Neuromodulation: Technology at the Neural Interface

Received: November 3, 2022 Revised: March 26, 2023 Accepted: April 10, 2023

https://doi.org/10.1016/j.neurom.2023.04.464

Burst Spinal Cord Stimulation as Compared With L2 Dorsal Root Ganglion Stimulation in Pain Relief for Nonoperated Discogenic Low Back Pain: Analysis of Two Prospective Studies

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ABSTRACT

Introduction: Chronic discogenic low back pain (CD-LBP) is caused by degenerated disks marked by neural and vascular ingrowth. Spinal cord stimulation (SCS) has been shown to be effective for pain relief in patients who are not responsive to conventional treatments. Previously, the pain-relieving effect of two variations of SCS has been evaluated in CD-LBP: Burst SCS and L2 dorsal root ganglion stimulation (DRGS). The aim of this study is to compare the effectivity in pain relief and pain experience of Burst SCS with that of conventional L2 DRGS in patients with CD-LBP.

Materials and Methods: Subjects were implanted with either Burst SCS ($n = 14$) or L2 DRGS with conventional stimulation ($n = 15$). Patients completed the numeric pain rating score (NRS) for back pain and Oswestry disability index (ODI) and EuroQoL 5D (EQ-5D) questionnaires at baseline, and at three, six, and 12 months after implantation. Data were compared between time points and between groups.

Results: Both Burst SCS and L2 DRGS significantly decreased NRS, ODI, and EQ-5D scores as compared with baseline. L2 DRGS resulted in significantly lower NRS scores at 12 months and significantly increased EQ-5D scores at six and 12 months.

Conclusions: Both L2 DRGS and Burst SCS resulted in reduction of pain and disability, and increased quality of life in patients with CD-LBP. L2 DRGS provided significantly increased pain relief and improvement in quality of life when compared with Burst SCS.

Clinical Trial Registration: The clinical trial registration numbers for the study are NCT03958604 and NL54405.091.15.

Keywords: Burst spinal cord stimulation, discogenic low back pain, L2 dorsal root ganglion stimulation, pain relief

Conflict of Interest: Elbert A. Joosten receives financial support for preclinical studies on the mechanism of SCS from Medtronic, Boston Scientific, Abbott Laboratories, and is on the advisory board for Saluda Medical. Jan-Willem Kallewaard is on the advisory board for Abbott Laboratories, Nevro Corporation, Saluda Medical, Medtronic, and Boston Scientific. The remaining authors reported no conflict of interest.

INTRODUCTION

Chronic discogenic low back pain (CD-LBP) is a condition caused by a damaged or degenerated intervertebral disk (IVD). This degeneration is marked by loss of disk height, ingrowth of sensory neurons, and development of an inflammatory environment inside the disk, causing chronic pain.[1–5] Recently, several promising publications applied neurostimulation for nonoperative LBP, which included patients with CD-LBP, but did not specifically target patients with CD-LBP.[6–10] These studies used conventional spinal cord stimulation (SCS) (con-SCS)[9,10] in addition to dorsal root ganglion stimulation (DRGS).[6–8]

Although these studies have shown con-SCS to be effective, novel forms of stimulation could provide enhanced pain relief. In 2010, De Ridder et al[11] introduced a passive recharge burst pattern waveform that was free of paresthesia sensation (referred to in this article as Burst SCS). It was in 2017 that Burst SCS was found to be superior to con-SCS in patients with chronic pain of the trunk and/or limbs.[12]

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Source(s) of financial support: The authors reported no funding sources.

