Clinical Policy Title: Deep brain stimulation

Clinical Policy Number: 09.03.02

Effective Date: July 1, 2015
Initial Review Date: February 18, 2015
Most Recent Review Date: February 6, 2018
Next Review Date: February 2019

Policy contains:
- Deep brain stimulation.
- Dystonia.
- Essential tremor.
- Parkinson’s disease.

Related policies:
None.

ABOUT THIS POLICY: Select Health of South Carolina has developed clinical policies to assist with making coverage determinations. Select Health of South Carolina’s clinical policies are based on guidelines from established industry sources, such as the Centers for Medicare & Medicaid Services (CMS), state regulatory agencies, the American Medical Association (AMA), medical specialty professional societies, and peer-reviewed professional literature. These clinical policies along with other sources, such as plan benefits and state and federal laws and regulatory requirements, including any state- or plan-specific definition of “medically necessary,” and the specific facts of the particular situation are considered by Select Health of South Carolina when making coverage determinations. In the event of conflict between this clinical policy and plan benefits and/or state or federal laws and/or regulatory requirements, the plan benefits and/or state and federal laws and/or regulatory requirements shall control. Select Health of South Carolina’s clinical policies are for informational purposes only and not intended as medical advice or to direct treatment. Physicians and other health care providers are solely responsible for the treatment decisions for their patients. Select Health of South Carolina’s clinical policies are reflective of evidence-based medicine at the time of review. As medical science evolves, Select Health of South Carolina will update its clinical policies as necessary. Select Health of South Carolina’s clinical policies are not guarantees of payment.

Coverage policy

Select Health of South Carolina considers the use of deep brain stimulation to be clinically proven and, therefore, medically necessary, for Parkinson’s disease, essential tremor, and primary dystonia (Pahwa, 2006; NICE, 2006; Bronstein, 2011).

Limitations:

Evidence is insufficient, and thus deep brain stimulation is considered investigational or experimental, for all other conditions, including, but not limited to, secondary Parkinson’s disease, secondary dystonia, depression, obsessive-compulsive disorder, epilepsy, and tardive syndromes.

Alternate covered services:

Medical management as discussed by diagnosis below.

Background
Deep brain stimulation is a surgically implanted device applying high-frequency stimulation to a targeted area. The implanted electrode is connected to a computerized pulse generator under the skin, analogous to the arrangement with a cardiac pacemaker, and produces electrical impulses that regulate abnormal impulses or affect certain cells and chemicals within the brain. The U.S. Food and Drug Administration (FDA) approved the Activa® Tremor Control System (Medtronic, Minneapolis) in 2002, contingent on post-approval studies to assess long-term results.

During deep brain stimulation, small holes are created in the skull to implant electrodes, along with an incision in the chest to implant batteries. Several weeks post-operatively, the device is activated, and caregivers attempt to identify the most appropriate settings, to avoid side effects (Mayo Clinic, 2017).

Essential tremor (also known as benign tremor, familial tremor, or shaky hand syndrome) is a common movement disorder of uncertain cause, typically involving hands, arms or fingers. An estimated seven million Americans, or about 2.2 percent of the population, have the disorder (Louis, 2014). It is distinct from Parkinson’s disease, although it is sometimes misdiagnosed as such, and a single individual may (rarely) experience both. In contrast to Parkinson’s disease’s resting tremor, essential tremor’s resting tremor occurs with sustained muscle activity. When symptoms are sufficiently troublesome to warrant treatment, beta-blockers and anti-epileptics may be used.

Parkinson’s disease is a chronic, progressive, neurodegenerative disorder with an estimated prevalence of one million people in the United States, most of whom are elderly (PDF, 2017). Parkinson’s disease is characterized by resting tremor, rigidity, bradykinesia, and postural instability. While treatment with levodopa can usually restore smooth motor function up to five to 10 years after onset, effectiveness gradually diminishes over time. No known treatment halts progression. There is no cure, and treatment-resistant symptoms (dementia; dystonia; dysautonomia; and motor symptoms affecting speech, swallowing, and gait) almost invariably occur.

Dystonia is the simultaneous uncoordinated contraction of opposing antagonistic muscles affecting about 200,000 persons in the United States. These movements are painful and can lead to abnormal positions and disability. There is no cure for the disease, so any potential treatment can only contain it. Dystonia, which can manifest at all ages, can be primary (develops on its own) or secondary (develops from an illness, medication, or toxin).

