Invasive spinal cord neuromodulation in the treatment of chronic musculoskeletal pain

A neuromodulação invasiva da medula espinal no tratamento de dor crônica musculoesquelética

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Abstract

Neuromodulation for analgesic purposes comprises non-ablative strategies that modulate neuronal and glial activity to treat refractory chronic pain. It can be pharmacological (gabapentin, antidepressants, medical cannabis), transcutaneous (acupuncture, transcutaneous electrical nerve stimulation (TENS), blocks), or invasive, with spinal cord stimulation (SCS) standing out. In SCS, epidural electrodes apply pulses to the dorsal cord, blocking spinal nociceptive pathways, activating descending systems, and modulating the sympathetic system, with clinically relevant efficacy in approximately two-thirds of cases. Complications include electrode migration or rupture, cerebrospinal fluid leak, hematomas, and spinal cord injury, especially in previously operated spines. Mechanistically, SCS has evolved from the Gate Control Theory to the concept of "neuromatrix", involving spinal and suprasegmental circuits; it restores GABAergic inhibition, reduces excitatory mediators (e.g., glutamate), and attenuates central and peripheral sensitization. The main indications cover persistent low back pain, complex regional pain syndrome (CRPS), peripheral vascular disease, diabetic neuropathy, and post-arthroplasty pain, with better outcomes in appendicular pain. Critical parameters include the number

Resumo

A neuromodulação para fins analgésicos compreende estratégias não ablativas que modulam a atividade neuronal e glial para tratar a dor crônica refratária. Pode ser farmacológica (gabapentinoides, antidepressivos, cannabis medicinal), transcutânea (acupuntura, TENS, bloqueios) ou invasiva, destacando-se a estimulação elétrica da medula espinal (EEE). Na EEE, eletrodos epidurais aplicam pulsos ao cordão posterior, bloqueando vias nociceptivas medulares, ativando sistemas descendentes e modulando o sistema simpático, com eficácia clinicamente relevante em cerca de dois terços dos casos. As complicações incluem migração ou ruptura de eletrodos, fístula liquórica, hematomas e lesão medular, sobretudo em colunas previamente operadas. Mecanisticamente, a EEE evoluiu da Teoria das Comportas para o conceito de "neuromatriz", envolvendo circuitos espinais e suprassegmentares; restaura a inibição gabaérgica, reduz mediadores excitatórios (p.ex., glutamato) e atenua a sensibilização central e periférica. As principais indicações abrangem lombalgia persistente, síndrome dolorosa regional complexa (SDRC), doença vascular periférica, neuropatia diabética e dor pós-artroplastia, com melhores resultados em dores apendiculares. Parâmetros críticos incluem número de

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of poles, frequency, pulse width, and amplitude. Tonic paresthesia-based waveforms (20–90 Hz) and paresthesia-free modalities (>200 Hz), such as burst, high-frequency, and differential target multiplexing (DTM), can be combined and individualized; evidence suggests benefits of high frequencies and burst stimulation, especially for the affective-emotional components of pain. Dorsal root ganglion (DRG) stimulation offers superior control in CRPS and certain low back pain conditions, acting as a peripheral ionic "filter". Current trends include closed-loop systems with target "electrical dose", AI customization, and miniaturized hardware. Although cost and limited knowledge hinder diffusion, neuromodulation is emerging as a scalable, cost-effective therapy for complex musculoskeletal pain.

Keywords: Chronic pain; Transcutaneous electric nerve stimulation; Complex regional pain syndrome; Spinal cord stimulation.

polos, frequência, largura de pulso e amplitude. Ondas tônicas parestésicas (20–90 Hz) e modalidades não parestésicas (>200 Hz), como burst, alta frequência e DTM, podem ser combinadas e individualizadas; evidências sugerem benefício das altas frequências e do burst, especialmente nos componentes afetivo-emocionais da dor. A estimulação do gânglio da raiz dorsal (DRG) oferece controle superior em SDRC e em certos quadros lombares, atuando como "filtro" iônico periférico. Tendências atuais incluem sistemas em circuito fechado com "dose elétrica" alvo, personalização por IA e hardware miniaturizado. Apesar do custo e do desconhecimento limitarem a difusão, a neuromodulação consolida-se como terapia custo-efetiva escalável para tratara dor musculoesquelética complexa.