Using the Burst waveform in an SCS pilot study for the treatment of CD-LBP, our group showed promising improvements in pain and function, resulting in significant reduction of back and leg pain in patients with CD-LBP and decrease in the level of disability.[13]

DRGS is a direct spin-off of SCS, which places an electrical field near the somata of afferent nerve fibers.[14] Receiving its Conformité Européenne mark in 2013 and US Food and Drug Administration approval in 2015, DRGS was designed for the treatment of focal pain, although there have been multiple indications of the efficacy of DRGS in multidermatomal pain syndromes, including LBP.[15–17] DRGS has also been used for nonoperative LBP and was studied prospectively in the treatment of CD-LBP.[6] Con-SCS is believed to use the pain gate theory mechanistically, with antidromic stimulation of dorsal columns Aβ-fibers to modulate nociceptive signaling in the dorsal horn.[18] DRGS is thought to modulate nociceptive signals locally at the dorsal root ganglion (DRG) for focal pain, and multidermatomal coverage potentially though convergence and orthodromic propagation of signals into the dorsal horn.[18,19]

Innervation of the lumbar disks has been shown to travel through the sympathetic nervous system, converging at the L2 level.[20–25] Huygen et al[15] first studied L2 DRGS lead placement in LBP with good but mixed results because seven patients had >50% pain relief, whereas five had poor results. We refined the study criteria from these data and proposed L2 DRGS for CD-LBP, excluding patients who responded to medial branch blocks and sacroiliac joint injections and who subsequently had a positive discogram result. This prospective study used DRGS at L2 to treat CD-LBP, resulting in significant, maintained improvements in pain, function, and quality of life.[6]

The increased availability of new waveforms for pain treatment in CD-LBP warrants an evaluation of their effectivity in helping to choose the optimal therapy. In previous research, we studied the efficacy of both L2 DRGS and Burst SCS in independent studies for the treatment of CD-LBP, using the same inclusion and exclusion criteria in similar patient populations.[6,13] The aim of this analysis is to compare the effectivity of Burst SCS with that of L2 DRGS in pain relief, function, and quality of life in patients with CD-LBP.

MATERIALS AND METHODS

This study analyzed data from two separate prospective studies with similar CD-LBP populations, using identical inclusion and exclusion criteria.[6,13] The Burst SCS study was conducted from June 2019 to December 2021 and the L2 DRGS study from November 2015 to November 2017, both in the Rijnstate Hospital, The Netherlands. The trials were conducted by the same staff. Both trial protocols were approved by the local medical ethics committee Arnhem-Nijmegen (trial reference numbers: NL67172.091.18 and NL54405.091.1), and all participants gave written informed consent.

Pre- and postprocedure protocols were identical in the studies and are available in these works.[6,13] Implant procedures of DRGS and SCS were according to international protocol.[26] For the Burst SCS study, patients (n = 14) were implanted using two eight-contact "Octrode" epidural leads and programmed with the BurstDR stimulation paradigm (Proclaim™, Abbott; Plano, TX). Briefly, the left electrode tip was placed at T8, and the right tip was placed at T9; no intraoperative testing was performed. For the L2 DRGS study, patients (n = 15) were implanted with a four-contact 50-cm MN 20550-50 lead (Spinal Modulation, Inc, Menlo Park, CA) placed over the L2 DRG and an Axium Mn 20200 internal pulse generator (Spinal Modulation, Inc). During intraoperative testing, all patients reported paresthesia coverage of the painful region.

Burst SCS stimulation settings were 40 Hz interburst frequency, 500 Hz intraburst frequency of five pulses, 1-millisecond pulse width, and 1.3 to 0.6 mA with on/off cycling. Amplitudes were adjusted to 60% of threshold levels. Average and individual burst cycling settings are presented in Supplementary Data Tables S1 and S2. L2 DRGS stimulation settings ranged from 4 to 40 Hz frequency and between 100 and 420 milliseconds pulse width, and then reduced to subthreshold levels. Individual and average DRGS stimulation parameters are displayed in Supplementary Data Tables S3 and S4.

For both studies, patients completed the numeric pain rating score (NRS) as an index of pain,[27] at baseline, after implantation trial, and, with a positive trial, at three, six, and 12 months after implantation. As a measure of disability, the Oswestry Disability Index (ODI)[28] was completed, and as a measure of quality of life and health status, the EuroQoL 5D (EQ-5D)–3L or EQ-5D-5L[29,30] was used. Both were completed at baseline and at three, six, and 12 months after implantation. Only data on matching follow-up moments between the two studies are reported in this study.

