boundary trimming, and complete separation of the one-second-window RMS ranges argue against a result caused solely by a single transient or filtering artefact.

Possible mechanisms

One possible explanation is afferent gating: cutaneous or deep-pressure input may alter excitability within spinal interneuronal networks or change supraspinal control of those networks. Such a mechanism is compatible with reports of sensory modulation of PSM and PLMT [9,10]. However, the striking dependence on who delivered the pressure is not readily explained by afferent intensity alone. Self-generated and externally generated touch differ in predictability, sensory attenuation, attention, and agency. The third-party-only response may therefore reflect interaction between somatosensory input and higher-order motor prediction rather than a purely segmental spinal reflex.

An internal forward model uses an efference copy of the motor command to predict the sensory consequences of self-generated action [12]. When the predicted and actual consequences agree, the resulting sensation is attenuated. The familiar inability to tickle oneself illustrates this process: an externally produced tactile stimulus generates stronger somatosensory activity and is perceived as more ticklish than a closely matched self-produced stimulus [13]. By analogy, self-applied pressure in this patient may have been predicted and attenuated, whereas externally applied pressure generated a larger sensory prediction error capable of perturbing the oscillatory motor state. This interpretation is biologically plausible but remains inferential; it should not be presented as proof of the mechanism.

The site specificity provides an important complementary observation. Several locations were tested during the same examination, but suppression occurred only over the right T7–T8 region. This argues against a nonspecific effect of being touched or observed, while still leaving open whether the effective variable was segmental afferent input, pressure geometry, attention directed to that body region, or an interaction among these factors. The manually applied force was not instrumented or matched between self- and externally applied conditions, and there was no randomized sham site, concealed onset, or expectation manipulation. The data justify a mechanistic hypothesis, not a definitive localization.

Diagnostic interpretation and functional features

The terminology surrounding PSM requires particular caution. Large electrophysiological series have shown that many clinically suspected cases demonstrate inconsistent polymyographic recruitment and a Bereitschaftspotential, supporting a functional motor mechanism [6,7]. Functional movement disorder is a positive neurological diagnosis involving altered motor control, attention, and agency; it does not require a specific psychiatric trait and is not equivalent to fabrication.

In this patient, surface polymyography demonstrated bursts longer than 100 ms with synchronous agonist–antagonist co-contraction, jerk-locked EEG back-averaging showed no time-locked cortical correlate, and video-polysomnography characterized sleep-onset-related movements. Together with the axial distribution and perceived spread of the jerks, these findings support PSM and argue against a demonstrated cortical generator [15,16]. They do not, in isolation, establish the anatomical generator or exclude functional modulation, particularly because a fixed multimuscle propagation sequence and conduction velocity were not available in the supplied summary.

Variability, modest attentional suppressibility, and dependence on externally administered pressure make a functional contribution plausible. Conversely, the persistent painful toe movements, distal sensory loss, absent Achilles reflexes, and coherent unstimulated frequency may indicate concomitant peripheral sensory pathology and maladaptive central reorganization. Organic and functional mechanisms are not mutually exclusive.

Clinical relevance

The observation may have practical value even before its mechanism is resolved. A safe, low-cost sensory manoeuvre that substantially reduces involuntary movement could support symptom management or help identify modifiable sensorimotor control variables. Nevertheless, this examination was designed only to demonstrate immediate movement suppressibility. No home intervention or longer-term follow-up was planned; consequently, the data do not establish durable therapeutic efficacy. The patient reported relaxation but no direct change in pain, and pain, function, sleep, and quality of life were not quantified. A future N-of-1 protocol should standardize pressure force and location, randomize active and sham conditions, conceal stimulus timing when feasible, acquire simultaneous multichannel surface EMG, and measure pain and patient-reported function alongside movement amplitude.

Strengths and limitations

Strengths include raw calibrated bivariate acceleration data, replication in two trials, consistent time- and frequency-domain effects, explicit sensitivity analyses, testing of several ineffective body locations, and supporting polymyographic, jerk-locked EEG, and video-polysomnographic characterization. No adverse effects were observed. Limitations include the single-patient design, only two short trials per condition, non-instrumented pressure, awareness of stimulus timing, absence of a randomized sham condition or blinding, lack of simultaneous objective pain ratings, and no home or longer-term follow-up. Accelerometry measured distal mechanical output but could not distinguish toe movement from movement transmitted through the foot or leg, nor could it identify a neural generator. The summarized polymyography did not provide a documented fixed rostrocaudal recruitment pattern or conduction velocity, so the precise anatomical generator remains uncertain. Results were reported descriptively without treating samples or one-second windows as independent observations.

