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Case Report

Neuromodulation in the Management of Pain from Brachial Plexus Injury

Silviu Brill, MD, and Itay Goor Aryeh, MD

From: Pain Clinic, Department of Anesthesiology, Tel Hashomer, Israel

Drs. Brill and Aryeh are with the Pain Clinic, Sheba Medical Center, Tel-Hashomer, Israel.

Address correspondence:
Silviu Brill, MD
Pain Clinic, Sheba Medical Center, Tel-Hashomer, Israel
E-mail: s_bril@yahoo.com

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We are presenting a paper on the effectiveness of spinal cord stimulation (SCS) in 2 patients suffering pain from brachial plexus injury (BPI).

After a traumatic brachial plexus lesion about 80% of patients develop pain in the deafferentated arm. This pain is considered very resistant to many forms of therapy. In the early 1970s, SCS was introduced in the treatment of BPI pain with disappointing results. There are only about 20 published cases of BPI pain treated with SCS. Many injuries are due to motorcycle accidents, so that patients are often young and require long-term pain relief.

During the SCS trial the pain relief was more than 50% with an absolute improvement in the quality of life and significant drug reduction. The results of the SCS were excellent in these 2 patients, defined as more than 50% pain relief at 6 and 18 months.

Key words: Spinal cord stimulation, brachial plexus injury, neurophatic pain.

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About 70% of brachial plexus injuries are of traumatic origin, with motorcycle accidents as the main cause. After a traumatic brachial plexus lesion, about 80% of patients develop pain in the deafferentated arm, which decreases to 20% of the patients after 3 years (1). This pain is considered very resistant to many forms of therapy.

The pain due to brachial plexus injury is of a burning and lancinanting type.

In the early 1970s, spinal cord stimulation (SCS) was introduced in the treatment of brachial plexus injury (BPI) pain with disappointing results. In the literature there are only about 20 reports of BPI pain treated with SCS.

The symptoms of BPI involve muscle atrophy and vasomotor and tropic changes. The roots most involved are C4–T1. According to the Millesi Classification of Brachial Plexus, both patients had a type 2 injury (infraganglionic). Spinal cord stimulation has been used to relieve chronic pain for 40 years. SCS is thought to produce its analgesic effect through activation of large A-fibers resulting in segmental pain inhibition as described by the gate control theory of Melzack and Wall (2). Other evidence suggests that SCS causes the release of endorphins and influences autonomic function, possibly through releasing segmental spinal reflexes and inhibiting sympathetic fiber discharge (3).

Methods

This is a report of 2 male patients suffering from brachial plexus injury of traumatic origin (see Table 1). The diagnosis of brachial plexus injury was made by clinical evaluation, MRI, and electrophysiologic studies (SSEP, EMG).

The EMG of patient 1 (Fig 1) shows reduced sensory amplitudes in the left median and radial nerve suggesting infraganglionic dysfunction.

The EMG of patient 2 shows complete paralysis of the brachial plexus.

Ultrasound examination of the brachial plexus of patient 1 shows complete disruption of C5, C6, and C7 after the foramen.

These patients suffered from total palsy and underwent many other treatments before SCS. Their treatment included opioids (Oxycontine), antiepileptics (Carbamazepine, Gabapentine, Pregabaline), SSRI (Paroxetine), SNRI (Duloxetine), TAD (Amytriptiline), sympathetic blocks, cervical selective nerve root blocks, and physiotherapy.

During the trial, the patients underwent a percutaneous implant of a 4-contact electrode in the cervical region. Insertion of the epidural electrodes was carried out under fluoroscopic control with the patient awake. The electrodes were inserted percutaneously via the thoracic spine, usually in the T6 and 7 interspaces, and advanced in a cephalic direction in the posterior epidural space. The patient was stimulated using an external pulse generator, and the position of the electrodes was finely adjusted according to verbal feedback from the patient with regard to the area of stimulation. The stimulation sites were chosen to induce parasthesia in the painful area at the lowest amplitude. Both patients report tingling on the area of stimulation during the trial.

Testing lasted 15 – 20 days with the patients at home. The patients reported significant pain relief during the trial period. The pain intensity was assessed by the Visual Analog Scale (VAS) and Leeds Assessment of Neuropathic Symptoms and Signs Scale (LANSS).

After the trial period, the trial electrode was removed and after 2 weeks the system was permanently implanted (8-contact electrode) (Medtronic, Minneapolis, MN, USA, and Advanced Neuromodulation Systems, Inc., Plano, TX, USA).

Following the implantation the patients were able to wean off all oral analgesics, including opioids. Both patients required stimulation 24 hours a day. One of the patients underwent epidural electrode repositioning after 16 months due to caudal migration.

Details of initial stimulation: frequency 20 to 40 Hz, pulse width 221 microseconds, intensity 5 to 6.5 amper, polarity (+-+00000). The patients got instruction on using 4 to 5 programs with different parameters.