Differences between time points of Burst SCS and L2 DRGS treatments were assessed using (non) parametric unpaired t-tests (GraphPad Prism). Differences between patient demographics were assessed using unpaired t-tests or chi-square test. Unless otherwise stated, error is displayed as SEM. Only data regarding patients who received permanent implants were evaluated.

RESULTS

Baseline characteristics did not differ between the patient populations with CD-LBP stimulated with either Burst SCS or L2 DRGS (Table 1). This analysis indicates that NRS scores are significantly lower (54%) for L2 DRGS (22.9 ± 5.7) than for Burst SCS (42.5 ± 5.2) after 12 months (p = 0.018) (Fig. 1). EQ-5D-5L scores were significantly increased at six (p = 0.013) and 12 months (p = 0.003) for L2 DRGS (0.82 ± 0.04, 0.84 ± 0.03) compared with Burst SCS (0.68 ± 0.04, 0.67 ± 0.04). No significant differences between Burst SCS and L2 DRGS were detected regarding ODI (Fig. 2). Burst SCS in addition to L2 DRGS significantly decreased NRS, ODI, and EQ-5D-5L rates over time, compared with baseline.[6,13] Data regarding the development of NRS, ODI and EQ-5D scores are presented in Table 2 and Supplementary Data Figures S1 to S6.

Table 1. Baseline Characteristics.

Characteristic	Burst SCS	DRGS	p Value
Age, y, ± SD	47.5 ± 13.4	49.0 ± 10.7	0.74
Sex, N (%)			0.65
Male	4 (27%)	8 (57%)	
Female	11 (73%)	6 (43%)	
BMI	26.2 ± 4.7	26.4 ± 3.8	0.92
Level of painful disk N (%)			
L3–L4	3 (18.8%)	1 (4.6%)	
L4–L5	7 (43.7%)	9 (40.9%)	
L5–S1	6 (37.5%)	12 (54.6%)	
Duration of discogenic LBP, y	9.0 ± 9.0	8.5 ± 1.4	0.83

BMI, body mass index.

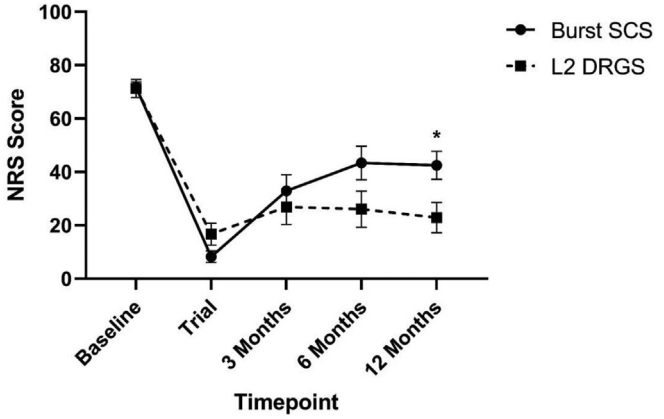

Figure 1. NRS scores for CD-LBP. DRGS provides significantly more CD-LBP relief at 12 months. Error bars display SEM.

DISCUSSION

This comparison of two prospective studies using neuromodulation for treatment of patients with CD-LBP indicates that L2 DRGS may be a better long-term treatment option than Burst SCS for CD-LBP. There may be inherent challenges that influence validity when comparing clinical studies for similar diagnoses, such as inclusion and exclusion criteria, age, sex, and cultural/social differences within patient populations. However, these two prospective pilot studies used a nearly identical inclusion criterion, patient populations, were performed by the same team, and measured the same outcomes. Both studies indicated a clinically significant efficacy in pain relief, function based on the ODI, and improvements in quality of life.

Pain Relief

Both Burst SCS and L2 DRGS were effective in the treatment of pain in patients with CD-LBP at all follow-up time points.[6,13] Although both groups showed early success, at 12 months, the L2 DRGS group revealed significantly improved pain relief of 22.9 on the NRS, on which Burst SCS pain relief only reached 42.5. In addition, pain relief associated with L2 DRGS remained stable after three months, whereas with Burst SCS, a trend of decreasing pain relief was shown after three months, eventually leading to statistical significance between the two treatments at 12 months (Fig. 1) (Table 2).

