

Ultrasound-Guided Cryoneurolysis for Peripherally Induced Dystonia After Non-neurological Surgery: A Case Series

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Current dystonia classifications define acquired dystonia through central nervous system (CNS)-centric etiologies without delineating how peripheral nerve injury following non-neurological surgery produces the dystonic phenotype. Dystonia is frequently confused with spasticity, contributing to misdiagnosis. Three female patients (ages 37–63) with refractory post-surgical presumed peripherally induced dystonia (PID) were evaluated. Ultrasound-guided diagnostic nerve blocks (DNBs) localized peripheral motor nerve generators, followed by targeted cryoneurolysis. All patients achieved NRS pain reduction of 5 to 6 points with sustained motor and functional improvement over 6 to 9 months. This is the first report of DNB-guided cryoneurolysis for PID. (A&A Practice. 2026;20:e02283.)

“Spasticity” has become a catch-all for involuntary muscle overactivity regardless of mechanism. Spasticity and dystonia are phenomenologically and pathophysiologically distinct, yet frequently confused.^{1,2} Spasticity is velocity-dependent hypertonia from stretch reflex hyperexcitability in upper motor neuron (UMN) lesions, whereas dystonia involves sustained involuntary contractions producing abnormal postures.¹ Even within UMN pathology, spastic dystonia has been called “the neglected phenomenon”; 74% of hypertonic wrist flexor muscles in stroke patients exhibited spastic dystonia rather than true spasticity.²

Beyond primary (genetic) and acquired central forms, dystonia has been reported following peripheral nerve injury, surgery, and immobilization, though these presentations lack formal classification.^{3–5} Peripherally driven dystonia following non-neurological surgery is systematically misclassified: patients receive antispasmodics, botulinum toxin, or neuromodulation, none targeting the peripheral nerve generator.^{1,2}

A systematic review of 713 peripherally induced movement disorder patients reported fixed dystonia as the most common phenotype, with nerve injury in 26% and complex regional pain syndrome (CRPS) in over one-third.⁵ Peripheral trauma is associated with dystonia risk (adjusted hazard ratio [HR] 3.12; 95% confidence interval [CI], 2.30 to 4.24).⁶

The 2025 Movement Disorder Society consensus dystonia classification retains “acquired” without specifying anatomical level or mechanism.³ Acquired etiologies are defined through CNS-centric frameworks (drugs, toxins, structural

CNS lesions, vascular diseases, immune disorders, infections),⁷ leaving no framework for phenotyping, staging, or treating peripheral nerve injury-induced dystonia. Computational modeling demonstrated that imbalanced Golgi tendon organ reflex sensitivity best explained CRPS-associated fixed flexion postures.⁸ The second hit hypothesis proposes peripheral nerve trauma can unmask dystonia in genetically susceptible individuals; sciatic nerve crush induced more dystonia-like movements in DYT-TOR1A and DYT-THAP1 mutant mice than wild-type controls.^{4,9} Peripherally induced dystonia (PID) likely represents a heterogeneous entity; no validated diagnostic criteria exist.^{5,10}

Diagnostic nerve blocks (DNBs) can identify peripheral nerve generators; a scoping review of 504 patients found DNBs consistently informed decision-making.^{11,12} Cryoneurolysis produces axonotmesis with Wallerian degeneration while preserving endoneurial architecture, permitting guided axonal regeneration over 3 to 6 months.¹³ To the authors’ knowledge, this is the first report of ultrasound-guided, DNB-directed cryoneurolysis for presumed PID after non-neurological surgery.

This article adheres to EQUATOR (CARE) guidelines and the Health Insurance Portability and Accountability Act (HIPAA) regulations. Written informed consent was obtained from all patients.

CASE DESCRIPTION

These patients were selected from consecutive patients presenting with refractory post-surgical motor dysfunction who had failed conventional therapies. All patients improved. A master summary is provided in the Table.

