SYSTEMATIC REVIEW

doi: 10.5455/medarh.2023.77.370-376
MED ARCH. 2023; 77(5): 370-376
RECEIVED: SEP 10, 2023
ACCEPTED: OCT 04, 2023

Imam Abdulrahman Bin Faisal University. ashar Ibn Burd St. Al Aqrabiyah, Al Khobar

Corresponding author: Faisal Meshael AlAbbas, Imam Abdulrahman Bin Faisal University. ashar Ibn Burd St. Al Aqrabiyah, Al Khobar 3444. E-mail address: fmabbas@iau.edu.sa ORCID ID: https://orcid.org/0000-0002-4367-1432

Surgical Neuromodulation of Traumatic Brachial Plexus Injuries a Systematic Review and Meta-analysis

Faisal Meshael AlAbbas

ABSTRACT

Background: Traumatic brachial plexus injuries are common among young adults, with a majority of patients succumbing to chronic pain syndromes. Conservative management is usually not satisfactory in these cases and surgical interventions areoften required. We have conducted a systematic review and meta-analysis examining one of the neurosurgical techniques, spinal cord stimulation (SCS), in chronic pain neuromodulation in cases of chronic pain syndrome after traumatic brachial plexus injuries. **Objective:** This systematic review aims toexplore the reported use of cervical spinal cord stimulation as a neuromodulator in patients with chronic pain syndromes following traumatic brachial plexus injury. **Methods**: A systematic literature search was conducted using MEDLINE through the OVID interface, ProQuest, Web of Science, The Cochrane Library, and Scopus. Our own files and reference lists of identified key articles were also searched. **Results:** A total of 13 studies (8 case reports and 5 case series), comprising 29 patients were included. Most brachial plexus injuries were sustained in motor vehicle accidents. 86% (25/29) of patients showed a good initial response to SCS, however, the response decreased over time, and 69% (20/29) of the patients reported a good response at theend of follow-up. Lead migration was the only complication reported in two studies. **Conclusion:** SCS is a less invasive procedure with significantly fewer neurological side effects. A trial period of SCS is suggested in patients who have failed conservative treatment modalities before other neurosurgical interventions are considered.

Keywords: Brachial plexus neuropathy; brachial plexus avulsion; chronic regional pain syndrome; neurostimulation; neuromodulation.

1. BACKGROUND

Brachial plexus injuries are devastating and disabling conditions commonly affecting young adults after motor vehicle accidents (1, 2). The injury may vary considerably fromthe involvement of the roots (either complete or partial) to individual peripheral nerves arising from the cords. Between 30% to 80% of patients with traumatic brachial plexus injuries develop chronic pain syndromes. (1, 2) In approximately 40% of cases, this pain is severe. (3) These syndromes include complex regional pain syndrome, brachial plexopathies, and secondary neuropathic pain due to direct nerve injury. For the most part, these syndromes are not responsive to medical therapy, and invasive interventions are common (4). Many patients are injured in motor vehicle accidents or at work, are quite young, and require effective long-term pain management. Surgical interventions to manage these conditions include nerve reconstruction procedures, such as brachial plexus nerve root transfer or sural nerve grafts. However, these techniques report inconsistent success and have variable incidences of persistent postoperative pain. (3,4) Dorsal root entry zone (DREZ) lesioning has been successfully used to manage deafferentation pain after brachial plexus avulsion. However, its utility in those patients reporting ongoing background pain has not been good (6).

Cervical spinal cord stimulation (SCS) is an intervention that has been successfully usedin some cases of traumatic brachial plexus injury (2, 7, 8).

