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SPINAL CORD STIMULATION (SCS): TECHNICAL ASPECTS AND NEW DIRECTIONS

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ABSTRACT

Introduction and purpose: Spinal cord stimulation (SCS) is a well-established implantable neuromodulation therapy for chronic pain. Over the last decade, the field has evolved rapidly due to the diversification of stimulation waveforms, expanded programming capabilities, and the emergence of data-driven paradigms such as ECAP-controlled closed-loop systems. This review aims to present SCS as a medico-technical system by summarizing key mechanisms of neuromodulation, core elements of system architecture, clinical indications and selection principles, and technological directions.
Materials and methods: A comprehensive literature search was performed using PubMed, BioMed Central, Scopus, and Google Scholar to identify relevant studies on spinal cord stimulation. The search included the following keywords and their combinations: spinal cord stimulation, SCS, neuromodulation, chronic pain, failed back surgery syndrome, and complex regional pain syndrome.
Results: Evidence supports that SCS analgesia arises from both segmental spinal mechanisms and supraspinal modulation, with waveform-dependent differences in sensory and affective pain processing. Modern SCS platforms integrate leads, IPG, and telemetry/programming layers, enabling multipolar field shaping and increasingly incorporating sensing channels for objective biomarkers (e.g., ECAP) and real-time dose stabilization. Indications remain centered on PSPS/FBSS and CRPS, with expanding applications across selected neuropathic pain syndromes.
Conclusion: SCS is transitioning from conventional open-loop neuromodulation toward biomarker-driven, adaptive, and remotely supported therapy. Future progress is likely to be shaped by closed-loop architectures, improved sensing and telemetry, and computational approaches enabling more consistent outcomes and scalable personalization.

KEYWORDS

Spinal Cord Stimulation, Neuromodulation, Chronic Pain, Neuropathic Pain, High-Frequency Stimulation, Pain Management

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Introduction

Chronic pain remains a major clinical and public health challenge due to its high prevalence, detrimental impact on quality of life, and the substantial costs associated with its management [1]. Neuromodulation represents an interface between interventional medicine and biomedical engineering, and ***Spinal Cord Stimulation*** (SCS) is among the most clinically established implantable neuromodulation therapies for chronic pain. In clinical practice, SCS outcomes depend not only on accurate lead implantation but also on appropriate patient selection, trial stimulation, and subsequent programming an approach reflected in contemporary consensus recommendations regarding candidacy assessment and the role of trialing.

Over the past decade, SCS has undergone rapid technological evolution, driven both by the expansion of stimulation waveforms and programming capabilities and by a shift toward data-driven approaches. Recent reviews emphasize the growing diversity of waveforms and their relevance to the continued development of neuromodulation [2]. In parallel, closed-loop systems have been developed that use objective neurophysiological biomarkers (e.g., evoked compound action potentials, ECAPs) to more precisely titrate the "dose" of stimulation; long-term results from the randomized EVOKE trial support the clinical rationale for this strategy. Another important direction is remote therapy management and monitoring, for which best practice recommendations have been proposed.

The aim of this paper is to present SCS as a medico-technical system by discussing mechanisms of neuromodulation, the key elements of system architecture, and the principal directions of technological development.

Methodology

A comprehensive literature search was performed using PubMed, BioMed Central, Scopus, and Google Scholar to identify relevant studies on spinal cord stimulation. The search included the following keywords and their combinations: spinal cord stimulation, SCS, neuromodulation, chronic pain, failed back surgery syndrome, and complex regional pain syndrome. Additionally, the reference lists of eligible articles were manually screened to identify further relevant publications.

Mechanisms of neuromodulation

The mechanisms of neuromodulation in spinal cord stimulation (SCS) are historically rooted in the gate control theory of pain proposed by Ronald Melzack and Patrick Wall in 1965, which posits that nociceptive transmission at the spinal level is dynamically modulated within the dorsal horn rather than being merely a passive relay of signals to the brain [3].