Palavras-chave: Dor crônica; Terapia de Neuromodulação Elétrica Percutânea; Síndrome da dor regional complexa; Estimulação da medula espinal.

Introduction

Chronic pain treatments can be ablative, through damage to parts of the central and/or peripheral nervous system, or non-ablative, providing analgesia while preserving the nervous system. Invasive neuromodulation is an expanding non-ablative method with several strategies that alter neuronal and glial activity in painful and psychiatric conditions.

Neuromodulation can be pharmacological with anticonvulsants such as gabapentin, dual and tricyclic antidepressants, medical cannabis, etc.; transcutaneous with acupuncture, dry needling, anesthetic nerve blocks, transcutaneous electrical nerve stimulation (TENS); trans- or intracranial; or invasive neuromodulation of the posterior spinal cord through implantation of electrical stimulation electrodes.

Spinal cord neurostimulation works by applying electrical pulses to the posterior cord (gracilis and cuneate funiculi) that block spinal pain pathways (dorsal horn), activate supraspinal descending analgesic pathways (brainstem and cortex), and modulate the sympathetic branch of the autonomic nervous system, among other effects.

Spinal cord stimulation is used for refractory chronic pain, with studies demonstrating analgesic efficacy in approximately 2/3 of patients (pain reduction and analgesic consumption > 30%). Stimulation is achieved with electrical impulses delivered through electrodes implanted in the posterior epidural space via a percutaneous approach (cylindrical leads) or a surgical approach (paddle leads). These are connected to a subcutaneously implanted generator, which is programmed and recharged via telemetry (Figure 1).

Trial electrodes can be used in cases of uncertainty or when required by the payer, usually for 3 to 7 days. The use of a trial is controversial, as it increases the risk of infection and placebo and nocebo effects.

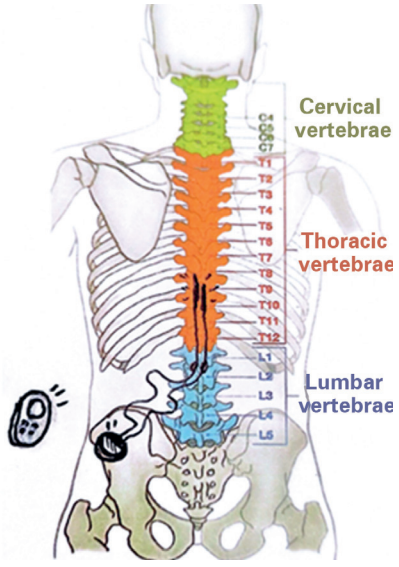

Figure 1. Representation of the spinal cord neurostimulator. Percutaneous electrodes are implanted via lumbar epidural route and advanced to the thoracic region, they are connected to a subcutaneous pulse generator, which is programmed and recharged via telemetry.

Electrode breakage or migration, cerebrospinal fluid leak, spinal cord injury, and epidural hematomas are dangerous complications, more frequent in cases with previous spinal interventions that cause fibrosis and/or loss of anatomical planes[1,2].

In this study, we will explore invasive spinal cord neurostimulation in the treatment of complex chronic musculoskeletal pain, discuss the types of stimuli and mechanisms of action of tonic or low density waves, called paresthesia (<125 Hz), high density waves, called non-paresthesia (>200 Hz) and burst waves, known as "burst" ("mixed" frequency of 40 Hz with intrapulses of 500 Hz).