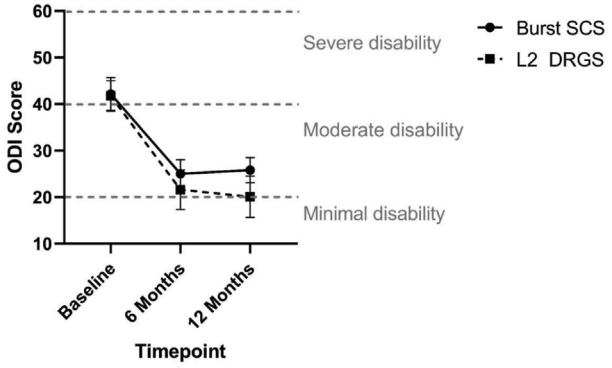

Figure 2. Oswestry Disability Index scores. Burst SCS and DRGS result in similar ODI scores. Error bars display SEM.

Table 2. Study Results.*

Questionnaire	Intervention	Baseline	Trial		3 mo		6 mo		12 mo	
NRS ± SEM	Burst SCS	71.7 ± 2.0	8.2 ± 2.2	$p = 0.094$	32.9 ± 6.0	$p = 0.518$	43.4 ± 6.3	$p = 0.073$	42.5 ± 5.2	$p = 0.018$*
	L2 DRGS	71.3 ± 3.4	16.7 ± 4.1		26.9 ± 6.1		26 ± 6.8		22.9 ± 5.7	
ODI ± SEM	Burst SCS	42.1 ± 3.5	–	–	–	–	25.0 ± 3.1	$p = 0.526$	25.8 ± 2.7	$p = 0.311$
	L2 DRGS	41.7 ± 3.3	–		–		21.6 ± 4.2		20.1 ± 4.4	
EQ-5D ± SEM	Burst SCS	0.5 ± 0.05	–	–	0.7 ± 0.02	$p = 0.360$	0.7 ± 0.04	$p = 0.012$*	0.7 ± 0.04	$p = 0.003$*
	L2 DRGS	64.7 ± 5.1	–		76.1 ± 4.0		78.7 ± 3.1		76.6 ± 3.3	

*Statistical significance $p < 0.05$.

Quality of Life

Both Burst SCS and L2 DRGS also showed clinically significant differences in quality of life in treating CD-LBP.[6,13] After parallel improvement up to the three-month point, DRGS resulted in significantly increased quality of life compared with Burst SCS at six and 12 months (Fig. 3) (Table 2).

Effects on Function and Disability

Both groups experienced statistically significant decreases in disability based on the ODI; there was no statistical significance between the treatments (Fig. 2) (Table 2).

Longevity of Therapy

Our data show a gradual increase in pain and gradual decrease in quality of life in the Burst SCS cohort starting at three months in contrast to L2 DRGS. Although underlying causation is unclear, we surmise that L2 DRGS may be more robust against loss of efficacy. This is supported by clinical evidence that indicated that in a pooled analysis of 249 DRGS cases, only a few explants were owing to inadequate pain relief, contrasting with the higher rates of explantation secondary to inadequate pain relief in SCS literature.[31–35]

Context

Previous works have evaluated both SCS and DRGs in prospective clinical settings, providing evidence for the success of these interventions in CD-LBP.[8,10,36] Differences between patient populations make it difficult to directly compare these works with the data described in this study. Our report is a step toward evaluating the differences between these treatment options.

We hypothesize that the mechanisms of action that underlie DRGS at L2 are responsible for our findings. As stated earlier, innervation of the ventrolateral IVDs and vertebral bodies occurs through fibers running in the sympathetic chain that converge at the L2 DRG.[20–23] Stimulation at the L2 DRG allows direct modulation of fibers transmitting CD-LBP by increasing its natural signal filtering effects.[19,37,38] In contrast, modulation of the dorsal columns via SCS occurs through a circuitous, multisynaptic path, targeting nonnociceptive dorsal column fibers and antidromically modulating the spinal nociceptive network to effect sympathetic transmission.[18,39] This may result in less specific targeting and blocking of the nociceptive C and Aδ fibers involved in CD-LBP.