From a segmental perspective, SCS primarily targets dorsal spinal structures, including pathways involved in tactile sensation and proprioception. It preferentially recruits large, myelinated Aβ afferent fibers, thereby increasing the activity of inhibitory interneurons within the dorsal horn and secondarily reducing the transmission of signals from Aδ and C fibers to projection neurons, including wide dynamic range (WDR) neurons, ultimately decreasing nociceptive input to ascending pathways. This "gate-closing" concept can be schematically represented as: Aβ fibers → inhibitory interneurons → suppression of C/Aδ transmission in the dorsal horn. Preclinical models have shown that SCS can attenuate the neuropathy-associated increase in excitatory amino acid release within the dorsal horn via a GABAergic mechanism, representing one of the most specific and biochemically demonstrable components of segmental inhibition [4,5].

At the same time, reviews emphasize that clinical efficacy does not arise solely from local spinal mechanisms, but rather from the interplay of spinal and supraspinal processes, including neural plasticity and modulation of the excitability of pain-processing networks [6, 7].

In parallel with the "gate control" mechanism, increasing emphasis is placed on descending pain control and supraspinal effects. Functionally, SCS may enhance the activity of descending antinociceptive pathways, including circuits involving the periaqueductal gray (PAG), the raphe nuclei, and the locus coeruleus (LC), which modulate sensory processing within the dorsal horn [8]. SCS influences pain transmission both at the spinal level and within modulatory networks, in part via serotonergic and noradrenergic systems. From this perspective, it is understandable why different stimulation waveforms may modify not only the sensory-discriminative component of pain but also its affective component: for example, tonic stimulation typically operates in the ~40-60 Hz range and often produces paresthesias, whereas burst stimulation delivers packets of pulses (e.g., ~500 Hz intra-burst at a burst rate of ~40 Hz), which is sometimes interpreted as a more "physiologic" pattern of neuronal activity and has been linked to effects on the affective dimension of pain (e.g., through thalamic circuits). In the case of kilohertz-frequency therapy (e.g., 10 kHz), an important

mechanistic insight is that paresthesia-free analgesia does not necessarily imply the absence of fiber activation. Rather, paresthesias may diminish because fibers discharge asynchronously, altering the subjective perception of stimulation while preserving analgesic efficacy [9,10].

Contemporary SCS systems increasingly implement closed-loop control architectures, in which stimulation parameters are adjusted in real time based on objective measures of neural activation (a data-driven approach), in contrast to traditional open-loop systems with fixed settings. In ECAP-controlled closed-loop systems, the evoked compound action potential (ECAP) is recorded as an objective marker of fiber recruitment, and an algorithm automatically modulates stimulation intensity to maintain the response within a predefined range despite changing conditions (e.g., variations in the electrode-spinal cord distance with postural shifts). In practice, neurophysiological mechanisms (what is actually being activated) are directly linked to "system-level" mechanisms (how the therapeutic dose is stabilized), providing an important bridge between physiology and device architecture as well as programming strategy [11].

SCS system architecture

The architecture of a spinal cord stimulation system constitutes an integrated implantable platform comprising the lead array, connecting components (extensions/connectors), an implantable pulse generator (IPG), and a programming and telemetry layer that enables real-time therapy configuration and long-term monitoring and control [12]. In clinical practice, both percutaneous approaches using cylindrical leads and open surgical techniques (e.g., paddle leads) are employed, and therapy is most often implemented in a two-stage manner: an initial trial with an external pulse generator followed by definitive IPG implantation with tunneling of the connections and lead anchoring [13]. The choice of lead type, contact geometry, and multipolar configurations determines the ability to "shape" the stimulation field, the selectivity of fiber recruitment, and the energy demand, thereby informing waveform selection and pulse parameterization during follow-up [12]. At the same time, the hardware architecture defines the risk profile of device-related complications: infections most commonly involve the IPG pocket or the connector/lead junction, with reported rates in the literature of approximately 2.5-14%; mechanical failures (e.g., lead fracture) have been reported at roughly ~3-9%, underscoring the importance of impedance diagnostics and verification of system positioning (e.g., radiographic documentation as a reference for suspected migration). The communication layer includes the clinician programmer and the patient interface (remote controller/app), and as device capabilities expand, remote management (monitoring and/or programming) has become increasingly relevant, with best-practice recommendations now available [14,15]. In more advanced architectures, dedicated sensing channels (e.g., ECAP recording) are also integrated, enabling objective quantification of fiber recruitment and implementation of closed-loop control strategies, thereby linking neurophysiological parameters with device design and programming algorithms [16].

