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The Implementation of Persistent Spinal Pain Syndrome (PSPS): Mechanism-Based Recommendations

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ABSTRACT

Background: In 2021, the term persistent spinal pain syndrome (PSPS) was introduced. PSPS type 2 (PSPS-T2) replaced the unsatisfactory term failed back surgery syndrome (FBSS). PSPS type 1 (PSPS-T1) is a clinical picture of signs and symptoms of FBSS but without prior surgery. PSPS applies to any spinal level. There are multiple underlying mechanisms and treatments within PSPS. Therapeutic choices are not based on a common nomenclature. One consequence is that the desired outcomes of interventions are not reliably achieved. There is a need to redefine diagnostic clusters of PSPS based on the pain mechanism rather than just anatomic factors or a history of previous surgery. This manuscript provides mechanism-based recommendations to improve treatment selection through the use of the PSPS concept.

Methods: An international task force of pain and neurosurgical specialists was established and met at the International Neuromodulation Society Congress 2024, at the North American Neuromodulation Society Congress 2024, and online.

Results: An overview of classification systems (i.e., PSPS; P), anatomical structure (A), neural mechanism (M), and degree of certainty (C), is discussed and combined to generate a novel categorization approach to assist with PSPS classification and diagnosis. The PAMC scale was developed to assist with the diagnosis clustering and consequently to improve treatment selection.

Conclusion: The proposed recommendations should support future research by better defining study populations and by assisting the clinician to select the appropriate therapy.

1 | Introduction

The term *persistent spinal pain syndrome* (PSPS) was introduced in 2021 to provide a replacement for the misleading and pejorative term *failed back surgery syndrome* (FBSS) [1]. By taking a holistic approach to chronic pain of spinal origin, based on the relevance of upright posture, PSPS avoids the often-inappropriate attribution of causation that was a major flaw of FBSS and of many proposed replacements. Its principles apply to the whole spine but do not directly incorporate precise pathophysiological mechanisms. Pain-mechanism-based recommendations are proposed which elaborate upon

the principles of PSPS to provide that level of detail. The primary aim of PSPS terminology was to improve the selection of patients for appropriate treatment, from conservative management to neural ablation, neuromodulation, and spinal surgery (decompression and/or fixation).

PSPS widens the focus beyond surgery and is subdivided into type 1 (PSPS-T1), where no spinal surgery that was intended to relieve pain had been performed, and type 2 (PSPS-T2) where there was a history of such surgery, and it had failed to relieve the pain or had increased or caused it. Type 2 includes, but does not equate to, the ICD-11 diagnosis *chronic pain after spinal surgery* (CPSS). The latter term is specific for pain caused by, or probably caused by, surgery [2]; PSPS-T2 includes pain that was not relieved by surgery. PSPS can apply to any spinal level, including cervical.

The most common manifestation of PSPS is chronic low back pain, with or without lower limb pain. This highly prevalent condition affected 619 million people worldwide in 2020 with its prevalence estimated to rise to 843 million people by 2050 [3]. Chronic low back pain is the single greatest cause of years lived with disability across the world [3]. A wide variety of treatments for low back pain is available. In addition to the practitioner's specialty and their available treatment methods, proven effectiveness, costs and invasiveness play a role in the choice of treatment, starting with the least costly and progressing to incrementally costly and invasive options as necessary [4]. When more conservative treatments including physical therapy and medication fail, a subset of patients undergoes surgery.

Spinal surgery is an invasive, irreversible and expensive treatment option for the management of low back pain. Only a small proportion of patients with new onset low back pain with or without lower extremity pain is estimated to receive spinal surgery (1.2%) [5]. In the United States these, however, account for 29.3% of total 12-month costs of care for low back pain following diagnosis ($784 million) suggesting that spinal surgery is a significant driver of healthcare expenditure [5]. In the United Kingdom, estimates suggest that 5000 new patients per year experience PSPS-T2 with each new cohort costing the healthcare system in excess of £70 million over the first 10 years alone [6]. Success rates of lumbar spine surgery performed primarily for pain are variable, with only about 60% of patients achieving reductions in pain of at least a minimal clinically important difference (MCID), and 20% experiencing persistent long-term pain after surgery [6–8].

