# CLINICAL RESULTS OF SPINAL CORD STIMULATION

By

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The first demonstration of improvement in neurological deficit as a result of dorsal column stimulation was by Cook and Weinstein (1) who reported improvement in five patients with multiple sclerosis following stimulation. Since that time there have been many reports of improvement in neurological deficit with this technique and although there has been a considerable amount of scepticism there seems to be no doubt that Cook and Weinstein's original observations were of a real phenomenon and one which is likely to alter views concerning rehabilitation of patients with chronic neurological deficit as well as ideas about the way in which the central nervous system functions. Although the electrodes in this technique are sited over the dorsal columns, the exact pathways which are stimulated by these electrodes and the shape and distribution of the electric field is unknown and we prefer, therefore, to term this procedure spinal cord stimulation rather than dorsal column stimulation. We reported our first cases of spinal cord stimulation in multiple sclerosis two years ago (2), confirming Cook and Weinstein's clinical results and demonstrating objective neurophysiological changes related to stimulation. We have carried out 26 periods of spinal cord stimulation in 14 patients. Five of these patients have had permanent implants and the longest follow-up in the permanent cases is 19 months. In this paper we present the clinical results of these patients.

The clinical data on the patients is summarised in Table 1. All the patients with multiple sclerosis fulfilled Schumacher's criteria. The mean age was about 44 years and the mean duration of illness was about 10 years. All but three of the patients had one or more previous courses of ACTH treatment with no improvement. The clinical manifestations, at the time of stimulation, are summarised using Kurtzke's scale. The motor deficit ranged from normal (0) to no useful function of the legs (5). Cerebellar disturbance ranged from mild (2) to severe ataxia (4); sensory function from mild disturbance (1-2) to total loss of sensation to the trunk (4+). Sphincter function ranged from normal (0) to no bladder sensation or control (5). All the multiple sclerosis patients had abnormal visual or brain stem evoked potentials.

Patients were evaluated as follows: All patients had an initial Out-Patient assessment. Some patients were excluded from the trial on the basis of fluctuating symptoms, intercurrent infection, or where the diagnosis was in doubt. Following Out-Patient assessment, patients were admitted to the Wessex Neurological Centre for repeated clinical and physiological examinations. Three patients had surface stimulation for three days, with cutaneous electrodes in the midline dorsal areas; two patients had electrodes inserted and connected but with no batteries in the apparatus for two days. There was no change in these patients. Electrodes were inserted percutaneously into the epidural space in the midline mid-thoracic area as described by Cook (3) and stimulation was carried out at 33 Hz with 200 microsec square wave pulses continuously for ten days at a voltage sufficient to produce a symmetrical tingling sensation below the level of stimulation. The electrodes were withdrawn after ten days. Details of the physiological tests and urodynamic studies will be given elsewhere. Patients were then discharged from hospital and followed-up at monthly intervals. Those patients with evidence of clinical and physiological improvement had a further period of spinal cord stimulation after three to six months. If a consistent result was obtained they were offered a permanent implant.

Figure 1 indicates the clinical response to initial stimulation. The vertical axis shows the degree of disability using the Kurtzke's scale. In each column the clear area is disability before stimulation and the shaded

after stimulation. The patients who improved are shown in red. The least responsive disability is cerebellar and the major response is in sphincter function:

> e.g. Patient S.E. could walk with great difficulty for a few yards using a Zimmer walking aid. She was unable to walk on her own and she was able to sit up in bed only with assistance and could remain sitting up unsupported for a maximum of 10-15 seconds. There was weakness of hand muscles. She was unable to take a bath on her own.
>
> 24 hours after stimulation she could sit up from the supine position with her arms folded across her chest. She could walk unaided. She was able to take a bath on her own. Hand movements were normal.
>
> Before stimulation she had no bladder sensation and had been permanently catheterised for one year. 72 hours after stimulation she had normal bladder sensation and control, and the catheter was removed.

All patients, after their initial period of ten days repetitive continuous stimulation, had the electrodes withdrawn. Those patients who showed improvement would usually maintain their improvement for up to four to six weeks and then reverted to their pre-stimulation state.