Author(s)	Year	Study design	Number of	Mechansm of injury	Priorinterventions	Intervention	Follow-up period
Garcia-March	1987	Case series	6	MVA, fall, industrial injury	Anticonvulsants, opioids, stellate ganglion block,	Cervical dorsal column	12 to 58 months
Bennett & Tai (2)	1994	Case series	5	4 – industrial accident1–MVA	Antidepressants, anticonvulsants, opioids, stellate ganglion blocks,	Cervical dorsal column spinal cord	13.5 months
Segal (8)	1999	Case report	1	MVA	Antidepressants, anticonvulsants, opioids, stellate	Cervical dorsal column	13 months
Teixei ra et al (10)	1999	Case series	4	MVA	Antidepressants, anticonvulsants, opioids, stellate	Cervical dorsal column	36 months
Piva et al (7)	2003	Case series	4	Undefined traumatic injury	Antidepressants, anticonvulsants, opioids, stellate ganglion block	Seven point cervical and thoracic dorsal	9 months
Brill & Aryeh	2008	Case series	2	MVA	Antidepressants, anticonvulsants, opioids, stellate	Cervical dorsal column	12 months
Lai et al (17)	2009	Case report	1	MVA	Anticonvulsants, opioids, DREZ surgery	Cervical dorsal column	12 months
Abdel-Aziz &	2014	Case report	1	MVA	Antidepressants, anticonvulsants, NSAIDs, opioids	Cervical spinal cord stimulation	1 month
Chien et al (13)	2014	Case report	1	MVA	Antidepressants, anticonvulsants, opioids, stellate	Cervical dorsal column	13 months
Choi et al (14)	2016	Case report	1	Iatrogenic after excision of superior mediastinal	Antidepressants, anticonvulsants, opioids, stellate ganglion blocks,	Cervical dorsal column spinal cord	6 and then 12 months
Lopez et al (18)	2016	Case report	1	Gunshot	Antidepressants, opioids	Cervical dorsal column	6 months andthen 6 months
Watan abe et al (20)	2018	Case report	1	MVA	Medication and nerve block	Cervical dorsal and ventral	6 months
Florid ia et al (15)	2018	Case report	1	Iatrogenic after plexus blockage during	Anticonvulsants, NSAIDs, cervical epidural corticosteroid injections	Cervical dorsal column spinal cord	6 months

Table 1. Characteristics of included studies investigating the efficacy of spinal cord stimulation to neuromodulate chronic neuropathic pain following brachial plexus avulsion Notes: Abbreviations DREZ = dorsal root entry zone lesion, MVA = motor vehicle accident, NSAID = nonsteroidal anti-inflammatory drug, TENS = transcutaneous electrical nerve stimulant

2. OBJECTIVE

This systematic review aims to explore the reported use of cervical spinal cord stimulation as a neuromodulator in patients with chronic pain syndromes following traumatic brachial plexus injury

3. MATERIAL AND METHODS

The review was conducted according to the Cochrane Handbook and reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analysis guidelines (PRISMA) (9,10). A PRISMA checklist was completed and can be viewed as Appendix A.

Eligibility criteria

All letters to the editor, case reports, case series, observational studies, or randomized controlled trials (RCTs) examining SCS aimed at chronic pain neuromodulation in patients with traumatic brachial plexus injuries were considered eligible for inclusion in this systematic review. We excluded studies where: a) no chronic pain condition was present (> 6 months of pain), b) only drug therapy was used for pain neuromodulation, c) radiofrequency ablation, deep-brain stimulation, or peripheral muscle or nerve stimulation was used, or d) Nerve transfer was performed.

Information sources and search strategy

The following electronic databases were searched for articles published up to December 23, 2021: PubMed, EMBASE, CT.gov, ICTRP, CINAHL, and The Cochrane Library. We also searched our files, consulted with experts, reviewed reference lists from identified articles, and searched for cited references of key publications. The following combination of keywords and Medical

