International Society for the Advancement of Spine Surgery Statement: Restorative Neurostimulation for Chronic Mechanical Low Back Pain Resulting From Neuromuscular Instability

Morgan Lorio, Kai-Uwe Lewandrowski, Domagoj Coric, Frank Phillips and Christopher I. Shaffrey

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ABSTRACT

This International Society for the Advancement of Spine Surgery statement has been generated to respond to growing requests for background, supporting literature and evidence, and proper coding for restorative neurostimulation for chronic low back pain. Chronic low back pain describes the diverse experience of a significant proportion of the population. Conservative management of these patients remains the predominant care pathway, but for many patients, symptom relief is poor. The application of new techniques in patients who have exhausted traditional care paradigms should be undertaken with a detailed understanding of the pathology being treated, the mechanisms involved, and the data supporting efficacy. This statement on restorative neurostimulation places this technology in the context of the current understanding of the etiology of mechanical low back pain and the currently available evidence for this technique. In an appropriately selected cohort with a specific subset of chronic low back pain symptoms, this technique may provide benefit to payers and patients.

MECHANICAL CHRONIC LOW BACK PAIN CLINICAL PRESENTATION

The Problem of Low Back Pain

As clinicians, we often recite, “low back pain is a symptom, not a diagnosis.” Still, despite this, the conflation of symptoms and diagnosis has led to the persistence of what we know as nonspecific low back pain (LBP), a term that arguably underserves our patients. The biopsychosocial model of LBP attributes the problem to a complex interplay of biological, psychological, and social factors.¹,²

In most cases of LBP persisting past 8 weeks, clinical investigation fails to diagnose a clear pathology other than common degenerative changes.³ These changes range from asymptomatic to painful, but the reliable identification of pain generators remains challenging.³ In many cases, more than 1 location may be suspected, but the complexity of the degenerative cascade overlaid with the biopsychosocial nature of chronic low back pain (CLBP) complicates focal diagnosis and moderates the efficacy of focal therapies.⁵ Our understanding of the biological mechanisms of LBP involving the generation and maintenance of pain signals has dramatically improved, as has the distinction between nociceptive, neuropathic, and nociplastic pain.⁶ Furthermore, the biological processes responsible for structural changes in the spine are now profoundly understood.⁷,⁸ Still, in many cases of persistent LBP, there needs to be more clarity between what can be observed diagnostically and what can be reliably targeted for treatment. In the absence of infection, inflammatory disease, malignancy, fracture, or neurological compression with concordant radiculopathy, identifying specific pain generators in the lumbar spine is difficult. It generally arises from the disc, the vertebral body, the zygapophysial joint (including the articular surface or capsule), ligaments, or the sacral iliac joint. Imaging such as magnetic resonance imaging (MRI) can easily visualize degenerative changes to these structures, but the sensitivity and specificity in predicting the effect of treatment could be
better. In most cases, there are multiple potential pain generators and limited therapeutic options that can universally address them all concurrently. In the absence of effective and durable interventions targeting the underlying pathomechanisms of CLBP, psychosocial interventions have become practically the only clinical practice guideline–recommended treatments.

One pathomechanism of particular interest is neuro-muscular instability or a loss in the fidelity of the motor control system proposed by Panjabi.9,10 In this model, normal function of spinal stability is an interactive process between the passive structures of the spine, the actuators (i.e., the muscles), and their neurological control (Figure 1). He predicted that dysfunction in these subsystems could lead to dysfunctional movement exceeding the normal range of motion, deviating from the neutral zone, and putting healthy and damaged tissues at further risk.

This dynamic approach to understanding the problem of LBP explains several clinical features that are not satisfactorily addressed by identifying and treating focal pain generators. It predicts that treatments with a multimodal action mechanism are more likely to be effective. Specifically, this includes the pattern of remission and recurrence observed early in the course of the disease, the lack of specificity and sensitivity of diagnostic imaging, and the lack of efficacy and durability in many treatments.

Scope of the Problem

LBP is the leading cause of years lived with disability worldwide and is often a determinant for chronic opioid use.11,12 In the United States, low back and neck pain are the most expensive health conditions, estimated at $134.5 billion, with 58.7% of this spending in ambulatory care and 26.3% as an inpatient or in the emergency department. Compared with other expensive health conditions such as diabetes or ischemic heart disease
ISASS Statement: Restorative Neurostimulation for Chronic Mechanical LBP

PREVALENCE

The prevalence of all CLBP in the adult population is estimated to be between 5% and 10%.\textsuperscript{15-17} Herman et al\textsuperscript{15} used Medical Expenditure Panel Survey survey data to conclude that 6.0% of US adults suffer from chronic spinal pain, and 2.2% of US adults suffer from high-impact chronic spinal pain. Johannes et al\textsuperscript{17} found in an internet survey of US adults that 8.1% reported having CLBP, with 5.5% reporting having this pain most of the time and 1.8% saying their LBP intensity was severe, consistent with high-impact CLBP. Yu et al\textsuperscript{18} analyzed 2016/2017 economic health records and found that among patients aged 35 years or older who recently visited their primary care physician for LBP, an estimated 26.0% of them had moderate to severe CLBP defined as LBP present on most or all days in the previous 6 months and an average intensity ≥5 on a 0–10 numerical rating scale (NRS). This finding is consistent with the proportion of roughly one-third of patients having CLBP found by Herman et al\textsuperscript{15} and Johannes et al.\textsuperscript{17}

CLBP resulting from motor control dysfunction and neuromuscular instability is a subset of this population. This phenotype can be segmented from the general low-back population by the application of several filters:

- No prior surgery and no indication for surgery: >87% of patients.\textsuperscript{18}
- Predominantly nociceptive pain: >85% of patients.\textsuperscript{19}
- Positive physical findings consistent with impaired neuromuscular control of the deep multifidus muscles: 65% of all CLBP patients.\textsuperscript{20}
- No widespread musculoskeletal pain.
  - Three main categories of pain that co-occur with persistent or CLBP are (1) axial pain (18%–58%), (2) extremity pain (6%–50%), and (3) multisite musculoskeletal pain (10%–89%).\textsuperscript{21}

Even after applying these criteria, the prevalence of patients meeting mechanical LBP from neuromuscular instability is estimated to be a significant proportion of the adult population.