Table 1. *Patient's demographics.*

	Age	Sex	Nerve roots involved	Time since accident (years)
Patient 1	48 years	Male	C5-6-7	4
Patient 2	39 years	Male	C5-6	2

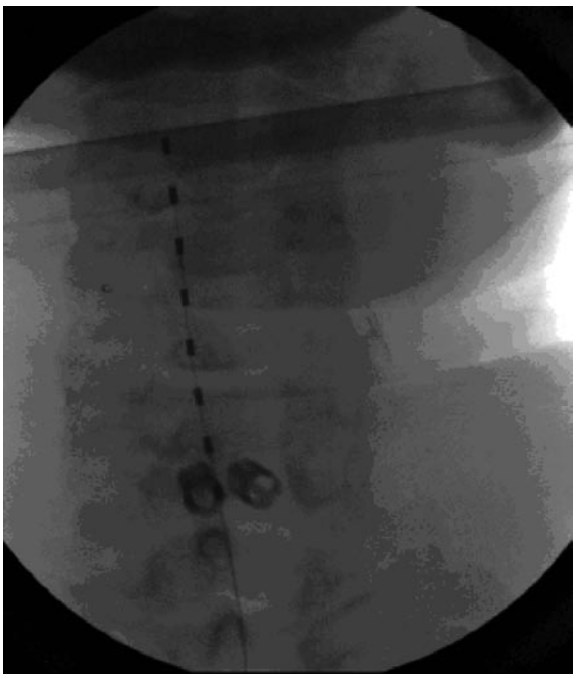

Fig. 1. *EMG of Patient 1.*

Results

The efficacy of the SCS was assessed by VAS and LANSS prior to implant and at 6 and 12 months (Table 2). There was a significant reduction in analgesic drug consumption (Table 3).

Table 2. *Efficacy of the SCS assessed by VAS and LANSS.*

	Patient 1	Patient 2
VAS before implant	10	9
LANSS before implant	20	19
VAS after 6 months	2	3
LANSS after 6 months	16	14
VAS after 12 months	2	3
LANSS after 12 months	13	10

Table 3. *Drug consumption.*

	Patient 1	Patient 2
Opioids (morphine equivalent) before SCS	100	60
Other drugs before SCS	Antiepileptics SSRI	Antiepileptics
Opioids (morphine equivalent) after 12 months	0	20
Other drugs after 12 months	0	NSAIDS