Author(s), year	Stimulator used	Stimulator settings	Electrode coverage	Trial before permeant implant?
Garci- March et al 1987 (16)	Bipolar stimulation	Frequency: 80 – 120 Hz Pulse width: 500 microsecondsIntensity: Suprathreshold to induce paresthesia	Positioned to match patients pain distribution	Yes
Bennett & Tai, 1994 (2)	Not reported	Not reported	C5 to C7	Yes
Segal, 1999 (8)	Medtronic–Resume TL thin line(Failure to place quad	Frequency: 55 HzPulse width: 180 microseconds Intensity: 1 Volt	C2-C3	Yes
Teixeira et al 1999 (19)	Medtronic – Pisces-Quad(4-contact electrode)	Frequency: 130 Hz Pulse width: not specified Intensity: adjusted to induceparesthesia	Positioned to match patients pain distribution	Yes
Piva et al 2003 (7)	Advanced neuromodulation systems–Cervitrode	Frequency: 50 to 100 Hz Pulse width: 400 microsecondsIntensity: 0.5 – 1 Volt	C2 to T1-2	Yes
Brill & Aryeh, 2008 (12)	Medtronic and Advanced Neuromodulation	Frequency: 20 to 40 Hz Pulse width: 221 microsecondsIntensity: 5 to 6.5 ampere	Patient directed – details not	Yes
Lai et al 2009 (17)	Medtronic MN	Not reported	C3-C5	No
Abdel- Aziz &	Not reported	Not reported	C3 to C6	Yes
Chien et al 2014 (13)	St Jude's Medical -Octode(8-contact electrode)	Frequency: 60 HzPulse width: 300 microseconds Intensity: 1.2 mA to 4.8 mA	Left C2-C5Right C4-7	Yes
Choi et al 2016 (14)	Not reported	Not reported	C3-6	No
Lopez et al 2016 (18)	St Jude's Medical -Lamitrode 3240 (4-contact electrode)	Frequency: 10 – 130 HzPulse width: 60 – 200 microsecondsIntensity: 5.0 to 13 mA	NR	No
Florida et al 2018	High frequency stimulator	Frequency: 10 kHz	C2-C3	Yes
Watanabe et al., 2018 (20)	Medtronic – Pisces-Quad(2 leads, one dorsal, one ventral)	Frequency: 25 HzPulse width: 120 microseconds Intensity: 2.7 Volts	C5/C6	No

Table 2. Characteristics of the spinal cord stimulators, settings, and cervical coverage used in the included studies investigating the efficacy of spinal cord stimulation to neuromodulate chronic neuropathic pain following brachial plexus avulsion

Subject Headings (MeSH) terms were used: "Brachial Plexus Neuropathies" AND "Treatment".

Eligibility assessment

We screened the titles and abstracts of each identified citation. Those reports possibly meeting the eligibility criteria were extracted for full-text review.

Outcomes of interest

The primary outcomes of interest were the success of SCS in modulating pain followingtraumatic brachial plexus injury. We further sought to identify the criteria used to select patients for SCS and the rate of postoperative complications

Quality and risk of bias analysis

We assessed the quality and risk of bias of each study using the following questions: 1) Is the study's objective clearly stated? 2) Is this a prospective study? 3) Is this a multicenter study? 4) Is the mechanism of injury stated? 5) Are adequate patient characteristics provided? 6) Is the stimulation intervention clearly described? 7) Are the outcomes measures defined? 8) Are the outcomes measures appropriate for the study aims? 9) Is the length of follow-up reported? 10) Is the number of patients lost to follow-up reported? 11) Are adverse events reported? 12) Are the study conclusions supported by the results? 13) Have competing interests and sources of support been reported?

Statistical analysis

A-priori, it was decided that no formal data meta-analysis would be attempted if less than three RCTs were included in the review.

4. STUDY CHARACTERISTICS

Table I details the design of the included studies representing 29 patients. The most were case reports (8,11,13-15,17,18,20) (n=8) with the remainder being case series (n=19).2,7,12,16,19 Most injuries were sustained in motor vehicle accidents, (2, 8,11- 13,16,17,19,20) industrial injuries contributed significantly (2,16) and two cases were due to iatrogenic intraoperative injuries. (14,15) Before undergoing SCS, most patients had been treated with combinations of antidepressants, anticonvulsants, opioids, stellate ganglion blocks, and in some cases, epidural corticosteroid injections. One of the patients had previously failed low-frequency SCS treatment 15 and another had two failed DREZ surgeries. (17) All studies used cervical dorsal column spinal cord stimulation, except for one that extended electrode coverage to include T1-T2.(7) and a second that included ventral stimulation. (20) Follow-ups ranged from 1 month (11) to 58 months (16) with the most common follow-up period being 12 months.