PSPS-T2 includes neuropathic, nociplastic, and nociceptive pain. The relative dominance of each category or mechanism may determine the success of therapy outcomes. Treatments such as spinal cord stimulation (SCS) which can address multiple mechanisms are superior both to comprehensive medical management and to revision spine surgery in appropriate cases. PSPS-T2 is the commonest indication for SCS [9, 10].

Single-arm studies [11, 12], randomized controlled trials (RCTs) [13–16], and systematic reviews [17, 18], have evaluated SCS systems used in clinical practice for patients with chronic low back pain with or without radiculopathy who have *not* undergone spinal surgery (PSPS-T1), showing that SCS is an effective treatment option for those patients by comparison with continued conventional medical management [19]. A major challenge is how to determine which individual patients who have not had surgery (PSPS-T1), but whose characteristics are otherwise comparable to those who have (PSPS-T2), would benefit from neurostimulation, potentially avoiding surgery in some cases. Attention has recently turned, for example, to a subgroup of patients with nociceptive low back pain caused by multifidus muscle dysfunction. This has been found to respond to multifidus nerve stimulation [20, 21]. While focusing on low back pain, it is important to recognize that PSPS is relevant to all spinal levels.

The current manuscript characterizes subpopulations of chronic secondary pain of spinal origin within the definition of PSPS (i.e., excluding certain specific conditions such as ankylosing spondylitis). We introduce a pain-mechanism-based approach that augments the classification by escaping the limitations of the traditional anatomically based system and that is more allied to therapeutic choices. It is hoped that a consensus can be achieved across a range of therapeutic stakeholders, thereby leading to improved and durable long-term treatment outcomes.

We are cognisant of the impact of psycho-social factors on the diagnosis, treatment and outcome from treatment [22]. However, the aim of this project is to focus on the biomedical factors, in which we attempt to shift away from solely pathological–anatomical classification considerations and to provide a framework that all PSPS—treating stakeholders (whatever their specialty) can use.

2 | PSPS Task Force

Authors of the first PSPS manuscript [1] were invited to take part in the current project. This was instigated by those who prepared a position statement regarding a need to further the proposed approach to implementation of PSPS. The first meeting took place on 19 January 2024 at the North American Neuromodulation Society Congress. Following the initial meeting, a number of case scenarios were provided for the group to assess how the proposed system would apply to the clinical presentation.

The second meeting occurred on 13 May 2024 at the International Neuromodulation Society Congress (attendees for both meetings are listed in the Acknowledgments section). A task force was established to encompass a multidisciplinary, international group who had held senior leadership positions within neuromodulation, pain and neurosurgery to develop a classification system based on pain mechanisms in the context of PSPS and its implementation.

Consensus was based upon a modified Delphi methodology. Following initial discussion concerning the important variables the lead author presented these via email following the first meeting. These were considered by the wider group via email which expressed their opinion and refined the variables that were represented at the second meeting and finally agreed upon by majority via email thereafter.

3 | Classification Systems

Several classification systems for low back pain have been proposed (Table 1). Differentiators between types of low back pain typically consider the presence or absence of radicular pain, prior spine surgery, and whether the pain experienced is limited to above the knee or also radiates below the knee [23]. Both limb pain and back pain can represent referred pain, that is, pain occurring in a location that is different from the site of pathology [25], but shares a nerve supply at the dermatomal, myotomal, or sclerotomal level. Limb pain can predominate over axial pain. The diagnostic term PSPS includes these cases [1]. The authors considered PSPS-T1 to be chronic axial pain and/or radicular symptoms of spinal origin in individuals who have not undergone any spinal surgery, while PSPS-T2 referred to pain despite, or as a consequence of, spine surgery [1]. In the definitions of low back pain [23], or nonspecific low back pain [24], there is no consideration of the neural mechanisms of the pain (i.e., neuropathic, nociceptive or nociplastic). The construct "nonspecific low back pain" refers to pain for which no specific cause or structure can be identified to account for the patient's symptoms [24]. These patients usually would not be considered for either surgical intervention or SCS therapy. The classification of PSPS considered the various known pathophysiologies but did not expand on neural mechanisms.