Generations and stimulation modes

In recent years, there has been rapid development in therapeutic programming strategies for SCS devices. SCS stimulation patterns are defined by frequency, pulse duration (pulse width), amplitude, and pulse shape. Commonly distinguished modalities include conventional (tonic) stimulation, high-frequency stimulation, burst stimulation, and differential target multiplex (DTM) spinal cord stimulation [17].

Conventional (tonic) stimulation operates at a steady frequency (typically 40-60 Hz) and, compared with other paradigms, generally uses a longer pulse width and higher amplitude, resulting in a greater electrical charge delivered per pulse. A characteristic feature of this mode is the induction of paresthesias, which arise from orthodromic activation of large myelinated Aβ afferent fibers. Numerous studies suggest that achieving paresthesia coverage over the painful area is an important determinant of analgesic efficacy in conventional SCS [17,18].

The development of novel stimulation paradigms has led to the emergence of high-frequency stimulation, burst stimulation, and DTM. These contemporary strategies are characterized by the fact that their clinical efficacy does not require the induction of paresthesias, and patients typically do not perceive subjective sensory sensations during therapy. Clinical adoption of these approaches has progressed more rapidly than a complete understanding of their biological mechanisms of action; consequently, their precise physiological underpinnings remain the subject of ongoing intensive investigation.

High-frequency stimulation is characterized by a very high frequency (10 kHz), a short pulse width (30 μs), and a low amplitude (1-5 mA), resulting in subthreshold stimulation that does not elicit paresthesias. The absence of paresthesias eliminates the need for intraoperative sensory mapping; instead, leads are positioned

anatomically based on the pain distribution. Kapural et al. demonstrated the superiority of this approach over conventional stimulation in the treatment of chronic low back pain as well as lower-limb pain [17,19].

Burst stimulation was designed to emulate a more physiologic firing pattern within the nervous system. It is characterized by a burst rate of 40 Hz, a pulse width of 1000 μs, and an intraburst pulse frequency of 500 Hz, followed by a quiescent period before the next burst cycle. The SUNBURST trial showed that burst spinal cord stimulation was more effective than tonic stimulation in the treatment of chronic pain [17,20,21].

Differential Target Multiplexed (DTM) spinal cord stimulation was developed with the intent of modulating glial cell activity within the nervous system. The DTM-SCS waveform employs multiplexed signals with frequencies ranging from 50 to 1200 Hz and pulse widths between 50 and 400 μs. A randomized clinical trial comparing DTM-SCS with conventional SCS demonstrated superiority of DTM-SCS for chronic low back pain; however, this advantage was not confirmed for lower-limb pain [22,23,24].

The precise mechanisms of action underlying the individual stimulation of modalities remain incompletely understood and warrant further investigation.

Table 1. Comparison of SCS modes.

Stimulation mode	Frequency	Pulse width	Amplitude	Paresthesias
Conventional (tonic) stimulation	40-60 Hz	200-500 μS	3.5-8.5 mA	Yes
High-frequency spinal cord stimulation (HF-SCS, 10 kHz)	10 000 Hz	30 μs	1-5 mA	No
Burst (BurstDR)	40 Hz (burst)	1000 μs	protocol-dependent	No
DTM (Differential Target Multiplexed SCS)	50-1200 Hz (multiplex)	50-400 μs	protocol-dependent	No

Closed-loop systems: sensors, ECAP, automatic adaptation

SCS is an established neuromodulation technique utilized in the treatment of conditions such as Persistent Spinal Pain Syndrome Type 2 (PSPS-2), Complex Regional Pain Syndrome (CRPS), and selected neuropathies. In conventional open-loop systems, pulse parameters remains constant, whereas the effective stimulation "dose" at the spinal cord level varies during activities of daily living. Physiological movements (breathing, heartbeat) and postural changes modify the electrode-to-spinal cord geometry and conduction conditions within the epidural space, which can lead to episodes of under- or over-stimulation. Clinically, this manifests as fluctuations in the analgesic effect, position-dependent paresthesia (for paresthesia-based therapies), and the need for frequent manual adjustments of settings and programming strategies [25,26].

