Narrative Review

Technical Strategies in Spinal Cord Stimulation for Refractory Angina: A Narrative Review

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Background: Refractory angina (RA) is a chronic condition characterized by persistent chest pain that remains even after the patient has received optimal medical and/or revascularization therapy. Spinal cord stimulation (SCS) has emerged as a minimally invasive adjunctive treatment for RA, yet the technical aspects of implanting SCS devices remain highly variable.

Objectives: To synthesize the existing literature on technical strategies used in SCS implantation for RA and to identify common practices and areas of variability in the clinical implementation of the procedure.

Study Design: A narrative review.

Methods: A comprehensive literature search of the PubMed and Embase databases was conducted to identify studies published between 1987 and 2025. Articles were included if they met the following criteria: 1) they reported on the use of SCS for patients with RA or chronic angina, and 2) they provided technical details regarding one or more aspects of the implantation process, such as epidural access level, lead positioning, or stimulation programming. Eligible articles included randomized trials, cohort studies, case series, and reviews.

Results: Lead placement was commonly reported at the upper thoracic or cervicothoracic junction (T1-T2 or C7-T1), with a few exceptions for cervical placement. Intraoperative paresthesia mapping was used in several studies to optimize electrode coverage of reported RA pain, though methodologies were described inconsistently. Of all stimulation paradigms, tonic stimulation remains the most well-studied and validated setting, though subthreshold, high-density, and burst stimulation show potential benefits for select patient populations. Trial phases were described variably, ranging from experiences that involved the implantation of transcutaneous electrical nerve stimulators to ones that included multiday spinal cord stimulators.

Limitations: Significant heterogeneity and inconsistent reporting of technical parameters are described in the literature. Few studies have directly compared outcomes related to various technical approaches or stimulation paradigms.

Conclusions: Technical aspects of the implantation of SCS devices for RA remain variable, particularly in terms of lead placement strategies, stimulation paradigms, and trial methodologies. More rigorous studies are needed to guide best practices for SCS use in this population.

Key words: Spinal cord stimulation, refractory angina, lead placement, stimulation paradigms

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Refractory angina (RA) is a clinical condition characterized by substernal chest pain due to reversible myocardial ischemia that persists for over 3 months despite optimal medical therapy and/or revascularization (1). RA represents a heterogenous patient population with varying underlying

pathophysiologies, comorbidities, and responses to treatment. The condition imposes a significant burden on patients and the health care system, contributing to reduced functional capacity, increased mortality risk, and frequent hospital admissions (2). In the United States, the prevalence of RA in people with coronary artery disease (CAD) is estimated to be as high as 15 percent, a number that is increasing due to the aging population and improved survival rates among patients with CAD (2,3).

Since the 1980s, spinal cord stimulation (SCS) has emerged as a promising, minimally invasive, adjunctive treatment for RA (4). By delivering low levels of electrical stimulation, SCS can inhibit the transmission of pain signals and modulate myocardial blood flow. The use of SCS alongside medical therapy has been well-documented, showing benefits such as reduced anginal episodes and nitrate use, as well as enhanced exercise tolerance and quality of life (5,6). Despite these potential advantages, the technical aspects of SCS-device implantation remain inconsistently reported in both clinical practice and literature. The absence of standardization for SCS complicates the interpretation and comparison of its outcomes, since differences in lead placement, stimulation parameters, and programming can produce inconsistent results. This variability also impedes the development of best practices and limits reproducibility across studies and clinical settings.

This narrative review aims to synthesize the existing literature on technical strategies used in SCS implantation for RA, with the goal of clarifying current practices and identifying emerging patterns.

Methods

This narrative review was conducted to summarize and analyze the current literature on technical strategies used in SCS for the treatment of RA. A comprehensive literature search was performed in the PubMed and Embase databases to identify relevant studies published between 1987 and 2025. Search terms included: "spinal cord stimulation," "refractory angina," "chronic angina," "technique," "lead placement," "programming," "stimulation parameters," and "neuromodulation strategies."