Case 1

A 52-year-old White woman presented with refractory left lower extremity dystonia and pain following foot reconstruction 16 years prior. She developed CRPS type I with neuropathic pain, allodynia, and fixed equinovarus deformity. Prior treatments including baclofen, tizanidine, diazepam, gabapentinoids, botulinum toxin, ketamine, spinal cord stimulation, bracing, and physical therapy all failed. Baseline numerical rating scale (NRS) 9/10. No UMN signs (Figure 1A).

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Table. Master Summary of Case Series

	Patient 1	Patient 2	Patient 3
Age/sex	52/F	37/F	63/F
Surgery	Left foot reconstruction	Revision left first rib resection	Right THA
Motor phenotype	CRPS-associated fixed equinovarus dystonia	Post-surgical ICN spasm, pectoral hypertonicity	Obturator-mediated adductor overactivity
Failed therapies	Baclofen, tizanidine, diazepam, gabapentinoids, BTX, IV ketamine, SCS, bracing, PT	Fentanyl, steroids, clonidine, nerve blocks, IV ketamine	NSAIDs, gabapentin, PF PT, BTX, pudendal decompression, steroid injections
DNB targets	Gastrocnemius, soleus, FDL, TP motor branches	T3–T6 ICN, LTN, TDN, MPN/LPN	Pudendal, PFCN, obturator (anterior + posterior)
DNB response	80%–90% equinovarus correction; plantigrade restored	Near-complete relief with pectoral blocks; 50%–100% with ICN blocks	>50% improvement in groin pain, adductor stiffness
Cryoneurolysis targets	Same as DNB targets	T3–T6 ICN, LTN, TDN, MPN/LPN, pectoral branches	Pudendal, PFCN, obturator (anterior + posterior)
NRS (pre → post)	9 → 3	8 → 3	8 → 2
Motor outcome	80%–90% sustained correction; plantigrade alignment	Reduced ICN spasm >80%; improved chest wall expansion and arm abduction	Reduced adductor overactivity; improved PF relaxation
Functional outcome	Improved gait, ambulation; reduced brace dependence	Off fentanyl; improved left arm ROM, sleep, activity	60%–70% improvement in posture, gait, sitting; reduced dyspareunia
Complications and motor safety	No complications. No transient motor effects	Transient neuritis (resolved). No scapular winging or shoulder weakness	Transient hip flexor spasm (resolved). No gait instability or adduction weakness
Repeat cryoneurolysis	Not required	LTN, TDN, MPN/LPN, T3–T6 ICN at 3 mo	Not required
Relief duration	>9 mo	>6 mo	>9 mo

Abbreviations: BTX, botulinum toxin; CRPS, complex regional pain syndrome; DNB, diagnostic nerve block; FDL, flexor digitorum longus; ICN, intercostal nerve; IV, intravenous; LPN, lateral pectoral nerve; LTN, long thoracic nerve; MPN, medial pectoral nerve; NRS, numerical rating scale; NSAIDs, nonsteroidal anti-inflammatory drugs; PF, pelvic floor; PFCN, posterior femoral cutaneous nerve; PT, physical therapy; ROM, range of motion; SCS, spinal cord stimulation; TDN, thoracodorsal nerve; THA, total hip arthroplasty; TP, tibialis posterior.