**Searches**

Select Health of South Carolina searched PubMed and the databases of:

- UK National Health Services Centre for Reviews and Dissemination.
- Agency for Healthcare Research and Quality’s National Guideline Clearinghouse and other evidence-based practice centers.
- The Centers for Medicare & Medicaid Services (CMS).
We conducted searches on December 14, 2017. Search terms were: “deep brain stimulation.”

We included:

- **Systematic reviews**, which pool results from multiple studies to achieve larger sample sizes and greater precision of effect estimation than in smaller primary studies. Systematic reviews use predetermined transparent methods to minimize bias, effectively treating the review as a scientific endeavor, and are thus rated highest in evidence-grading hierarchies.

- **Guidelines based on systematic reviews.**

- **Economic analyses**, such as cost-effectiveness, and benefit or utility studies (but not simple cost studies), reporting both costs and outcomes — sometimes referred to as efficiency studies — which also rank near the top of evidence hierarchies.

**Findings**

A 2006 practice parameter from the American Academy of Neurology (AAN) addressed treating Parkinson’s disease with deep brain stimulation, to improve motor function and reduce off time, dyskinesia, and medication usage (Pahwa, 2006). That same year, the National Institute for Health and Care Excellence (NICE) recommended that deep brain stimulation be used for essential tremor and dystonia (NICE, 2006). An AAN guideline recommends deep brain stimulation as a highly efficacious treatment for essential tremor (Zesiewicz, 2011).

A panel of more than 50 experts published an expert consensus on use of deep brain stimulation in 2011. The consensus identified symptoms and characteristics that make patients good candidates for the treatment, declared that the procedure is best performed by an experienced neurosurgeon working with an expert team, that optimal results can take three to six months, and that benefits appear to be long-lasting in many motor domains (Bronstein, 2011).

The Health Information and Quality Authority in Ireland produced a health technology assessment in 2012 on deep brain stimulation. The assessment found deep brain stimulation more effective than the best medicinal therapy for advanced dopamine-responsive Parkinson’s disease not controlled by medication, but found limited evidence for effectively treating dystonia and essential tremor (HIQA, 2012).

Parkinson’s disease has been the subject of a number of meta-analyses and systematic reviews of deep brain stimulation treatment:

- One meta-analysis of seven studies (n = 45) found deep brain stimulation significantly improved postural instability and gait disturbance, but not motor function, in Parkinson’s disease patients (Wang, 2016).

- A systematic review of 38 studies (n = 1,443) of Parkinson’s disease found sub-thalamic deep brain stimulation had a positive effect on sleep quality, even though measures of sleep/wake quality are often heterogeneous (Eugster, 2016).
A meta-analysis of 27 studies of Parkinson’s disease patients found unilateral and bilateral deep brain stimulation linked with a significant improvement in gait speed (Roper, 2016).

A meta-analysis of 10 trials found sub-thalamic nucleus deep brain stimulation decreased global cognition, memory, verbal fluency, and executive function in Parkinson’s disease patients compared with controls (Xie, 2016).

Deep brain stimulation was linked with improved depression and anxiety in Parkinson’s disease patients in the short term, an improvement that appeared to wane over time, in a systematic review and meta-analysis of 63 studies (Couto, 2014).

A trial of 366 Parkinson’s disease patients compared efficacy of those on medical therapy alone with those on medical therapy and surgery (lesioning or deep brain stimulation). After one year, the mean improvement in the PD questionnaire was 5.0 points in the surgery group versus 0.3 in the non-surgery group (+4.7). The surgical groups had greater difference in other measures of functioning: mobility domain score (+8.9), activities of daily living domain score (+12.4), and bodily comfort domain score (+7.5). Other differences were not significant (Williams, 2010).

While the literature concludes that deep brain stimulation improves motor symptoms of Parkinson’s disease, little is known to date about non-motor symptoms (Kurtis, 2017).

Dystonia has also been the subject of literature reviews addressing deep brain stimulation efficacy. A systematic review of 43 papers (n = 487) of persons with dystonic tremor found deep brain stimulation of the globus pallidus internus, thalamus, or sub-thalamic areas led to marked improvement of dystonic axial or appendicular tremors in most cases. Botulinum injections were similarly effective, but medicinal treatment was generally ineffective (Fasano, 2014).