Articles were included if they met the following criteria: 1) they reported on the use of SCS in patients with RA or chronic angina, and 2) they provided technical details on one or more aspects of the implantation process, such as epidural access level, lead positioning, or stimulation programming. Study types included randomized clinical trials, cohort studies, case series, case reports, and review articles. Animal studies, and reports lacking technical descriptions, and articles written in languages other than English were excluded.

Relevant data were extracted from each eligible study, including level of epidural access, electrode positioning, type and parameters of stimulation, and use of trial outcomes. Emphasis was placed on identifying variations in technique and assessing their reported or potential clinical implications.

Mechanistic Rationale and Device Components

Understanding the mechanisms by which SCS alleviates RA is essential to optimizing the technical application of the treatment. The therapeutic rationale stems from its ability to modulate neural activity at the spinal level. The primary mechanism is thought to involve the gate control theory, whereby low-intensity electrical stimulation of the spinal cord enhances the release of inhibitory neurotransmitters like GABA (7). This process inhibits nociceptive signaling in the dorsal columns, therefore reducing the perception of RA pain (8). Beyond its impact on nociceptive transmission, SCS may also modulate autonomic function. Evidence suggests that SCS can reduce sympathetic tone, which leads to improved myocardial perfusion by increasing blood flow to ischemic regions and decreasing myocardial tissue oxygen demand (9,10). Additionally, some preliminary experimental studies suggests that SCS may attenuate neuroinflammation by reducing microglial activation in the spinal cord (11).

A detailed understanding of these mechanisms informs the technical approach to SCS implantation. The device consists of 2 main components: an array of leads, or electrodes, and an implantable pulse generator (IPG). The leads are introduced in the epidural space and positioned along the dorsal columns of the spinal cord. The IPG is implanted subcutaneously in the abdominal wall or gluteal region and generates electrical impulses according to programmed settings (12).

Primary Literature on Technical Techniques (Table 1)

Epidural Access Level

Few studies report on the specific spinal level at which the epidural space is entered with the Tuohy needle prior to lead placement. According to the rare instances in which this information is provided, the

Table 1. *Summary of primary literature describing technical strategies for the implantation of SCS for RA.*

Author	Year	Study Type	Population	Lead Placement Level	SCS/Lead Type	Stimulation Parameters	Trial Approach
Murphy & Giles	1987	Prospective case series	10 patients with severe RA referred by cardiology to pain relief clinic	Cervical/thoracic (upper thoracic or lower cervical spinal level that produces electrical stimulation over area where angina pectoris is perceived)	Medtronic® Pisces Sigma® 3483	3-7 V, pulse rate 80-100 beats/set, pulse width 250-500 psec, cycle on: 20 set, cycle off: 40 sec	Not reported
De Jongste et al	1994	Prospective feasibility study	22 patients with angina refractory to standard therapies	Electrode tip mostly at T1 and slightly left of midline (final position guided by paresthesia in area of angina pain); epidural access at T4-T5	Unipolar system: Itrel I Model 7421 (Medtronic®) connected to a Sigma®/Pisces model 3483S electrode, using an extension lead model 7491 with 7432 Console programmer Bipolar system: Itrel II model 7424 stimulator (Medtronic), Pisces-Quad® model 3487A electrode, extension lead model 7495	Intermittent stimulation with standardized pulse width of 210 μs and pulse frequency 85 Hz	Not reported
Sanderson et al	1994	Prospective interventional cohort study	23 patients with angina unresponsive to standard therapy	Mid-thoracic region, adjusted to produce paresthesia in area of anginal pain	Medtronic® Itrel I, Itrel II and Cordis	One hour 3 times a day	Transcutaneous electrical nerve stimulation
Mannheimer et al	1998	RCT	104 patients with severe angina randomized to SCS (53) or CABG (51)	Electrode tip at level of T1 to T2; adjusted to produce paresthesia covering area of radiation of anginal pain, epidural access at level of T6	Medtronic®	Two preset stimulation strengths, one stronger that was used in case of established anginal pain and one weaker that was used as prophylactic treatment (latter was used for at least 2 hours 4 times daily)	Not reported
Hautvast et al	1998	RCT	25 patients with chronic intractable angina pectoris; 13 randomized to treatment and 12 to control	Thoracic (T1) midline; adjusted to produce paresthesia in area where angina usually felt; epidural access at T3-4	Medtronic® Itrel II connected to a quadripolar electrode by an extension lead	Continuous mode; pulse width 210 μs and frequency 85 pps; treatment group instructed to use 3 times per day for one hour each time and additionally when anginal symptoms occurred	Not reported
Di Pede et al	2003	Prospective registry study	104 patients with severe RA implanted with SCS	Usually at C6-T2 level; positioned at level in which patient felt paresthesia in the chest region (usual referred angina pain)	Not reported	Patients instructed to use stimulation protocol (continuous, cyclic, intermittent) that they believed would effectively control their symptoms	2-session implant (permanent IPG implanted after 2-to-3-week trial period with temporary, external neurostimulator connected to electrode)