Mechanisms of action of spinal cord stimulation

Spinal cord stimulation was based on Melzack and Wall's Theory of Behaviors[3] published in 1965, which postulated competition between large (faster in transmitting stimuli) and small (slower) fibers. The former preferentially transmit non-painful (proprioceptive) stimuli, while the smaller ones (with little or no myelination) transmit painful stimuli.

When both fibers (large and small) are stimulated, the larger ones mainly activate non-painful connections and block pain (Figure 2). Based on this, Shealy et al.[4] in 1967 implanted an electrode over the dorsal column of a patient's spinal cord, achieving analgesia and reporting for the first time successful pain control with this therapy.

Epidural electrical stimulation acts directly on the large fibers of the dorsal column, and indirectly on the glia and neurons of the dorsal horn of the spinal cord, with GABAergic activation of interneurons and anti-inflammatory glial activation, cholinergic modulation of the sympathetic nervous system, and descending analgesic activation of the brainstem and brain[5] (Figure 3).

After many criticisms of the simplistic gate control model, Melzack[6] in 1999 revised his hypothesis on fiber competition in the dorsal horn of the spinal cord and included suprasegmental circuits, calling it the "neuromatrix" or "neurosignature" of pain, which involves multiple limbic, cognitive-emotional connections shaped by painful experiences throughout life, conferring uniqueness to each individual's pain[7,8].

During pain chronification, there is loss of GABAergic neurons in the dorsal horn, thus weakening inhibitory circuits. This loss increases the activity of excitatory glutamatergic neurons with a wide dynamic range (WDR), which causes hyperexcitability in the dorsal horn of the spinal cord. Both medications, such as baclofen or gabapentin (GABA analog), and spinal stimulation activate these remaining GABAergic circuits by inhibiting pain[9,10].

Intense or repeated painful stimuli increase concentrations of excitatory mediators such as glutamate, aspartate, tachykinins (A, B, and Substance P), and Calcitonin Gene-Related Polypeptide (CGRP) in the dorsal horn of the spinal cord, hyperexciting interneurons of the pain pathways.

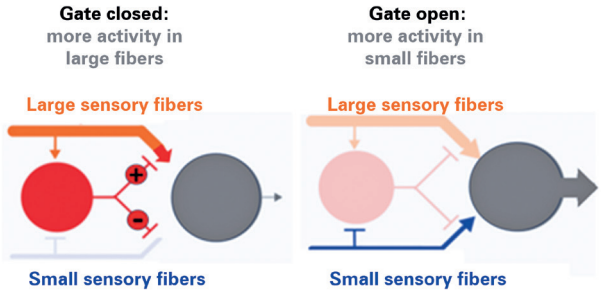

Figure 2. Large, non–pain-related fibers conduct stimuli faster, activating tactile and proprioceptive circuits that inhibit pain circuits mediated by small fibers when both are activated.

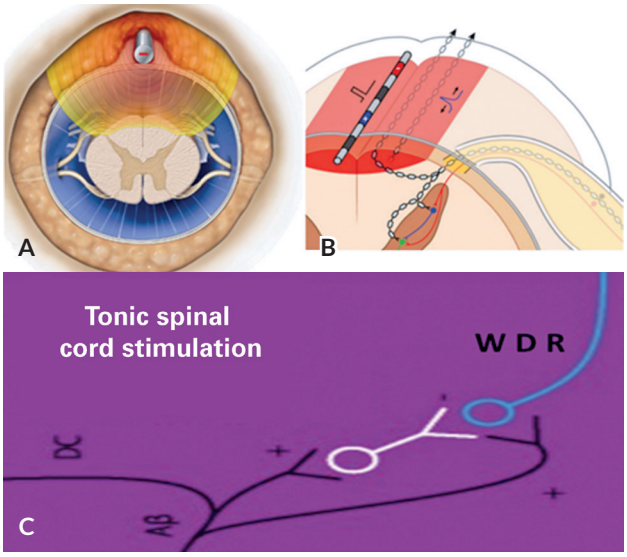

Figure 3. Percutaneous electrode at the midline (A) and paramedian (B) during dorsal column (DC) stimulation, activating large fibers (Aβ - black) with branches that activate (+) inhibitory interneurons (white) in the dorsal horn of the spinal cord, thereby preventing activation of the WDR spinotalamic neuron (blue) (C).

