

PARKINSON DISEASE

Spinal cord stimulation gets movement going in Parkinson disease

Spinal cord stimulation might be a valuable alternative to electrical deep-brain stimulation (DBS) in patients with Parkinson disease (PD), suggests a study by researchers at Duke University Medical Center (Durham, NC, USA). Romulo Fuentes and colleagues used two well-established animal models of PD to show that electrical stimulation of nerve pathways in the spine can restore movement. “It is desirable to identify a less invasive method to electrically stimulate neuronal circuits to obtain beneficial effects similar to those of DBS,” comments Fuentes.

Chronic and progressive movement problems in PD are currently treated with levodopa, which is associated with many adverse effects. Over time, higher doses are required to maintain movement, and this can lead to tremor and dyskinesia. Motor deficiencies in patients with PD can be improved by electrical stimulation of the basal ganglia. However, implantation of the electrodes deep within the skull is a highly invasive surgical procedure that carries considerable risks.

To test whether stimulation of afferent somatic pathways could have similar effects to DBS, Fuentes *et al.* stimulated epidural electrodes in the dorsal column of the spinal cord in

both acute pharmacologically induced dopamine-depleted mice and chronic 6-hydroxydopamine-lesioned rats. Movement was restored in both models. Electrical stimulation produced activity patterns in the dorsolateral striatum and in the primary motor cortex that were similar to those that occur just before spontaneous initiation of movement in normal animals. “This suggests that spinal cord stimulation helps the motor areas of the brain shift into a state that allows initiation of movements,” notes Fuentes.

Further experiments revealed that spinal cord stimulation abolished the aberrant, synchronous, low-frequency oscillations that are thought to block the patterns of neuronal activity needed for locomotion. “Suppressing these low-frequency oscillations is particularly important for amelioration of motor symptoms in human patients with PD,” comments senior author Miguel Nicolelis.

When used in combination with levodopa in severely dopamine-depleted animals, spinal cord stimulation facilitated the recovery of motor function at much lower doses of levodopa than are usually required. “Reducing the long-term dosage of levodopa by using spinal cord stimulation in addition to drug therapy in the early stages of PD



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could have several advantages,” concludes Fuentes. The authors now recommend that the technique is explored in primate models of PD to evaluate its long-term potential viability.

Kathryn Senior

Original article Fuentes, R. *et al.* Spinal cord stimulation restores locomotion in animal models of Parkinson's disease. *Science* 323, 1578–1582 (2009).