Conclusion

Continuous focal pressure applied directly to the skin over the right T7–T8 region by another person markedly and reproducibly suppressed movement amplitude and the coherent 5.2–5.5 Hz oscillatory component in a patient with PLMT and electrophysiologically supported PSM. Pressure at other tested locations, self-pressure, and TENS were ineffective. The dependence on both anatomical site and external agency is compatible with an interaction between localized afferent input and predictive attenuation of self-generated sensation. The diagnostic studies support a noncortical/propriospinal classification of the myoclonus, but the mechanism by which pressure modulated the movement remains uncertain and may be spinal, supraspinal, functional, or mixed. Controlled replication is needed before therapeutic or causal conclusions can be drawn.

Patient perspective

“I am fully committed to collaborating with my healthcare team to manage my myoclonus. My goal is to participate actively in a non-invasive treatment approach. I am ready to follow recommended therapies, track my symptoms, and make the lifestyle modifications needed to help reduce the frequency and severity of my muscle spasms.”

Declarations

Informed consent

Written informed consent was obtained from the patient for publication of this case report and accompanying figures.

Ethics Approval

The case report was reviewed and approved by the Commission cantonale d'éthique de la recherche sur l'être humain (CER-VD), Lausanne, Switzerland.

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Conflicts of Interest

The author declares no conflicts of interest.

Author Contributions

Jo Van Vaerenbergh is the sole author and takes responsibility for the conception, clinical observation, data acquisition, interpretation, manuscript preparation, and approval of the final work.

Data Availability

De-identified acceleration data and the analysis protocol may be made available by the corresponding author on reasonable request, subject to patient consent and applicable data-protection requirements.

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References

1. Spillane JD, Nathan PW, Kelly RE, Marsden CD. Painful legs and moving toes. Brain. 1971;94(3):541–556. doi.org/10.1093/brain/94.3.541

2. Hassan A, Mateen FJ, Coon EA, Ahlskog JE. Painful legs and moving toes syndrome: a 76-patient case series. Arch Neurol. 2012;69(8):1032–1038. doi.org/10.1001/archneurol.2012.161

3. Alvarez MV, Driver-Dunckley EE, Caviness JN, Adler CH, Evidente VGH. Case series of painful legs and moving toes: clinical and electrophysiologic observations. Mov Disord. 2008;23(14):2062–2066. doi.org/10.1002/mds.22272

4. Termsarasab P, Thammongkolchai T, Frucht SJ. Spinal-generated movement disorders: a clinical review. J Clin Mov Disord. 2015;2:18. doi.org/10.1186/s40734-015-0028-1

5. van der Salm SMA, Erro R, Cordivari C, et al. Propriospinal myoclonus: clinical reappraisal and review of literature. Neurology. 2014;83(21):1862–1870. doi.org/10.1212/WNL.0000000000000982

6. Erro R, Bhatia KP, Edwards MJ, Farmer SF, Cordivari C. Clinical diagnosis of propriospinal myoclonus is unreliable: an electrophysiologic study. Mov Disord. 2013;28(13):1868–1873. doi.org/10.1002/mds.25627

7. Williams DR, Cowey M, Tuck K, Day B. Psychogenic propriospinal myoclonus. Mov Disord. 2008;23(9):1312–1313. doi.org/10.1002/mds.22072

8. Lopez R, Barateau L, Dauvilliers Y. Painful legs and moving toes et myoclonies propriospinales: une association fortuite? Médecine du Sommeil. 2022;19(2). doi.org/10.1016/j.msom.2022.01.188

9. Maltête D, Verdure P, Roze E, et al. TENS for the treatment of propriospinal myoclonus. Mov Disord. 2008;23(15):2256–2257. doi.org/10.1002/mds.22315

10. Vanegas-Arroyave N, Schneider SA, Bhatia KP, et al. Possible functional moving toes syndrome. Tremor Other Hyperkinet Mov. 2016;6:352. doi.org/10.5334/tohm.295

11. Stone J, Erro R. Functional (psychogenic) painful legs moving toes syndrome. Mov Disord. 2014;29(13):1701–1702. doi.org/10.1002/mds.26023

12. Wolpert DM, Ghahramani Z, Jordan MI. An internal model for sensorimotor integration. Science. 1995;269(5232):1880–1882. doi.org/10.1126/science.7569931

13. Blakemore SJ, Wolpert DM, Frith CD. Central cancellation of self-produced tickle sensation. Nat Neurosci. 1998;1(7):635–640. doi.org/10.1038/2870

14. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D; CARE Group. The CARE guidelines: consensus-based clinical case reporting guideline development. J Med Case Rep. 2013;7:223. doi.org/10.1186/1752-1947-7-223

15. Liu YL, Lu MK, Tsai CH. Propriospinal myoclonus: report of a case. Acta Neurol Taiwan. 2007;16(4):238–241. pubmed.ncbi.nlm.nih.gov/18220019/

16. Roze E, Bounolleau P, Ducreux D, et al. Propriospinal myoclonus revisited: clinical, neurophysiologic, and neuroradiologic findings. Neurology. 2009;72(15):1301–1309. doi.org/10.1212/WNL.0b013e3181a0fd50