A meta-regression of 157 papers documented that in patients with primary generalized dystonia, significantly shorter duration of symptoms, lower baseline severity score, and torsin-1A, or dystonia 1 protein (DYT1) were associated with a higher improvement from surgery. Other classifications of dystonia failed to achieve these consistent results, making prediction of patient response difficult (Andrews, 2010). A systematic review of 17 studies (n = 50) found that in patients with deep brain stimulation, mean improvement of tardive dyskinesia and dystonia was 77.5 percent, three to 76 months after treatment, with just two exacerbations of depression or psychosis (Mentzel, 2012). A trial of 40 patients with severe dystonia followed subjects for five years after deep brain stimulation, and found significant improvements compared with baseline plus relative safety (Volkmann, 2013).

Pallidal deep brain stimulation for primary cranial, cervical, writing, and complex cervical dystonia has been found effective in relieving symptoms after conservative approaches (medication or BoNT (botulinum neurotoxin therapy and dystonia)) have proven ineffective (Albanese, 2011). Deep brain stimulation can cause side effects in segmental dystonia, including a Parkinsonian gait or bradykinesia (Cappelle, 2009).
The effects of DBS on essential tremors have also been covered in literature reviews. One review concluded that DBS is an established, safe, and efficacious treatment for essential tremors, particularly in the ventral intermediate nucleus and posterior sub-thalamic area. Adverse effects were found to be typically mild (Chopra, 2013). This review cited an earlier study that found DBS to be more effective than nucleus ventralis intermedius stimulation (Hamel, 2007).

Reviews of deep brain stimulation efficacy in disorders other than Parkinson’s disease, essential tremors, and dysphoria have found limited or mixed evidence. The 2007 guideline by the American Psychiatric Association mentions several studies that found deep brain stimulation reduced symptoms in patients with obsessive-compulsive disorder (APA, 2007). A Hayes review of five studies that used deep brain stimulation to treat treatment-resistant depression found response rates varying from 29 to 55 percent, and remission rates from 19 to 36 percent after 12 months; the studies suffered from small samples, lack of control groups, and potentially conflicting financial incentives from manufacturers among authors (Hayes, 2012). Results were generally similar in an evidence review by Health Quality Ontario the following year (Health Quality Ontario, 2013). A guideline on tardive syndrome treatment found insufficient evidence, mostly case reports, on the efficacy of deep brain stimulation to recommend treatment (Bhidayasiri, 2013).

A recent meta-analysis of 14 studies on deep brain stimulation’s effects on treatment-resistant depression found significant reductions/improvements in the depression scale compared to baseline for subcallosal cingulate gyrus (P<.00001), medial forebrain bundle (P<.0001), ventral capsule/ventral striatum (P<.005), and nucleus accumbens (pP.003). Treatment had high response rates and was effective for all 12 months of the study (Zhou, 2017).

Policy updates:

A total of four new practice guidelines/other and 16 new peer-reviewed references have been added to the 2017 version of the policy.

A total of two new practice guidelines/other and three new peer-reviewed references have been added to, and one practice guideline/other and two peer-reviewed references removed from, this version of the policy.

Summary of clinical evidence:

<table>
<thead>
<tr>
<th>Citation</th>
<th>Content, Methods, Recommendations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roper (2016)</td>
<td>Key points:</td>
</tr>
</tbody>
</table>
| DBS effect on gait velocity in persons with PD | ● Meta-analysis of 27 studies on PD patients undergoing DBS, measuring gait speed before and after treatment.  
          ● DBS significantly improved gait speed.  
          ● Speed improved regardless of whether patients were on or off medication.  
          ● Both bilateral and unilateral DBS associated with gait speed improvement. |
Citation | Content, Methods, Recommendations
--- | ---
Xie (2016) | **Key points:**
Cognitive changes following bilateral DBS in PD patients | • Meta-analysis of cognitive changes from sub-thalamic nucleus DBS for PD patients, vs. controls.
• 10 studies included (three randomized controlled trials, seven non-randomized).
• DBS results in decreased global cognition, verbal fluency, memory, and executive function compared to controls — all other categories are similar.

Fasano (2014) | **Key points:**
Comparison of various interventions on dystonic tremors | • Systematic review of 43 papers (n = 487), effects on tremor severity of various therapies.
• Drug efficacy disappointing for tremors other than dystonia.
• Botulinum toxin injections provided a marked improvement, especially for axial tremors.
• DBS of globus pallidus internus, thalamus, or sub-thalamic area led to a marked improvement of dystonic axial or appendicular tremors.

Kisely (2014) | **Key points:**
Efficacy of DBS on obsessive-compulsive disorder | • Five randomized controlled trials (n = 44).
• Patients on DBS had significantly lower symptom scores (partial remissions) but also higher rate of adverse effects.
• Promising but insufficient evidence for other psychiatric conditions.