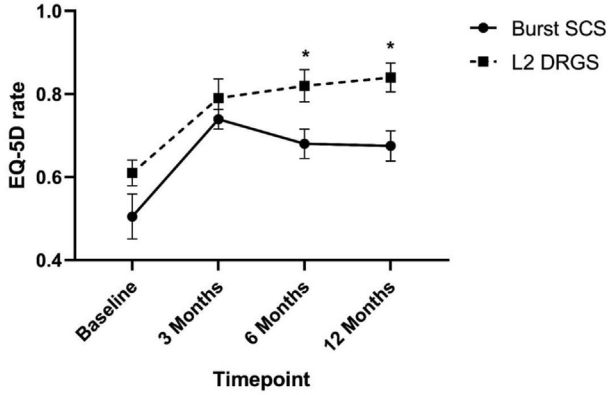

Figure 3. EQ-5D quality-of-life score. Burst SCS and DRGS result in similar EQ-5D rates. Error bars display SEM.

Limitations

Although this analysis presents what we believe is the first insight into efficacy of L2 DRGS compared with Burst SCS in the treatment of patients with nonoperated CD-LBP, its limitations must be noted. These are small prospective studies that cannot be compared with large randomized controlled trials, which are a more definitive means of comparing treatments. In addition, research has shown that lower Burst SCS amplitudes may result in increased pain relief.[40] However, because this has not yet been widely introduced in the clinic, this study reflects the current state of Burst SCS programming. Moreover, it is now common practice to standardize the cycling programs for Burst SCS, which was not previously the case.[41] Burst SCS has an established effect on the affective component of pain via the medial pathway, and we did not measure this.[42–45] Translational preclinical studies are needed to provide further mechanistic insight into the possible differences in mechanism of action between L2 DRGS and Burst SCS.

CONCLUSIONS

Comparison of two small-scale patient cohorts shows that L2 DRGS provided better long-term pain relief and increases in quality of life than did Burst SCS for patients with CD-LBP. Patients treated with L2 DRGS show significantly more pain relief at 12 months than do those treated with Burst SCS, and increased quality of life at six and 12 months.

Authorship Statements

Martijn R. Mons wrote the manuscript and performed data analysis; Jan-Willem Kallewaard designed and conducted the study, including patient recruitment; Chris Terwiel conducted the study, recruited patients, and performed data collection; Jan-Willem Kallewaard, Kenneth B. Chapman, and Elbert A. Joosten provided editorial support. All authors approved the final version of the manuscript.

How to Cite This Article

Mons M.R., Chapman K.B., Terwiel C., Joosten E.A., Kallewaard J.W. 2023. Burst Spinal Cord Stimulation as Compared With L2 Dorsal Root Ganglion Stimulation in Pain Relief for Nonoperated Discogenic Low Back Pain: Analysis of Two Prospective Studies.
Neuromodulation 2023; ■: 1–6.

SUPPLEMENTARY DATA

To access the supplementary material accompanying this article, visit the online version of *Neuromodulation: Technology at the Neural Interface* at www.neuromodulationjournal.org and at https://doi.org/10.1016/j.neurom.2023.04.464.

REFERENCES

1. Navone SE, Marfia G, Giannoni A, et al. Inflammatory mediators and signalling pathways controlling intervertebral disc degeneration. *Histol Histopathol*. 2017;32:523–542.
2. Fournier DE, Kiser PK, Shoemaker JK, Battié MC, Séguin CA. Vascularization of the human intervertebral disc: a scoping review. *JOR Spine*. 2020;3:e1123.

