

Neuromodulation for Refractory Neuropathic Pain Following Critical Illness: A Narrative Review and Phenotype-Based Clinical Framework

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Citation: Suresh Srinivasan, Kiran Khurshid, Uzam Hussain, Joseph Pathakamuri, Mohanraj Palani Selvam. Neuromodulation for Refractory Neuropathic Pain Following Critical Illness: A Narrative Review and Phenotype-Based Clinical Framework. Clin Med and Med Res. 2026;4(1):1-23.

Received Date: 02 September, 2026; **Accepted Date:** 06 October, 2026; **Published Date:** 07 October, 2026

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ABSTRACT

Background: Persistent pain can complicate recovery after critical illness, but neuropathic pain remains incompletely characterized. Critical illness polyneuropathy, small-fiber injury, and focal peripheral nerve injuries may produce persistent pain alongside weakness, cognitive impairment, and psychological distress.

Objective: To critically synthesize the rationale and evidence for peripheral nerve stimulation (PNS), dorsal root ganglion stimulation (DRGS), and spinal cord stimulation (SCS) after critical illness and propose a framework for prospective evaluation.

Methods: This narrative review integrates critical-care survivorship literature, neurological diagnostic frameworks, selected neuromodulation trials and evidence syntheses, and procedural guidance. References were checked against accessible PubMed-indexed records and journal sources through October 4, 2026. Evidence concerning ICU survivors is distinguished from indirect evidence in analogous pain populations; no systematic-review or PRISMA-ScR compliance is claimed.

Findings: Post-ICU chronic-pain estimates encompass multiple mechanisms and cannot be equated with neuropathic-pain prevalence. Neuromodulation trials in peripheral nerve pain, postamputation pain, complex regional pain syndrome, and painful diabetic neuropathy provide a translational rationale, but do not establish efficacy after critical

illness. A proposed framework considers PNS for anatomically localized peripheral nerve pain, DRGS for selected focal regional pain, and SCS for selected bilateral or multisegmental peripheral neuropathic pain. Medical stabilization, neurological diagnosis, rehabilitation, psychological assessment, and procedural safety evaluation precede consideration of invasive treatment.

Conclusions: Neuromodulation warrants prospective investigation for persistent neuropathic pain after critical illness. Multidisciplinary follow-up, phenotype-specific enrollment, comparative studies, and patient-centered functional outcomes are needed before routine use for this indication can be supported.

Keywords: Critical illness; Post-intensive care syndrome; neuropathic pain; critical illness polyneuropathy; peripheral nerve stimulation; dorsal root ganglion stimulation; spinal cord stimulation.

INTRODUCTION

Recovery after critical illness extends beyond survival and hospital discharge. Survivors may experience persistent physical, cognitive, and psychological impairments within the spectrum of post-intensive care syndrome (PICS). Pain can interact with these impairments, restricting mobility, sleep, participation in rehabilitation, and return to valued activities.^[1] An effective recovery strategy therefore requires both recognition of pain and identification of its underlying mechanisms.

Critical illness-associated weakness includes critical illness polyneuropathy (CIP), critical illness myopathy (CIM), and overlapping polyneuromyopathy (CIPNM). These disorders share clinical features, particularly weakness and prolonged recovery, but differ in the tissues affected. CIP involves predominantly axonal injury to motor and sensory nerves; CIM involves muscle pathology and does not, by itself, establish a neuropathic pain diagnosis.^[2] Positioning-related neuropathy, plexopathy, procedural nerve injury, and small-fiber pathology may contribute additional, anatomically distinct pain phenotypes.

Persistent pain is common in ICU survivorship studies. A systematic review reported frequencies ranging from 28% to 77%, with substantial variation in populations, definitions, and assessment periods.^[3] A 2026 meta-analysis of 20 studies involving 4,726 patients estimated chronic-pain incidence of 47.0% at 3 months, 29.4% at 6 months, and 30.9% at 12 months.^[4] These time-specific pooled estimates arise from heterogeneous studies and should not be interpreted as a single cohort's recovery trajectory. They also describe chronic pain broadly, rather than neuropathic pain specifically.

A prospective multicenter cohort illustrates this distinction. Among 814 patients assessed 3 months after ICU admission, 388 (47.7%) reported significant persistent pain, defined as a numerical rating scale (NRS) score of at least 3. Within that pain subgroup, 34 (8.7%) had symptoms compatible with neuropathic pain on the ID-Pain questionnaire.^[5] Screening results do not confirm a somatosensory lesion, and the follow-up interval was measured from admission, rather than discharge. These findings support systematic assessment while cautioning against equating post-ICU pain with neuropathy.

For patients whose neuropathic pain remains disabling despite appropriate multimodal care, neuromodulation is a plausible treatment category. PNS targets peripheral nerves, DRGS targets selected dorsal root ganglia, and SCS delivers epidural stimulation that modifies spinal and broader pain-processing networks. Their clinical evidence bases differ by indication and technology. This review examines how that evidence may inform research after critical illness and develops an explicitly unvalidated framework linking pain phenotype, procedural readiness, and modality selection.

SCOPE AND APPROACH TO THE EVIDENCE

This article is a narrative review and clinical framework. It addresses adults with persistent neuropathic pain arising or worsening after critical illness, including CIP-associated pain and focal ICU-related nerve injury. The clinical questions concern diagnostic phenotyping, the relevance of PNS, DRGS, and SCS evidence from analogous populations, timing and safety, and prospective-study design.

The synthesis integrates critical-care pain epidemiology, neurological diagnosis and recovery, pivotal neuromodulation studies, relevant evidence syntheses, and procedural guidance. Accessible PubMed-indexed records, PubMed Central content, and journal webpages were consulted through October 4, 2026. Targeted queries combined critical illness, ICU or post-ICU, polyneuropathy, neuropathic pain, and the names of the stimulation modalities; study titles and identifiers were used to verify numerical and bibliographic claims. These checks were selective and were not a reproducible, exhaustive database search. Independent duplicate screening, formal risk-of-bias assessment, and de novo meta-analysis were not performed.