The evolution of SCS systems is moving from the rigid parameters of open-loop control, through posture- and activity-responsive solutions (e.g., automatic amplitude adjustment based on position sensors), to the modern closed-loop approach driven by neurophysiological markers. A key breakthrough is the utilization of Evoked Compound Action Potentials (ECAPs) as an objective biomarker of fiber recruitment in the dorsal columns. This enables real-time, automatic modulation of current intensity to maintain the response within a predefined range, despite changes in patient positioning or epidural space dynamics. In practice, this represents a transition from regulating "output parameters" (preset mA/V/μs) to regulating the "effect at the neural input" (measurable response), which aligns more closely with the concept of dose control in other device-based therapies [17,26,27].

An ECAP is defined as the total electrical response of an axon population excited by a single stimulation pulse. In epidural recordings, a characteristic triphasic morphology (P1-N1-P2) is typically observed, and the response amplitude in control applications is most commonly expressed as the potential difference between N1 and P2 (N1-P2 / P2-N1). It is worth noting that early components may be partially masked by the stimulation artifact; therefore, reliable measurement requires precise signal acquisition and processing (including blanking windows, artifact reduction, and averaging) to isolate the relatively weak neuronal component from the dominant stimulation artifact [17,25,28].

The primary parameter in closed-loop systems is the ECAP amplitude, treated as an objective indicator of fiber activation levels (and thus, the stimulation "dose"). In this architecture, electrodes serve a dual role: delivering the stimulus and utilizing separate recording configurations, measuring the response for real-time stabilization via a feedback loop algorithm. Clinical observation data demonstrate that during daily use, it is possible to maintain objective metrics of stable recruitment (e.g., a high percentage of pulses above the ECAP threshold and low dose accuracy error). This establishes a direct link between physiology (actual recruitment) and system engineering (stabilization of recruitment despite changing conditions) [11].

The strongest clinical evidence supporting the efficacy of closed-loop stimulation systems comes from the landmark, multicenter EVOKE study. This was the first randomized controlled trial (RCT) in the history of neuromodulation to be conducted using a double-blind method, directly comparing ECAP-controlled closed-loop technology with traditional open-loop stimulation. The results, published in *The Lancet Neurology*, not only confirmed the safety of the new method but, most importantly, demonstrated its significant superiority across key therapeutic parameters [29].

Table 2. Comparison of the efficacy of Open-loop and Closed-loop stimulation systems based on the 12-month results of the EVOKE study [29].

Parameter/Feature	Open loop	Closed loop	Clinical Benefit
Overall pain ≥50% reduction (primary outcome)	61%	83%	Higher overall therapeutic efficacy
High responder rates Overall, back and leg pain ≥80% reduction (hierarchical secondary outcome)	37%	56%	Profound pain relief
Time within the therapeutic window	49%	95%	Consistent stimulation stability
Reduction / discontinuation of opioids	40%	55%	Increased probability of reducing pharmacotherapy

Indications and patient selection

Patient selection for SCS is critical and often the most challenging aspect of care, as both medical factors and social determinants of health can influence treatment effectiveness. Therapeutic success depends not only on the technical aspects of the procedure but also on the patient's active engagement in follow-up care, including scheduled visits and device management [30].

SCS is most commonly used when other pain management strategies have failed to provide adequate relief. The primary indication is failed back surgery syndrome (FBSS), with complex regional pain syndrome (CRPS) being the second most common indication.

Failed back surgery syndrome (FBSS) is a clinical entity defined as persistent pain despite an apparently successful spinal operation, or recurrence of pain after a procedure that was initially intended to relieve it. Early studies of SCS reported long-term efficacy of approximately 50% pain reduction, along with a 41-84% decrease in the use of analgesic medications [31]. Complex regional pain syndrome (CRPS) is characterized by persistent, disproportionate pain accompanied by sensory, vasomotor, and sudomotor disturbances, as well as motor and trophic changes in the affected region. SCS may be effective in selected patients following a successful trial phase. A systematic review including 30 studies found that SCS reduces perceived pain and improves quality of life [32,33].