3. Groh AMR, Fournier DE, Battié MC, Séguin CA. Innervation of the human intervertebral disc: a scoping review. *Pain Med*. 2021;22:1281–1304.
4. Raj PP. Intervertebral disc: anatomy-physiology-pathophysiology-treatment. *Pain Pract*. 2008;8:18–44.
5. Kallewaard JW, Terheggen MAMB, Groen GJ, et al. 15. Discogenic low back pain. *Pain Pract*. 2010;10:560–579. https://doi.org/10.1111/j.1533-2500.2010.00408.x.
6. Kallewaard JW, Edelbroek C, Terheggen M, Raza A, Geurts JW. A prospective study of dorsal root ganglion stimulation for non-operated discogenic low back pain. *Neuromodulation*. 2020;23:196–202. https://doi.org/10.1111/ner.12937.
7. Alo KM, Yland MJ, Redko V, Feler C, Naumann C. Lumbar and sacral nerve root stimulation (NRS) in the treatment of chronic pain: a novel anatomic approach and neuro stimulation technique. *Neuromodulation*. 1999;2:23–31.
8. Mehta V, Bouchareb Y, Ramaswamy S, Ahmad A, Wodehouse T, Haroon A. Metabolic imaging of pain matrix using 18 F fluoro-deoxyglucose positron emission tomography/computed tomography for patients undergoing L2 dorsal root ganglion stimulation for low back pain. *Neuromodulation*. 2020;23:222–233.
9. Al-Kaisy A, Palmisani S, Smith TE, et al. Long-term improvements in chronic axial low back pain patients without previous spinal surgery: A cohort analysis of 10-kHz high-frequency spinal cord stimulation over 36 months. *Pain Med*. 2018;19:1219–1226.
10. Vallejo R, Zevallos LM, Lowe J, Benyamin R. Is spinal cord stimulation an effective treatment option for discogenic pain? *Pain Pract*. 2012;12:194–201. https://doi.org/10.1111/j.1533-2500.2011.00489.x.
11. de Ridder D, Vanneste S, Plazier M, van der Loo E, Menovsky T. Burst spinal cord stimulation: toward paresthesia-free pain suppression. *Neurosurgery*. 2010;66:986–990.
12. Deer T, Slavin KV, Amirdelfan K, et al. Success using neuromodulation with BURST (SUNBURST) study: results from a prospective, randomized controlled trial using a novel burst waveform. *Neuromodulation*. 2018;21:56–66. https://doi.org/10.1111/ner.12698.
13. Mons MR, Chapman KB, Terwiel C, Joosten EA, Kallewaard JW. A prospective study of BurstDR™ spinal cord stimulation for non-operated discogenic low back pain. *Pain Pract*. 2023;23:234–241. https://doi.org/10.1111/papr.13181.
14. Liem L, Russo M, Huygen FJPM, et al. A multicenter, prospective trial to assess the safety and performance of the spinal modulation dorsal root ganglion neurostimulator system in the treatment of chronic pain. *Neuromodulation*. 2013;16:471–482 [discussion: 482].
15. Huygen F, Liem L, Cusack W, Kramer J. Stimulation of the L2–L3 dorsal root ganglia induces effective pain relief in the low back. *Pain Pract*. 2018;18:205–213.
16. Deer TR, Levy RM, Kramer J, et al. Dorsal root ganglion stimulation yielded higher treatment success rate for complex regional pain syndrome and causalgia at 3 and 12 months: a randomized comparative trial. *Pain*. 2017;158:669–681.
17. D'Souza RS, Kubrova E, Her YF, et al. Dorsal root ganglion stimulation for lower extremity neuropathic pain syndromes: an evidence-based literature review. *Adv Ther*. 2022;39:4440–4473.
18. Joosten EA, Franken G. Spinal cord stimulation in chronic neuropathic pain: mechanisms of action, new locations, new paradigms. *Pain*. 2020;161(suppl 1):S104–S113.
19. Koopmeiners AS, Mueller S, Kramer J, Hogan QH. Effect of electrical field stimulation on dorsal root ganglion neuronal function. *Neuromodulation*. 2013;16:304–311 [discussion: 310].
20. Ohtori S, Takahashi K, Chiba T, Yamagata M, Sameda H, Moriya H. Sensory innervation of the dorsal portion of the lumbar intervertebral discs in rats. *Spine*. 2001;26:946–950.
21. Nakamura SI, Takahashi K, Takahashi Y, Morinaga T, Shimada Y, Moriya H. Origin of nerves supplying the posterior portion of lumbar intervertebral discs in rats. *Spine*. 1996;21:917–924.
22. Aoki Y, Takahashi Y, Takahashi K, et al. Sensory innervation of the lateral portion of the lumbar intervertebral disc in rats. *Spine J*. 2004;4:275–280.
23. Morinaga T, Takahashi K, Yamagata M, et al. Sensory innervation to the anterior portion of lumbar intervertebral disc. *Spine*. 1996;21:1848–1851.