This "excitatory soup" in the dorsal horn affects thousands of synapses within the spinal interstitium, stimulating neighboring synaptic receptors. This central sensitization also modifies pseudounipolar neurons (dorsal root ganglia), both by enlarging the peripheral receptor field and by reducing nociceptor activation thresholds (peripheral sensitization).

Ascending and descending proximal axonal branches of sensory neurons transmit action potentials to cranial and caudal levels, thereby sensitizing neurons beyond the somatotopic segment, a phenomenon known as pain "spread" (progressive expansion of the painful area). Desensitization induced by spinal cord stimulation reduces pain elicited by stimuli known to be nonpainful[11,12].

Experimental studies have shown decreased glutamate levels in the dorsal horn of rodents undergoing spinal cord stimulation, increasing pain tolerance. Electrical stimulation of glial cells induces neuroinflammatory changes that contribute to another mechanism of chronic pain control[13,14].

Clinical indications of spinal cord neuromodulation

Posterior epidural electrical stimulation of the spinal cord is indicated for chronic pain refractory to more conservative treatments. Patient selection is important for outcomes; however, as with any chronic pain treatment, outcomes vary. The main indications are persistent low back pain, CRPS, peripheral vascular disease and painful diabetic neuropathy. The best outcomes occur in appendicular pain[15,16.]

Spinal cord stimulation has been shown to reduce chronic neuropathic pain and improve quality of life at 6, 12, and 24 months[17]. Rigoard et al.[18] also demonstrated significant improvement in pain relief over 24 months in patients with Persistent Spinal Pain Syndrome (former term for Failed Back Surgery Syndrome) with spinal cord stimulation compared to the best alternative medical care, establishing spinal cord neurostimulation as the best treatment for "untreatable" chronic low back pain.

Low back pain persistence syndrome is the main indication of spinal cord stimulation. In the USA[19,20], for example, the patient with low back pain undergoes three surgeries before the neurostimulator. Kumar et al.[20] reported an 85% good response in early implants (less than 2 years), but only 9% in late implants with more than 10 years of pain. Thus, early implantation in cases of "intractable" pain is essential for better outcomes.

Huttunen et al. (2022)[21] studied 150,000 patients 30 years after lumbar spine surgery and found that repeat surgery was the main risk factor for persistent pain and an indication for spinal cord stimulation (3.6% in the second surgery and 11.9% after the fifth surgery), suggesting caution when considering repeated reoperations in the lumbar spine.

Spinal cord stimulation controls chronic pain well in limbs affected by neuropathy, ischemia, and/or complex regional pain syndrome[22-27].

Kumar et al. (2006)[22] reported a 74% improvement in painful feet, improvement maintained even after eight years of spinal cord stimulation in 410 patients. Cameron[28] reviewed 68 publications and enrolled 747 patients with chronic foot pain, showing a mean improvement in 62% of patients (more than 50% relief) over three years. It is noteworthy that the target for spinal cord stimulation for chronic axial low back pain is T9/T10, whereas for lower-limb pain, the target is lower, usually T11/T12.

In complex regional pain syndrome, the initial soft-tissue injury or fracture does not correspond to the final clinical picture, which is associated with sympathetic nervous system hyperactivity, including a temperature difference (at least 1°C) between the affected and unaffected extremity; skin, nail, and hair changes; joint stiffness; tactile hypersensitivity; and local edema[29].