The literature also describes additional applications of SCS, including the treatment of refractory angina pectoris, peripheral vascular disease, phantom limb pain, lumbar spinal stenosis, post-thoracotomy pain syndrome, chronic head and neck pain, chronic abdominal visceral pain, and even diabetic neuropathy.

The most common indications for spinal cord stimulation are presented in Table 3. [15,30]

Table 3. Indications for Spinal Cord Stimulation (SCS).

Indications
Failed back surgery syndrome (FBSS)
Complex regional pain syndrome (CRPS)
Peripheral vascular disease and critical limb ischemia
Lower Back Pain (LBP)
Refractory angina pectoris
Diabetic neuropathy

Implantation technique

Implantation of a spinal cord stimulation system can be performed using two main surgical techniques: a percutaneous approach or an open (surgical) method. The choice of the appropriate technique depends on multiple factors, including clinical indications, the operator's experience, the patient's anatomical conditions, and the planned type of electrode [34].

In clinical practice, the percutaneous technique is used particularly frequently, as it is less invasive and allows the procedure to be performed in settings with limited surgical access. The percutaneous spinal cord stimulator implantation procedure can be divided into two main stages, comprising first the implantation and positioning of the electrode, followed by subsequent steps related to preparing the stimulation system. The first stage of the procedure involves performing a trial spinal cord stimulation in the operating room setting. In a patient previously qualified for treatment, electrodes are implanted into the epidural space, and their position is established at the appropriate anatomical level of the spine, in accordance with the location of pain symptoms and the planned area of stimulation. Once the optimal electrode position is achieved, it is secured using a surgical suture or adhesive material in order to minimize the risk of displacement. Subsequently, the electrode is connected to an external pulse generator, which is attached to the patient's body. The device undergoes initial programming, with stimulation parameters adjusted to the individual needs of the patient. The purpose of the trial phase is to evaluate the effectiveness of the therapy by analyzing the degree of pain reduction, improvement in functioning, and the patient's overall clinical response to stimulation. The results obtained form the basis for the decision to proceed to the second stage, namely implantation of a permanent stimulation system [30,35].

Similar to the percutaneous technique, the open method is preceded by a trial stimulation, and permanent stimulation uses paddle-type electrodes. The procedure is performed under general anesthesia with the patient positioned prone on a Jackson table. The midline of the spine is marked with reference to the spinous processes, and the incision site for lateral implantation of IPG is also indicated. After preparing the surgical field, maintaining asepsis, and performing a formal "timeout," fluoroscopy is used to identify the inferior lamina of the T9 vertebra, which will be removed to facilitate further dissection. The paraspinal muscles are dissected subperiosteally up to the facet joint using a Weitlaner retractor, allowing broad exposure of the vertebra. The dissection is extended laterally to ensure sufficient visualization while preserving the joint capsules. The inferior lamina of T9 is verified fluoroscopically and then removed using standard laminectomy techniques. At this point, efforts are made to position the paddle electrode beneath the T8 lamina, aiming for a central and proper alignment within the spinal plane. A Woodson instrument is used to clear the epidural space. If a mechanical obstruction prevents the electrode from passing, a partial laminotomy can be performed depending on the site of the blockage. In the case of electrode placement not being possible, laminoplasty is performed. Using an ultrasonic bone scalpel, bilateral cuts are made in the T8 lamina, just medial to the facet line. The lamina is then completely removed with a surgical curette and Rongeur forceps and kept in saline. The inferior lamina of T7 may be partially trimmed to provide additional space for the paddle electrodes. Any adhesions on the exposed dura are meticulously dissected under microscopic guidance. At the T8 level, a 16-channel paddle electrode is placed directly on the dura. Proper placement is confirmed fluoroscopically, and the electrode lead is anchored to the dura using 6-0 prolene sutures, leaving partial tissue thickness to minimize the risk of dural injury and cerebrospinal fluid leakage. This serves as a temporary measure to maintain electrode position while the lamina is repositioned. The excised lamina is restored to its original position on the vertebra and stabilized with screws. A lateral subcutaneous pocket is fashioned to accommodate the IPG, and the lead is tunneled beneath the skin from the IPG to the paddle electrode. Prior to closing the incision, generator impedance is assessed [36].