Chopra (2013) | **Key points:**
DBS effects on essential tremor | • Literature review of current evidence of efficacy and safety of DBS in essential tremors
• Ventral intermediate (VIM) nucleus and posterior subthalamic area (PSA) treatment are safe and efficacious in reducing tremors and improving activities of daily living
• DBS-related adverse events are typically mild and related to the stimulation.

References

Professional society guidelines/other:


[https://www.cadth.ca/media/pdf/htis/aug-](https://www.cadth.ca/media/pdf/htis/aug-}


Peer-reviewed references:


Berlim MT, McGirr A, Van den Eynde F, et al. Effectiveness and acceptability of deep brain stimulation (DBS) of the subgenual cingulate cortex for treatment-resistant depression: a systematic review and


Louis ED, Ottman R. How many people in the USA have essential tremor? Deriving a population estimate based on epidemiological data. *Tremor Other Hyperkinet Mov (NY)*. 2014;4:259.


**CMS National Coverage Determinations (NCDs):**

Deep brain stimulation for essential tremor (ET) and Parkinson’s disease (PD). (NCD 160.24). Effective April 1, 2003..https://www.cms.gov/medicare-coverage-database/details/ncd-details.aspx?NCDId=279&ncdver=1&CoverageSelection=Both&ArticleType=All&PolicyType=Final&s=All&KeyWord=deep+brain+stimulation&KeyWordLookUp=Title&KeyWordSearchType=And&bc=gAAAACAAAA
Local Coverage Determinations (LCDs):

Psychological and Neuropsychological Testing (L34646). Effective February 1, 2016.  


Commonly submitted codes

Below are the most commonly submitted codes for the service(s)/item(s) subject to this policy. This is not an exhaustive list of codes. Providers are expected to consult the appropriate coding manuals and bill accordingly.