Spinal cord neurostimulation is an excellent indication for treatment[30,31], and more recently, direct neurostimulation of the DRG appears to be even more effective[32,33] (Figure 4). Although less frequent, there are cases of complex regional pain syndrome in adolescents and children, and the use of spinal cord stimulation or DRG can be used in cases refractory to more conservative treatments[34-36].

Persistent pain after joint arthroplasty is not rare, affecting 10% of patients after total hip arthroplasty (THA) and 20% after total knee arthroplasty (TKA)[37-39].

Oral polypharmacy, nerve blocks, and periarticular injections can reduce pain in most cases, but neurostimulation is indicated in refractory cases, obviously after ruling out infection, loosening, or prosthesis failure[40]. Spinal cord stimulation offers pain reduction, improved mobility, and less dependence on analgesics[41,42].

Neuromodulation parameters

Neurostimulation parameters include the number of poles (cathode - and anode+), frequency (activations

per second – Hertz), pulse width (distance traveled by the current, in microseconds), and energy amplitude (mVolt or voltage/resistance = Ampere). Adjusting these parameters is essential for treatment efficacy (Figure 5).

Tonic or paresthetic stimulation (tingling) occurs at low density, usually between 20 and 90 Hz. It activates ascending proprioceptive spinal tracts in an orthodromic manner (i.e., in the physiological direction of axonal conduction) and antidromically (i.e., in the reverse direction), thereby activating inhibitory interneurons in the dorsal horn of the spinal cord, DRG, and the sympathetic nervous system[43,44].

Over the past decade, new waveforms known as nonparesthetic stimulation have begun to be used in clinical practice, including burst stimulation, high-frequency stimulation (10 kHz), and differential target multiplexed (DTM) stimulation. Both paresthetic and nonparesthetic waveforms activate inhibitory GABAergic interneurons in the dorsal horn, but only tonic waveforms (< 200 Hz) activate the dorsal fibers of the proprioceptive tract[45-50].

Although clinically effective, tonic therapy is associated with 30%-50% long-term failure rate. With the emergence of nonparesthetic waves (> 200 Hz), efficacy rates improved[51-53]. North et al.[51] observed that patients with loss of response to tonic stimuli improve when switching to high-frequencies of 1 kHz. The conversion of stimulation from low to high frequency can restore analgesic control in patients with loss of response[54].

High-frequency 10-kHz stimulation is not available in Brazil. This ultra-high frequency requires a very low pulse width (30 µs) and amplitude (1–5 mA) because it consumes excessive energy[52,53].

Some studies suggest that 1 kz can be as efficient as 10 kHz. North et al. (2020)[55] studied 99 patients with chronic low back pain treated with 10 KHz or 1.5 KHz stimulation, with 92% and 84% pain improvement, results much higher than the 50% typically reported for conventional tonic stimulation.

Burst stimulation arises from the observation of two distinct patterns of impulses in the CNS: tonic and burst[56]. The "BurstDR" frequency consists of 40 Hz