Cost Drivers

In the United States, 40% of chronic pain patients have high-impact chronic pain (ie, chronic pain with high symptom severity that frequently limits life or work activities for more than >6 months).\textsuperscript{22} These patients are more likely to be frequent health care users for pain, reported substantially higher average pain intensity, and described higher pain-related interference with life- and work-related activities and lower quality of life.\textsuperscript{23,24} Longitudinal studies of CLBP patients suggest that the level of pain interference with daily activities (ie, disability) is a stronger predictor of health-related quality of life and costs than pain intensity.\textsuperscript{23-27} The National Pain Strategy has recommended operationalizing this relationship to focus health care resources on chronic patients with high-impact chronic pain, that is, significant levels of life interference with work, social, and self-care activities.\textsuperscript{24,28}

Due to the limited effectiveness and durability of available treatments prognosis for this group remains poor.\textsuperscript{15,27,29-31} Inception cohort studies and latent class analyses demonstrate that patients with a declared high CLBP burden will likely remain severely affected.\textsuperscript{30,32-39} Patients in this group tend to follow a pain trajectory with very few fluctuations over long periods and have a high probability of still reporting persistent severe pain after 7 years.\textsuperscript{33,35,40-43} Although less than 28% of LBP cases fall in the “severe” categories, they are responsible for 77% of all years lived with disabilities.\textsuperscript{32} A 2010 US chronic pain prevalence study showed that 32% of CLBP patients reported severe pain, and 47% had daily pain.\textsuperscript{17}

In patients seeking multidisciplinary spine care for CLBP, quality of life and workability are poor, and health care-related costs are twice as high as costs for those seeking primary care.\textsuperscript{31} In a published analysis of a moderate-sized US commercial insurer of enrollees with CLBP, mean health care costs reached almost double that of the population mean ($11,932 vs $6034).\textsuperscript{27} A recent claims analysis study found that after a diagnosis of nonspecific CLBP, median total annual health care cost almost doubled from $3732 (Q1 $1292 and Q3 $9072) to $6590 (Q1 $2710 and Q3 $13,922) with the highest increases. The total cost among patients with Medicare supplemental insurance was $10,156 (Q1 $5481 and Q3 $18,570).\textsuperscript{44} Review of the interquartile ranges revealed that approximately half of the patients generate three-quarters, and a quarter of the patients are responsible for half of the total incremental expenditure related to CLBP. The most significant opportunity to reduce disease burden and associated costs lies with
treatments potent enough to reduce activity limitations in patients with high-impact CLBP sustainably.

Impact on Work Performance

CLBP and the accompanying psychological and sociological problems commonly associated with it are the second most common pain condition contributing to lost productive time at work, experienced as either diminished work capacity, paid absenteeism, or permanent exit from the workforce. Many episodes of acute back pain resolve with time and may result in a short duration of absenteeism or reduced employment capacity during the recuperation period. However, in many patients, pain recurs, inter-episode span shrinks, and occupational performance diminishes as pain transitions to a chronic, intractable phenotype. This is especially so for those with activity-limiting LBP, most of whom experience episodes that are likely to be progressively worse, with increasing disability and little, if any, remission from symptoms.

Significant factors associated with poor work outcomes include previous work absence, activity limitation, worsening of pain, and disability. A strong predictor of future CLBP is a history of LBP; therefore, one might presume workplace absence due to LBP impacts future work for many patients. When a worker exits paid employment due to CLBP, the pathway to return to work is often unsuccessful. The occupational impact of pain is manifested in three main domains listed in order of economic implications to employers:

Presenteeism: A decrease in the capacity to perform the designated job or a reduction in the employer’s expectation of performance (i.e. reduced duties).

Absenteeism: Increase in the number of days of work missed for health-related reasons.

Health-Related Exit: Inability to participate in the workforce.

Presenteeism is the concept of attending work despite health issues or complaints that should prompt rest and sickness absence. For those living with chronic pain, sickness absence or delaying re-entry into the workforce are not always an option, particularly so in the United States, where many workers do not have paid sick leave in their jobs, and most disability insurance coverage comes with limits on length and amount of income covered. Chronic pain patients already face an increased financial burden associated with health care costs for their condition and are more likely to be socio-economically disadvantaged.

This dynamic presents as an issue of “pain presenteeism,” defined as the capacity to remain at work less productively despite the continued experience of pain. Presenteeism is a largely overlooked factor contributing to the economic burden of chronic pain despite the increasing evidence that lost productivity in the workforce is primarily a result of reduced work effectiveness rather than absenteeism. In a large, cross-sectional study of workers in the United States that captured both absenteeism and presenteeism, over 75% of the lost productive time due to common pain conditions was found to occur due to reduced work performance.

LBP and other musculoskeletal disorders are among the most common causes of absenteeism. The evidence for CLBP and absenteeism, however, is much less clear but possibly more visible in workers with more physically demanding jobs and where there is a greater risk of CLBP-related disability. In a systematic review and meta-analysis of work absence and return-to-work data from 45 studies across different countries, it was reported that while a high proportion (68%) of workers with LBP return to work by the 1 month mark, the remainder are at a significantly increased risk of frequent or long-term work absences without any intervention. Population studies have shown that the transition from work to sickness absence, and then to disability pension, is increased in workers with chronic disease.

Health-related work exit significantly contributes to the socioeconomic burden of CLBP. This includes exit accompanied by receiving health-related benefits or disability pension, involuntary transition to unemployment, and early retirement. In an extensive 28-year follow-up of workers with back pain, recurrent pain was associated with health-related exit, whereas occasional, single reports of pain were not. While a consistent body of evidence supports physical activity and exercise programs in improving LBP, there are limited data regarding work-related outcomes such as presenteeism and absenteeism. There is some evidence of reduced work absence in nonspecific LBP patients who undergo formal physical therapy (ie, physical conditioning and graded-activity interventions). Similarly, for spinal cord stimulation (SCS) therapy, evidence of effectiveness mainly focuses on reductions in pain intensity, while work-related outcomes are often overlooked or neglected. A few studies of conventional SCS for failed back surgery syndrome patients have reported a poor return to work outcomes.
ROLE OF MUSCLES IN LBP

Natural History of the Aging Spine

Both genetics and environmental factors predispose patients to the onset of degenerative changes in the spine. While not all degenerative changes induce pain, they can be closely aligned to mechanisms by which nociception or neuropathy can occur. Early proteoglycan loss in the nucleus pulposus has been studied in depth. It is often considered an index event leading to observable disc degeneration, loss of disc height, and annular tears. These changes, in turn, have mechanical consequences that tend to result in segmental instability, which both offload and overload the facet joints. The degenerative cascade continues as these altered mechanics at the articular joint surface result in osteoarthritic changes and cartilage degeneration. Facet osteoarthritis and hypertrophy may lead to foraminal and central stenosis. Direct nociception due to poor disc and facet biomechanics or neurological impingement from hypertrophic tissues are the more commonly recognized sources of spinal disorders.