Table 1 cont. *Summary of primary literature describing technical strategies for the implantation of SCS for RA.*

Author	Year	Study Type	Population	Lead Placement Level	SCS/Lead Type	Stimulation Parameters	Trial Approach
Yu et al	2004	Retrospective study	24 patients with RA implanted with SCS	Thoracic (T1-T2), adjusted so that paresthesia in chest covered area of RA	Medtronic®	Low stimulation intensity intermittently or constantly and high stimulation intensity during an acute angina attack for 5-10 min	Temporary electrode or TENS unit
McNab et al	2006	RCT	68 patients randomized to SCS (34) or percutaneous myocardial laser revascularization (34)	High thoracic and low cervical to obtain adequate paresthesia C6 (n =10) C7 (n =16) T1 (n =6)	Medtronic® Itrel 3	Not reported	Not reported
Deer & Raso	2006	Case report	58-year-old white man with anginal pain at rest despite previous surgical revascularization	2 octrode leads: one at C7-T1 midline and one at T1-2 5 mm left of midline	Genesis (Advanced Neuromodulation Systems , Inc.)	Preferred frequency 80 Hz, and amplitudes <3 mAmps	Not reported
Eddicks et al	2007	RCT	12 patients with previously implanted SCS who already responded to treatment, randomized to treatment arms of various stimulation parameters	Cervical/thoracic (C7-T1)	Medtronic® Itrel and Synergy neurostimulators; Pisces-Quad® epidural electrode (Medtronic®)	Conventional stimulation parameters: amplitude 3-5.5 V, pulse width 210-300 ms, and frequency 75-85 Hz, adjusted for each patient Subthreshold output: 85% (range 2.1-4 V) of minimum stimulation output (voltage) causing paresthesia Control: 0.1 V output	Not reported, previously implanted
Bondesson et al	2008	Observational study	153 patients with RA (44 SCS, 79 enhanced external counter pulsation, 30 controls)	T1-T2, adjusted to produce paresthesia covering area of radiation of angina pain	Medtronic®	Primary setting of frequency 31 Hz and pulse width of 210 µs, adjusted if needed; 2 preset stimulation strengths (one stronger in case of establishing angina pain and one weaker used as prophylactic treatment)	High-intensity TENS
Anselmino et al	2009	Prospective cohort study	8 patients with RA implanted with SCS	Varied according to where patients described anginal pain (inserted in mid-to-upper thoracic area) C5-C6 (n = 1) C6 (n = 3) C6-C7 (n = 1) C7 (n = 1) C7-T1 (n =2)	Synergy Surgical™ (Medtronic®)	Median amplitude of 1.6 (1.3-2.4) mV, frequency of 80 (70-85) Hz, and pulse duration of 210 (180-210) ms	Not reported