24. Quinones S, Konschake M, Aguilar LL, et al. Clinical anatomy of the lumbar sinuvertebral nerve with regard to discogenic low back pain and review of literature. *Eur Spine J*. 2021;30:2999–3008.
25. Breemer MC, Malessy MJA, Notenboom RGE. Origin, branching pattern, foraminal and intraspinal distribution of the human lumbar sinuvertebral nerves. *Spine J*. 2022;22:472–482.
26. Chapman KB, Spiegel MA, Dickerson DM, et al. A paramedian approach for dorsal root ganglion stimulation placement developed to limit lead migration and fracture. *Pain Pract*. 2021;21:991–1000.
27. Perrot S, Lantéri-Minet M. Patients' Global impression of Change in the management of peripheral neuropathic pain: clinical relevance and correlations in daily practice. *Eur J Pain*. 2019;23:1117–1128.
28. Fairbank JCT, Pynsent PB. The Oswestry disability index. *Spine (Phila Pa 1976)*. 2000;25:2940–2952 [discussion: 2952].
29. Devlin NJ, Brooks R. EQ-5D and the EuroQol Group: past, present and future. *Appl Health Econ Health Policy*. 2017;15:127–137.
30. Rabin R, de Charro F. EQ-5D: A measure of health status from the EuroQol Group. *Ann Med*. 2001;33:337–343.
31. Chapman KB, Yang A, Mogilner AY, et al. Dorsal root ganglion stimulation device explantation: a multicenter pooled data analysis. *Pain Pract*. 2022;22:522–531. https://doi.org/10.1111/papr.13113.
32. Dupré DA, Tomycz N, Whiting D, Oh M. Spinal cord stimulator explantation: motives for removal of surgically placed paddle systems. *Pain Pract*. 2018;18:500–504. https://doi.org/10.1111/papr.12639.
33. Simopoulos T, Aner M, Sharma S, Ghosh P, Gill JS. Explantation of percutaneous spinal cord stimulator devices: a retrospective descriptive analysis of a Single-Center 15-year experience. *Pain Med*. 2019;20:1355–1361.
34. Thomson SJ, Kruglov D, Duarte R v. A Spinal cord stimulation service review from a single centre using a single manufacturer over a 7.5 year follow-up period. *Neuromodulation*. 2017;20:589–599.
35. Wang VC, Bounkousohn V, Fields K, Bernstein C, Paicius RM, Gilligan C. Explantation rates of high frequency spinal cord stimulation in two outpatient clinics. *Neuromodulation*. 2021;24:507–511.
36. Mehta V, Poply K, Ahmad A, et al. Effectiveness of high dose spinal cord stimulation for non-surgical intractable lumbar radiculopathy - HIDENS Study. *Pain Pract*. 2022;22:233–247.
37. Kent AR, Min X, Hogan QH, Kramer JM. Mechanisms of dorsal root ganglion stimulation in pain suppression: a computational modeling analysis. *Neuromodulation*. 2018;21:234–246.
38. Esposito MF, Malayil R, Hanes M, Deer T. Unique characteristics of the dorsal root ganglion as a target for neuromodulation. *Pain Med*. 2019;20(suppl 1):S23–S30.
39. Smits H, van Kleef M, Joosten EA. Spinal cord stimulation of dorsal columns in a rat model of neuropathic pain: evidence for a segmental spinal mechanism of pain relief. *Pain*. 2012;153:177–183.
40. Leong SL, de Ridder D, Deer T, Vanneste S. Potential therapeutic effect of low amplitude burst spinal cord stimulation on pain. *Neuromodulation*. 2021;24:574–580.
41. Deer TR, Patterson DG, Baksh J, et al. Novel intermittent dosing burst paradigm in spinal cord stimulation. *Neuromodulation*. 2021;24:566–573. https://doi.org/10.1111/ner.13143.
42. de Ridder D, Plazier M, Kamerling N, Menovsky T, Vanneste S. Burst spinal cord stimulation for limb and back pain. *World Neurosurg*. 2013;80:642–649.e1.
43. Hagedorn JM, Falowski SM, Blomme B, Capobianco RA, Yue JJ. Burst spinal cord stimulation can attenuate pain and its affective components in chronic pain patients with high psychological distress: results from the prospective, international TRIUMPH study. *Spine J*. 2022;22:379–388.
44. Yearwood T, de Ridder D, Yoo HB, et al. Comparison of neural activity in chronic pain patients during tonic and burst spinal cord stimulation using fluorodeoxyglucose positron emission tomography. *Neuromodulation*. 2020;23:56–63.
45. de Ridder D, Vanneste S. Burst and tonic spinal cord stimulation: different and common brain mechanisms. *Neuromodulation*. 2016;19:47–59.