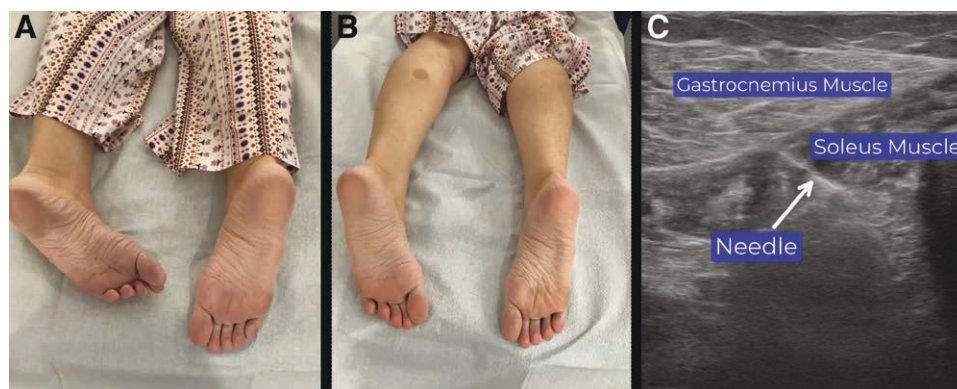


Figure 1. Patient 1: Complex regional pain syndrome-associated fixed equinovarus dystonia. Patient is prone in all panels. A, Preprocedure fixed equinovarus deformity. The left foot (left side of image) demonstrates fixed plantarflexion and inversion with the sole facing medially, consistent with dystonic posturing of the posterior compartment musculature. The right foot is in neutral position for comparison. B, Plantigrade alignment restored after ultrasound-guided diagnostic nerve block with electrical stimulation of tibial nerve motor branches. Note corrected left foot position with resolution of inverted tone and restored physiologic alignment comparable to the unaffected right foot. C, Ultrasound image showing cryoprobe needle (arrow) positioned between gastrocnemius (superficial) and soleus (deep) muscles, targeting an intramuscular motor branch. Each motor branch (gastrocnemius, soleus, flexor digitorum longus, tibialis posterior) was targeted independently in separate passes (flexor digitorum longus and tibialis posterior not shown).

Ultrasound-guided DNBs targeting intramuscular tibial nerve branches to gastrocnemius, soleus, flexor digitorum longus, and tibialis posterior produced 80% to 90% immediate equinovarus correction with restored plantigrade alignment (Figure 1B).^{11,12}

With the patient prone, cryoneurolysis was performed using the Iovera nitrous oxide–based system (Pacira Biosciences Inc).¹³ Area sterilized with 70% isopropyl

alcohol. She received nitrous oxide/oxygen (50:50) for anxiolysis. Under ultrasound guidance (15–4 MHz, sterile sheath), 2 mL of 1% lidocaine was injected for skin wheal. The Iovera needle (Smart Tip 2190) was advanced via an 18-gauge angiocatheter to the same motor branches identified during the DNB. Cryoneurolysis was performed at –88 °C for four cycles of 106 seconds (1 second prewarming, 60 seconds cooling, 45 seconds post-cooling) (Figure 1C).

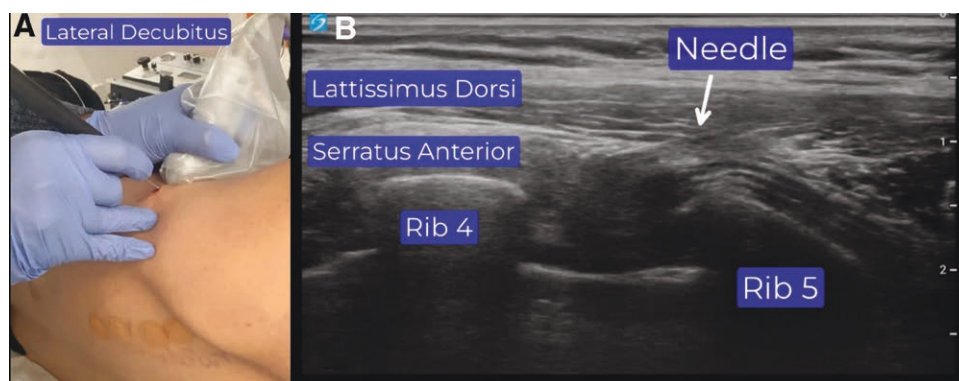


Figure 2. Patient 2: Long thoracic nerve cryoneurolysis. A, Patient in lateral decubitus positioning; the operator's right hand holds the Iovera cryoprobe (black handle) with the needle penetrating the skin inferior to the left scapula; the left hand holds a linear ultrasound probe (15-4 MHz) with a sterile sheath for real-time guidance. Four circular adhesive skin markers are visible along the paravertebral region, corresponding to the T3 to T6 intercostal nerve generator targets. B, Ultrasound image showing cryoprobe needle (arrow) positioned between rib 4 and rib 5, with latissimus dorsi (superficial) and serratus anterior (deep) visible. The long thoracic nerve courses along the superficial surface of the serratus anterior at this level.