3.1 | Therapeutic Options (Surgery Versus Neuromodulation)

For patients presenting with chronic low back pain, there are few absolute indications for spinal surgery. Progressive and/or potentially disabling neurological deficit due to a compressing lesion is an absolute indication, but there are alternative treatments for pain.

Until recently, most surgeons, referring physicians and many patients believed that when conservative management fails, and provided there is a concordant lesion, open surgery to the spine is indicated and provides good outcomes in most cases with a reasonable degree of safety. Some question whether this applies to all spine surgeries and the durability of the outcomes achieved. For example, surgical decompression with microdiscectomy for back pain with radiculopathy of less than 1-year duration was

TABLE 1 | Terms from guidelines or consensus-based classifications of low back pain.

Source	NASS EB Clinical Guideline 2020 [23]	Maher et al. [24]	Christelis et al. [1]
Term	Low back pain	Nonspecific low back pain	Persistent spinal pain syndrome Type 1
Definition	Pain of musculoskeletal origin extending from the lowest rib to the gluteal fold that may at times extend as somatic referred pain into the thigh (above the knee)	Pain in which no specific cause or structure can be identified to account for the patient's perceived symptoms	Chronic axial pain and/or radicular symptoms of spinal origin who have not undergone any spinal surgery (no surgically remediable pathology, or unfit for or declined surgery)
Inclusion	Low back pain limited to somatic referred pain/nonradicular pain limited to above the knee only	Any cases not mentioned in the exclusion	Recurrent pain of spinal origin, paresthesia, numbness, stiffness, muscle spasms and weakness, and, in some cases, sphincter disturbance. The distribution is variably axial and/or radicular, and most commonly lumbosacral, but can be cervical. It may also be thoracic but less commonly; splinting by the ribcage affords a degree of protection to this region
Exclusion	Low back pain due to tumor, infection, metabolic disease, inflammatory arthritis, fracture; patients with a diagnosed deformity, including spondylolisthesis, spondylolysis and scoliosis; pain experienced below the knee; extraspinal conditions (i.e., visceral, vascular, genitourinary); patients who have undergone prior lumbar surgery; presence of neurological deficit; back pain that is associated with widespread multisite pain (> 2 sites); or pregnancy	Cases in which the pain arises from either problems beyond the lumbar spine (e.g., leaking aortic aneurysm); specific disorders affecting the lumbar spine (e.g., epidural abscess, compression fracture, spondyloarthropathy, malignancy, cauda equina syndrome); or radicular pain, radiculopathy, or spinal canal stenosis	Chronic primary musculoskeletal pain defined by ICD-11 as pain located in muscles, bones, joints, or tendons that lasts or recurs for longer than three months, that is associated with significant emotional distress and/or functional disability, and that cannot be better accounted for by another diagnosis. Underlying specific conditions, for example, ankylosing spondylitis

Abbreviations: EB, evidence-based; ICD, International Classification of Diseases; NASS, North American Spine Society.

not shown to be superior to targeted nerve root injections at 1-year follow-up and there was a greater number of adverse events in the surgical group [26]. Prior to the body of RCT evidence in surgical patients, many would consider discectomy to be one of the most successful spinal interventions for the relief of pain in comparison with other decompression procedures, with or without instrumental fusion or revisional surgery.

Surgeons are frequently faced with patients with radicular or referred spinal pain of spinal origin where there is little concordance with the anatomical findings. This may be because the degenerative changes are widespread, but it is often due to the emerging dominance of neuropathic or nociplastic mechanisms, or a combination. Over 50% of orthopedic patients have mixed phenotypes [27, 28]. Anatomical correction will either fail to relieve the symptoms, or the symptoms will likely recur within months. All procedures are associated with risk and some patients will have significant co-morbidity, adding to the risk of unsuccessful surgical outcome. Surgical outcomes are not reliably predictable [29, 30], and adverse and serious adverse events occur regularly (e.g., rates range from 10.8% to 13.3% for any complication, 1.3% to 3% for new or worsening neurological deficit, 0.9% to 2.6% for direct nerve root injury, 3.1% to 4.4% for recurrent disc complications, and 3.7% to 10.2% for reoperations) [31]. Balanced consideration should be given to less invasive, reversible therapies.

