*Paraplegia* 28 (1990) 399–405

0031–1758/90/0028–0399 $10.00

Paraplegia

# Neural Stimulation for Spinal Spasticity

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## Summary

*The knowledge of neural plasticity is used to improve motor function of the spinal cord by chronic stimulation of the dorsal columns. We achieved this with an improvised technique. The electrodes are exteriorised and stimulated by using a Grass stimulator.*

*Five paraplegic patients with different aetiologies causing their paraplegia were treated by this method, and the details are enumerated. Four had good improvement and relaxation was maintained for varying periods after cessation of the stimulation. Mode of action is discussed.*

**Key words:** *Electrical neural stimulation; Neural plasticity; Spinal spasticity; Grass stimulator.*

Electrical stimulation of the brain (ESB) has been used for centuries. Delgado (1977), a pioneer in the modern method of electrical stimulation, states that Galvani (1791), Du Bois Reymond (1948) and Fritsch and Hitzig (1870) used electrical stimulation to modify local functions of specific neuronal structures.

Although electrical stimulation of the brain is comparatively widely used, spinal stimulation is rarely used. Utilising the 'Gate Control Theory' of pain postulated by Melzack and Wall (1965), Shealy *et al.* (1970) reported that chronic stimulation of the spinal cord relieves 'pain'. Cook and Weinstein (1973) observed that in one of their patients with multiple sclerosis (MS), dorsal column stimulation (DCS) relieved the pain and also improved voluntary motor control and sensory appreciation. After prolonged observation of this patient they stimulated the dorsal columns in 4 patients with MS whose complaint was deficient motor function and not pain. This experience led them to conclude and report in 1973 that DCS is an effective procedure to improve voluntary motor control and sensory appreciation in patients with MS. Further workers utilised this observation gainfully. Reports confirming Cook and Weinstein's observation have been reported by Illis *et al.*

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(1976), Abbate *et al.* (1977), Cook (1978), Cook *et al.* (1979), Richardson *et al.* (1979), Sherwood (1986, 1988), Dimitrijevic (1986), Dimitrijevic *et al.* (1986, 1987) and Campos *et al.* (1987). These reports encourage us to utilise this method for our patients with hypertonus of limbs from varying causes.

## Methods

Apart from routine investigations the patient's hypertonus was assessed by surface electromyography. The spasticity was assessed clinically and by surface electromyography. Clinically the hypertonus is graded as follows:

Grade 1: Dynamic hypertonus, i.e. hypertonus increasing with movements.
Grade 2: Hypertonus enabling the patient to walk without support.
Grade 3: Hypertonus enabling the patient to walk with support only and with limitation in the distance covered.
Grade 4: Patient confined to bed with ability to move the limbs to limited extent passively or actively with effort.
Grade 5: Severe hypertonus with inability to move the limbs passively or actively.

Surface electromyography is carried out by placing silver button electrodes on the agonists and antagonists. The recording is taken at rest, during voluntary movement and during stretch of the tendons. The recording is done on an EEG machine. Surface electromyography is a useful objective method to record improvement after treatment. Surface EMG helped the classification of hypertonus into rigidity, rigidospasticity, spasticity and spastorigidity. All were given intensive physiotherapy. No specific antispastic drugs were given because the patients could not afford financially long term treatment with expensive antispastic drugs.

The absolute indications for neurostimulation were: (1) Severe hypertonus which impedes physiotherapy; (2) Hypertonus not yielding to physiotherapy.

The aim of neurostimulation was to help the patient to obtain maximum benefit from physiotherapy.

The criteria for selection of patients for neurostimulation were:

1. Patients intelligent enough to co-operate for neurostimulation.
2. Patients who were feeble minded were avoided.
3. Patients with a cardiac pacemaker were avoided.
4. Patients should be fit for general anaesthesia.
5. Patients with severe hypertonus which was interfering with physiotherapy were chosen for this procedure.

### *Operative procedure*

As the conventional method of stimulation is too expensive for our patients, we used an improvised, less expensive method. With the patient under general anaesthesia, and placed in the prone position, through a vertical midline incision the spinous processes and laminae were exposed at three consecutive levels; cervical or thoracic as planned. A limited laminectomy was carried out. A pair of copper electrodes indigenously made were placed posteriorly extradurally on either side of midline and were fixed to the laminae. The other end of the electrodes were

exteriorised about 2 cm from the main wound. Four or 5 days later, stimulation was started using a Grass stimulator. Model SD 5 and 9 Grass stimulators (manufactured by Grass Instruments Company, Quincy, Mass., USA). Our present stimulator was donated by Lady Grass. The parameters set were 200 microseconds, 100 to 200 Hz with voltage varying from 2 to 10 volts. The duration of stimulation and the parameters of stimulation varied depending on the patients' response. The stimulation was given every day initially and later on alternate days. Stimulation was continued for 1 to 3 months.