The inherent complexity of the spine, overlayed with concurrent multiple degenerative processes often means that attempting to identify a single pain generator can be futile. The degeneration of various structures may result in pain at the same segment or different motion segments, or it may refer pain to other regions. CLBP without precise pain generators is a frustratingly common presentation that is poorly understood and often managed palliatively. These patients are generally not considered surgical candidates; thus, the current treatment guidelines include therapies that are temporary at best and ineffective at worst. It has been argued that some of these patients have apparent pathology—motor control dysfunction secondary to degenerative spine changes.

Sensory Motor Control of the Spine

Motor control of the lumbopelvic region serves to both move and stabilize the trunk. Without muscles, the spine is inherently unstable. Thus, the muscles of the trunk must have not only capacity and endurance but the fidelity of timing and activation to choreograph the complex responses required to move, maintain stability, and anticipate external perturbations. The controller of this feedback/forward network is the central nervous system (CNS). The CNS compiles sensory input from diverse sources, including visual and vestibular systems and peripheral mechanoreceptors in spinal muscle and discoligamentous tissues. This network ensures that the appropriate postural responses are generated at the proper time and magnitude. The complexity of truncal motion to maintain spinal stability throughout daily activity requires multiple interdependent muscle actions to control multiplanar movement (rotation, translation, and flexion-extension) over single segments that combine to achieve the desired functional goal.

Functional goals, whether they are simple like flexion or complex like sitting or locomotion, require multiple inputs from the various aspects of the motor control “matrix” (Figure 3). Planning originates in the motor cortex, and execution to the plan relies on input from mechanoreceptors, found in muscle (muscle spindles) and tendon (Golgi tendon organs), facet capsules (Ruffini corpuscles) to regulate paraspinal muscle load and reflex responses.

Figure 3. Motor control feedforward and feedback loops. Source: Adapted from Holm et al. 74
Each lumbar paraspinal muscle (Figure 4) generates moments, compressive and shear forces dependent on its relative location and insertions. The more superficial muscles tend to span multiple segments and have a mechanical advantage to generate movement rather than stability. The mechanical advantage of deeper, shorter, more medial muscles is to provide compressive stabilizing forces over individual motion segments. The multifidus is of particular interest in understanding the motion and stability of the lumbar spine as the most medial and, therefore, most important for intersegmental stabilization. Morphologically, the multifidus spans the entire lumbar spine from the laminae and the spinous processes cranially, and inserting at the caudal mammillary process, arranged in deep intermediate and superficial fascicles. The deep multifidus spans 2 motion segments, the intermediate 3 motion segments, and the superficial 4 motion segments. All of the fascicles originating at a single level are innervated by the medial branch of the dorsal ramus immediately caudal to that level.

The proper functioning of the back muscles, particularly the multifidus, is essential for motor control of the trunk. There is a deficiency in back muscle function in many cases of CLBP. In a vicious cycle, pain and injury affect muscle structure and function, and muscle dysfunction and structural changes can elicit pain or injury. Injury and degeneration of spinal tissues result in lesions and inflammation that alter spinal mechanoreceptors' proprioceptive and reflex function. The sensitization of these nerve endings elicits either reflex activation or inhibition of various paraspinal muscles, which may directly or indirectly induce pain by altering biomechanical function.

Structural and Functional Changes in the Spinal Muscles During LBP

Some paraspinal muscles respond to painful stimuli and unstable biomechanics via a reflex activation intended to provide stability to the spine. In contrast, others, particularly the deep multifidus, respond with compromised activation. This underactivation of the multifidus is related to inhibitory reflexes originating from stretch receptors in the facet capsule and intervertebral disc and initiates a cascade of degenerative processes resulting in structurally and functionally compromised spinal function. Changes in the muscle correspond with multiple underlying mechanisms that have been described over 3 temporal phases (Figure 5).

Acute/Inhibitory

The index event in an acute episode of LBP is generally thought to be an excursion of spinal tissues outside the safe range of motion, which may occur due to high-strain trauma or a low-strain injury in the presence of

![Figure 4](https://example.com/figure4.png)  
**Figure 4.** Key muscles of the lumbar trunk.

![Figure 5](https://example.com/figure5.png)  
**Figure 5.** Progression of acute to chronic low back pain. Source: Adapted from Hodges et al.
altered biomechanics resulting from ongoing degenerative processes. Irrespectively, the mechanical deviation initiates pain and sensory neural processes, including spinal reflex inhibition and altered descending drive that reduces multifidus activation. This process is rapid and initially functionally reversible, but there is strong evidence that persistent pain and inflammation result in changes to muscle structure over multiple episodes. During the acute phase, the multifidus undergoes significant atrophy, though these structural changes are not hypertrophic and are likely a result of sympathetic vasoconstriction or other mechanism.81

The reversibility of these changes in the early disease state suggests that targeted therapies to alleviate pain, overcome inhibition, and downregulate inflammatory processes can be beneficial in the resolution of muscle dysfunction. General activity and targeted motor control exercise have both demonstrated some benefit for these patients.82

**Subacute/Inflammatory**

Acute LBP commonly improves spontaneously or with limited intervention in many cases; however, the consensus increasingly is that this is a long-lasting condition with a variable course rather than unrelated episodes. This consensus is mechanistically consistent with the alteration of biomechanics resulting from the ongoing degenerative processes predisposing to multiple acute episodes. Each episode results in further structural and functional compromise of the paraspinal muscles. In turn, this dysfunction results in a propensity for further injury and perpetuation of the cycle of pain, inhibition, and dysfunction.39 Many people presenting to primary care with LBP have either persistent and severe pain or are in a cycle of symptom and remission. Over time, the symptomatic phase becomes more disabling and the remission phase shorter.34

The structural changes to the multifidus are believed to result from a dysregulated inflammatory process resulting in adaptive changes to muscle tissue, including fibrosis, fat infiltration, and transition in the proportion of muscle fiber types.83–85 Inhibition and inflammation of the deep multifidus lead to a protective strategy of superficial muscle activation, relying on less well-adapted structures like the erector spinae for stability and control. In many other disease states, exercise has been shown to rebalance the inflammatory milieu, reverse fibrosis, and change muscle fiber type distribution. Still, the deep muscle inhibition and overarching protective strategy of the superficial muscles make this a challenging functional task for rehabilitation.86,87 This suggests that specific activation of the multifidus by alternative methods, such as electrical stimulation, may address inhibition and degenerative changes.