Patients with PSPS-T1, that is, chronic axial pain and/or limb symptoms of spinal origin who have not undergone spinal surgery, may be considered ineligible for spinal surgery for various reasons, including:

- patient does not give consent for surgical procedure following unbiased information;
- significant co-morbidity that will adversely affect the risk/benefit of the procedure;
- better outcomes and safety can be achieved with nonsurgical management;
- evidence of chronic neuropathy with neuropathic pain;
- potentially remediable pathology is nonconcordant with symptoms.

Further surgery for patients with PSPS-T2 may be considered inappropriate for the same reasons.

RCTs of SCS for patient populations with low back pain without prior spine surgery have been conducted following the emergence of novel types of stimulation and reports of case series. Varied terminology has been used, though the definitions align with the description of PSPS-T1 (Table 2). Eligibility criteria for these RCTs considered the absence of spine surgery, chronic axial low back pain with or without leg pain and a neuropathic component. In addition, all RCTs specify ineligibility or unwillingness to undergo (nonneuromodulatory) spine surgery as a criterion. The detailed exclusion criteria (N.B., Table 2 presents only a summary of the exclusion criteria) indicate knowledge of patients for whom SCS will not work. Identification of those patients with PSPS-T1 who will respond to SCS remains a challenge that our proposed approach may help to address. Treatment with SCS of PSPS-T1 is infrequent but is increasing in certain health economies where SCS reimbursement is available, such as the United Kingdom and Australia. SCS for PSPS-T1 is not reimbursed in the Netherlands and it is not routinely covered by commercial insurers within the USA making the therapy difficult to access for patient populations outside of Medicare.

4 | Pathophysiology of Pain

4.1 | Neural Mechanism

Nociceptive pain has been defined as arising from actual or threatened damage to nonneural tissue and is due to the activation of nociceptors [33]. It is typically intermittent/mechanically evoked pain confined to the area of primary hyperalgesia.

Nociplastic pain arises from altered nociception despite no clear evidence of actual or threatened tissue damage causing the activation of peripheral nociceptors or evidence for disease or lesion [34]. Typically, nociplastic pain is intermittent/mechanically evoked nociceptive pain with evidence of sensitisation with pain beyond the area of primary hyperalgesia. It may have central mechanisms, as in fibromyalgia (top-down nociplastic pain of central origin), but the term includes patients where, following prolonged c-fiber activation there is central sensitisation, an adaptive neuroplasticity (bottom-up sensitisation of peripheral origin). The pain is maintained by both continued nociceptive activation and central sensitisation. To date there are no definitive approaches to determine the dominant mechanism. In some patients however, a simple local anesthetic nerve block can produce prolonged pain relief, long exceeding the duration of effect of the local anesthetic and is thus potentially useful as treatment [35]. Prognostic or diagnostic nerve blocks, however, lack specificity [36].

Neuropathic pain is caused by a lesion or disease of the somatosensory nervous system that requires a demonstrable lesion or a disease that satisfies established neurological diagnostic criteria [33]. Typical neuropathic symptoms within a named nerve distribution are confirmed by clinical testing of sensory and motor abnormalities. Patients may also present with typical neuropathic symptoms involving, but not confined to, a nerve distribution with or without sensory/motor changes. This can be regarded as probable "neuropathic-like" with nociplastic mechanism.

Mixed pain has no clear pathophysiology as it involves more than one of the above mechanisms simultaneously or sequentially.

4.2 | Anatomical Structure and Pain Generators of PSPS

The traditional approach for the diagnosis of pain of spinal origin has relied upon the identification of structural pathology thought to relate to the pathogenesis of pain but also attempts to identify specific anatomical pain generators. Over-reliance on the identification of structural abnormalities and pain generators alone has not been successful in providing routinely effective therapies. There are many treatment

TABLE 2 | Terms used in RCTs of SCS.