Complications

From a surgical perspective, the SCS implantation procedure is widely regarded as safe, particularly considering that it is typically performed in two stages, allowing for preliminary assessment of therapeutic efficacy before permanent system implantation. This staged approach enables careful patient selection and reduces the likelihood of unnecessary permanent implantation in non-responders, thereby contributing to the overall safety profile of the therapy [37].

Among the most commonly reported surgical complications are surgical site infections and hematoma formation. Infections may involve the generator pocket, lead insertion site, or deeper tissues, and in some cases may necessitate partial or complete system removal. Hematomas, including epidural hematomas, are less frequent but represent potentially serious complications due to the risk of neural compression and subsequent neurological deficit. Cerebrospinal fluid (CSF) leakage may occur as a result of inadvertent dural puncture during epidural access. Although often self-limiting, persistent CSF leak may require additional intervention. Neurological injury, including nerve root irritation or, rarely, spinal cord injury, has also been described. While such events are uncommon, they represent the most serious potential complications of the implantation procedure. Isolated reports have additionally described rare events such as syrinx formation, epidural fibrosis, and foreign body reactions. However, these remain exceptional findings in the literature [38,39].

Hardware-related complications are among the most frequently reported adverse events in spinal cord stimulation therapy. These events are primarily associated with the implanted device, including leads, pulse generators, and connection systems. Unlike surgical complications, hardware issues may arise weeks to years after implantation and can affect the efficacy of therapy as well as patient safety and comfort [40]. The most frequently encountered hardware complications include lead migration, lead fracture, and lead disconnection, which can compromise pain coverage and necessitate revision procedures. Other device-related issues include generator failure, loss of charge, generator flipping, hardware-related pain, and paresthesia intolerance [40].

In addition, SCS therapy may be associated with functional or clinical complications that primarily affect treatment efficacy and patient experience. The most common functional complications include loss of analgesic effect, recurrence of pain, uncomfortable paresthesias, and device-related pain. These events can occur even when the implanted system is mechanically intact and properly positioned. Reported data indicate that the overall rate of functional complications ranges from approximately 10% to 25%, with loss of efficacy being the leading cause of treatment failure [41]. Although functional complications do not typically pose direct neurological risk, they can significantly impact patient satisfaction and the perceived success of therapy. Prompt recognition and management, including reprogramming, optimization of stimulation parameters, and patient counseling, are essential to maximize long-term outcomes. Understanding the prevalence and nature of these complications helps clinicians anticipate potential challenges and improve individualized care plans for patients undergoing SCS therapy.

It is worth noting that hardware-related complications, while possible, are largely manageable and rarely permanent. The majority of hardware issues are reversible or can be successfully managed, with permanent adverse events being rare. Even when complications are minor or manageable, they may still result in significant costs over time, impacting healthcare systems and society. Awareness of potential device-related complications allows clinicians to optimize patient selection, select the most appropriate device, and implement meticulous perioperative management. Early identification and timely intervention are critical to maintaining long-term therapy effectiveness and maximizing patient outcomes. Importantly, recent studies indicate that the overall rate of hardware-related complications has decreased over the past years, likely due to advances in device design, improved materials, more reliable leads, and enhanced implantation techniques. These trends highlight the evolving safety profile of SCS and reinforce its value as a long-term treatment option for chronic neuropathic pain [39,40,42].

Future directions and the “SCS of the future”

Despite the high level of technological sophistication in current SCS systems, significant further evolution is anticipated in the near future, fundamentally reshaping the standards of care for chronic pain patients. The foundation of these advancements remains closed-loop technology, which, according to the ASPN guidelines (NEURON Project), shifts the clinical focus from subjective patient reports to objective electrophysiological data, such as Evoked Compound Action Potentials (ECAPs). This shift enables precise stabilization of the "neural dose," which is critical for mitigating therapy failures caused by postural changes. As noted by Levy et al., the next milestone in neuromodulation will be the integration of these systems with Artificial Intelligence (AI) and Machine Learning. AI is poised to become an indispensable tool, capable of

predictive therapy management by analyzing a patient's daily routines and habits. This will allow for the autonomous optimization of stimulation parameters even before the onset of pain.