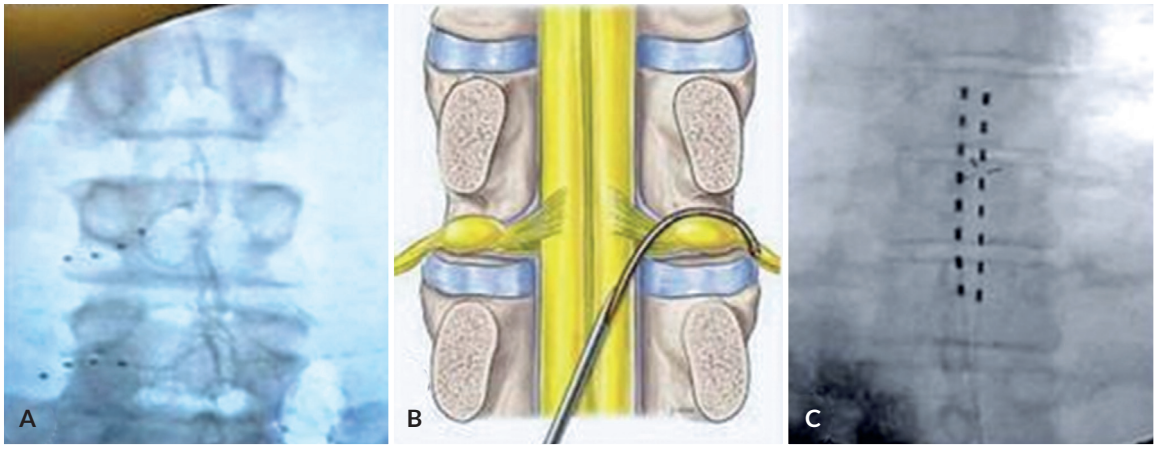

Figure 4. Percutaneous electrodes in the intervertebral foramina on the left radiograph (A), drawing in the center (B), and epidural placement on the right radiograph (C).

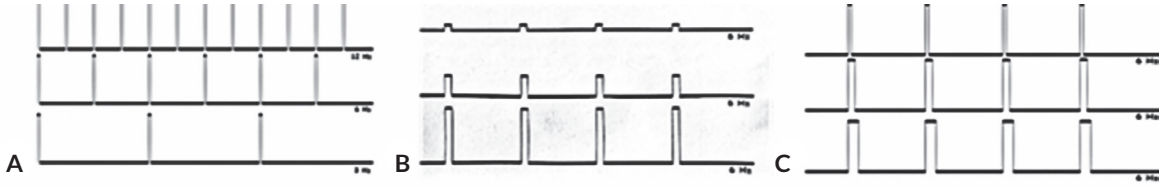

Figure 5. We can increase the "number" of waves per second to 3, 6, or 12 Hz (A); increase the stimulus intensity (amplitude or "wave height") (B), or maintain the same frequency and amplitude but increase the pulse duration ("wave width") (C).

pulses with five "intra-peaks" of 500 Hz, followed by a 1 ms pause. Studies on chronic pain control were clinically superior to conventional stimulation[57,58].

Severe psychological stress, such as depression and catastrophizing, is part of the overall management of patients with chronic pain. Burst waveform therapy appears to attenuate some emotional aspects by targeting the medial pain pathways (paleospinothalamic tract) that project to the anterior cingulate cortex and insula—structures involved in pain chronification (Figure 6)[59,60].

The lateral tract of small fibers (neospinothalamic) projects to the lateral cortex (S1/S2) and is primarily discriminative, whereas the affective-emotional component of pain ascends through the medial tract of small fibers (paleospinothalamic). The anterior cingulate cortex potentiates excitatory synapses in the spinal cord, thereby facilitating chronification—another example of a circuit within the pain neuromatrix[61-63].

Hagedorn et al. (2022)[64] followed 128 patients receiving burst stimulation for pain control for two years; patients meeting criteria for severe stress achieved a 71% improvement in catastrophizing and a 58% improvement in depression, with reduced or discontinued antidepressant use.

Yearwood et al. (2020)[65] analyzed PET-CT images from seven patients treated with tonic stimulation, followed by burst stimulation, and demonstrated medial and lateral spinothalamic activation during burst stimulation, but only lateral activation during conventional tonic stimulation.

Despite the recent emergence of subperception waveforms (> 200 Hz), some patients still prefer conventional paresthesia-based stimulation. Because different biological processes are involved, the ideal therapy may require a combination of paresthetic and nonparesthetic waveforms (combined waveforms)[66-73].

For several reasons, patients prefer different programming settings, and each patient may require an individualized program. In Brazil, the combined-waveform platforms Evolve (Medtronic) and Wave-Writer (Boston Scientific) are currently available. Burst stimulation (Abbott) is also available and is considered a combined waveform because it uses 40 Hz with intra-peaks of 500Hz.