Source	SENZA-NSRBP [32]	DISTINCT [14]	NOVA [15]	EU DTM SCS vs. CMM [16]
Term	NSRBP	Refractory Low Back Pain, also mentions PSPS-T1	Chronic refractory axial low back pain, mentions PSPS-T1	PSPS-T1
Definition	Patients with moderate-to-severe refractory back pain who have not had previous major lumbar spine surgery, are not candidates for back surgery as assessed by a spine surgeon, or decline surgery	Back pain was the primary complaint; leg pain also could be present, but it could not exceed back pain in severity. Subjects reported moderate-to-severe back pain-related disability. Lack of an identifiable pathology that could effectively be treated with surgery	PSPS-T1 subjects with CLBP who were deemed ineligible to undergo spinal surgery (NB not specified how ineligibility for surgery was assessed)	PSPS-T1 patients ineligible for spine surgery
Inclusion criteria	Chronic, refractory axial low back pain and not a candidate for surgery based on a spine surgeon's assessment Pain should have a predominant neuropathic component as per the investigator's clinical assessment Have not had any surgery for back or leg pain, or any surgery resulting in back or leg pain	Chronic (≥ 6 months), refractory axial low back pain with a neuropathic component and is not a candidate for spine surgery Patient has back pain for ≥ 6 months inadequately responsive to supervised conservative care Patient has not had spine surgery for back or leg pain	Candidate for SCS system as per labeled indication (back pain with or without leg pain) Refractory axial CLBP with a neuropathic component and not eligible for spine surgery (e.g., lumbar fusion, discectomy, laminectomy, laminotomy)	Diagnosis of neuropathic CLBP with or without leg pain Ineligible to undergo spinal surgery treatment based on a multidisciplinary medical review and consultation Mechanical spine instability
Exclusion criteria	Diagnosed back condition with inflammatory causes of back pain (e.g., ankylosing spondylitis or diseases of the viscera) Have a medical condition or pain in other areas, not intended to be treated with SCS, that could interfere with study procedures or accurate pain reporting, and/or confound evaluation of study endpoints, as determined by the investigator	Pathology seen on imaging tests obtained within the past 12 months that is clearly identified and is likely the cause of the CLBP, that can be addressed with surgery Primary symptom of leg pain, or leg pain is greater than back pain Back pain is due to any of the following: • spinal instability defined as > 2 mm translation on radiographic imaging • visceral causes (e.g., endometriosis or fibroids) • vascular causes (e.g., aortic aneurysm) • spinal infection (e.g., osteomyelitis) • inflammation or damage to the spinal cord (e.g., arachnoiditis or syringomyelia) • tumor or spinal metastases Has widespread pain (e.g., fibromyalgia) or pain in other area(s), not intended to be treated in this study (e.g., neck pain, shoulder pain)	Previous lumbar spinal surgery A medical, anatomical, and/or psychosocial condition that contraindicated the commercially available study neurostimulation system Mechanical spine instability Diagnosis of a condition with inflammatory causes of back pain (e.g., onset of severe pain with activity), serious spinal pathology and or neurological disorders	Previous lumbar spinal surgery A medical, anatomical and/or psychosocial condition that contraindicates the neurostimulation system

Abbreviations: CLBP, chronic low back pain; NSRBP, nonsurgical refractory back pain; PSPS-T1, persistent spinal pain syndrome type 1; SCS, spinal cord stimulation.

failures or treatments that lack durability. For example, postoperative persistent pain affects one in five patients following lumbar spine surgery [6]. Pain relief after radiofrequency ablation of the medial branch of the posterior ramus of the segmental nerve is time-limited with recurrence of pain often within 12-months [37]. Further, studies have reported a drop-off in the success rates of this procedure and it is not recommended to repeat it more than two times per year, and only in those patients who obtain ≥ 50% pain relief at 3 or ideally 6-months [37].

Typically, we consider structural anatomical abnormalities in radiographic or magnetic resonance imaging terms and pain generators in terms of anterior, middle or posterior spinal elements (Table 3). Such descriptors still have a role in defining the subtype of PSPS along with the mechanism and location of pain.

TABLE 3 | Anatomical structural causes and pain generators.