Evidence is considered in two streams: literature describing neurological injury or pain after critical illness, and neuromodulation literature in analogous pain populations. The verified literature does not establish the efficacy of PNS, DRGS, or SCS in a prospectively defined post-critical-illness neuropathic pain population. This statement describes the limits of this synthesis and does not establish that no relevant case reports or studies exist. Table 2 presents selected indirect evidence, rather than a complete inventory or a cross-modality ranking. The patient-selection criteria and algorithms are proposals for evaluation, not validated clinical recommendations.

Table 1: Neurological complications following critical illness and associated pain phenotypes

Complication / phenotype	Clinical pattern and pain features	Diagnostic considerations	Implication for pain management
Critical illness polyneuropathy (CIP)	Diffuse, usually symmetrical axonal sensorimotor neuropathy; weakness and sensory loss. Burning, dysesthetic or distal neuropathic pain may coexist.	Clinical trajectory and EMG/NCS support diagnosis. Do not infer neuropathic pain from weakness alone; document concordant sensory signs.	Rehabilitation and medical management remain central. SCS is an investigational analogy only for selected refractory peripheral neuropathic pain.

Complication / phenotype	Clinical pattern and pain features	Diagnostic considerations	Implication for pain management
Critical illness myopathy (CIM)	Predominantly muscle weakness, reduced endurance and possible muscle pain; sensation generally preserved.	Differentiate muscle pathology from neuropathy using examination and appropriate electrodiagnostic testing.	CIM alone is not a neuropathic-pain indication for PNS, DRGS or SCS. Address muscle recovery and nociceptive contributors.
Critical illness polyneuromyopathy (CIPNM)	Overlapping nerve and muscle dysfunction; mixed weakness, sensory impairment and possible mixed pain.	Identify the neural component and the contribution of pain versus weakness to disability.	Treat each component; an analgesic response does not establish recovery of the neuropathy or myopathy.
Focal mononeuropathy or plexopathy	Pain and sensory or motor deficits in a nerve or plexus distribution following positioning, compression, trauma or procedures.	Correlate exposure, pain map and examination; consider EMG/NCS and targeted imaging. Evaluate ongoing compression or a repairable lesion.	PNS may be a research option for an accessible, concordant nerve target. Plexus pain may require more complex target assessment.
Small-fiber pathology	Burning pain, altered thermal sensation, allodynia or autonomic symptoms may occur; large-fiber studies can be normal.	Specialist assessment; selective skin biopsy or other testing when clinically justified. Symptoms alone do not confirm the diagnosis.	Evidence of small-fiber injury provides a mechanistic rationale, not proof that any stimulation modality will help.
CRPS / causalgia after injury	Regional disproportionate pain with sensory, vasomotor, sudomotor/edema and motor/trophic features.	Apply Budapest criteria; exclude alternative explanations. CRPS is not synonymous with post-ICU neuropathy.	Established CRPS evidence may inform care when criteria are met; DRGS trial evidence is strongest for lower-extremity CRPS/causalgia.
Central neurological injury or mixed non-neuropathic pain	Stroke or spinal cord lesions may produce central neuropathic pain; joint, muscle and wound pain may coexist after ICU care.	Investigate lesion-specific findings and progressive deficits. Separate neuropathic, nociceptive and nociplastic contributions.	Do not automatically assign diffuse or central pain to SCS. Use a diagnosis-specific multidisciplinary plan.

Illustrative diagnostic distinctions, not prevalence estimates or validated treatment-selection rules. Sources: [2,7–9,12,16]. CIPNM, critical illness polyneuromyopathy; CRPS, complex regional pain syndrome; DRGS, dorsal root ganglion stimulation; EMG, electromyography; ICU, intensive care unit; NCS, nerve conduction studies; PNS, peripheral nerve stimulation; SCS, spinal cord stimulation.

Table 2: Selected evidence for PNS, DRGS and SCS in analogous neuropathic pain populations

Modality / study	Population and design	Reported outcome and time point	Main limitations	Relevance after critical illness
PNS Helm et al., 2021 [13]	Systematic review: 102 studies identified; 5 randomized trials and 4 case series included. Several peripheral pain indications.	Review authors estimated approximately two-thirds of patients with peripheral neuropathic pain achieved $\geq 50\%$ sustained pain relief; level II evidence under their framework.	Heterogeneous populations, technologies and follow-up; few higher-quality studies. Not a uniform pooled success rate.	Indirect rationale for focal nerve pain; no post-ICU response probability can be derived.
Percutaneous PNS Gilmore et al., 2019 [14]	Small randomized placebo-controlled trial of chronic postamputation pain; 28 participants enrolled; primary comparison included 12 active and 14 control participants.	During weeks 1–4, $\geq 50\%$ average pain reduction: 7/12 (58%) with PNS versus 2/14 (14%) with placebo.	Small sample and short blinded comparison. Amputation biology differs from ICU-associated nerve injury.	Supports study of peripheral targeting; does not establish efficacy in CIP or positioning neuropathy.
Implantable PNS COMFORT, 12-month report [15]	Peripheral nerve pain at several anatomical sites; 89 initially randomized. Twelve-month treated cohort included crossover recipients.	At 12 months, $\geq 50\%$ pain reduction in 53/61 assessed participants (87%); mean pain reduction 69%.	Later follow-up lacked a continued randomized control; trial enrichment, attrition and device-specific findings.	Contemporary indirect evidence; cannot be generalized to temporary PNS or a post-ICU cohort.
DRGS vs conventional SCS ACCURATE [16]	Randomized comparative trial; 152 participants with lower-extremity CRPS or causalgia; follow-up through 12 months.	At 3 months, composite treatment success: 81.2% with DRGS versus 55.7% with conventional SCS; success included $\geq 50\%$ pain relief and neurological safety.	Specific diagnoses and comparator; does not compare all modern SCS waveforms.	Most relevant to a confirmed regional CRPS/causalgia phenotype, not diffuse CIP.
DRGS Deer et al., 2020 [17]	Systematic review of DRG neurostimulation across pain indications, including CRPS and focal neuropathic pain.	Supports benefit in selected indications; evidence strength varies substantially across diagnoses.	Observational evidence predominates outside the pivotal comparative indication.	Supports hypothesis generation for focal regional pain; no validated post-ICU selection rule.
10-kHz SCS + CMM SENZA-PDN [18]	Open-label randomized trial; 216 participants with refractory painful diabetic neuropathy.	At 3 months, $\geq 50\%$ pain relief without neurological worsening: 75/95 assessed with SCS + CMM (79%) versus 5/94 with CMM (5%).	Unblinded intervention; eligibility and trial selection; outcome denominators differ from total randomized.	Bilateral neuropathic limb pain offers an analogy; diabetes and CIP remain distinct conditions.
10-kHz SCS 24-month follow-up [19]	Implanted cohort from SENZA-PDN; 142 followed for 24 months, including 58 crossover recipients.	At 24 months, 90.1% achieved $\geq 50\%$ pain relief; mean pain reduction 79.9%.	Treated follow-up cohort after crossover; no preserved randomized comparison at this time point.	Durability signal in diabetic neuropathy; not evidence of nerve regeneration or CIP efficacy.