Table II details the characteristics of the spinal cord stimulators used, their settings, and the field of electrode coverage. Manufactures included Medtronic (Resume TL, Pisces- Quad, 8-contact electrodes) (8,12,13,19,20)

Author(s), year	Pain scoring system	Response to SCS	Complications
Garci-March et al., 1987 (16)	Not described	**Initial**: 2 excellent, 2 good, 2 fair responses**Follow-up:** 1 pain free, 2 fair responses, 3 failure of treatment	None reported
Bennett & Tai, 1994 (2)	VASVerbal rating scale	Mean VAS reduction: 4.0 Reduction in oral analgesic intake Improved mood score (1.9 p>0.01)	None reported
Segal, 1999 (8)	Not described	Compete pain resolution No analgesia use	None noted
Teixeira et al 1999 (19)	VAS	**Initial:** 2 good responses, 2 no response**Follow-up:** 1 regression to baseline	None noted
Brill & Aryeh, 2008 (12)	VAS LANSSOpioid use	Mean VAS reduction: 9.5 to 2.5Mean LANSS reduction: 19.5 to 12.5 Mean morphine reduction: 80 mg daily to 10 mg	Catheter migration
Piva et al., 2003 (7)	VAS	Mean VAS reduction: 9.0 to 5.9	None reported
Lai et al., 2009 (17)	Not described	Compete pain resolution No analgesia use	None reported
Abdel-Aziz & Ghaleb, 2014 (11)	NRS	NRS reduction: 50%	None reported
Chien et al 2014 (13)	NRS	Mean NRS reduction: 9 to 3 Reduction in tramadol useImprovements in mood and sleep quality	Catheter migration (caudal)
Choi et al 2016 (14)	NRS	**Initial:** NRS reduction: 8.5 to 3.5**Follow-up:** return to baseline pain scores requiring	None reported
Lopez et al 2016 (18)	VAS	**Initial:** VAS reduction: 10 to 6**Follow-up:** regression to baseline pain scores	None reported
Floridia et al 2018 (15)	NRS	Complete pain resolution: NRS=0 No analgesic use	None reported
Watanabe et al 2018 (20)	VAS	VAS reduction: 8.9 to 5.5	None noted

Table 3. Patient pain scoring systems and outcomes of included studies investigating the efficacy of spinal cord stimulation to neuromodulate chronic neuropathic pain following brachial plexus avulsion. Notes: Abbreviations LANSS= Leeds Assessment of Neuropathic Symptoms and Signs Scale, NRS = numerical rating scale, SCS = spinal cord stimulation, VAS = visual analog pain scale.

Advanced Neuromodulation Systems (7,12), and St Jude's Medical (13, 18) The remainder of the studies did not report the type of stimulator used. (2,11,14,15) Low-frequency settings (20 to 130 Hz) were used in all but one study where a high-frequency stimulator at 10 kHz was used.15Pulse width ranged from 60 to 500 microseconds and pulse intensity from 1.2 to 13 mA. In most studies electrode position was determined by initial imaging of the injury and then fine-tuned with direct patient feedback. A temporary pulse generator was used for a trial period in nine of the studies. (2,7,8,11-13,15,16,19)

RISK OF BIAS

A summary of each risk of bias, presented as a percentage across all included studies, can be found in Figure 2. Figure 3 reports the risk of bias for the individual bias items for all included studies. All included studies were case reports or case series – no RCTs were identified or included. None of the studies were prospectively designed or were multicenter in nature. Generally, patient details, mechanism of injury, outcome measures, length of follow-up, and loss of follow-up were well reported. Many studies failed to report on the presence of adverse events specifically, and most did not report competing interests or sources of financial support.

IMPACT OF SCS NEUROMODULATION

Table III describes the patient pain scoring systems, postintervention pain score changes, and reported adverse events after implementing SCS. All studies showed a good (>40% reduction in pain scores, or a cessation or reduction in oral analgesia use) immediately after initiation of SCS, except for Garci-March et al., where two of the six patients were reported to have a "fair" response, (16) and Teixeira et al. where two of the four patients did not have any response to SCS. (19) Thus, a total of 86% (25/29) patients showed a good initial response to SCS. In addition, some studies further reported associated improvements in quality of life after SCS (2,13,15) Importantly, three of the studies. (4,16,19) representing nine patients (31%), reported deterioration in pain control over the follow-up period, irrespective of their initial response to SCS. Choi et al. reported worse pain scores at six months, which required the addition of peripheral nerve stimulation. (14) In the series reported by Garci-March et al., only one of the six patients was pain-free at 28 months, and three were considered to have failed treatment. (16) All three of these patients progressed to DREZ treatment. Teixeira et al.found that two of the four patients having SCS had no sustained reduction in pain scores, and one patient who had initially shown "considerable" pain reduction experienced a significant pain relapse at 14 months. (19) Thus, by the end of follow-up, 69% (20/29) of patients retained a good response to SCS.