Pathological anatomical structural causes	Stenosis • Central canal • Foraminal • Lateral recess • Facet joint arthropathy • Ligamentum flavum thickening Spondylolisthesis Degenerative disc disease • Annular tear • Disc herniation • Inflammatory Fibrosis Facet joint arthropathy Multifidus fat infiltration and dysfunction
Anatomical pain generators	Posterior spinal elements • Multifidus and other muscle groups • Facet joints • Cluneal nerve and other sensory nerves Middle spinal elements • Pars interarticularis dysfunction/instability • Radicular Anterior spinal elements • Vertebral body • Discogenic Sacro-iliac joint elements • Inflammatory • Dysfunction

4.3 | Degree of Certainty

Finnerup et al. [38] introduced a grading system to determine the level of certainty that the pain experienced by a patient is neuropathic:

- Possible neuropathic pain—history of relevant neurological lesion, pain distribution neurologically plausible;
- Probable neuropathic pain—sensory signs confirmed by sensory testing or diagnostic test;
- Definite neuropathic pain—confirmed lesion and sensory signs.

A similar grading system could be adopted for all types of pain and subtypes of PSPS. For example, hospital coding systems often use the terms possible, probable and definite when applying the appropriate ICD-11 codes. "Possible" usually indicates a degree of certainty based on the clinical suspicion of a lesion or disease and its anatomical distribution. "Probable" indicates a greater level of certainty, and "Definite" indicates a high level of certainty. The degree of certainty of the diagnosis and mechanism of pain can fluctuate as information is accrued.

4.4 | Diagnosis Based on Pathophysiology of Pain

Consideration of the degree of certainty for the prevalent mechanism of action while factoring in location and anatomical pain generators assists with PSPS classification and diagnosis (Table 4). We propose a novel categorization to assist with diagnostic clustering—a PAMC scale—which is similar to the internationally accepted TNM classification of malignant tumors [39]. Use of the PAMC scale in routine practice would consist of consideration of the different components to assist with diagnosis and potentially the best course of treatment.

- P(SPS) = PSPS-T1 (1) or PSPS-T2 (2).
- A(natomical) = bodily region affected—cervical: neck and occipital (1); cervical: neck > arm (2); cervical: arm > neck (3); upper/mid-back (4); lumbar: back only (5); lumbar: back only + multifidus dysfunction (6); lumbar: back > leg (7); lumbar: leg > lumbar (8); lumbar: leg only (9).
- M(echanism) = dominant mechanism of pain; neuropathic (1), nociplastic (2), nociceptive (3), mixed (4).
- C(ertainty) = degree of certainty; definite (1), probable (2), possible (3).

5 | Discussion

This presentation of the proposed model has focused on low back pain, but similar considerations apply to all spinal regions and associated limb pain, reflecting a fundamental principle of PSPS. The introduction of PSPS broke the impasse over the ambiguity of causation which had impeded the replacement of the unsatisfactory terms FBSS and *failed neck surgery syndrome* (FNSS) and provided a logical, coherent and neutral classification [1]. It covers the majority of cases of chronic pain of spinal origin, not just those where surgery was unsuccessful. Intentionally, it did not expand on detailed pathophysiology. The aim of the proposed pain-mechanism-based model is to provide a vehicle to

TABLE 4 | Clinical PSPS scenarios aligning with PAMC coding.

PSPS type (P)	Predominant pain location (A)	Dominant MoA (M)	Degree of certainty (C)[a]	Descriptor	Traditional diagnosis	PAMC
Type 1	Arm > neck pain	Neuropathic	Definite	Cervical neuropathic pain	Cervical radicular neuropathy	$P^1A^3M^1C^1$
Type 1	Low back	Nociceptive	Definite	Posterior spinal nociception	Lumbar facet-mediated pain	$P^1A^5M^3C^1$
Type 1	Low back	Nociceptive	Probable	Multifidus dysfunction with posterior spinal nociception	Clinical diagnosis LBP	$P^1A^6M^3C^2$
Type 1	Low back > Leg	Nociplastic	Probable	Anterior spinal nociception with some radicular pain	Nonsurgical DDD	$P^1A^7M^2C^2$
Type 1	Low back	Nociplastic	Possible	Anterior spinal nociception	Nonsurgical DDD	$P^1A^5M^2C^3$
Type 1	Upper/mid back	Nociplastic	Probable	Anterior spinal nociception	Vertebral fracture	$P^1A^4M^2C^2$
Type 2	Arm > Neck	Neuropathic	Definite	Neuropathic with nerve dysfunction	PSPS T2 (FNSS)	$P^2A^3M^1C^1$
Type 2	Upper/mid back	Mixed	Possible	Unable to define dominant mechanism	Post vertebroplasty	$P^2A^4M^4C^3$
Type 2	Leg > Low back	Nociplastic "neuropathic-like"	Probable	Neuropathic symptoms with secondary hyperalgesia	PSPS T2 (FBSS)	$P^2A^8M^2C^2$
Type 2	Low back > Leg	Mixed	Possible	Unable to define dominant mechanism	PSPS T2 (FBSS)	$P^2A^7M^4C^3$
Type 2	Leg	Neuropathic	Definite	Neuropathic with nerve dysfunction	PSPS T2 (FBSS)	$P^2A^9M^1C^1$