Modality / study	Population and design	Reported outcome and time point	Main limitations	Relevance after critical illness
SCS Awad et al., 2026 [20]	Meta-analysis of randomized studies in painful diabetic neuropathy.	Synthesis supports improvements in pain and quality of life compared with conventional medical management.	Different technologies and trial methods; overlap with the primary studies above.	Updates the indirect evidence base; does not supply independent post-ICU evidence.

All studies in this table provide indirect evidence for post-critical-illness neuropathic pain. Response percentages must not be ranked across studies; endpoints, denominators, comparators, technologies and follow-up differ. Reviews may include trials also listed separately, so sample sizes must not be added. CMM, conventional medical management; CIP, critical illness polyneuropathy; CRPS, complex regional pain syndrome; DRGS, dorsal root ganglion stimulation; PNS, peripheral nerve stimulation; SCS, spinal cord stimulation.

NEUROLOGICAL COMPLICATIONS AND PAIN MECHANISMS

Critical illness polyneuropathy and myopathy

CIP typically presents with diffuse, symmetrical weakness, reduced reflexes, and sensory abnormalities in the setting of severe illness. Axonal dysfunction reflects interacting inflammatory, microvascular, metabolic, and bioenergetic disturbances. CIM may coexist but predominantly affects muscle, with sensory responses often preserved. Clinical examination and appropriately selected electrodiagnostic studies help distinguish these conditions from alternative causes of weakness.^[2] A diagnosis of ICU-acquired weakness alone is insufficient to classify pain as neuropathic.

Recovery can continue for months and varies with the underlying neuromuscular diagnosis. A scoping review of 29 studies involving 788 patients reported full recovery in 70.3%, although outcome definitions, follow-up, and study quality were heterogeneous.^[6] This estimate cannot predict an individual patient's course or serve as a device-selection threshold. Serial assessment is needed to separate improving neurological impairment from persistent pain that independently limits function.

Small-fiber injury and focal peripheral nerve injury

Small-fiber pathology has been documented in critically ill patients and provides a possible substrate for persistent sensory symptoms.^[7] However, its contribution to post-ICU pain frequency and severity remains uncertain. Routine nerve conduction studies principally assess large fibers; normal results do not exclude small-fiber pathology. Specialist-directed investigation may be appropriate when symptoms, examination findings, and conventional testing are discordant.

Prolonged immobilization, compression, prone positioning, surgery, and vascular or other procedures can produce focal mononeuropathy or plexopathy. Peripheral nerve injuries have been described in survivors of severe COVID-19-associated acute respiratory distress syndrome exposed to prone positioning.^[8] Such injuries should be

differentiated from diffuse CIP because a focal, anatomically accessible nerve may offer a more discrete treatment target. Attribution requires consideration of the ICU course, symptom onset, examination, imaging or electrodiagnostic findings, and pre-existing neurological disease.

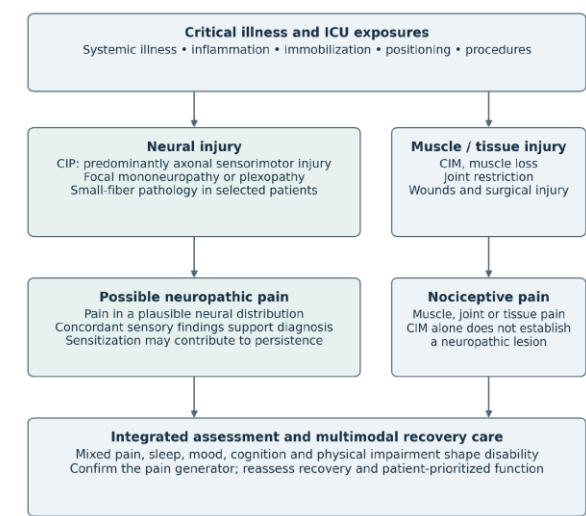
Mixed pain and altered pain processing

Persistent post-ICU pain may combine neuropathic, nociceptive, and nociplastic mechanisms. Muscle injury, joint restriction, pressure injury, surgical wounds, and deconditioning may generate pain without a somatosensory lesion. Psychological distress and disturbed sleep may increase pain interference regardless of its initiating mechanism. Peripheral sensitization and altered central processing are plausible contributors, but their presence and relative importance must not be assumed in every ICU survivor.

Figure 1 summarizes this conceptual continuum while preserving a separate muscle and musculoskeletal pathway. Table 1 distinguishes major neurological and neuromuscular complications, associated pain patterns, and diagnostic implications. The model depicts potential relationships; progression to chronic neuropathic pain is neither inevitable nor uniformly linear.

Figure 1 | Pathways to persistent pain after critical illness

Conceptual relationships; recovery and mixed mechanisms remain possible



CIP, critical illness polyneuropathy; CIM, critical illness myopathy; ICU, intensive care unit.

Figure 1: Pathophysiological continuum from critical illness to persistent pain.

Conceptual model distinguishing neural injury and neuropathic pain from muscle or musculoskeletal injury and nociceptive pain. Mixed mechanisms and recovery are possible at every stage. Proposed peripheral and central amplification mechanisms are not demonstrated in every ICU survivor, and neuromodulation is not shown as a treatment for isolated myopathy. Conceptual synthesis informed by [2,7–9]. CIP, critical illness polyneuropathy; CIM, critical illness myopathy; ICU, intensive care unit.