Table 1 cont. *Summary of primary literature describing technical strategies for the implantation of SCS for RA.*

Author	Year	Study Type	Population	Lead Placement Level	SCS/Lead Type	Stimulation Parameters	Trial Approach
Lanza et al	2011	RCT	25 patients with RA randomized to paresthesia SCS, subliminal SCS, or placebo	Location that induced paresthesia over area of anginal pain	Not reported	Classical SCS with satisfactory paresthesia coverage of the anginal pain zone of the chest (group PS) Continuous SCS with current intensity between 75% and 80% of the sensory threshold, resulting in subliminal stimulation (group SS) "Sham" or no stimulation: one hour daily of SCS at a current intensity of 0.05 mV (i.e., a very low intensity that is considered ineffective [group NS])	Not reported
Lee et al	2012	Case report	40-year-old man with RA and anginal episodes 2-3 times per day	Tip at the C7-T1 intervertebral disc level; epidural access at T4-5 interlaminar space	Octad Lead 3778 (Medtronic®)	Pulse width of 270 μs, amplitude of 2.0 mA, and frequency of 50 Hz	10-day trial
Zipes et al	2012	RCT	68 patients with SCS implanted randomized to high stimulation or low stimulation	Location that induced paresthesia over area of anginal pain	Itrel III neurostimulator (Medtronic®)	2 groups: high stimulation (>2 hours, 4x/day) and low stimulation (one minute, one time/day)	Not reported, previously implanted
Eldabe et al	2016	RCT	29 patients with RA randomized to SCS (15) and usual care (14)	Epidural access in thoracic epidural interspace; adjusted to obtain optimum coverage of painful area C7/T1 (n = 7) T11/T12 (n = 1) T1/T2 (n = 5) C2 (n = 1)	Not reported	Patient adjusted device for "comfortable" paresthesia level for 2 hours, 3x/day to terminate any angina attack or before any exertion known or anticipated to generate angina pain	On-table trial, if <80% of paresthesia coverage of painful area, or if generation of painful sensations, then considered "failed trial" and leads removed with no full implantation
Abusabha & Slotty	2019	Case report	60-year-old man with RA and partial response to tonic conventional stimulation, switched to burst stimulation	T2	2 paramedian Octrodes™ with permanent stimulator Eon Mini™	Tonic stimulation with 50 Hz, pulse-width 412 μs, perception 5.1 mA, comfort 6.80 mA, continuous stimulation Burst stimulation 500 Hz, 40 bursts/s, PW 1000 μs, cyclic stimulation 3600 sec. on/15 sec. off	TENS followed by one-week trial

Table 1 cont. *Summary of primary literature describing technical strategies for the implantation of SCS for RA.*

Author	Year	Study Type	Population	Lead Placement Level	SCS/Lead Type	Stimulation Parameters	Trial Approach
Yun et al	2023	Case report	52-year-old man with RA implanted with SCS	T1-T2, epidural access at T6-T7	Proclaim XR™ (Abbott Neuromodulation)	Tonic (frequency of 20 or 30 Hz, pulse width of 500 µs, and amplitude of 1.0–2.0 mA) and burst (5 pulses at 500 Hz, 40 bursts/s, and amplitude of 0.3 mA)	14-day trial program testing tonic and burst stimulation
Vervaat et al	2023	RCT	18 patients with RA	Cephalad tip positioned left paramedian at C7-T1; sedation temporarily lifted and use of external programmer to paresthesia coverage achieve >80% of target area; epidural access at T7-8	Lead: Vectris Compact™ SureScan™ MRI (Medtronic®) IPG: PrimeADVANCED™ SureScan™ MRI (Medtronic®)	High-density stimulation	Standardized TENS treadmill test screening tool (pulse width of 200µs and frequency of 2 Hz)

needle is typically introduced at a mid-to-upper-thoracic level (4,13,14), between T7-T8 (15) and as high as T3-T4 (16) and T4-T5 (17,18). These levels are accessed under fluoroscopic guidance using the loss-of-resistance technique.