Recent multicenter studies have demonstrated this waveform-based "individualization." Rigoard et al. (2024)[74] followed 58 patients for seven years and observed the best response with tonic stimulation in 34.4%, subperception stimulation in 44.8%, and combined waveforms in 20.7%.

Another European study including 188 patients followed for 12 months reported a better clinical response with subperception stimulation in 24% of patients, tonic stimulation in 14%, burst/microburst mode in 18%, and combined waveforms in > 40%.[75] Piedade et al. (2022)[76] studied 22 patients with

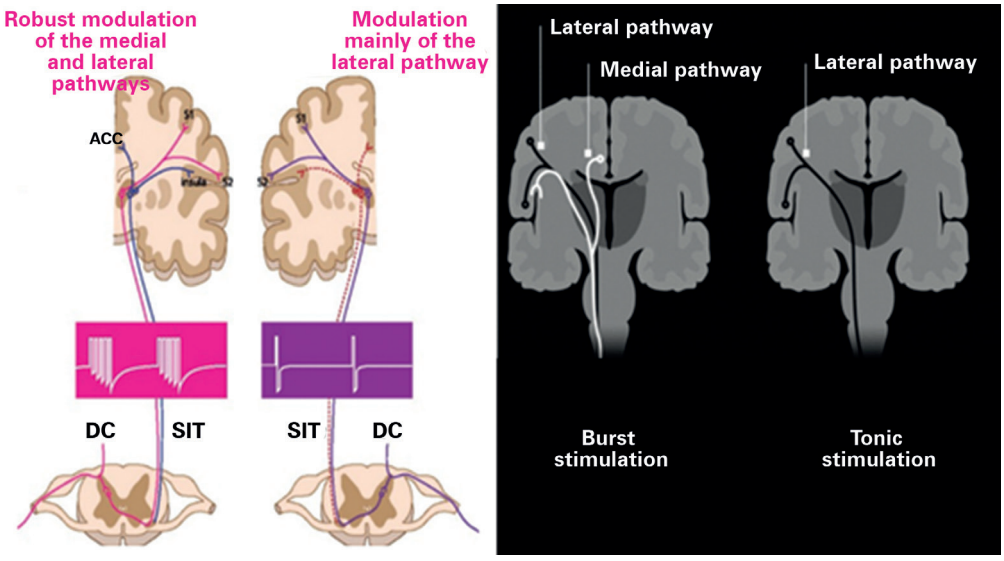

Figure 6. Burst stimulation can activate the lateral (discriminative) and medial (affective) pathways of the spinothalamic tract. Conventional (tonic) stimulation stimulates only the lateral (neospinothalamic) pathway.

"intractable" chronic pain who underwent epidural stimulation implantation. After three months, 25% chose microburst mode, 25% tonic waveforms, 20% combined waveforms, and 20% preferred burst stimulation.

Direct stimulation of the DRG places the electrode in the intervertebral foramen rather than in the epidural space and has shown excellent results (Figure 7), with greater reduction of sympathetic hyperactivity, which is common in complex regional pain syndrome[77-80] and in intractable chronic low back pain[81-84].

The DRG is composed of pseudounipolar neurons—a neuronal type unique in the entire nervous system. Its T-shaped axonal anatomy allows the formation of an ionic channel "filter" that blocks most impulses that would otherwise proceed to the dorsal horn of the spinal cord. This T-junction is considered by some authors to be the "current portal of pain" (Figure 8)[85,86].

The advent of closed-loop stimulation was another major advance in spinal cord stimulation[87-90]. Levy et al. (2024)[91], using closed-loop stimulation, established 2.8 μV as the ideal value for pain relief in 180 patients. This study provided, for the first time, a specific dose for optimizing spinal cord stimulation intensity.