After a period of three to six months the patients who had shown improvement had a repeated episode of stimulation and if a consistent response was obtained then they went on to permanent implant and these are summarised in Figure 2. These figures show the improvement with initial stimulation, the decline to pre-stimulation state on stopping stimulation, and the consistent response with permanent stimulation.

As can be seen, patients vary as to their long term response but short term effects are a good guide to further response. Of our five patients with permanent implants, improvement in bladder function has been maintained, motor deficit has either been maintained or has decreased to about 50 per cent of the improvement produced by spinal cord stimulation and mobility has been improved. There is one exception and that is a patient who had a dramatic improvement with spinal cord stimulation but after several months he deteriorated to the pre-stimulation state clinically and physiologically at the same time as the stimulator sensation changed. X-ray showed that one electrode had moved 5 mms from the midline. When this was replaced, stimulator sensation and clinical and physiological changes reverted to the improved state and this was maintained for a further two months. He subsequently deteriorated progressively and is now more or less in the pre-stimulation state except that mobility is worse and sphincter function is slightly better. We think that the present deterioration is probably due to an exacerbation of multiple sclerosis.

Originally our patients who went on to permanent implant had electrodes sutured to the dura via laminectomy but in more recent patients we have left the electrodes free in the epidural space.

The follow-up time in our patients is too short and the number of patients is too few to make any precise claims. The reason for the small numbers in our series is partly because of the protocol which we have worked to which

requires patients who improve to have two episodes of consistent change before we offer permanent implant, and for these two episodes to be separated by three to six months, partly because of the considerable time spent on physiological testing, and mostly because of the shortage of beds which we have available for this procedure. We now have enough clinical and physiological data to make it clear that we are dealing with a real phenomenon. We intend to continue with this procedure and help to extend it into conditions other than multiple sclerosis.

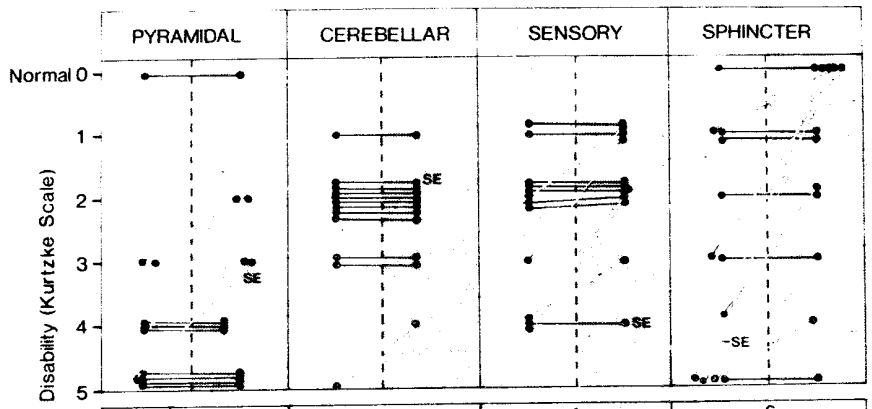
## REFERENCES


1. Cook, A.W. and Weinstein, S.P. Chronic Dorsal Column Stimulation in Multiple Sclerosis. New York State Journal of Medicine. 2868-2872 1973.

2. Illis, L.S., Sedgwick, E.M., Oygar, A.F. and Sabbahi Awadalla, M.A. Dorsal Column Stimulation in the Rehabilitation of Patients with Multiple Sclerosis. Lancet. 1383-1386. June 1976.