Lead Placement (Level)

Placement of leads for RA is variable, though most studies follow the originally described approach of targeting the lower cervical and upper thoracic spinal levels (4). The most commonly reported positions are C7-T1 (13,15,17-23) or T1-T2 (12,24-26), typically referring to the uppermost contact of a multi-contact lead. Thoracic placement is the most frequently studied position, since the thoracic levels are thought to influence cardiac afferent pathways and modulate autonomic tone (5,8). Cervical placement is less common; only 3 studies positioned the electrode tip at C5 or C6 in some patients, without reporting outcomes stratified by level (13,20,22). No studies directly compared cervical lead placement to its thoracic counterpart.

Lead positioning strategies differ. Some studies use empiric placement at prespecified levels, while others employ intraoperative adjustments based on paresthesia mapping to the patient's anginal symptoms (4,20,24,27,28). However, the specific techniques used for intraoperative mapping and adjustment are often not well described. The degree of paresthesia coverage required for optimal outcomes remains unclear. One study by Vervaat et al targeted > 80% coverage of the region with reported RA pain (15), though how this threshold was measured or defined was not detailed.

Stimulation Paradigms

Tonic (Conventional) Stimulation (<One kHz)

This widely used setting applies low-frequency stimulation, typically 60-80 Hz, to elicit paresthesia in the targeted region. In the SCS-ITA trial they conducted, Lanza et al reported that paresthesia-inducing stimulation was associated with significantly greater reductions in anginal attacks and nitroglycerin usage than the placebo was (27). Eddicks et al also found that conventional stimulation improved both symptom burden and functional status in patients with RA (21).

Subthreshold Stimulation

Subthreshold stimulation delivers a current below the threshold of perception, producing no paresthesia. Lanza et al compared tonic (paresthesia-inducing), subliminal (75%-80% of sensory threshold), and placebo stimulation (27). Tonic stimulation was superior in reducing patients' anginal episodes and nitroglycerin use and in improving the individuals' quality of life, while the effects seen in association with subliminal stimulation did not differ significantly from those seen in association with

the placebo. In a separate study, Eddicks et al compared conventional stimulation, subthreshold stimulation (85% of paresthesia threshold), and a placebo. In agreement with Lanza et al, Eddicks et al demonstrated that conventional stimulation resulted in the greatest improvements in functional abilities, such as walking distance and quality of life scores. However, the findings of Eddicks et al also showed that subthreshold stimulation was more effective than the placebo was (21).

High-Density (HD) Stimulation

HD stimulation delivers high-frequency pulses (500-1200 Hz). Vervaat et al reported on patients who transitioned from conventional to HD stimulation due to the progression of RA symptoms. On average, patients remained on conventional stimulation for an average of 40 months before switching, suggesting HD stimulation may offer benefits in cases of waning response (29).

Burst Stimulation

Burst stimulation is a paresthesia-free modality with limited evidence, primarily restricted to case reports, in situations involving RA. Abusabha and Slotty described a patient with a partial response to conventional stimulation who experienced a marked reduction in anginal attacks (from 3-4 per day to 4-5 per month) after 12 months of burst stimulation (30). Similarly, Yun et al reported a case in which a patient alternated between tonic and burst stimulation. Although the patient initially preferred tonic stimulation, that patient's intolerance to paresthesias led to a change in approach, with burst stimulation used for background pain and tonic stimulation for breakthrough episodes (31). This strategy reduced both the frequency of breakthrough pain and intensity of pain.

High-Frequency Stimulation (10 kHz)

No studies were found on the use of high-frequency stimulation for RA.