Parallel to algorithmic progress, SCS hardware is evolving toward device miniaturization and the development of leadless systems, aimed at minimizing mechanical complications. This evolution necessitates continuous monitoring of safety and efficacy, as well as the expansion of medical indications to challenging clinical areas, such as cervical stimulation for upper extremity CRPS or thoracic pain. The intensive development of these technologies is imperative; as they become more widespread, unit costs are expected to decrease, improving accessibility for a broader patient population. From a healthcare economics perspective, effective closed-loop neuromodulation offers tangible benefits: it reduces the reliance on chronic pharmacological management, directly translating into a lower incidence of complications, such as opioid dependency and life-threatening gastrointestinal bleeding (GIB). Ultimately, the synergy of intelligent algorithms, novel biomarkers, and remote programming (telemedicine) will lead to the creation of an autonomous neuroprosthesis, capable of restoring physiological balance within the pain conduction system in a highly personalized manner [43,44],

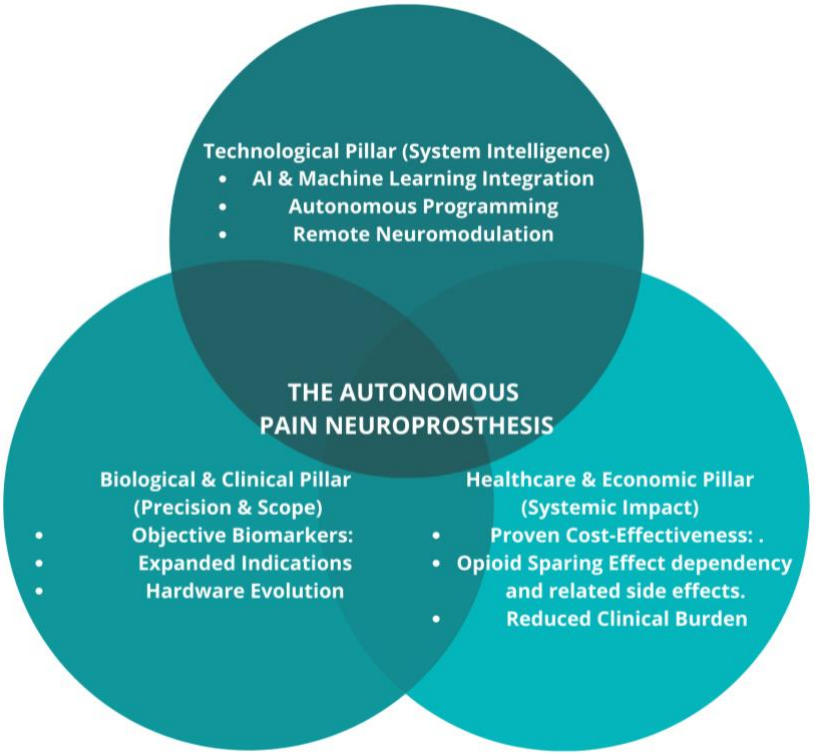

Fig. 1. *Own elaboration based on [43,44]*

Discussion

Spinal Cord Stimulation (SCS) remains one of the best-established methods of implantable neuromodulation for the treatment of selected chronic pain syndromes. Its development over the last decade has clearly shifted the emphasis from "simple stimulation" toward therapies that are increasingly programmable, adaptive, and integrated with telemetry [10]. From a mechanistic perspective, the efficacy of SCS does not stem solely from segmental "gate control" at the dorsal horn level, but rather from the overlap of spinal and supraspinal processes, neurotransmitter modulation, and the plasticity of pain networks. This multi-level mechanism of action provides a coherent justification for the clinically observed variability in patient responses and for the fact that different stimulation patterns (tonic, burst, high-frequency, DTM) can affect not only the sensory-discriminative but also the affective components of pain [10].

