

Perceiving Parkinson's

Deep Brain Stimulation (Day 80)

Surgical methods have been used to treat Parkinson's since the 1920s. Earlier surgical treatments for Parkinson's focused on **stereotactic ablation**, in which a small target in the brain was located and removed. These procedures were often partly or wholly successful, but a major downside was that in removing part of the brain, the effects were permanent.

The era of ablative surgery ended when **levodopa** appeared in the 1960s. The honeymoon period of levodopa seemed like magic to many people with Parkinson's. However, the eventual appearance of motor fluctuations and dyskinesias dampened some of the initial enthusiasm for levodopa.

Surgical therapies for Parkinson's made a comeback when the French neurosurgeon Alim Benabid introduced a new surgical therapy called **deep brain stimulation** in 1987. In the right people, Benabid's new procedure was a godsend.



Alim Benabid introduced deep brain stimulation in 1987.

Deep brain stimulators consist of **three components** - electrode, extension wire, and pacemaker. The **electrode** is a device implanted deep into the brain; it sends out electrical signals that stimulate surrounding brain tissue. The **extension wire** is an insulated cable; it connects the electrode to the pacemaker. The **pacemaker** is a box-shaped device implanted under the skin near the person's chest; it generates the electrical signals that are sent to the electrode. The patient uses a handheld device to communicate with their pacemaker, allowing them to control the "dosage" of deep brain stimulation.

Even after 30 years, nobody knows how deep brain stimulation works. Yet somehow it profoundly influences the **abnormal basal nuclei** of a person with Parkinson's. One possibility is that deep brain stimulation **overrides** the abnormal basal nuclei in Parkinson's, "drowning out" irregular patterns of brain activity. Another possibility is that deep brain stimulation **inhibits** the abnormal basal nuclei in Parkinson's, "blocking" irregular patterns of brain activity. There are other theories.



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Several randomized controlled trials have attempted to identify which basal nuclei region is the best one to stimulate. Stimulating either the **subthalamic nucleus** or the **globus pallidus** produces an equal improvement in motor symptoms. However, while stimulating the subthalamic nucleus allows people to reduce their oral dopaminergic medications more, stimulating the globus pallidus interna produces fewer mood disorders and cognitive difficulties. There is a third target called the **thalamus**, which is a good region to target for people who have tremor as the main disabling feature.

Whichever brain region is chosen, randomized controlled studies have demonstrated that in carefully selected people with Parkinson's, deep brain stimulation is **better than the best medical therapy to date** for improving motor symptoms, motor complications, and quality of life. These benefits have been shown to last for ten years or more, although the Parkinson's still progresses.

By far, the most important factor for a successful outcome in deep brain stimulation is **careful patient selection**. To be considered, prospective candidates must be evaluated by a movement disorders neurologist, a neurosurgeon, a neuropsychologist, and a psychiatrist. The ideal Parkinson's candidate for deep brain stimulation can be described as follows:

- (1) Age no more than 70 years at the time of surgery.
- (2) Suffers from medically refractory motor fluctuations, dyskinesias, or tremor.
- (3) Still has an excellent "on" response to levodopa.
- (4) Does not have a significant mood disorder or cognitive difficulty.
- (5) Does not have another serious medical condition.

The potential complications of deep brain stimulation can largely be classified as surgery-related, hardware-related, or neuropsychiatric. **Surgery-related** complications appear during or shortly after the operation and include stroke, clots in the lung, and pneumonia. **Hardware-related** complications

include usually appear within three months of the operation and include electrode breakage, extension wire failure, and pacemaker malfunction. **Neuropsychiatric** complications often appear within the first year after the operation and include post-surgery depression and an increased risk of suicide.

It is important to realize that deep brain stimulation is **not a cure** for Parkinson's - like all the medical therapies discussed so far, it only masks the symptoms of the condition. However, in the right person, it can be a godsend. If you would like more information about deep brain stimulation in New Zealand, see the link below.

<http://www.parkinsons.org.nz/sites/default/files/page/Publications/DBS.pdf>

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References

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- (3) Pollak. 2013. Deep brain stimulation for Parkinson's disease - patient selection. *Handbook of Clinical Neurology* 116, 97-105.