DIAGNOSTIC PHENOTYPING AND CONVENTIONAL MANAGEMENT

A neuropathic pain diagnosis requires a plausible lesion or disease of the somatosensory system and a compatible pain distribution. Diagnostic confidence increases when examination demonstrates concordant sensory abnormalities and testing confirms a relevant lesion.^[9] Burning, electric-shock pain, allodynia, and paresthesias are useful clues but do not independently establish the diagnosis. DN4 or other validated screening instruments can support case finding; they supplement, rather than replace, clinical assessment.^[10]

The assessment should document pre-ICU pain, neurological diagnoses, diabetes and other neuropathy risks, the critical illness and procedural exposures, and the trajectory of symptoms. Pain drawings, sensory mapping, strength, reflexes, gait, and region-specific examination help distinguish a single-nerve pattern from plexus, dermatomal, distal symmetric, or mixed pain. Electrodiagnostic studies and imaging should answer specific clinical questions. New or progressive weakness, myelopathic signs, rapidly evolving sensory loss, or bowel or bladder dysfunction requires prompt evaluation before an elective pain procedure.

Conventional management combines rehabilitation, education, psychological support, sleep optimization, and individualized pharmacotherapy. Evidence from general neuropathic pain populations supports consideration of gabapentinoids, serotonin–norepinephrine reuptake inhibitors, or tricyclic antidepressants, with selected topical treatments for localized peripheral pain.^[11] Renal and hepatic function, orthostatic symptoms, cognition, respiratory status, and concurrent sedating medications can constrain tolerability after critical illness. Medication choice and titration should account for these factors. Opioid escalation is not a prerequisite for neuromodulation evaluation, and medication reduction should be individualized.

Treatment adequacy is better described by the interventions attempted, duration, tolerated doses, adherence, adverse effects, and participation barriers than by a fixed count of failed medications. Rehabilitation goals should reflect the patient's baseline abilities and priorities. A patient whose main limitation is painless weakness requires a different strategy from one whose otherwise improving mobility is repeatedly interrupted by focal neuropathic pain.

Regional pain with disproportionate symptoms and autonomic or trophic features warrants assessment for complex regional pain syndrome (CRPS), using established diagnostic criteria.^[12] CRPS should not be inferred solely from severe pain after immobilization. Identifying an established pain diagnosis may inform management, but does not convert indirect evidence into evidence for CIP.

NEUROMODULATION: EVIDENCE FROM ANALOGOUS POPULATIONS

Peripheral nerve stimulation

PNS is anatomically attractive when pain maps to one or a small number of accessible peripheral nerves. A 2021 systematic review identified 102 studies, of which five randomized trials and four case series met its inclusion criteria. The authors classified evidence for refractory peripheral nerve injury as level II within their grading system and estimated that approximately two-thirds of patients with peripheral neuropathic pain achieved at least 50% sustained relief.^[13] This estimate reflects heterogeneous indications and should not be presented as a post-ICU response probability or a pooled rate applicable to every PNS system.

In a randomized placebo-controlled study of chronic postamputation pain, 7 of 12 participants receiving percutaneous PNS (58%) achieved at least 50% average pain reduction during weeks 1–4, compared with 2 of 14 controls (14%).^[14] This small trial supports investigation of targeted peripheral stimulation, but amputation-related pain differs from positioning neuropathy and diffuse CIP.

More recent implantable-system evidence includes the COMFORT study. In its 12-month report, 89 participants had been randomized initially, and 53 of 61 participants assessed after treatment, including crossover recipients, achieved at least 50% pain reduction (87%).^[15] This later outcome was a treated-cohort assessment after crossover, not a continued randomized comparison. The population included several anatomical pain regions, and trial selection and device-specific characteristics constrain generalization.

Temporary percutaneous treatment and permanently implanted PNS are distinct strategies. Lead configuration, target, stimulation delivery, duration, and external hardware requirements vary. A temporary approach could be studied for selected focal pain during ongoing recovery, but neither its superiority nor its optimal timing has been established after critical illness. Risks include infection, skin irritation, lead migration or fracture, retained fragments, and local nerve or vascular injury. Technical feasibility and the patient's ability to manage the system matter alongside anatomical fit.

Dorsal root ganglion stimulation

DRGS may provide a focused approach for selected regional neuropathic pain when coverage is difficult to achieve with a peripheral nerve target. In the ACCURATE randomized trial, 152 participants with lower-extremity CRPS or causalgia received DRGS or conventional SCS. At 3 months, treatment success was achieved in 81.2% of

the DRGS group and 55.7% of the SCS group; the trial also assessed outcomes through 12 months.^[16] Treatment success combined at least 50% pain relief with the study's neurological safety criterion. This comparison does not establish superiority over all contemporary SCS waveforms or efficacy in post-ICU neuropathy.

A systematic review supports DRGS in selected focal pain populations while identifying differences in evidence strength across diagnoses.^[17] Post-ICU applications remain extrapolations. A dermatomal distribution alone does not establish candidacy: radiculopathy, compressive pathology, and surgically correctable lesions must be evaluated. Pain can also cross dermatomes, and regional CRPS patterns may not correspond neatly to a single root. Selection therefore requires clinical diagnosis and technical assessment, not a rigid anatomical label. Neuraxial access introduces bleeding, infection, neurological, and lead-related risks.

Spinal cord stimulation

SCS offers a possible research strategy for selected bilateral distal or multisegmental peripheral neuropathic pain. The most relevant randomized analogy is painful diabetic neuropathy. In SENZA-PDN, 216 participants were randomized to conventional medical management (CMM) or 10-kHz SCS plus CMM. At 3 months, the composite endpoint of at least 50% pain relief without neurological worsening was achieved by 75 of 95 assessed participants receiving SCS plus CMM (79%), compared with 5 of 94 receiving CMM (5%).^[18]

The 24-month report followed 142 implanted participants, including crossover recipients. At that assessment, 90.1% achieved at least 50% pain relief, and mean pain reduction was 79.9%.^[19] These durability findings represent an implanted follow-up cohort, not a preserved randomized comparison at 24 months. A 2026 meta-analysis of randomized painful-diabetic-neuropathy studies further supports pain and quality-of-life benefits within that indication.^[20]

Diabetic neuropathy differs from CIP in etiology, neurological recovery, comorbidity, and disease trajectory. These studies do not demonstrate axonal regeneration or reversal of post-critical-illness neuropathy. SCS should not be assumed suitable for nonspecific widespread pain, isolated weakness, or central post-stroke pain merely because peripheral targeting is difficult. In post-ICU research, eligibility should require a confirmed, clinically coherent peripheral neuropathic phenotype and a justified spinal target.