COMMENTS

This is an interesting comparison between two different neuromodulation techniques to treat virgin low back pain, both provided by the same company so that at least industry bias is removed. The authors conclude that DRG of L2 is superior to BurstDR SCS. Although the data do suggest this, there are two caveats that limit the generalizability of this conclusion. The first caveat is that the study was not a priori set up to compare the two technologies, which limits the scientific value because there was no randomization of patients, the settings were different, and potentially, the clinical indications were not exactly the same. The second, more important caveat is that BurstDR SCS was provided in a nonstandardized way, and the data suggest that many patients could potentially have been overstimulated; that is, the amplitudes may have been too high. The progressive loss of efficacy in time hints at this. Unfortunately, the SCS amplitudes could not be provided by the authors, which precludes a subanalysis that correlates the efficacy of pain suppression of BurstDR with delivered amplitudes. Indeed, it has previously been shown that lower BurstDR amplitudes yield superior pain suppression. Therefore, the conclusion must be considered somewhat premature, and a true prospective randomized comparison study is required to determine whether one of the two techniques is superior. Nevertheless, even with its limitations, the study is beneficial in that it paves the way for much needed comparison studies between different neuromodulation approaches to help pain physicians decide what technique or technology may offer the patient most chances of successful pain suppression.

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This report compares two neurostimulation approaches to treating primary discogenic pain: dorsal column stimulation with BurstDR and DRGS. The first technique is more regional in effect, while the second is known to be more focal in effect. The authors derive their conclusions from 12-month data from two separate prospective studies conducted by the same team, using identical patient selection criteria to focus on non-operative discogenic pain by excluding facet arthropathy or radiculopathy as components of axial low back pain. Positive discographic evidence was also a requirement for subject selection.

This report comparing the results of prospective studies on two different devices from the same manufacturer is rare in the neuromodulation literature, if not unique. Both techniques provided patients with sub-perception stimulation. DRGS fared slightly better than BurstDR at the 12-month mark, though the numbers of participants in each study were small and possibly clouded the statistical relevance. There could be confounding issues with each study, especially regarding programming optimization. As noted previously, the potential exists for a slow drift towards overstimulation with BurstDR. Also, the authors do not provide data on the paresthesia overlap of the painful areas using the treating mode of stimulation (BurstDR). Targeting therapeutic BurstDR programming using a "Tonic" stimulation paradigm may not truly represent the paranesthesia pattern of the therapy in operation. Anatomic placement of the contacts used for DRGS is not available, and there is no indication if the active contacts are at the DRG, near the DRG, proximal to the DRG or distal to the DRG. At best, DRGS, as presented here, can only be assumed to be transforaminal. Despite the low numbers involved, and the inherent difficulties associated with targeting and programming optimization for both therapeutic paradigms, this report suggests the DRGS may be a superior technique in terms of treatment durability. It would be interesting to see if a larger prospective study of combined focal and regional techniques provided superior therapy to either technique in isolation. This report helps establish the technique and durability question for this specific phenotype and its remarkable sensory neural network. Similar efforts on other pain phenotypes (ie, visceral vs nociceptive; or complex regional pain syndrome Type I vs Type II) should also be considered.

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