**Chronic/Disuse**

The progression from the subacute to the chronic phase is accompanied by a proliferation of structural changes to the muscle as function is adapted to compensate for the loss of motor control. The altered movement patterns preferentially recruit superficial muscles, and the dysfunctional deep stabilizers are offloaded.88 These altered movement patterns are perpetuated by both pain and fear avoidance behaviors and are associated with the loss of discrete cortical organization of inputs to back muscles.89 The disuse of the deep stabilizing muscles results in a bilateral reduction in cross-sectional area and further fat tissue deposition.

Therapies applied to this LBP process stage have not been broadly successful. Similarly to the inflammatory phase, the neural and structural changes to the muscle are difficult to overcome with traditional exercise and physical medicine. Numerous well-constructed exercise trials have failed to produce compelling outcomes for these patients, not because activating the multifidus in CLBP patients is incorrect but because the reduction to practice is challenging.90 Specific strategies that target full physiological contractions of the multifidus are necessary to reverse physiological processes of inhibition, inflammation, and disuse.

**CURRENT TREATMENTS FOR CLBP**

**Early Treatment Options**

**Physical Medicine and Rehabilitation**

The most recent Cochrane review91 concludes that there is moderate certainty that exercise-based physical therapy is effective for the treatment of CLBP compared with no treatment, usual care, or placebo, though the treatment effect size is smaller than the threshold for a minimally clinically important difference. Due to the heterogeneity of the patient presentation, a generalizable understanding of the efficacy of physical therapy is limited.

Physical medicine approaches remain guideline-recommended treatments for LBP, though there is a variable quality of evidence for the timing and nature of the intervention. The North American Spine Society guidelines92 gave detailed recommendations based on specific trial results. They proposed a consensus statement:
In the absence of reliable evidence for patients with nonspecific back pain, based on abundant data for other spinal disorders that result in back pain, it is the work group’s opinion that remaining active is preferred and likely results in better short-term outcomes than does bed rest. This advice is consistent with guidelines from other societies.93

Despite the small effect, physical therapy is a safe and relatively inexpensive intervention and remains at the forefront of the treatment and triage of CLBP patients.

Pharmacological Management

Various drugs are routinely prescribed to manage CLBP, and the efficacy has been the subject of multiple systematic reviews and society guidelines. Reviewing pharmacological options in detail is well beyond the scope of the present article; however, guidelines from the relevant US and European Societies require more evidence for their general application. We recommend that interested readers consult the appropriate guidelines and reviews.92–97

Psychological Therapies

Psychological therapies consistently receive the most robust recommendations from multiple society guidelines due to the emphasis on the biopsychosocial nature of CLBP. Guidelines developed under the European Cooperation in Science and Technology Framework95 make strong recommendations for behavioral treatments compared with placebo, waiting list control, and usual care. However, they also cite strong evidence that there is no difference between the treatment modalities. The North American Spine Surgery (NASS) guidelines92 make strong recommendations for applying cognitive behavioral therapy to augment physical therapy outcomes. A Cochrane systematic review by Henschke et al.98 concluded that these behavioral interventions were more effective than usual care over the short term. However, over the longer term, there was no incremental benefit. Table 1 summarizes the relative benefits of some psychological treatments.

Interventional Treatment Options

Epidural Steroid Injections

The European95 or NASS92 guidelines do not support epidural steroid injection for treating mechanical LBP. Several high-quality systematic reviews assess the evidence for radicular pain but provide limited evidence for mechanical pain. The latest Cochrane review99 supports these findings, concluding that there is “insufficient evidence to support or refute the use of injection therapy, regardless of type and dosage, for patients with subacute and [CLBP] without radicular pain.” The UK’s National Institute of Clinical Excellence (NICE) only recommends epidural injections of local anesthetic and steroid in people with acute and severe sciatica.100

Intra-articular Facet Injection

In appropriately selected patients, intra-articular facet injections have moderate quality evidence for no effect in the medium term (6 months), according to the NASS and European guidelines. A recent systematic review101 assessed 42 studies for eligibility and provided qualitative evidence based on 6 studies based on heterogeneity of treatment, outcome, and follow-up period preventing a formal meta-analysis. By way of example, a randomized controlled trial conducted by Lakemeier et al.102 compared intra-articular injection with radiofrequency ablation (RFA) and concluded that both techniques provided limited relief over the short term but were similar. The injection cohort in this study showed improvements in visual analog scale (VAS) from 7.0 ± 1.7 to 5.4 ± 2.1 and improvement in Oswestry Disability Index (ODI) from 38.7 ± 18.4 to 33.0 ± 17.4, consistent with a clinically insignificant treatment effect.

Facet Joint RFA

RFA remains at the forefront of clinical interventions for CLBP. It is well tolerated by patients and has few complications. Despite being in clinical practice for decades, this procedure still has concerns regarding efficacy and durability. The most recent Cochrane review103 summarized the published evidence through 2015 and suggested that there was moderate quality evidence that

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suggests that RFA might better relieve facet joint pain and improve function over the short term when compared with placebo. Not included in this review was the minimal interventional treatments for patients with chronic low back pain (MINT) trials report,104 a high-quality prospective randomized control trial comparing a standardized exercise program to RFA. Of the patients being treated for facet-related problems, there was no difference in outcomes between denervation and exercise. Some criticism has been leveled at the trial design and interpretation,105–107 including a lack of standardization of patient selection and diagnostic blocks.

The durability of the effect of RFA is often questioned. There is conflicting low-quality evidence (Grade II and III) of the long-term effect, for example, including a study by McCormick et al,108 which included a wide range of follow-up times (median 39 months [IQR 21]) and which reported that a greater than 50% improvement in pain and function was maintained in 58% of patients. Alternatively, in a retrospective review of 500 patients at 1 year, Yadav et al109 showed responder rates of ≤30% reduction in VAS of 60%, 44%, and 24% at 3, 6, and 12 months, respectively.