Abbreviations: DDD, degenerative disc disease; FBSS, failed back surgery syndrome; FNSS, failed neck surgery syndrome; LBP, low back pain; MoA, mechanism of action; PSPS, persistent spinal pain syndrome; RF, radiofrequency neurolysis (ablation).
[a]Possible = less than 50% probability (i.e., based upon clinical likelihood); probable = more than 50% probability (i.e., augmented with neurological examination concordance and/or response to treatment); definite = 95% or more probability (i.e., objective neurophysiological concordance and/or complete pain relief with targeted treatment).

assist and optimize the implementation of PSPS, through a composite approach which combines four variables: pain location; mechanism; degree of certainty; and structural pain generator.

The primary aim of PSPS was to improve the selection of the most appropriate treatment, whether behavioral, pharmacological, physical, surgical (excluding surgery for neuromodulation), neural ablation or neuromodulation [1]. The pain-mechanism-based model should facilitate the achievement of this aim, leading to a reduction in the incidence of spinal surgery and poor outcomes, and an increased availability of neuromodulation and other less invasive procedures, thereby reducing the transition of patients from PSPS-T1 to T2. It should also inform decisions regarding treatment for patients with PSPS-T2 who had not responded to conservative measures: to reoperate or to select a neural ablation or a neurostimulation procedure.

Adoption of the PAMC scale amongst the multiple stakeholders who treat PSPS would serve as a checklist, not only to complete a more accurate diagnosis of the pain mechanism but also to show the way towards more effective treatment. Its use encourages consideration of different components to assist with a diagnosis. For example, PSPS-T2 patients with clear evidence of neuropathy might not be best managed with revision spinal surgery as continues to occur in some routine practice, but rather with neuromodulation. Potential barriers to adoption may include resistance to change and a lack of awareness. The PAMC methodology will require further research and dissemination but can be used alongside existing classification systems that may be pertinent to reimbursement.

The model should aid the design and communication of research by better defining the patient populations and should aid coding. PSPS is compatible with ICD-11 and its three levels [1]. ICD-11 coding is rarely used in everyday practice and PSPS itself is not recognized as an ICD-10-CM or ICD-11 code but it can be tracked to a number of codes (Table 5). Similarly, the subtypes of PSPS can also be tracked. ICD-11 still gives anatomical considerations primacy [40]. ICD-11 is cumbersome to use in routine or even research practice and is essentially a coding methodology. PSPS can still track to ICD-11 but is more relevant to routine and research practice. Future ICD revisions could consider PSPS as a broader classification code within chronic pain.

6 | Conclusion

A composite approach combining location, mechanism, degree of certainty, and structural pain generator

TABLE 5 | Mapping PSPS-T1 to ICD-11 and ICD-10-CM.