Noninvasive approaches and limits of comparison

Peripheral magnetic stimulation and other noninvasive approaches may be investigated alongside rehabilitation. A 2024 review of 23 studies found short-term signals of benefit for chronic peripheral neuropathic pain, but evidence certainty was low or very low and sustained benefit remained uncertain.^[21] Neuromuscular electrical stimulation used to address weakness and nerve stimulation used to monitor neuromuscular blockade are distinct from analgesic PNS and should not be counted as post-ICU pain-treatment evidence.

Response percentages in [Table 2](#) cannot be compared as if derived from a common trial. Populations, controls, blinding, responder definitions, enrichment through stimulation trials, crossover, and follow-up denominators differ. Small samples, incomplete follow-up, and industry involvement require attention. The appropriate inference is that each modality has indication-specific evidence that supports a testable post-ICU hypothesis; the data do not establish a preferred modality for the post-critical-illness population as a whole.

PROPOSED PATIENT-SELECTION FRAMEWORK AND TIMING

The proposed framework begins with diagnostic confidence, the functional burden attributable to pain, and medical readiness. [Table 3](#) organizes these considerations. Neuromodulation is intended to modulate symptoms and support rehabilitation; improvement in muscle strength or neurological recovery should be measured separately and cannot be assumed.

PNS may be considered for prospective evaluation when a focal nerve lesion and pain territory are concordant and an accessible peripheral target exists. DRGS may be evaluated in selected focal regional or lower-extremity CRPS/causalgia phenotypes when the relevant indication and anatomy justify its use. SCS may be evaluated for selected bilateral distal or multisegmental peripheral neuropathic pain after an appropriate neurological workup. Overlapping patterns require multidisciplinary judgment; they do not necessarily require multiple devices. [Figure 3](#) presents these options as branches for specialist assessment, rather than automatic treatment assignments.

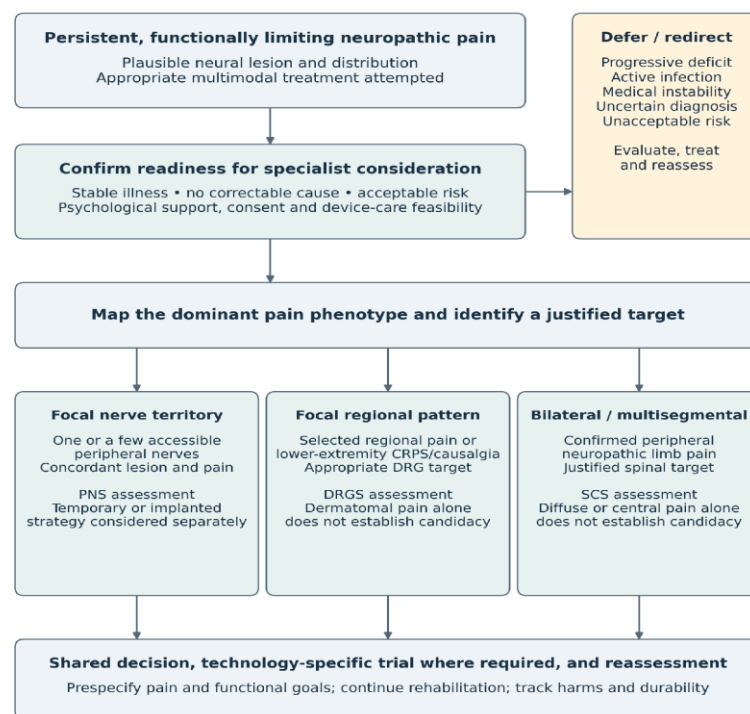
Pain duration of at least 3 months can define a chronic-pain study population, but does not establish readiness for implantation. The draft's 3–6-month post-ICU assessment window is retained only as a research hypothesis. Referral and treatment optimization may occur earlier, while implantation decisions depend on medical stability, the recovery trajectory, diagnostic clarity, and a tolerable conservative-care trial. No optimal post-ICU waiting interval is established by the evidence reviewed here.

Psychological assessment should identify expectations, coping, depression, post-traumatic stress symptoms, substance-use concerns, and practical barriers to device management. Such findings guide support and treatment planning; depression or a history of ICU-related distress should not automatically exclude a patient. Established SCS selection guidance supports evaluation of psychosocial factors and a multidimensional assessment of trial benefit.^[22] Its use after critical illness requires individualized judgment and should not be described as a validated post-ICU selection rule.

For technologies requiring a stimulation trial, prespecified goals should include pain reduction, improvement in a patient-prioritized activity, tolerability, and ability to manage the device. Trial response alone does not guarantee durable benefit. Temporary PNS treatment is also not necessarily equivalent to a screening trial for a permanent system. Device-specific indications, anatomical constraints, and jurisdictional requirements should be checked before treatment; the framework does not imply regulatory approval or payer coverage for post-critical-illness neuropathic pain.

Figure 3 | Phenotype-based neuromodulation selection

Unvalidated post-ICU algorithm; modality evidence is indirect for this population



Overlapping phenotypes require individualized judgment; the branches do not rank the modalities.

Figure 3: Proposed neuromodulation selection algorithm: PNS, DRGS and SCS.

Phenotype-specific branches identify options for specialist assessment, not automatic treatment recommendations. Overlapping patterns require individualized multidisciplinary judgment. Evidence from analogous populations [13–20] is indirect for post-critical-illness neuropathic pain; no modality has established comparative superiority in this population. A dermatomal distribution alone does not establish DRGS eligibility, and nonspecific widespread or central pain does not automatically establish SCS eligibility. CRPS, complex regional pain syndrome; DRGS, dorsal root ganglion stimulation; PNS, peripheral nerve stimulation; SCS, spinal cord stimulation.