SCS complications

Two studies reported lead migration at the last follow-up. (12,13) No studies reported any postoperative infections.

5. DISCUSSION

Brachial plexus avulsion is a complex and devastating injury. The injury includes direct nerve root injury and

may often cause preganglionic injury. (21) The avulsion may be partial or complete, including all five roots. In cases of partial brachial plexus avulsion, it is unclear whether the avulsed roots play a role in the chronic pain complexes or whether the preserved roots are more important in the pathophysiology (1, 6, 22).

The spinal ganglia are the interface between the central and peripheral nervous systems. This provides the mechanism behind the complex chronic pain phenomena described bypatients, including burning, shooting, crushing, and phantom limb pain (22). Complex pain syndromes are managed using a broad and diverse range of interventions. Non- surgical interventions include non-steroidal anti-inflammatory drugs, antidepressants, antiepileptics, local anesthetic blocks, infusions of lidocaine or ketamine, and experimental drugs such as cannabinoids. Gebreyohanes et al. classifies surgical interventions for managing brachial plexus avulsions as ablative, modulatory, or reconstructive (22). Ablative interventions include thalamotomy stereotactic mesencephalotomy and spinal cord anterolateral cordotomy, and DREZ lesioning. Modulatory interventions include electrical motor cortex stimulation, thalamic deep brain stimulation, and spinal cord stimulation. Most reconstructive techniques have focused on nerve transfer.

The ideal management approach for patients with chronic pain after traumatic brachial plexus injuries remains unclear. Current practices focus on the use of SCS or DREZ lesioning, with a tendency to favor DREZ.

SCS stimulation is thought to have its effect through A-fiber modulation. Low-frequency SCS has sought to induce paresthesia in the target area, described by patients as a tingling or uncomfortable sensation of vibration. Anterograde stimulation of the large-diameter fibers belonging to the dorsal column is thought to mediate this sensation. In addition, pain relief may further be mediated by the inhibition of wide-dynamic-range neurons in the lamina V – a critical factor in driving neuropathic pain (23, 24) Chien et al. provide an excellent discussion of the possible physiological mechanism underlying pain modulation in SCS (13).

This analysis has shown that the long-term response to traditional low-frequency SCS isunpredictable. While most patients experience early benefits, approximately 30% see a reduction in efficacy after 6 to 12 months. Still, other studies have reported a loss of efficacy up to 2 years after surgery. Therefore, it is essential to appreciate that most of the studies reported in this analysis had follow-up periods shorter than two years.

Recently, studies have reported success with high-frequency SCS in patients with chronic back and limb pain. (25-27). High-frequency SCS uses frequencies of 10,000 Hzinstead of traditional low-frequency stimulation with frequencies of between 30 and 150Hz. High-frequency stimulation uses short-duration pulses, approximately 30 microseconds, with an amplitude of 2 to 5 mA. This stimulation does not seem to cause paresthesia by initiating an action potential in the lemniscal pathway. Instead, it is postulated that high-frequency stimulation may entrain the small and medium-diameter dorsal column fibers, thereby causing pain modulation (15).

The most common alternative to SCS is DREZ lesioning surgery, and studies have reported good results in more than 75% of these patients. (22) However, DREZ lesioning may not reach lamina IV to VI, thereby causing treatment failure. Further, dueto atrophy and distortion of the cervical cord, DREZ procedures may not reach the targeted sites. Finally, DREZ's semi-blind landmark technique may not be preciseenough to target the substantia gelatinosa accurately. As a result of these limitations, DREZ studies report pain recurrence within 6 to 12 months, good pain relief in approximately 75% of patients five years after treatment, and a high incidence of paralysis and proprioceptive disorders (7).