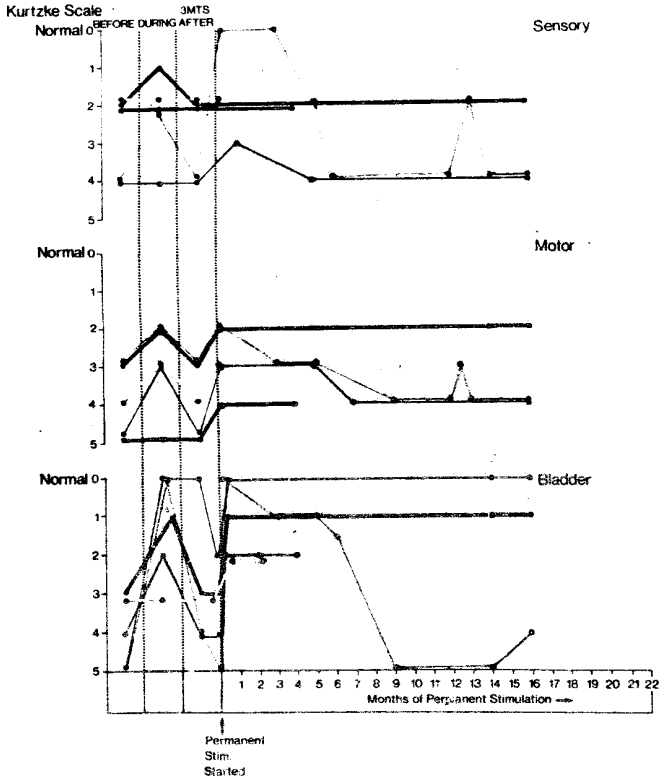
3. Cook, A.W. Electrical Stimulation in Multiple Sclerosis. Hospital Practice. 2, No. 4. 51-58. April 1976.

RESPONSE OF MS PATIENTS TO INITIAL STIMULATION




| | PYRAMIDAL | CEREBELLAR | SENSORY | SPHINCTER |
|---|---|---|---|---|
| Improved | 4 | 1 | 4 | 6 |
| No Change | 8 | 11 | 8 | 6 |
| Worse | 0 | 0 | 0 | 0 |
| Total | 12 | 12 | 12 | 12 |

Effect of Spinal Cord Stimulation on Function in MS Patients Receiving Permanent Stimulation



PATIENTS RECEIVING STIMULATION

| Patient | Age | Sex | Diagnosis | Type of MS | Duration of Illness (years) | Severity of manifestations at time of first stimulation: Motor | Cerebellar | Sensory | Sphincter | ACTH Treatment | V.E.P. X=Abnormal | BSEP X=Abnormal |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. C.P. | 36 | M | MS | Relapsing | 5 | 3 | 2 | 4+ | 5 | No change | X | X |
| 2. D.S. | 41 | M | MS | Progressive | 2 | 3 | 2 | 2 | 3 | No change | X | X |
| 3. S.E. | 44 | F | MS | Relapsing | 2 | 4+ | 2x | 4 | 5 | No change | X | X |
| 4. E.M. | 22 | F | MS | Progressive | 5 | 0 | 4+ | 2 | 0 | —— | X | X |
| 5. J.M. | 50 | F | MS | Relapsing | 8 | 5 | 2x | 2 | 1 | Worse | X | Normal |
| 6. O.B. | 40 | F | MS | Relapsing | 20 | 5 | 2x | 1 | 2 | —— | X | Normal |
| 7. S.B. | 46 | F | MS | Relapsing | 9 | 5 | 3x | 2 | 4 | No change | X | X |
| 8. C.F. | 57 | F | MS | Progressive | 20 | 4 | 2x | 2 | 1 | No change | X | X |
| 9. M.R. | 35 | F | MS | Relapsing | 12 | 5 | 2x | 3 | 5 | No change | X | X |
| 10. A.M. | 59 | M | MS | Progressive | 16 | 5 | 1x | 4 | 3 | —— | X | X |
| 11. N.P. | 53 | F | MS | Progressive | 16 | 5 | 3x | 2 | 5 | No change | not done | X |
| 12. M.W. | 40 | F | MS | Relapsing | 6 | 4 | 2 | 1 | 1 | Shortened Acute relapse | X | X |
| Mean | 43.6 ± 10.3 | | | | 10.1 ± 6.6 | | | | | | | |
| 13. R.W. | 39 | M | M.N.D. | | 3 | 4 | 0 | 0 | 0 | | | |
| 14. D.E. | 50 | F | Spinal arachadiotis | | 20 | 4 | 0 | 3 | 3 | | | |