Table 3: Proposed patient-selection criteria for neuromodulation after critical illness

Domain	Proposed assessment	Reason to defer or redirect
Neuropathic diagnosis	Probable or definite neuropathic pain with a plausible lesion, compatible distribution and concordant clinical findings; selective confirmatory testing.	Unexplained or progressive deficit; uncertain phenotype; predominantly muscle, joint or nonspecific widespread pain.
Pain-related burden	Persistent symptoms with meaningful interference in sleep, mobility, daily activities or rehabilitation; specify an index pain and patient-prioritized goal.	Minimal pain burden or disability primarily attributable to painless weakness or another untreated impairment.
Conventional care	Document appropriate, tolerable pharmacological, rehabilitation and psychological interventions, their duration, benefit, adverse effects and access barriers.	Incomplete assessment or avoidable barriers to appropriate care. Opioid escalation is not required before evaluation.
Recovery and timing	Underlying illness stabilized; serial neurological and functional assessment. A 3–6-month post-ICU review may be tested prospectively.	Ongoing acute systemic illness or a recovery trajectory that makes an elective implant premature; no validated waiting interval exists.
Procedural readiness	Assess infection, skin and wounds, nutrition, bleeding/thrombotic risks, organ function and overall procedural tolerance.	Active infection, unresolved medical instability or an unacceptable procedure-specific risk pending correction or further assessment.
Psychological and cognitive needs	Assess expectations, coping, mood, trauma symptoms, substance-use concerns, consent capacity and device-management ability; provide support.	An untreated condition that prevents informed participation or safe management. Distress or depression alone is not an automatic exclusion.
Target and technology	Justify an accessible nerve, DRG or spinal target; consider anatomy, system-specific MRI conditions, external components and follow-up access.	No coherent target, a correctable structural cause or inability to meet essential device-care requirements despite support.
Shared decision and trial	Discuss uncertain post-ICU efficacy, alternatives and treatment burden. Where a trial is required, prespecify pain and functional goals and adverse-event criteria.	No acceptable benefit–risk balance or meaningful trial benefit. Temporary PNS treatment is not necessarily a trial for a permanent system.

Author-proposed framework; not validated criteria, a guideline, or a statement of regulatory approval or coverage. Diagnostic and procedural principles are informed by [9,22–24]; the post-ICU application and timing hypothesis are proposals. DRG, dorsal root ganglion; ICU, intensive care unit; MRI, magnetic resonance imaging; PNS, peripheral nerve stimulation.

MULTIDISCIPLINARY ICU-TO-PAIN-CLINIC PATHWAY

Figure 2 proposes a coordinated pathway beginning during the ICU stay and continuing through outpatient recovery. Critical-care teams document pain, weakness, sensory abnormalities, and relevant positioning or procedural exposures. At transition, the handoff should include the neurological trajectory, pre-existing pain, analgesic and sedative exposure, anticoagulation, wounds, infection history, and a named follow-up clinician.

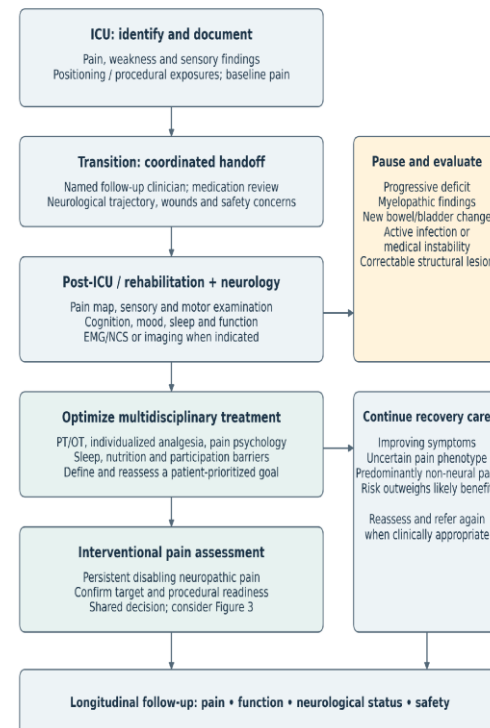
Post-ICU or rehabilitation services assess pain interference, physical function, cognition, mood, and sleep, with neurology involvement when the diagnosis is unclear or deficits persist. Physical and occupational therapists identify whether pain, weakness, joint restriction, cardiopulmonary limitation, or cognitive impairment primarily constrains recovery. Pharmacy and primary-care input can improve medication reconciliation and tolerability. Pain psychology supports coping and expectation setting.

Persistent disabling neuropathic pain despite appropriate treatment prompts interventional pain assessment. The team confirms the target, excludes correctable causes, reviews procedural readiness, and discusses the uncertainty of post-ICU efficacy with the patient. Neuromodulation, when selected, remains integrated with rehabilitation and longitudinal reassessment. Patients with improving pain, an uncertain neuropathic diagnosis, a predominantly non-neuropathic phenotype, or unresolved safety concerns continue individualized care with a clear reassessment plan.

The pathway assigns responsibility across transitions and allows feedback from pain services to critical care, neurology, and rehabilitation. Its effectiveness should be evaluated through referral completion, diagnostic agreement, time to appropriate care, access across demographic and geographic groups, patient experience, and outcomes—not solely through implantation rates.

Figure 2 | Multidisciplinary ICU-to-pain-clinic pathway

Proposed framework for prospective evaluation



Neuromodulation remains integrated with rehabilitation; referral does not imply implantation.

Figure 2: Proposed multidisciplinary ICU-to-pain-clinic clinical pathway.

Coordinated assessment, rehabilitation and medical management precede consideration of analgesic neuromodulation. Progressive neurological findings and unresolved medical or procedural concerns redirect the pathway to evaluation and treatment. Follow-up continues whether or not a device is selected. The pathway is an author-proposed framework for prospective evaluation, not a validated guideline. Diagnostic and procedural principles are informed by [9,22–24]. EMG/NCS, electromyography/nerve conduction studies; ICU, intensive care unit; PT/OT, physical and occupational therapy.

SAFETY CONSIDERATIONS AFTER CRITICAL ILLNESS

Residual organ dysfunction, frailty, malnutrition, immunosuppression, anticoagulation, impaired wound healing, and cognitive or psychological impairment may alter procedural risk. These factors should be assessed individually; prior ICU admission alone does not quantify risk. Active infection, unresolved systemic instability, unexplained progressive neurological deficits, or an untreated correctable lesion should interrupt an elective implantation pathway.