From our analysis of these papers, it was instructive to see the progressive development of standardized approaches to screening and implementation of SCS (25) Appropriate patient selection lays the foundation of clinical success when using SCS. The primary eligibility criterion for SCS is the presence of chronic pain that has been refractory to at least three months of conservative management. Conservative management should include pain medication, pharmacological and behavioral interventions, physical therapy, and possibly epidural injections or nerve blocks. Pain should have a VAS intensity of 5 or more and be accompanied by high disability index scores (e.g., Oswestry Disability Index 40 to 80 out of 100). (25) Care should be taken to meticulously document all conservative management modalities, as well as baseline pain and disability scores. Critical exclusion criteria should be sought. These include thepresence of active disruptive psychiatric or psychological disorders. Other conditions that may affect pain perception or the inability to comply with postoperative follow-up plans may need to be identified as well.

We identified two new aspects related to SCS. The first was the combination of both dorsal and ventral leads as described by Watanabe et al. (20). This method of stimulationmay induce transverse spinal cord stimulation in a dorsoventral direction, thereby potentially generating a wider range of paresthesia and greater neuromodulation. However, it is likely that standard SCS limitations and possible loss of neuromodulation over time would still apply to this technique. Further, there is insufficient data to understand if routinely placing ventral leads would provide improved neuromodulation or if it would improve the duration of pain modulation. The second aspect is the use of high-frequency SCS (15, 25). Its utility for chronic back and limb pain continues to be demonstrated and we believe this holds exceptional promise in patients with traumatic brachial plexus injuries. Based on this review, we wish to offer the following suggestions for managing patients with traumatic brachial plexus injuries. First, we suggest that SCS be considered the first-line treatment in patients with chronic pain after brachial plexus avulsion who havefailed conservative treatment modalities. SCS is less invasive than DREZ, allows for a trial period of temporary stimulation, and has significantly less permeant neurological side ef-

fects. Its two major risk factors are a failure to provide adequate pain modulation and catheter migration, both of which are not permanent. Second, the benefits of SCS may be further enhanced if high-frequency SCS is used as a first-line intervention.Third, we suggest that a period of trial stimulation be used in all patients before generator implantation. Fourth, in patients who fail high-frequency SCS, DREZ

lesioning should be considered the next step. Finally, we suggest the establishment of aninternational register to track the efficacy of patients receiving high-frequency SCS for brachial plexus avulsion. Again, SCS is less invasive than DREZ, allows for a trial period of temporary stimulation, and has significantly less permeant neurological side effects.

Limitation of the study

Our analysis carries inherent limitations. First, all studies reported here are retrospectiveand therefore suffer from inherent bias. This is essentially the result of the small numberof SCS studies that have been published. Second, the follow-up period of many of these studies is less than two years. This is important as many of the studies with longer-term follow-up have shown that the efficacy of SCS may wane dramatically over time. Thus, these results must be understood to reflect a cohort of patients with a follow-up period of less than two years.

Third, there is wide heterogeneity across time and methodology between these studies. There is a significant risk in comparing studies done decades apart that have used very different equipment and methodologies. (16, 20) This difference will become moreimportant as high-frequency SCS stimulation begins to enter clinical use. Fourth, the pain scale measurements vary considerably across studies and cannot be readily compared. Fifth, in contrast with the broader literature, very few studies in this review have used high-frequency SCS in brachial plexus injuries.

6. CONCLUSION

SCS has been reported to successfully and safely neuromodulate the chronic pain experienced by patients with traumatic brachial plexus injuries. The reported response is very good (86%) initially but the response decreases (69%) over the follow-up period. High-frequency SCS may hold significant potential in treating patients with traumatic brachial plexus injury. Because of a better safety profile, we suggest giving an early trial of SCS as a first-line neuro intervention in patients who have failed to respond to conservative management

- **Author's contribution**: The author was involved on all steps of preparation this article including final proofreading.
- **Conflicts of Interest**: The author have no conflicts of interest to disclose.
- **Financial support and sponsorship**: No specific funding was received in relation to this study.

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