Outcomes. NRS 9 to 3. Sustained plantigrade correction with improved gait and reduced brace dependence. No complications. Relief exceeded 9 months. No repeat cryoneurolysis was required during follow-up.

Case 2

A 37-year-old White woman with post-traumatic stress disorder presented with refractory left chest wall neuropathic pain, intercostal spasm, pectoral hypertonicity, and decreased left upper extremity range of motion (ROM) following left first rib resection with nerve grafting for neurogenic thoracic outlet syndrome and subsequent rib reintervention 3 years before, complicated by hematoma and lymphatic leak requiring three reoperations. Previous treatments included transdermal fentanyl, steroids, clonidine, serial nerve blocks, and intravenous ketamine. Baseline NRS 8/10. No UMN signs.

Ultrasound-guided DNB of left T3 to T6 intercostal, long thoracic, thoracodorsal, and medial/lateral pectoral nerves confirmed focal peripheral generators.^{11,12} Cryoneurolysis targeted the same motor branches using the technique in case 1¹³ (Figure 2).

Outcomes. NRS 8 to 3. Fentanyl discontinued. Improved ROM, chest wall expansion, and activity tolerance. No scapular winging or shoulder weakness observed following long thoracic nerve cryoneurolysis. Relief exceeded 6 months. Repeat cryoneurolysis was performed approximately 3 months after the initial treatment, consistent with expected axonal regeneration.¹³

Case 3

A 63-year-old white woman presented with refractory right groin pain, pelvic floor hypertonicity, vulvar dysesthesia, dyspareunia, and severe sitting intolerance following a sacral fall and right total hip arthroplasty (THA) 4 years before. Treatments including nonsteroidal anti-inflammatory drugs (NSAIDs), gabapentin, pelvic floor physical therapy, botulinum toxin, pudendal decompression, and steroid injections all failed. Severe gluteus medius/minimus wasting was consistent with superior gluteal nerve injury.¹⁴ Baseline NRS 8/10. No UMN signs.

Pudendal and posterior femoral cutaneous nerve (PFCN) DNBs confirmed focal generators; cryoneurolysis targeted the same nerves, yielding partial relief. Obturator nerve DNB (anterior and posterior divisions) produced greater than 50% improvement, redirecting treatment. Obturator cryoneurolysis at -88°C (2–3 cycles) yielded 60% to 70% functional improvement.^{11–13}

Outcomes. NRS 8 to 2. Improved pelvic floor relaxation, posture, gait, sitting tolerance, and dyspareunia. Transient hip flexor spasm resolved. No gait instability or adduction weakness observed following obturator nerve cryoneurolysis. Relief exceeded 9 months. No repeat cryoneurolysis was required during follow-up.

DISCUSSION

These cases illustrate that DNB-guided cryoneurolysis can achieve durable functional improvement in refractory post-surgical presumed dystonia. All three patients achieved NRS improvement of 5 to 6 points alongside motor correction.^{12,13}

Patient 1 is consistent with CRPS-associated fixed dystonia possibly driven by aberrant Golgi tendon organ (GTO) force feedback rather than stretch reflex hyperexcitability, as described in computational modeling.⁸ Cryoneurolysis produced sustained 80% to 90% equinovarus correction, improved gait, and NRS reduction from 9 to 3 over 9 months.¹³