The conflicting evidence for durability and efficacy has resulted in society guidelines recommending this procedure for temporary relief in chronic mechanical LBP patients with only strict diagnostic protocols.92,95

Late Treatment Options

Surgical Interventions

The Spine Patient Outcomes Research Trial110 comparing surgical and nonsurgical treatments for patients with degenerative spondylolisthesis with CLBP with or without leg pain demonstrated that spinal fusion outcomes (pain and disability) were better compared with outcomes of patients who received nonsurgical treatments in an as-treated analysis. This finding was also compared with a nonrandomized observational cohort to overcome the methodological challenges of surgical trials and demonstrated similar results. Patients in this study had neurological claudication or radicular leg pain and back pain and thus are not a direct comparison to the CLBP alone phenotype discussed here. Still, the results show significant improvements in outcome and demonstrate the benefit of fusion in a well-selected patient. Other studies from the early 2000s, such as the Swedish Lumbar Spine Study111 and the Maine Lumbar Spine Study,112,113 employed open surgical techniques and compared spinal fusion to nonsurgical treatments for CLBP alone. Neither were able to identify significant differences in patient-reported outcomes and showed higher complication rates in the surgical cohort. Modern minimally invasive surgical techniques may improve these outcomes, but that has yet to be effectively demonstrated.

More recently, ISASS has taken a pragmatic stance on the role of fusion and disc arthroplasty for degenerative disc disease with back and leg pain based on the results of several RCTs and systematic reviews. Long-term fusion outcomes have been assessed from the control arms of arthroplasty investigational device exemption (IDE) studies. The Charité Food and Drug Administration (FDA) IDE trial114 has published 5-year outcomes on 43 anterior lumbar interbody fusion control patients and reported a 17-point improvement in ODI and a success rate per the IDE protocol of 51%. Similarly, the Pro-Disc FDA IDE trial has published the 2-year outcomes on 2-level circumferential fusion patients.115 A minimum 15-point improvement in ODI was seen in 60% of the fusion patients, and the mean VAS pain score dropped from 75 mm at pre-op to 38 mm at 2 years. In the fusion arm from a prospective study by Berg et al,116 72 patients with degenerative disc disease received posterior fusion (posterior lumbar fusion or posterior lumbar interbody fusion), and mean VAS back pain decreased from 59/100 at before the operation to 29/100 at 2 years, while ODI decreased from 41 to 23. This study shows that good clinical outcomes can be achieved in many patients. Brox et al117 compared fusion to nonsurgical therapy and showed that the fusion cohort improved by 4 years from 44.1 to 29.7. However, this was not significantly different from the cognitive and exercise-based interventions (43.4–27.0).

NASS,92 NICE,100 and the European Guidelines95 do not suggest spinal fusion for LBP without radicular symptoms, except in extraordinary circumstances.

REVIEW OF RESTORATIVE NEUROSTIMULATION

Target Anatomy; Medial Branch Motor Nerve Stimulation

The dorsal ramus nerve is a mixed sensory and motor nerve formed from the nerve roots and gives rise to the lateral and the medial branches on the superior border of the transverse process (Figure 6). The medial branch runs in the groove at the junction of the transverse and the superior articular process. Then it descends caudally and posteriorly, accompanying the vessels arising from the lumbar artery and vein. The medial branch innervates the capsules of the facet joints, skin, muscles, and
ligaments medial to the facet joint line. The paraspinal multifidus muscles are critical for lumbosacral stability and are innervated solely by the medial branch of the dorsal ramus, which is of particular clinical significance.

**Device, Procedure, and Therapy Description**

The system consists of an Implantable Pulse Generator, two stimulation leads, an radio frequency telemetry programmer, and a patient-controlled activator (Figure 7).

The stimulator and leads are implanted in a minimally invasive, single-stage procedure involving 2 skin incisions in the posterior lumbosacral region down through the adipose tissue. The patient is positioned in a prone position on a pillow to flatten out lordosis. A 1- to 2-cm incision for lead placement is made midsagittal, approximately at the level of the L4 vertebral body. From this incision, leads are percutaneously placed bilaterally, with the electrodes positioned adjacent to the medial branch of the dorsal ramus as it crosses the transverse process at L3 (Figure 8). Under fluoroscopic visualization, the leads are introduced percutaneously and advanced through the L2/3 intertransversarii using a needle, guide wire, and delivery sheath with a dilator (modified Seldinger approach). Withdrawal of the sheath deploys 2 opposing sets of flexible tines, which bracket either side of the intertransversarii for distal anchoring. A 4- to 5-cm incision is made near the iliac crest, and a pocket is created to implant the IPG. After confirming the placement of the leads through fluoroscopy, the IPG is attached to the leads to test for multifidi response through a trial (Figure 9). Response is confirmed through trial stimulation of the nerve and observation or palpation of the contractions of the multifidus.

Leads are tunneled subcutaneously between the lead implant incision and the IPG pocket, and once tunneled, the lead terminals are cleaned, dried, and inserted into the IPG header. Like other implantable neurostimulation systems, the IPG is implanted in a subcutaneous pocket, either in the low back or high buttock region. The IPG is placed in a location deemed appropriate by the implanting physician and considering the patient’s ability to reach the IPG location with the activator to initiate stimulation sessions.

The patient initiates stimulation sessions via the application of the activator. During stimulation sessions, the system repetitively delivers 10-second trains of electrical stimulation twice per minute for 30 minutes. During programming, the parameters stored in the IPG control the precise timing and stimulation intensity. Amplitude, pulse width, and rate may be adjusted in the clinic to control the energy (charge) and stimulation rate. Stimulation sessions are recorded within the IPG and can be accessed by the health care professional to assess compliance and use of the system.

In contrast to SCS, leads are placed outside the spinal canal, avoiding the risk of spinal cord injuries. And the risk of lead migration, which represents the most common adverse event reported in neurostimulation clinical trials, has been effectively mitigated with the lead design incorporating flexible fixation tines.

**Mechanisms of Action**

The mechanism of the reversibility of motor control dysfunction of the multifidus by the activation of muscles is achieved by a combination of changes to the muscle architecture and neurological control. Changes in muscle composition function have mainly been observed in animal and human models of targeted exercise rather than direct electrophysical stimulation. However, at face value, electrical stimulation is a more robust and consistent way of achieving specific activation.