*Patients*

Five patients were treated by DCS since 1981. They were admitted to the Institute of Neurology, Madras. All the patients were examined neurologicially and were treated initially with intensive physiotherapy, this was also continued after the surgery.

No antispastic drugs were given as they are expensive. Analgesics and diazepam were given before physiotherapy if the patients had severe spasms and pain interfered with physiotherapy.

### Case report 1

A 24-year-old male paraplegic suffering from cerebral palsy was admitted on 5 November 1981. Spastic paraparesis with grade 2 hypertonus and rigidospasticity. Investigations were not contributory. Electrodes were implanted at T4 and T5 levels and electrical stimulation was carried out.

### Case report 2

A 26-year-old man was admitted on 12 December 1981 with the history of a sudden onset of paraplegia following hyperpyrexia 6 months earlier. He had also had generalised seizures. The CT scan, EEG and lumbar myelogram were normal. CSF Protein was 50 mg. On 3 April 1981 electrodes were implanted extradurally at T3 and T4 levels. Stimulation was started 1 week later.

### Case report 3

A 35-year-old man was admitted on 24 March 1983 with a history of progressive spastic weakness of both lower limbs of 5 years duration. Laminectomy and decompression carried out 4 years earlier elsewhere did not improve his condition. Clinical examination indicated a motor and sensory level at T6 spinal segment. Lumbar CSF Protein was 85 mg% with positive Pandy. A lumbar myelogram revealed a total hold-up of the dye at T6 vertebral level. At surgery, extradural granulation tissue was seen and was excised. Extradural electrodes were placed at T1, T2, T3 levels. Electrical stimulation was started on the fourth post-operative day.

### Case report 4

A 25-year-old man was admitted on 18 September 1986 with spastic paraplegia following a fracture of D12 L1 sustained 1 year previously. On 6 October 1986, electrodes were implanted at C6, C7 and T1 levels. Electrical stimulation was started on the seventh post-operative day.

## Case report 5

A 36-year-old man was admitted on 28 January 1986. He had spastic paraplegia caused by a fracture of T11 sustained 2 years earlier. Electrodes were placed at T11, T12 and chronic stimulation was instituted. There was infection. He had no relief of hypertonus, and the electrodes were extruded within 15 days after surgery. On 25 November 1986 the electrodes were placed at C6, C7, T1 levels. Once again there was no improvement with stimulation, therefore a median myelotomy was done on 3 March 1987, and he was discharged on 10 April 1987.

## Results

Assessment of the results was done by a team consisting of a neurosurgeon, an orthopaedic surgeon, a physiotherapist and the patient. This is subjective assessment whereas objective assessment depended on surface electromyography and the functional improvement.

Case 1 was given the electrical stimulation for nearly a month and his gait was improved at the time of discharge. The improvement was maintained for 1 year.

Case 2 had stimulation for nearly 2 months, every day during the first month, and later on alternate days. During stimulation the limbs became hypotonic and hence the stimulation was given on alternate days. A 6-year follow-up report was that his limbs were relaxed enough for him to walk independently.

Case 3 volunteered relaxation of the limbs and subsidence of flexor spasms. On examination better volitional movement was seen in the right lower limb than in the left lower limb, and some return of sensory function was also noted. The improvement persisted for only 1 year.

Case 4 showed improvement in that he could stand and walk with support, and this improvement persisted when he was seen at a recent follow-up 2 years after surgery.

Case 5 did not improve with any of the procedures employed. We had to modify stimulation to produce optimal reduction of spasticity.

Stimulation was given for 4 to 8 hours continuously, daily altering the duration of stimulation and the interval between stimulation depending on the patient's response. Stimulation was discontinued when the last pair of electrodes extruded. This occurred in 2 to 3 months after implantation. Even after cessation of stimulation the functional improvement was maintained from 6 months to 1 year or more. Sherwood (1988) observed that the common features of SCS were:

1. Modification of motor control in patients with abnormal functions.
2. SCS did not influence normal activity.
3. Inappropriate stimulation may result in worsening of abnormal function.
4. Modification induced by SCS was confined to the period of stimulation.
5. Permanent changes do not result.

## Discussion

Previous experimental work predicted that patients with a chronic neurological deficit should respond to 're-education' of the intact central nervous system (Illis *et al.*, 1976).

Cook (1978) initially used subdural bipolar electrode placement through a

laminectomy. Two receivers and two transmitters were used for a short while but were given up in favour of extradural electrodes with a receiver and transmitter. As this method involved general anaesthesia and elaborate surgical procedure, he changed over to the placement of the electrode extradurally introducing it percutaneously using fluoroscopic control.