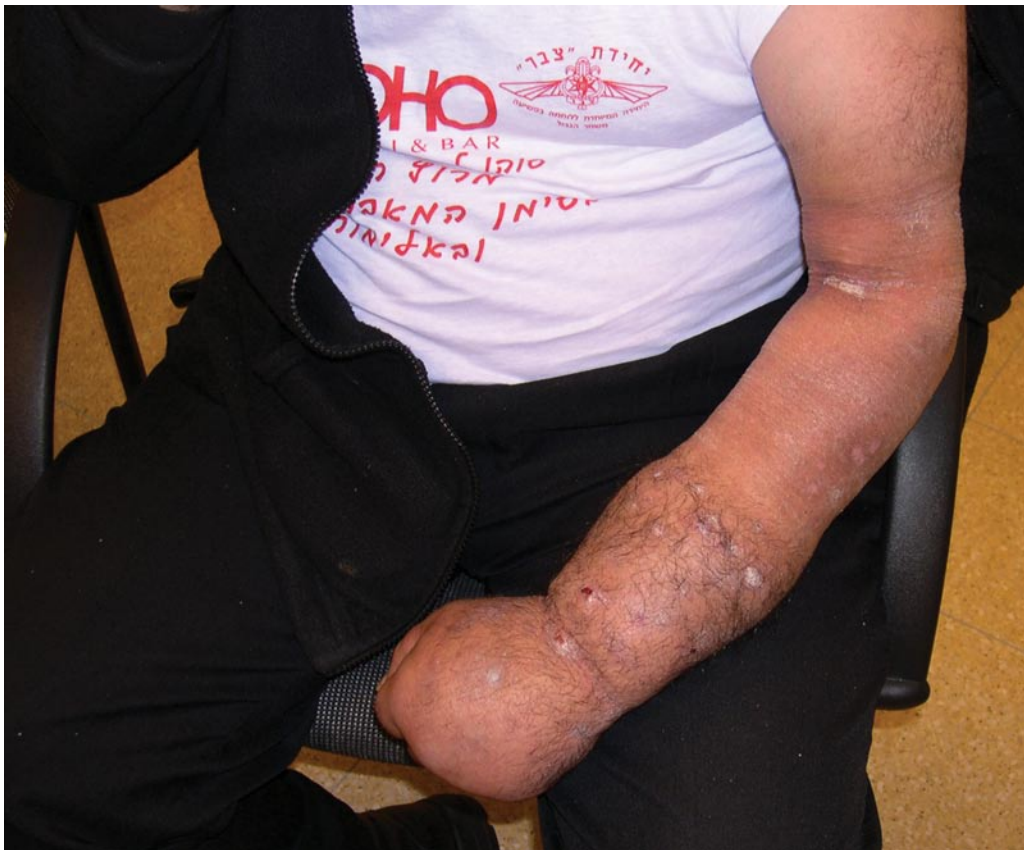

Fig. 2. *Photo of the patient's hand, wrist, and arm.*

Sensory Nerves	Lat (ms)	SD	Amp (uV)	SD	CV (m/s)	SD	Amp% (%)	SD
Right Median Wrist, Digit 2	2.8	5.4	28		59/5			
Left Median Wrist, Digit 2	2.6	4.3	18		61.9			
Right Ulnar Wrist, Digit 5	2.9	6.0	8.4		54.5			
Left Ulnar Wrist, Digit 5	2.4	3.1	6.0		57.1			
Right Radial Forearm, Digit 1	2.1		10		67.3			
Left Radial Forearm, Digit 1	1.83		4.8	-3.3	65.2	1.3		

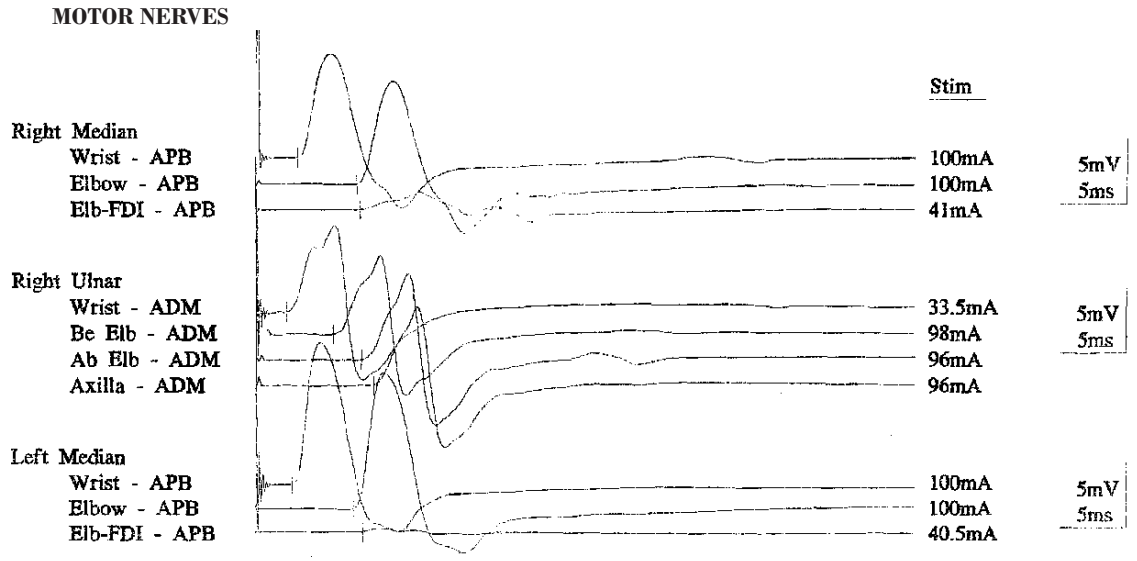

Fig. 3. *Parameters of stimulation.*

Discussion

The pathogenesis of pain after BPI is unclear but is thought to be central in origin, probably related to changes in the substantia gelatinosa secondary to traumatic afferent disconnection of the dorsal nerve roots. At the level of injury the action of large myelinated fibers is lost, leaving the substantia gelatinosa without inhibition, resulting in central pain. Chronic deafferentation of dorsal horn interneuronal pools leads to segmental hyperactivity in the dorsal horn cells

Neuromodulation has been used to treat pain for thousands of years (2) but has been improved technically only over the last 3 decades. This method was used in the early seventies for the treatment of BPI with controversial results. In the literature, there are only about 20 cases reported of BPI pain treated with SCS (1,3-5). Knowledge among physicians about this method of treatment is still limited.

Our patients obtained good or excellent relief from pain beginning with the time of the electrode implantation. This contrasts with the series published by Zorub et al and Garcia-March et al (1,3). The failure of SCS in these series was probably due to a loss of sensibility in the affected arm and, thus, no activation of intact myelinated fibers could occur to produce analgesia (6). Furthermore, the technical evolution of the stimulators and our knowledge of the patterns of

stimulation may play important roles. A complete lesion that affects the roots proximal to the ganglion will produce a degeneration of the corresponding dorsal column fibers that are thus no longer available for stimulation. Probably our patients had the roots partially injured, and the corresponding dermatome is covered by overlapping projective fields from adjacent roots (7).

Pain after traumatic BPI is very resistant to treatment. Many other procedures have shown limited efficacy in long-term follow-up.

In our experience, a trial of SCS should be considered earlier in the treatment of pain due to brachial plexus injury even in patients with severe sensory deficit. However because prior studies and case reports showed that the SCS does not work in BPI further prospective-large-sample-size studies needed to further prove the utility of SCS in this setting.

The trial procedure carries minimal risk for the patients, it is completely reversible, and the efficacy can be assessed before implant.

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