Bleeding risk depends on the procedure and target. Epidural SCS and DRGS differ from superficial peripheral procedures, while some PNS targets lie near deep or noncompressible vascular structures. Antithrombotic management requires a procedure-specific plan that considers both bleeding and thrombosis risk, coordinated with the prescribing clinician.^[23] The present review does not provide medication-interruption intervals or replace relevant procedural guidance.

Infection prevention, wound assessment, and prompt investigation of concerning symptoms are essential for any implanted system.^[24] Previously healed infection should be distinguished from active infection, and additional infectious-disease input may be appropriate. Procedural technique, lead placement, and follow-up should account for mobility limitations, skin integrity, falls, and the ability to observe or report complications.

Device-management requirements should be assessed directly. Charging, programming, external components, adhesive tolerance, travel, and access to troubleshooting can determine feasibility. Cognitive impairment may require supported decision-making and reliable caregiver involvement. MRI access must be evaluated for the complete implanted system and its specific conditions; compatibility should never be assumed from the modality name alone. Adverse-event reporting should include local complications, neuraxial events where relevant, neurological deterioration, revision, explantation, and treatment burden.

EVIDENCE GAPS AND PROSPECTIVE RESEARCH PRIORITIES

The central evidence gap concerns the defined post-critical-illness population. Clinical plausibility and positive trials in analogous conditions cannot establish comparative effectiveness, cost-effectiveness, or the optimal timing of treatment after an ICU stay. [Table 4](#) links these gaps to practical research priorities.

An initial multicenter inception cohort should enroll patients with persistent pain after critical illness regardless of eventual treatment. This permits estimation of neuropathic phenotypes, spontaneous recovery, referral patterns, and barriers to access. Embedded neuromodulation cohorts can then describe feasibility and safety without restricting the entire registry to implanted patients. A proposed treatment-study population might include adults with probable or definite neuropathic pain, clinically meaningful pain

interference, persistent symptoms despite appropriate conservative care, and medical readiness. A threshold such as NRS ≥ 5 can be tested as an enrollment criterion, but should not be presented as a universal clinical rule.

ICU duration, sepsis, organ failure, ventilation, prone positioning, and neurological injury should be recorded as exposures. An arbitrary 48–72-hour ICU threshold could exclude brief but severe critical illness and should therefore be justified by the study question. Pre-existing pain and neuropathy require explicit documentation, with adjudication of new, worsened, or unrelated symptoms. Diagnosis should distinguish diffuse CIP, focal nerve injury, small-fiber pathology, CRPS, and non-neuropathic or mixed pain.

Comparative studies should be designed within clinically overlapping eligibility groups. A three-modality comparison across dissimilar focal and diffuse syndromes would be difficult to interpret because anatomy influences selection. Feasibility work may support phenotype-specific randomized studies of a justified modality plus optimized care versus optimized care alone, with blinded outcome assessment where feasible. Trials should document waveform or stimulation parameters, nerve or spinal target, hardware, trial requirements, duration, programming changes, rehabilitation, medication adjustments, and industry funding.

For a proposed pain-efficacy study, a prespecified primary endpoint could be the proportion achieving at least 50% reduction in average index-pain intensity at 6 months. Twelve-month response and durability through 24 months can be key secondary endpoints. At least 30% improvement should also be reported, alongside absolute pain change and a functional endpoint.^[25,26] The assessment schedule should distinguish time since ICU discharge from time since stimulation initiation. Pain relief accompanied by worsening neurological status must be identified rather than obscured within a responder rate.

Table 5 proposes a measurement set covering pain, interference, function, health status, mood, sleep, medications, neurological findings, safety, and patient priorities. PROMIS Pain Interference and EQ-5D-5L offer established instruments for relevant domains, although their measurement properties require consideration in this population.^[27,28] This is not an established post-ICU core outcome set. Instruments and language versions should be chosen before enrollment, with licensing and accessibility checked. Region-specific disability measures should be used only where appropriate; a back-pain instrument is not a substitute for global function in diffuse neuropathy.

Analyses should retain clear denominators for patients screened, eligible, referred, trialed, implanted, treated temporarily, and assessed at follow-up. Trial failures, crossover, explantation, nonresponse, deaths, and losses to follow-up must be reported. Missing-data assumptions and sensitivity analyses should be prespecified. Attrition related to

persistent disability is particularly relevant, and a complete-case response rate should not be presented as the response rate of all initially enrolled patients. Patient and caregiver involvement can improve both outcome selection and the practical burden of follow-up.

Table 4: Current evidence gaps and priorities for future research

Evidence gap	Priority question	Suggested study or minimum design feature
Uncertain phenotype frequency	Which survivors develop clinically confirmed neuropathic pain, and how does it evolve?	Prospective inception cohort with pre-ICU pain history, standardized examination and repeated assessment; include nonimplanted patients.
Limited direct treatment evidence	Is analgesic neuromodulation feasible, effective and acceptably safe after critical illness?	Phenotype-specific feasibility studies, followed by controlled trials; distinguish analgesic stimulation from muscle rehabilitation or monitoring.
Selection and target uncertainty	Which clinical and anatomical features predict meaningful benefit?	Prespecified phenotyping and target documentation; evaluate prediction models with independent validation rather than retrospective thresholds.
Unknown optimal timing	When does intervention offer benefit beyond spontaneous recovery and optimized care?	Record time since illness, ICU discharge and treatment; compare timing strategies with serial neurological and functional assessments.
Lack of interpretable comparisons	Which modality is preferable when more than one target is clinically justified?	Compare only overlapping eligibility groups; document waveform, hardware, trial enrichment and co-interventions.
Incomplete functional and safety data	Does pain relief translate into valued activity, and what harms occur?	Prespecify pain interference, function, participation, neurological status and adverse events; follow through at least 12–24 months.
Attrition and treatment-selection bias	How do trial failures, crossover, explantation and loss to follow-up alter estimates?	Report all denominators and reasons for attrition; prespecify estimands, missing-data analyses and relevant untreated comparators.
Access, burden and costs	Who reaches specialist care, and is benefit worth the treatment burden?	Measure referral completion, inequities, caregiver burden, visits, device support, healthcare use and costs; involve patients in study design.

Research priorities proposed from the narrative synthesis; they are not a formal consensus statement or evidence-certainty assessment. ICU, intensive care unit.