Muscle activation has demonstrated changes in hypertrophy, muscle fiber type distribution, inflammation, mechanoreceptor function, adipogenesis, and...
fibrosis. The role of the central and peripheral nervous system is also mechanistically relevant as patients commonly develop potentially reversible changes in proprioception, motor cortex representations, excitation of the pain matrix, and peripheral and central hypersensitivity.

Danneels et al.\textsuperscript{118} measured significant changes in the multifidus muscle area following stabilization training combined with progressive dynamic-static resistance training. Berglund et al.\textsuperscript{119} demonstrated that specific motor control exercise reduced multifidus asymmetry in patients with nociceptive mechanical LBP. The severity of the disease in these patients was relatively mild (VAS 4–4.8/10). As such, the methodology may not be applicable, but these studies highlight that functional activation of the paraspinal muscles can elicit hypertrophy.

While the reversibility of back pain-induced loss of type I (slow twitch) muscle fibers\textsuperscript{120} in the multifidus has yet to be proven, studies in other muscles clarify the physiology involved. Muscle biopsy studies of endurance training, that is, multiple low-load contractions, demonstrated an increased proportion of type I fibers relative to type II/IIX. In human and animal studies, different muscles have shown variable propensities for fiber-type transition in various muscles and locations within those muscles\textsuperscript{121} and highlighted the role of appropriate neural input to maintain the fiber population\textsuperscript{122}.

Increased muscle fatiguability stems from altered motor control, and changes in the muscle architecture induce a persistent and dysregulated inflammatory process.\textsuperscript{83} These inflammatory processes drive further degenerative changes, including fibrosis and adipogenesis, and impact the function of tissue mechanoreceptors.\textsuperscript{123,124} The current hypothesis based on animal experiments is that improving muscle activity downregulates this inflammatory process and attenuates or reverses these changes.\textsuperscript{83,125,126}

Neurological inhibition and alterations to motor planning can develop from changes to the central and peripheral nervous system. Studies have shown an absence in the differentiation of motor cortex regions representing the erector spinae and multifidus, suggesting a central homogenization of control strategies.\textsuperscript{89,127} With appropriate simulation/muscle activation, the distinction between these regions can be reestablished,\textsuperscript{128} at least over short timeframes, but it is suspected that long-term patterning associated with cortical plasticity may reestablish fidelity in descending control strategies.\textsuperscript{129} The adverse effect that degenerative changes and persistent inflammation have on peripheral input into motor control via muscle spindles is also believed to be a reversible phenomenon when properly timed sensory neuron-intrafusal muscle fiber interaction is present.\textsuperscript{130}
Ultimately improvements at the cellular- and structural-level result in functional improvements in the motor control system. Restoration of appropriate motor planning at the cortical level, accurate proprioceptive input, and functional activation, both timing and durability of contractions, improve dynamic spinal stability. This stability restores the interactions between the active, passive, and control subsystems described by Panjabi and allows for the complicated tradeoff between spinal movement and spinal stability required for pain-free activities of daily living.

Role of Trialing in Restorative Neurostimulation

Although the economic cost and clinical benefit remain questionable, trialing is common in many neuromodulation procedures. The objective of a trial is to enrich the clinical success rate by determining whether an appropriate response relative to the mechanism of action can be elicited. Thus a trial must replicate the mechanism of action or demonstrate that the mechanism by which the permanent implant elicits efficacy can be achieved in a specific individual. In palliative approaches such as SCS, where the outcome of interest is pain, a trial should elicit a clinically meaningful treatment effect within the trial period. Where the mechanism is restorative and an accumulation of therapy is needed to elicit a treatment effect, it is not feasible for a trial to establish a measurable effect during a short-term trial period. In this case, the trial should show that the mechanism of action (MOA) is viable (or not). For example, a demonstration that stimulation of the medial branch during on-table intraprocedure testing does or does not invoke a contraction of the multifidus would be a way of excluding people unlikely to respond to restorative neurostimulation. In restorative neurostimulation, on-table testing is a predictor of outcomes outside of robust identification of the patient with appropriate physical examination and clinical workup.

PATIENT SELECTION CHARACTERISTICS

Consultation

Diagnosing mechanical CLBP resulting from neuromuscular instability begins by developing a complete clinical history of the patient’s pain presentation. The evaluation of neuropathic LBP has been detailed by Freynhagen et al. and Cook et al proposed several patient-reported signs related to clinical instability and mechanical LBP that, when combined, provide insight into the underlying pathophysiology for the treating physician. After ruling out red flags suggestive of a potentially serious cause for pain such as malignancy, fracture, infection, and cauda equina syndrome, Table 2 highlights some essential factors that should be investigated.

### High-Impact Pain

High-impact pain refers to pain experienced by a specific subset of patients that has significant negative consequences on activities of daily living. Work is ongoing on reaching a consensus and validating the categorization of pain impact states. Several approaches have been proposed and studied, including responses to various patient-reported outcomes score items. Regardless of categorization methodology, high-impact pain is classified based on the duration and severity of symptoms and the impact those symptoms have on the ability to work, perform usual activities, perform self-care activities, and find overall enjoyment of life. High-impact LBP is associated with a poor prognosis for recovery and a tendency for high health care resource utilization. A subanalysis of the ReActiv8 B population demonstrated that at baseline, 71% of patients presented with high-impact LBP, and by 2 years, 85% of patients reported little or no impact.

### Physical Examination for Multifidus Motor Control Dysfunction

**Prone Instability Test**

The Prone Instability Test (PIT) has adequate interrater reliability (reported as \( \kappa = 0.87 \)) and good face validity. A positive PIT was 1 of the 4 variables shown to predict success with a stabilization exercise.
program for patients with subacute LBP (a sample of 40 subjects with an average duration of 75 days) that included exercises designed to reactivate the lumbar multifidus. In another study, of 105 patients with an average duration of 65 days, positive PITs and aberrant movement patterns were shown to have reduced disability and pain following a course of motor control retraining exercises when compared with patients who did not have these clinical findings.

The PIT is performed with the patient prone in a relaxed and neutral spine posture. The tester applies posterior to anterior glides (pressure) over each lumbar segment. If 1 or more glides produce pain, the glides are repeated when the subject’s posterior spinal muscles are activated, extending the hips by lifting the feet off the floor. If the pain is significantly diminished when the glides are performed during muscle activation, the test is considered positive and suggestive of a motor control deficit, including multifidus dysfunction (Figure 10).