Broseta (1985) reported that he found no improvement in 10 patients (5 spastic, 3 dystonic, 2 torticollis) treated with high frequency cervical spinal cord stimulation, but in 30 patients with peripheral vascular disease with spinal cord stimulation, he observed improvement in blood flow and symptoms.

Sharkey (1985) placed epidural electrodes from C2 to T12 in 67 patients with hypertonia after spinal cord injury. He stimulated the cord at 3 to 5 MA amplitude, 0·2 MS duration at 30 Hz. Eighteen had a pronounced reduction of muscle hypertonia, 24 had moderate effect and 25 had no effect.

Sogabae (1985) reporting on experience with 124 spinal implants stated that displacement of the electrode was the commonest complication, and this could be avoided by using bipolar electrodes and by restricting movement in the first month after surgery.

Extensive use of epidural spinal cord stimulation in spinal injury has been reported by Sherwood (1986, 1988), Dimitrijevic *et al.* (1986), Dimitrijevic *et al.* (1986, 1987) and Campos *et al.* (1987) of the Division of Restorative Neurology and Human Neurobiology, Baylor College of Medicine, Houston. All the reports stress the necessity for the proper selections of patients.

Neurostimulation is probably the first practical application of the concept of plasticity in the central nervous system. The concept of neural plasticity introduced for the treatment of spinal cord injuries in about 1960 by Liu and Chambers and McCouch *et al.* (Tsubokawa, 1985) raised expectations amongst neurosurgeons.

The term 'synaptic plasticity' was formally introduced by Konorski (1948) to describe the hypothesis that long term changes in the efficacy of synaptic transmission can be induced by short periods of neuronal activity. In recent years the concept of plasticity has been broadened to include (1) regenerative and collateral sprouting of axons, (2) the formation of new synapses, (3) synaptic unmasking and (4) cellular dedifferentiation and change in transmitter phenotype (Sutin, 1985).

Spinal cord stimulation may act on the three different aspects of CNS function (Illis *et al.*, 1976) (1) An effect on the lesion itself; environmental changes can alter conduction in damaged fibres but our knowledge at present does not suggest that spinal cord stimulation can significantly affect this aspect of function. However, repetitive stimulation may modify the molecular enironment sufficiently to alter conduction through the lesion or part of the lesion. (2) Stimulation may modify the functional and anatomical reorganisation. (3) Stimulation can be seen to increase the central excitatory state which, by producing movement, further increases afferent inflow. Neurons are then more likely to reach their firing thresholds and inhibitory mechanisms have some activity upon which to operate.

These factors are acting on an altered CNS, i.e. a nervous system which has already reacted to a partial lesion as described above. Without sensory feedback (with a decreased central excitatory state) no further improvement in function can occur and the clinical picture is static. Spinal cord stimulation provided the increase in feedback and produces a new clinical picture.

Dimitrijevic *et al.* (1986) hypothesise that spinal cord stimulation (SCS) controls spasticity by modification of the activity of spinal-brainstem-spinal loops and by suppression of segmental excitation through antidromic activation of propriospinal pathways.

Their criteria of selection of cases for SCS were spasticity, clinical evidence of sensation below the lesion and clinical neurophysiological evidence of residual descending pathways.

Better results were obtained by stimulation below the level of the lesion in the spinal cord and in patients with incomplete lesions.

Using SCS at 30 Hz with a pulse width of 200 MS and an amplitude ranging from 2 to 8 MA continuously, Dimitrijevic *et al.* (1987) found that even after cessation of stimulation the beneficial effects were carried on for zero to several hours.

The severity of the hypertonus did not influence the result. Sherwood (1986) stressed that quantification of the effectiveness of SCS should take into consideration the nature of the patient's neural control as the measurement of hypertonia often fails to correlate clinical improvement. Dimitrijevic *et al.* (1986) reported that 63% of their 59 patients on SCS improved markedly or moderately. They found that SCS was effective if the electrodes are properly positioned below the lesion over the dorsal aspect of the spinal cord.

Parameters of stimulation have an important role to play in the functional improvement. The use of low frequency, low voltage square wave output not only relieves the hypertonus but also prevents episodes of autonomic hyperreflexia (Richardson *et al.*, 1979).

Whatever the mechanism of the stimulation, our experience so far is that it is beneficial and confirms the work of Cook. The changes seen in these patients have been dramatic, and clearly it is important to discover the fundamental mechanisms involved in rehabilitation with spinal cord stimulation.

We conclude that sophisticated equipment, and a fully implanted system can certainly offer permanent relief in these patients.

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