Trial Phase

A trial phase is often used to identify patients most likely to benefit from permanent implantation. In the RASCAL study, Eldabe et al used an intraoperative ("on-table") trial, adjusting lead placement to achieve at least 80% of the painful area associated with RA. If patients had less than 80% of paresthesia coverage, or if the sensations from the on-table trial were too painful, the leads were removed (19). Other studies have reported alternative trial strategies, including a 10-day trial with temporary electrodes or preliminary assessment using a transcutaneous electrical nerve stimulation (TENS) unit (17,24,32). Some reports also describe trialing different stimulation paradigms to determine the most effective setting for a given patient (31).

Discussion

Although growing evidence supports the safety and efficacy of SCS as an RA treatment, technical approaches to implantation and programming remain highly variable across the literature and clinical practice. This review summarizes major technical considerations, including epidural access, lead positioning, stimulation paradigms, and the role of a trial phase, highlighting both current practices and important gaps.

Lead placement levels are reported inconsistently, but most studies describe positioning at the upper thoracic or cervicothoracic junction, typically T1-T2 or C7-T1 (4,21,24,26,29). No studies have compared cervical placement to thoracic placement directly. However, stimulation at different spinal levels may have distinct physiological effects: C1-C2 has been associated with increased cerebral blood flow and reduced sympathetic activation, while C3-C6 increases perfusion to the upper extremities (33). These differences may be relevant for patients with anginal radiation to the arms, although this possibility has not been formally evaluated in the context of RA.

Intraoperative paresthesia mapping is often used to tailor lead placement to the patients' reported regions of anginal pain. This personalized approach may improve outcomes, since inadequate coverage is a potential cause of treatment failure. Studies by Eldabe et al and Vervaat et al targeted ≥ 80% coverage of the painful region, though methods for assessing this threshold were not described consistently (19,29).

A trial phase can help identify patients most likely to respond to treatment, yet the utilization and methodology of these phases vary widely. Some studies use TENS-based assessments (24), others conduct intraoperative testing (19), and still others perform week-long trials (17). A systematic review by Gazzeri et al suggests that a "direct-to-implant" approach may be growing more standard across institutions, due to the decreased use of resources compared to trial phases (12).

Stimulation paradigms also vary across studies. Tonic stimulation remains the most well-validated first-line option. For patients who no longer respond

to tonic stimulation, subthreshold and high-density stimulation may offer additional relief (21,29). With advances in coronary disease management and longer patient lifespans, an increasing number of individuals who receive SCS for RA may become less responsive to conventional stimulation over time and require adjustments to their programming. Other paradigms, such as burst stimulation and high-frequency stimulation, have demonstrated efficacy in treating low back pain and offer the added advantage of being free from paresthesia (34). Although no studies have evaluated the effects of high-frequency stimulation on RA, burst stimulation may be useful for patients who are either unresponsive to tonic stimulation or intolerant of the associated paresthesias (30,31).

As with all interventional procedures, patient selection and periprocedural management remain critical for optimizing outcomes and minimizing complications. Because patients with RA may be maintained on antiplatelet and/or antithrombotic therapy, the risk of periprocedural bleeding, particularly epidural hematoma, requires careful assessment. Coordination with the managing cardiology team is advised for the balancing of thrombotic and bleeding risks (35,36).

Limitations

This review is limited by the paucity of recent studies that report technical parameters in sufficient detail to inform best practices. Future research should aim to systematically evaluate these technical variables through prospective registries, randomized trials, and standardized reporting frameworks to report best practices for RA-specific SCS.

Conclusions

SCS represents a valuable treatment option for patients with refractory angina, demonstrating benefits in symptom relief, functioning, and quality of life. However, the technical aspects of the implantation and programming of SCS remain inconsistently reported, limiting the reproducibility and impeding the development of standardized approaches.

This review highlights major areas of variability, including lead placement strategies, stimulation paradigms, and trial methodologies, and calls for more rigorous studies to guide practice. As newer stimulation technologies emerge and patient survival improves, it is increasingly important to define best practices for SCS as an RA treatment.

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