**Multifidus Lift Test**

Hebert et al reported the reliability of the multifidus lift test (MLT), a palpation technique designed to test for multifidus function (Figure 11). Herbert et al used the MLT procedure and compared the results of palpation to determine diminished multifidus response compared with normal multifidus contraction to measure multifidus muscle thickness change via sonography. Inter-rater reliability of the MLT at the L4–L5 was reported to be $\kappa = 0.75$ with 86% agreement and at the L5–S1 level demonstrated a $\kappa = 0.81$ with 91% agreement. To establish the validity of the test, they assessed the correlation between the outcome of the MLT and the ultrasound measure of thickness change. The correlation coefficients demonstrated a consistent relationship (0.59–0.73, $P < 0.01$) between the MLT findings and the ultrasound measures of lumbar multifidus function at L4–L5 and L5–S1.


![Figure 11. Multifidus lift test. Source: Reprinted from Chakravarthy et al. Restorative neurostimulation: a clinical guide for therapy adoption. *J Pain Res.* 2022;15:1759–1774 under the Creative Commons Attribution Non-commercial license. The negative and positive responses have been reversed from the published version.](image)
Aberrant Movements

Observing the quality of truncal motion is essential to a spinal physical examination and can highlight deficits in motor control and multifidus function (Figure 12). Five specific movement patterns are relevant: altered lumbopelvic rhythm, Gower’s sign, sagittal plane deviation, instability catch, and painful arc of motion. The observation by trained clinicians has a high interobserver reliability ($\kappa = 0.35–0.89$) and was associated with patients with CLBP compared with healthy controls or patients with remission of symptoms.

Imaging Characteristics

Changes to the multifidus muscle apparent with MRI are strongly associated with back pain. However, since back pain of any cause can lead to changes in the multifidus cross-sectional area and amount of fat infiltration, the diagnostic value of such muscle changes alone is limited in individual patients. There appears to be no relationship between multifidus function and the amount of fat infiltration. There is some evidence that the severity of fat infiltration correlates with decreased range of motion in flexion and that the amount of fat infiltration may be a predictor for continued CLBP, but the diagnostic utility of imaging alone is unclear.

Figure 13 highlights the MRI classifications of fatty infiltration.

CLINICAL EVIDENCE SUMMARY

Current Publications

The evidence supporting the clinical effect of restorative neurostimulation comes from several multicenter and 1 single-center study (Table 3).

The key points of these studies include the following:

- Studies consistently show that most patients report substantial and durable improvements in pain, disability, and quality of life.
- Clinical benefits accrue over time, consistent with the restorative mechanism of action.
- 71% of the patients (36 of 51) had voluntarily discontinued (49%) or reduced (22%) opioid
intake at 3-year follow-up in the ReActiv8-B study.

ReActiv8-B Pivotal Trial

The ReActiv 8-B Pivotal trial is a recent randomized, sham-controlled, double-blinded clinical trial (clinicaltrials.gov NCT02577354) that included 204 patients, and outcomes provided safety and effectiveness evidence that supported FDA approval restorative neurostimulation using the ReActiv8 Device.35

Participants had a mean age of 47 ± 9 years, and 54% were women. The mean duration of CLBP was 14 ± 11 years (7 months to 50 years) from the onset of the first occurrence, and the mean percentage of days with LBP in the previous year was 97% ± 8%. The mean VAS was 7.3 ± 0.7 cm, the mean ODI was 39 ± 10, and the mean EQ-5D-5L index was 0.585 ± 0.174. All participants had undergone physical therapy with an average of 31 ± 52 sessions. Of all participants, 12% had undergone medial branch rhizotomy (>1 year before enrollment), 49% had received spinal injections (>30 days before enrollment), and 37% were taking opioid analgesics for LBP. Although the primary endpoint was inconclusive, overall data from this trial’s (successfully) blinded phase are consistent with a clinically meaningful benefit at 120 days. After unblinding and switching from sham to therapeutic stimulation in the sham-control group, improvements increased to the 1 year mark in the combined cohort. At 1 year, 64% of patients reported an improvement in VAS of ≥50%, 52% of patients reported LBP resolution (VAS ≤2.5), and 69% reported an improvement of ≥15 points in ODI. No lead migrations were reported, and the overall safety profile was favorable compared with other implantable neurostimulator therapies for chronic pain.

Three-Year Outcomes

Three-year peer reviewed evidence from the ReActiv8-B152 study suggests that the clinical benefits are maintained and show durable, statistically significant, and clinically substantial help in this cohort. Consistent with the restorative mechanism and improvements in neuromuscular control, participants derived benefits that increased with treatment duration. To date, 4-year outcomes have been presented at national meetings. Although these outcomes have not been peer reviewed yet, they are included here for completeness.153

ReActiv8-PMCF Study

The ReActiv8-PMCF is a postmarket clinical follow-up of 37 patients from 5 sites in the United

Table 3. Current published evidence for restorative neurostimulation for CLBP.

<table>
<thead>
<tr>
<th>Clinical Study</th>
<th>Number of Participants</th>
<th>LOF</th>
<th>Evidence Level</th>
<th>Publication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feasibility study</td>
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<td>3 mo</td>
<td>II-1</td>
<td>Deckers et al 2015145</td>
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<tr>
<td>ReActiv8-A</td>
<td>53</td>
<td>12 mo</td>
<td>II-1</td>
<td>Deckers et al 2018146</td>
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<td>ReActiv8-PMCF</td>
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<td>II-1</td>
<td>Mitchell et al 2021147</td>
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<td>24 mo</td>
<td>II-1</td>
<td>Thomson et al 2021148</td>
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<td></td>
<td>36 mo</td>
<td>II-1</td>
<td>Thomson et al 2021149</td>
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<tr>
<td>ReActiv8-B</td>
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<td>120 d/12 mo</td>
<td>II-1</td>
<td>Gilligan et al 2021150</td>
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<td></td>
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<td>24 mo</td>
<td>II-1</td>
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<td>48 mo</td>
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<tr>
<td></td>
<td>Subanalysis</td>
<td>12 mo</td>
<td>II-1</td>
<td>Shaffrey et al 2022155</td>
</tr>
</tbody>
</table>

Abbreviation: LOF, length of follow-up.
This study represents real-world clinical outcomes as the inclusion/exclusion criteria aligned with the device instructions for use at the treating physician’s discretion. These criteria resulted in demographically very similar patients to the pivotal trial, with an average age of 47.2 ± 11 years and an LBP history of 13.7 ± 10.2 years. However, at baseline, the mean NRS pain score was 7.0 ± 0.2, mean ODI score was 46.6 ± 2.2, and mean quality-of-life index (EQ-5D-5L) was 0.426 ± 0.035, indicating there was worse disability and HRQoL than the ReActiv8-B cohort. After 2 years of therapy, average NRS scores had reduced to 3.5 ± 0.3 (P < 0.001), ODI scores decreased to 29.2 ± 3.1 (P < 0.001), and the EQ5D-5L index improved to 0.680 ± 0.030 (P < 0.001). The improvement in mean NRS between 1 and 2 years also met the threshold for statistical significance (P < 0.001). The recent publication of the 3-year outcomes shows continued durable and meaningful benefit in this patient group.\(^{149}\)

ReActiv8-C Registry

The ReActiv8 C Registry is a postmarket study undertaken in Germany. One publication has arisen from this, a single center consecutive cohort, although more results will likely be forthcoming.