Table 5: Recommended outcome measures for prospective studies in this population

Domain	Suggested measure	Role / interpretation	Proposed timing
Index-pain intensity	0–10 NRS for average pain over the preceding 7 days; identify the target region.	Candidate primary endpoint: $\geq 50\%$ reduction at 6 months. Also report $\geq 30\%$ response and absolute change. [25, 26]	B; 1, 3, 6, 12, 24 months
Pain interference	PROMIS Pain Interference short form or Brief Pain Inventory interference scale.	Key secondary endpoint; select one prespecified version and report change with uncertainty.[25,27]	B; 1, 3, 6, 12, 24 months
Neuropathic phenotype	Pain map, sensory examination and diagnostic grade; DN4 for screening support.	Baseline eligibility/stratification and reassessment; screening-score change alone is not proof of nerve recovery.[9,10]	B; 6 and 12 months; if symptoms change
Physical function	PROMIS Physical Function or an appropriate regional measure; a feasible performance test such as 6-minute walk or sit-to-stand.	Separate pain-related limitation from weakness and cardiopulmonary impairment. Record walking aids and testing conditions.	B; 3, 6, 12, 24 months
Activities and participation	Patient-prioritized activity goal; ADLs; rehabilitation attendance; return to work or usual roles where applicable.	Define success before treatment and distinguish nonapplicability from missing data.	B; 3, 6, 12, 24 months
Health-related quality of life	EQ-5D-5L index and health visual analogue scale.	Use an appropriate country value set; do not assume a universal clinically important difference.[28]	B; 3, 6, 12, 24 months
Global improvement	Patient Global Impression of Change (PGIC).	Patient-rated overall benefit; interpret alongside pain, function and adverse effects.[25,26]	1, 3, 6, 12, 24 months
Mood, trauma and sleep	PHQ-9, GAD-7, a validated PTSD measure such as PCL-5, and PROMIS Sleep Disturbance or another prespecified sleep measure.	Screening and secondary outcomes; positive screens require clinical follow-up, not automatic diagnostic labeling.	B; 3, 6, 12 months; as indicated
Medication exposure	Analgesic class/dose and daily opioid morphine equivalents, with prespecified conversion rules.	Report absolute and relative changes, adverse effects and reasons for tapering; avoid forced medication reduction.	B and every follow-up
Neurological recovery	Strength, reflexes, sensory findings and gait; EMG/NCS when clinically indicated.	Keep neurological outcomes separate from analgesic response; record new or worsened deficits.	B; 6, 12, 24 months; if indicated
Safety and treatment burden	Infection, bleeding, neurological events, lead/device problems, revision, explant, visits and discontinuation.	Report severity, timing, relatedness and denominators; include trial and temporary-treatment phases.	Continuously; summarize at each visit
Feasibility and retention	Referral, eligibility, trial, implantation and follow-up rates; crossover, reasons for withdrawal and deaths.	Essential for interpreting response estimates and designing a subsequent comparative trial.	At each study stage

Proposed measurement set, not a validated post-ICU core outcome set. B = baseline before treatment; follow-up months are measured from treatment initiation, with time since ICU discharge recorded separately. Add protocol-specific early safety visits. Check instrument versions, validated language translations, accessibility and licensing before use. Prespecify one primary endpoint, key secondary endpoints and missing-data handling. ADLs, activities of daily living; DN4, Douleur Neuropathique en 4

Questions; EMG/NCS, electromyography/nerve conduction studies; GAD-7, Generalized Anxiety Disorder-7; NRS, numerical rating scale; PCL-5, PTSD Checklist for DSM-5; PHQ-9, Patient Health Questionnaire-9; PROMIS, Patient-Reported Outcomes Measurement Information System; PTSD, post-traumatic stress disorder.

DISCUSSION AND LIMITATIONS

The rationale for post-critical-illness neuromodulation is strongest when a persistent pain generator can be linked to a coherent neurological phenotype and an anatomically justified target. Focal ICU-related nerve injury offers a relatively direct anatomical analogy to peripheral nerve pain studies. Regional CRPS or causalgia may overlap with populations investigated using DRGS, while bilateral distal neuropathic pain provides a possible analogy to diabetic-neuropathy SCS trials. These relationships are clinical hypotheses, not evidence that one modality is uniformly superior.

A symptom-focused approach must remain connected to the broader recovery process. Pain, weakness, cognition, psychological distress, and cardiopulmonary impairment can contribute independently to disability. Even substantial analgesia may produce limited functional improvement when another impairment remains dominant. Conversely, a moderate reduction in pain interference may be valuable if it restores participation in rehabilitation or a personally important activity. This supports multidimensional outcomes and shared decision-making rather than reliance on pain intensity alone.

Several limitations constrain the conclusions. This selective narrative synthesis cannot quantify the total direct evidence base, rule out missed studies, or support formal certainty ratings for post-ICU efficacy. Much of the neuromodulation evidence concerns biologically distinct populations. Trial enrichment, unblinded comparisons, crossover, device variation, industry involvement, and follow-up attrition complicate translation. Critical-care pain studies use heterogeneous definitions and may exclude survivors unable to provide self-report. The proposed criteria, timing window, pathway, and algorithm have not been validated prospectively.

Future formal evidence mapping should use reproducible searches and transparent screening, while preserving the distinction between direct post-ICU evidence, analogous pain evidence, muscle rehabilitation stimulation, and diagnostic nerve stimulation. Prospective clinical work should determine whether phenotype-based targeting improves outcomes beyond optimized multidisciplinary care and which patients experience an acceptable balance of benefit, harm, and treatment burden.

CONCLUSIONS

Persistent neuropathic pain can complicate recovery after critical illness, arising from diffuse or focal neural injury within a broader mixture of pain mechanisms. PNS, DRGS, and SCS have indication-specific evidence in analogous populations, providing a rationale for carefully designed post-ICU studies. That evidence does not establish routine use for post-critical-illness neuropathic pain or recovery of the underlying neuropathy.

A coordinated pathway incorporating neurological diagnosis, rehabilitation, psychological support, individualized analgesia, and procedural safety assessment can support appropriate specialist evaluation. Prospective research should test phenotype-specific selection and timing, retain untreated and nonimplanted comparators where feasible, and prioritize meaningful function, safety, and durable benefit alongside pain relief.

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