From a clinical practice perspective, a key conclusion is that SCS should be treated as a medico-technical system, where the therapeutic outcome depends simultaneously on indication and patient selection, implantation technique, hardware architecture (leads-IPG-connections), and the quality and continuity of the programming process. In this sense, the neuromodulation "dose" is not a constant value set in mA or µs, but a

result of the electrode-tissue geometry, impedance, body position, and the preferred stimulation strategy. This explains why traditional open-loop systems are susceptible to fluctuations in clinical effect during daily activities, and why systems capable of stabilizing neural activation despite anatomical and behavioral changes are becoming increasingly important.

In this context, data-driven approaches and closed-loop architectures represent more than just "another mode"; they constitute a qualitative shift in therapeutic philosophy. A control algorithm based on an objective marker (e.g., ECAP) allows for the real-time linking of neurophysiological parameters with programming decisions. In the technological discussion, this serves as an important "bridge" between physiology and engineering: the system not only delivers a stimulus but also measures the response and corrects the stimulation to maintain fiber recruitment within a defined therapeutic window. However, it should be emphasized that the effectiveness of such an approach depends on the quality of the measurement path (reduction of stimulation artifacts, recording stability, proper selection of thresholds/targets) and whether the biomarker truly represents a clinically relevant dimension of neuromodulation for a given patient [11].

The dynamic development of programming strategies (expansion of waveforms and mapping capabilities) has, in practice, progressed faster than a full understanding of the underlying biological mechanisms. On one hand, this has brought tangible benefits, including greater flexibility in tailoring therapy to the pain phenotype and patient tolerance; on the other hand, it creates a need for cautious interpretation. "Different modes" are not equivalent in terms of neurophysiological goals, energy profiles, or predictability of response. Therefore, it is worth emphasizing in the discussion that further optimization of SCS will likely depend on better integration of clinical data with objective activation metrics (sensing) and the standardization of reporting settings and outcomes (which will facilitate comparisons across studies, manufacturers, and centers) [2].

Financial and implementation aspects

SCS, especially in variants featuring advanced telemetry and closed-loop systems, involves a significant initial cost (procedure, device, perioperative care). At the same time, potential economic benefits may emerge in the long term through the reduction of pain burden, improved functioning, decreased pharmacotherapy (including opioids), fewer visits due to "dose fluctuations," and, in the case of remote solutions, a partial shift of care toward a hybrid model [43].

New directions

In the perspective of "new directions," the most prominent trends include: (1) further development of waveforms and programming tools (including automation and algorithm-assisted decisions), (2) expansion of sensing functions (ECAPs and other potential signals), (3) miniaturization and improved energy management, (4) telemetry and remote therapy management, and (5) adaptive architectures combining device data, patient-reported outcomes, and functional parameters. A potential "bottleneck" remains the need to balance system complexity (and the risk of failure/artifacts) with ease of use and safety (including cybersecurity and data quality control) [43].

Applications beyond classical neuropathic pain: cardiology

Although the core indications for SCS remain chronic pain of neuropathic etiology (e.g., PSPS/FBSS, CRPS), the literature also describes applications in "non-neuralgic" areas, including cardiology, primarily in refractory angina pectoris. Mechanistically, autonomic modulation and the reduction of ischemic pain perception are considered, potentially impacting exercise tolerance and quality of life. However, it should be noted that this area is more heterogeneous in terms of evidence, and the position of SCS in treatment algorithms depends on the availability of other methods (revascularization, pharmacotherapy, other devices) and local recommendations [45].

Limitations and directions for further research

A limitation of much of the data regarding new SCS strategies is population heterogeneity, differences in response definitions, a lack of comprehensive head-to-head comparisons between waveforms, and the influence of psychosocial factors and social determinants of health (SDOH) on outcomes and therapy maintenance. Further research should focus on identifying predictors of response, standardizing metrics (both clinical and neurophysiological), assessing long-term effect durability, and developing care models that integrate programming, remote monitoring, and rehabilitation.

In conclusion, SCS is evolving from fixed-setting, open-loop therapy toward adaptive, biomarker-driven neuromodulation supported by telemetry. The ultimate clinical value of these technologies will depend on the quality of implementation (selection, implantation, programming, follow-up), reliable long-term evaluation, and whether objective metrics (e.g., ECAPs) translate into more stable, predictable, and scalable treatment outcomes.

Disclosure

Author's contribution:

All authors contributed to the article.

All authors have read and agreed with the published version of the manuscript.

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