**Single Center Interim Analysis**

Ardeshiri et al\(^{154}\) published 12-month outcomes from 44 consecutive patients. Patients in this cohort were, on average, slightly older than in previous studies (54 years) and had a shorter reported history of CLBP (5.8 years). While baseline outcome measures were worse than other reports, the magnitude of improvement and responder rates were comparable to those seen in other publications. Pain scores (NRS) improved from a baseline of 7.6 ± 0.2 to 3.9 ± 0.4 at 1 year. Disability at baseline improved from 43.0 ± 2.8 to 25.8 ± 3.9 and HRQoL (EQ-5D) from 0.504 ± 0.034 to 0.755 ± 0.039.

**COMBINED OUTCOMES OF REACTIV8-A, REACTIV8-B, REACTIV8-C, AND REACTIV8-A-PMCF—1- TO 4-YEAR COMPLETERS**

The cumulative data supporting restorative neurostimulation from several well-constructed prospective studies and real-world data through 4 years suggest the durability of the treatment effect in these patients. Figure 14 compares the published outcomes across studies in (a) pain, (b) disability, and (c) HRQoL.
showing that the magnitude of benefit is sustained and that real-world outcomes are similar to those achieved in the regulated clinical trials.

**GUIDANCE ON APPROPRIATE COVERAGE CRITERIA**

Patients who have documented evidence of the following clinical indications may be eligible for coverage:

- Patient has severe, disabling, CLBP that has persisted for more than 12 months with pain on more than 50% of the days.
- Patient has failed all conservative medical management, including a minimum of both:
  - Physical therapy
  - Medication (nonsteroidal anti-inflammatory drugs, muscle relaxants, and other).
- Documented multifidus dysfunction through physical examination (PIT/MLT or aberrant movement patterns) or imagery (MRI)
- Pain is consistent with a predominant mechanical/nociceptive origin.

Patients who are candidates for spine surgery are excluded.

**CURRENT COSTS ASSOCIATED WITH THE TREATMENT OF CLBP**

LBP accounts for 7.6% or 42.5 million years lived with disability, and a recent study estimated that the United States spent US$134.5 billion on health care for low back and neck pain in 2016, leading the top 154 conditions. The data on the economic impact such as this is often stratified across the severity of the disease spectrum. However, the actual consumption of health care resources is disproportionately skewed toward those with more severe disease states. Patients with high-impact chronic pain were demonstrated to use more than double the overall and spine-related health care costs compared with patients less severely affected.

Using data from the Medicare Expenditure Panel Survey, Herman et al estimated overall and spine-related annual health care costs for individuals with high-impact chronic pain to be $14,661 and $5979, respectively. These were significantly greater than other chronic pain patients that did not have a high impact of their pain on work, self-care, and activities of daily living ($6371 overall and $2300 spinal).

Spears et al attempted to differentiate the cost of care for CLBP patients who were not candidates for spine surgery using a claims analysis from the IBM Marketscan Database. Of the approximately 56,000 patients identified with chronic refractory LBP, the upper quartile (14,000 patients) all experience costs greater than $14,000 in the first- and second-year postindex diagnosis, with the majority of costs coming from outpatient services.

To estimate the economic impact of restorative neurostimulation, Shaffrey and Gilligan stratified patients to pain impact status as a predictor of health care resource utilization. In a completer analysis of 146 patients, 103 (71%) were in a high-impact pain state at baseline. By the second year of therapy, 15 (10%) remained high impact and 124 (85%) reported no or low impact pain only. The previous studies suggest that utilization and costs are directly proportional to chronic pain impact; in combination with these data, these findings provide some evidence for cost efficacy.

**CODING FOR RESTORATIVE NEUROSTIMULATION**

**ICD-10-CM Codes for Coverage**

Restorative neurostimulation is a new approach to managing mechanical LBP specific to patients who suffer from neuromuscular instability from multifidus dysfunction. The resulting characteristics often appear as atrophy and wasting of the lower back muscles. Atrophy and weakening of muscles can result in weakness, decreased range of motion, and other functional impairments that are causal for LBP.

Typical International Classification of Diseases, 10 Revision, diagnosis codes that indicate medical necessity are as follows:

- Primary diagnosis: M62.5A2 (atrophy and wasting of muscles, not elsewhere classified, back, lumbosacral)
- Secondary diagnosis: M54.59 (low back pain, other)

**Current Procedural Terminology**

The procedures invoking restorative neurostimulation may include the use of a peripheral nerve stimulator and placement of percutaneous neurostimulator electrodes for which category 1 Current Procedural Terminology (CPT) codes are available to report. These codes include:
CPT1 - 64590—Insertion or replacement of peripheral or gastric neurostimulator pulse generator or receiver, direct or inductive coupling.

CONCLUSION

Restorative neurostimulation for chronic mechanical LBP is supported by several clinical studies that show robust and durable clinical effects over the pretreatment condition. The totality of evidence suggests that in a well-selected patient population who have exhausted conventional care paradigms, the potential benefits outweigh the risks and costs. These patients tend to be exposed to multiple therapies with limited durability, resulting in a continuous cycle of high-cost health care utilization. Restorative neurostimulation should be considered for clinically appropriate patients who have exhausted reasonable conservative approaches.

LIMITATIONS

1. Industry funding is a potential source of study bias for the available data reviewed.
2. A limited number of studies is available.

REFERENCES

ISASS Statement: Restorative Neurostimulation for Chronic Mechanical LBP
International Journal of Spine Surgery, Vol. 17, No. 5