ICD-11 classification	ICD-11 code	ICD-10-CM classification	ICD-10-CM code
Intervertebral disc disorders			
Radiculopathy due to intervertebral disc disorders	8B93.6	Degenerative intervertebral disc thoracic (with) neuritis, radiculitis or radiculopathy	M51.14
		Degenerative intervertebral disc lumbar (with) neuritis, radiculitis, radiculopathy or sciatica	M51.16
		Disorder of disc (intervertebral) cervical with neuritis, radiculitis or radiculopathy	M50.10
Myelopathy in intervertebral disc disorders	8B42	Disorder of disc (intervertebral) with myelopathy cervical	M50.00
		Disorder of disc (intervertebral) with myelopathy thoracic	M51.04
		Disorder of disc (intervertebral) with myelopathy lumbar	M51.06
Other specified conditions associated with the spine	FB1Y	—	—
Intervertebral disc degeneration			
Intervertebral disc degeneration	FA80	—	—
Intervertebral disc degeneration of cervical spine with nervous system involvement	FA80.3	Disorder of disc (intervertebral) cervical	M50.90
Intervertebral disc degeneration of thoracic spine with nervous system involvement	FA80.7	Degenerative intervertebral disc thoracic region	M51.34
Intervertebral disc degeneration of lumbar spine with nervous system involvement	FA80.B	Degenerative intervertebral disc lumbar region	M51.36
Other specified intervertebral disc degeneration	FA80.Y	—	—
Spondylosis			
Spondylosis	FA81	Spondylosis without myelopathy or radiculopathy	M47.819
		Cervical region	M47.812
		Thoracic region	M47.814
		Lumbar region	M47.816
		Spondylosis with radiculopathy	M47.20
		Cervical region	M47.22
		Thoracic region	M47.24
		Lumbar region	M47.26
Spinal stenosis	FA82	Spinal stenosis	M48.00
		Cervical region	M48.02
		Thoracic region	M48.04
		Lumbar region (NOS) (without neurogenic claudication)	M48.061
		Lumbar region (NOS) (with neurogenic claudication)	M48.062

(Continues)

TABLE 5 | (Continued)

ICD-11 classification	ICD-11 code	ICD-10-CM classification	ICD-10-CM code
Spondylolisthesis	FA84	Spondylolisthesis (acquired) (degenerative)	M43.10
		Cervical region	M43.12
		Thoracic region	M43.14
		Lumbar region	M43.16
Degenerative condition of spine, unspecified	FA8Z	—	—
Radiculopathy			
Radiculopathy due to compression	8B93.0	—	—
Radiculopathy due to intervertebral disc disorders	8B93.6	Degenerative intervertebral disc thoracic (with) neuritis, radiculitis or radiculopathy	M51.14
		Degenerative intervertebral disc lumbar (with) neuritis, radiculitis, radiculopathy or sciatica	M51.16
		Disorder of disc (intervertebral) cervical with neuritis, radiculitis or radiculopathy	M50.10
Radiculopathy due to spondylosis	8B93.8	Spondylosis with radiculopathy	M47.20
Chronic painful radiculopathy	MG30.51	—	—
Other specified radiculopathy	8B93.Y	Radiculopathy, cervical region	M54.12
		Radiculopathy, lumbar region	M54.16
		Radiculopathy, lumbosacral region	M54.17
Spinal pain			
Low back pain	ME84.2	Low back pain	M54.50
Lumbago with sciatica	ME84.20	Low back pain with sciatica	M54.4
Sciatica	ME84.3	—	—
Back syndrome without radiating pain	ME86.20	—	—
Back syndrome with radiating pain	ME86.21	—	—

descriptors provides a model for the application of PSPS, both T1 and T2, which aims to improve diagnosis and the selection of appropriate and effective treatment, and to enhance research.

Author Contributions

S.T. initiated and led the current work. S.T., B.S., F.J.H., and R.V.D. wrote the initial draft of the manuscript. All authors contributed to discussions, drafts, and critical revision of the manuscript. All authors approved the final version of the manuscript. Both the principle of persistent spinal pain syndrome and its relationship to ICD-11 were proposed by B.S.

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Ethics Statement

The authors have nothing to report.

Consent

The authors have nothing to report.

Conflicts of Interest

S.T. discloses consulting agreements with Boston Scientific, Galvani Bioelectronic, Mainstay Medical, and Saluda Medical outside the submitted work. F.J.H. discloses consulting agreements with ABBOTT, Saluda Medical, and Grunenthal outside the submitted work. F.J.H. is an Editorial Board member of *Pain Practice* and a co-author of this article. To minimize bias, he was excluded from all editorial decision-making related to the acceptance of this article for publication. R.B.N. serves as an unpaid officer of the nonprofit Neuromodulation Foundation Inc., to which grants and support have been provided by Abbott, Boston Scientific Corp, Medtronic Ltd, Nevro Corp, Nuvectra, and Stimwave Inc. outside the submitted work. G.B. discloses a consulting agreement

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Data Availability Statement

Data sharing not applicable to this article as no datasets were generated or analyzed during the current study.

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