Patient 2 illustrates post-surgical presumed PID where sequential insults (rib resection, nerve graft, hematoma, lymphatic leak, three reoperations) may be analogous to the gene–environment interaction in the second hit hypothesis, though genetic susceptibility was not assessed.^{4,9} Pectoral hypertonicity produced sustained involuntary contraction at rest, not velocity-dependent resistance, meeting dystonia criteria.^{1,2} Cryoneurolysis restored arm abduction ROM, reduced intercostal spasm by greater than 80%, allowed fentanyl discontinuation, and achieved NRS reduction from 8 to 3 beyond 6 months.¹³

Patient 3 is consistent with how peripheral nerve injury may initiate aberrant motor compensation beyond the original biomechanical deficit. Superior gluteal nerve injury produced gluteus medius/minimus wasting,¹⁴ disrupting

proprioceptive feedback and triggering compensation that propagated to distinct territories.^{5,8} DNB identification of the obturator nerve as dominant generator redirected treatment, confirming spread beyond index injury territory.^{11,12} Cryoneurolysis achieved NRS reduction from 8 to 2 with 60% to 70% functional improvement over 9 months.

Cryoneurolysis of functionally important motor nerves (long thoracic, obturator) caused no clinically significant motor deficits, consistent with selective reduction of pathologic tone.

Improvement was sustained 6 to 9 months, compatible with axonal regeneration timelines.¹³ Current therapies (antispasmodics, botulinum toxin) target spinal reflex circuits or the neuromuscular junction, not the peripheral nerve generator. In UMN-mediated motor dysfunction, the causative central lesion persists; cryoneurolysis requires repetition as axons regenerate.¹³ In PID, cryoneurolysis targets the pathologic nerve directly, inducing Wallerian degeneration and guided regeneration through preserved endoneurial architecture, reflecting potentially neuroregenerative rather than purely palliative intent.^{13,15} Whether early intervention may prevent central reorganization consolidating the dystonic phenotype remains unresolved.^{4,5,8}

Limitations

This retrospective three-patient series lacks a control group. Placebo-controlled blocks were not performed; territory-dependent motor corrections argue against a placebo effect, though not excluded. Electromyography/nerve conduction studies (EMG/NCS) were not performed, as the workup was framed within a spasticity paradigm. Dystonia is a clinical diagnosis; no diagnostic test with high accuracy exists for dystonia confirmation. EMG with co-contraction analysis would have strengthened diagnostic certainty and is recommended for future studies. No validated diagnostic criteria exist for PID; existing dystonia scales (Fahn-Marsden Dystonia Rating Scale, Unified Dystonia Rating Scale [UDRS], Global Dystonia Rating Scale [GDRS]) lack validation for peripheral forms, and spasticity tools (Modified Ashworth, Tardieu) measure velocity-dependent resistance irrelevant to PID.^{5,10} Outcome assessment relied on NRS, clinical examination, photographic documentation, and patient-reported improvements; standardized goniometric ROM or validated functional instruments were not systematically used. The treating clinician performed diagnosis, intervention, and outcome assessment, introducing potential observer bias. Retrospective reclassification by a coauthor from a different specialty, validated by a movement disorder specialist, provides partial mitigation.

CONCLUSIONS

In CNS-mediated acquired dystonia, the causative lesion is fixed; in PID, the pathologic driver is a peripheral nerve generator amenable to direct intervention. DNB-guided cryoneurolysis provided sustained improvement in three patients with refractory post-surgical presumed dystonia.^{12,13} Based on these observations and existing preclinical and epidemiological literature, PID may represent a progressive spectrum of aberrant motor compensation involving peripheral nerve dysfunction, maladaptive spinal plasticity, and cortical reorganization.^{4-6,8} These findings propose the

peripheral nerve generator as a therapeutic target. Future studies should incorporate EMG/NCS, placebo-controlled blocks, genetic screening for dystonia susceptibility alleles, inflammatory biomarkers, and standardized assessments to determine whether early intervention may alter PID natural history. Longer-term follow-up is needed to determine durability beyond the reported period. ■■

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