<table>
<thead>
<tr>
<th>CPT Code</th>
<th>Description</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>61863</td>
<td>Twist drill, burr hole, craniotomy, or craniectomy with stereotactic implantation of neurostimulator electrode array in subcortical site (e.g., thalamus, globus pallidus, subthalamic nucleus, periventricular, periaqueductal gray), without use of intraoperative microelectrode recording; first array</td>
<td></td>
</tr>
<tr>
<td>+61864</td>
<td>Twist drill, burr hole, craniotomy, or craniectomy with stereotactic implantation of neurostimulator electrode array in subcortical site (e.g., thalamus, globus pallidus, subthalamic nucleus, periventricular, periaqueductal gray), without use of intraoperative microelectrode recording; each additional array (List separately in addition to primary procedure)</td>
<td></td>
</tr>
<tr>
<td>61867</td>
<td>Twist drill, burr hole, craniotomy, or craniectomy with stereotactic implantation of neurostimulator electrode array in subcortical site (e.g., thalamus, globus pallidus, subthalamic nucleus, periventricular, periaqueductal gray), with use of intraoperative microelectrode recording; first array</td>
<td></td>
</tr>
<tr>
<td>+61868</td>
<td>Twist drill, burr hole, craniotomy, or craniectomy with stereotactic implantation of neurostimulator electrode array in subcortical site (e.g., thalamus, globus pallidus, subthalamic nucleus, periventricular, periaqueductal gray), with use of intraoperative microelectrode recording; each additional array (List separately in addition to primary procedure)</td>
<td></td>
</tr>
<tr>
<td>61880</td>
<td>Revision or removal of intracranial neurostimulator electrodes</td>
<td></td>
</tr>
<tr>
<td>CPT Code</td>
<td>Description</td>
<td>Comments</td>
</tr>
<tr>
<td>----------</td>
<td>-----------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>61885</td>
<td>Insertion or replacement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to a single electrode array</td>
<td></td>
</tr>
<tr>
<td>61886</td>
<td>Insertion or replacement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to 2 or more electrode arrays</td>
<td></td>
</tr>
<tr>
<td>95970</td>
<td>Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude, pulse duration, configuration of wave form, battery status, electrode selectability, output modulation, cycling, impedance and patient compliance measurements); simple or complex brain, spinal cord, or peripheral (i.e., cranial nerve, peripheral nerve, sacral nerve, neuromuscular) neurostimulator pulse generator/transmitter, without reprogramming</td>
<td></td>
</tr>
<tr>
<td>95971</td>
<td>Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude, pulse duration, configuration of wave form, battery status, electrode selectability, output modulation, cycling, impedance and patient compliance measurements); simple spinal cord, or peripheral (i.e., peripheral nerve, sacral nerve, neuromuscular) neurostimulator pulse generator/transmitter, with intraoperative or subsequent programming</td>
<td></td>
</tr>
<tr>
<td>95974</td>
<td>Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude, pulse duration, configuration of wave form, battery status, electrode selectability, output modulation, cycling, impedance and patient compliance measurements); complex cranial nerve neurostimulator pulse generator/transmitter, with intraoperative or subsequent programming, with or without nerve interface testing, first hour</td>
<td></td>
</tr>
<tr>
<td>+95975</td>
<td>Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude, pulse duration, configuration of wave form, battery status, electrode selectability, output modulation, cycling, impedance and patient compliance measurements); complex cranial nerve neurostimulator pulse generator/transmitter, with intraoperative or subsequent programming, each additional 30 minutes after first hour (List separately in addition to code for primary procedure)</td>
<td></td>
</tr>
<tr>
<td>95978</td>
<td>Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude and duration, battery status, electrode selectability and polarity, impedance and patient compliance measurements), complex deep brain neurostimulator pulse generator/transmitter, with initial or subsequent programming; first hour</td>
<td></td>
</tr>
<tr>
<td>+95979</td>
<td>Electronic analysis of implanted neurostimulator pulse generator system (e.g., rate, pulse amplitude and duration, battery status, electrode selectability and polarity, impedance and patient compliance measurements), complex deep brain neurostimulator pulse generator/transmitter, with initial or subsequent programming; each additional 30 minutes after first hour (List separately in addition to code for primary procedure)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>ICD 10 Code</th>
<th>Description</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>G20</td>
<td>Parkinson's disease</td>
<td></td>
</tr>
<tr>
<td>G24.1</td>
<td>Genetic torsion dystonia (includes familial and idiopathic)</td>
<td></td>
</tr>
<tr>
<td>G25.0</td>
<td>Essential/familial tremor</td>
<td></td>
</tr>
<tr>
<td>HCPCS Level II Code</td>
<td>Description</td>
<td>Comment</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----------------------------------------------------------</td>
<td>----------------------------------------------</td>
</tr>
<tr>
<td>C1767</td>
<td>Generator, neurostimulator (implantable), nonrechargeable</td>
<td></td>
</tr>
<tr>
<td>C1778</td>
<td>Lead, neurostimulator (implantable)</td>
<td></td>
</tr>
<tr>
<td>C1816</td>
<td>Receiver and/or transmitter, neurostimulator (implantable)</td>
<td></td>
</tr>
<tr>
<td>C1883</td>
<td>Adaptor/ extension, pacing lead or neurostimulator lead (implantable)</td>
<td></td>
</tr>
<tr>
<td>C1897</td>
<td>Lead, neurostimulator test kit (implantable)</td>
<td></td>
</tr>
<tr>
<td>E0745</td>
<td>Neuromuscular stimulator, electronic shock unit</td>
<td></td>
</tr>
<tr>
<td>L8680</td>
<td>Implantable neurostimulator electrode, each</td>
<td></td>
</tr>
<tr>
<td>L8681</td>
<td>Patient programmer (external) for use with implantable programmable neurostimulator pulse generator, replacement only</td>
<td></td>
</tr>
<tr>
<td>L8682</td>
<td>Implantable neurostimulator radiofrequency receiver</td>
<td></td>
</tr>
<tr>
<td>L8683</td>
<td>Radiofrequency transmitter (external) for use with implantable neurostimulator radiofrequency receiver</td>
<td></td>
</tr>
<tr>
<td>L8685</td>
<td>Implantable neurostimulator pulse generator, single array, rechargeable, includes extension</td>
<td></td>
</tr>
<tr>
<td>L8686</td>
<td>Implantable neurostimulator pulse generator, single array, non-rechargeable, includes extension</td>
<td></td>
</tr>
<tr>
<td>L8687</td>
<td>Implantable neurostimulator pulse generator, dual array, rechargeable, includes extension</td>
<td></td>
</tr>
<tr>
<td>L8688</td>
<td>Implantable neurostimulator pulse generator, dual array, non-rechargeable, includes extension</td>
<td></td>
</tr>
<tr>
<td>L8689</td>
<td>External recharging system for battery (internal) for use with implantable neurostimulator, replacement only</td>
<td></td>
</tr>
<tr>
<td>L8695</td>
<td>External recharging system for battery (external) for use with implantable neurostimulator, replacement only</td>
<td></td>
</tr>
</tbody>
</table>