By processing data from clinical studies with artificial intelligence programs, it may become possible to

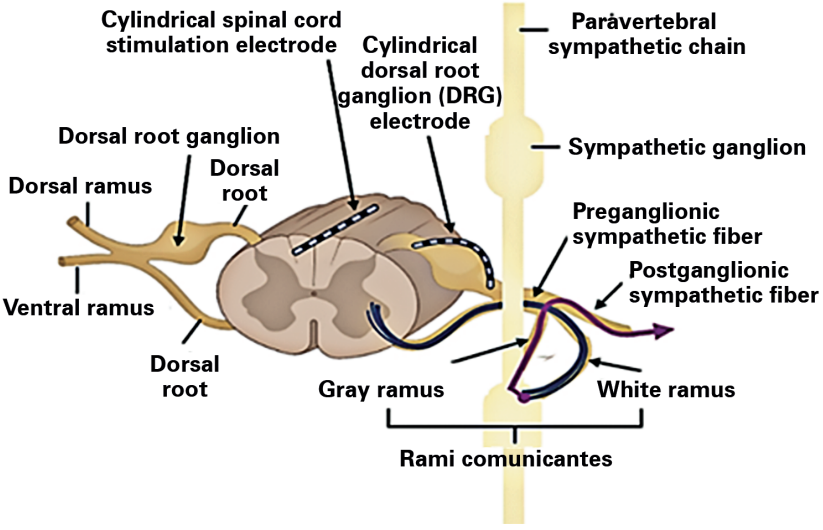

Figure 7. The illustration shows the positioning of the spinal cord and dorsal root ganglion electrodes and their anatomical relationship with the sympathetic system.

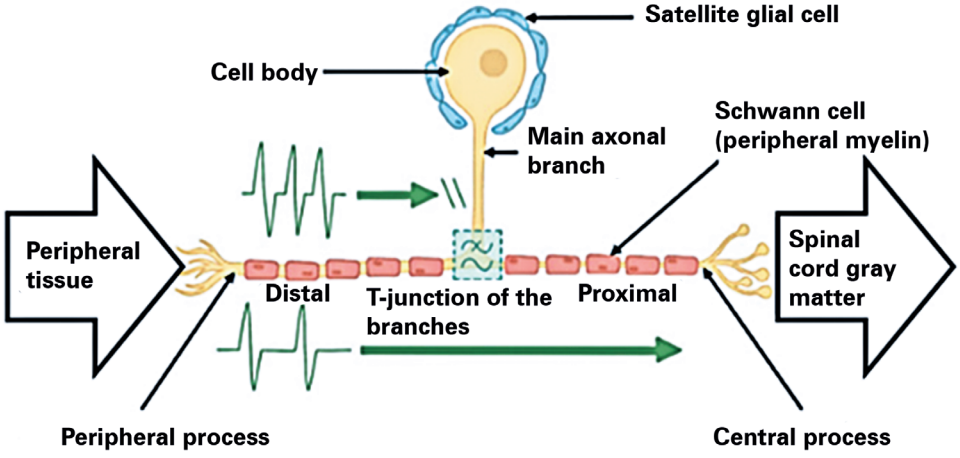

Figure 8. Pseudounipolar neuron in the dorsal root ganglion illustrating the "sensory filter" function of the axonal "T-junction".

develop algorithms capable of eliminating the need for trial electrodes[92], using wireless trial electrodes to avoid infection[93], personalizing waveform selection[94], identifying biomarkers that may improve responder selection, or determining the reasons for loss of function after months or years[95].

The miniaturization of generators, long-lasting rechargeable batteries, and new waveforms has led to greater patient acceptance of implantation. High cost and lack of awareness of the therapy remain the main barriers to its more widespread use for chronic pain relief in our country.

Authors' contributions: ASG: conceived and planned the activities that led to the study, wrote the paper, participated in the reviewing process, approved the final version; JOOJ: interpreted the results of the study, participated in the reviewing process and approved the final version.

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