Cigna Medical Coverage Policy

Subject **Deep Brain and Motor Cortex Stimulation**

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Coverage Policy

Deep Brain Stimulation

Cigna covers deep brain stimulation (DBS) as medically necessary for the treatment of ANY of the following:

- chronic, medically intractable primary dystonia (including generalized and/or segmental dystonia, hemidystonia, or cervical dystonia/torticollis) for an individual seven years of age or older when used in accordance with the Humanitarian Device Exemption (HDE) specifications of the U.S. Food and Drug Administration (FDA)
- chronic, medically intractable Parkinson disease (PD) when ALL of the following criteria are met:
  - The individual has intractable motor fluctuations, dyskinesia or tremor.
  - The individual is levodopa-responsive.
  - The individual does not have a significant mental impairment (e.g., dementia, severe depression) or a medical (e.g., stroke, cardiovascular disease) or surgical (e.g., previous ablative surgery such as thalamotomy, pallidotomy) contraindication to DBS.
- chronic, medically intractable essential tremor (ET)

Cigna does not cover DBS for any other indication including, but not limited to, obsessive-compulsive disorder because it is considered experimental, investigational or unproven.

Motor Cortex Stimulation

Cigna does not cover motor cortex stimulation for any indication because it is considered experimental, investigational or unproven.

General Background

Deep Brain Stimulation
Deep brain stimulation (DBS) involves the delivery of continuous, high-frequency electrical impulses to an area in the brain responsible for movement. The procedure is reversible and causes no permanent damage. Prior to implantation, a stereotactic rigid frame, or frame based system, is secured to the patient's skull, and the initial targeted area is selected using an imaging technique (e.g., magnetic resonance imaging [MRI], computed tomography [CT] or ventriculography). An alternative to the frame-based system is the frameless stereotactic system which may use external fiducial markers and/or internal anatomic landmarks. An electrode is introduced into the brain and test simulations are performed to evaluate and adjust tremor amplitude, diffusion of stimulation and determination of the threshold for paresthesias and speech disturbances. The electrode is connected to a computerized pulse generator which is typically implanted underneath the skin near the collarbone. The DBS system may be implanted either unilaterally or bilaterally, depending on the distribution of the patient's symptoms. When the intended targets include both sides of the brain, two separate systems are implanted. The system also includes a handheld therapy controller and a control magnet. Batteries in the generators typically last from three to five years and are replaced in an outpatient procedure. Some newer devices may have a rechargeable battery (Medtronic, 2013; Dystonia Medical Research Foundation, 2010; Weintraub, et al., 2007; Holloway, et al., 2005).

DBS is used for a carefully selected subset of individuals with chronic primary dystonia including generalized and/or segmental dystonia, cervical dystonias (i.e., torticollis), and hemidystonia. In addition, DBS is considered an established intervention for the treatment of medically refractory essential tremor (ET) and Parkinson disease (PD). DBS is not a first line therapy and is generally considered when the individual cannot tolerate or has failed pharmacotherapy or when pharmacotherapy is no longer effective.

U.S. Food and Drug Administration (FDA)
The Activa® Dystonia Therapy System (Medtronic Neurological, Minneapolis, MN) was FDA-approved under the Humanitarian Device Exemption (HDE) process. The device was approved for "unilateral or bilateral stimulation of the internal globus pallidus (GPi) or the subthalamic nucleus (STN) to aid in the management of chronic, intractable (i.e., drug-refractory) primary dystonia, including generalized and/or segmental dystonia, hemidystonia, and cervical dystonia (i.e., torticollis) in patients seven years of age or above" (FDA, 2003).

The Activa® Tremor Control System (Medtronic) was approved by the FDA under the premarket approval process (PMA) for "unilateral thalamic stimulation for the suppression of tremor in the upper extremity in patients who are diagnosed with essential tremor or Parkinsonian tremor not adequately controlled by medications and where the tremor constitutes a significant functional disability" (FDA, 1997).

The Activa® Parkinson's Control Therapy System (Medtronic) is FDA approved as a PMA device for "bilateral stimulation of the internal globus pallidus (GPi) or the subthalamic nucleus (STN) for adjunctive therapy in reducing some of the symptoms of advanced, levodopa-responsive Parkinson's disease that are not adequately controlled with medication" (FDA, 2002).

In February 2009, the Medtronic Reclaim™ DBS™ Therapy for OCD system was FDA approved as a HDE device and is "indicated for bilateral stimulation of the anterior limb of the internal capsule (AIC) as an adjunct to medications and as an alternative to anterior capsulotomy for treatment of chronic, severe, treatment-resistant obsessive-compulsive disorder (OCD) in adult patients who have failed at least three selective serotonin reuptake inhibitors (SSRIs)".

The HDE labeling for the Reclaim system stated that "The safety and probable benefit of DBS for the treatment of OCD has not been established for the following:

- patients with Tourette's syndrome
- patients with primary subclassification of hoarding
- patients whose OCD is documented to be less than five years duration
- patients whose Yale-Brown Obsessive-Compulsive Scale (YBOCS) score is less than 30

- patients who have not completed a minimum of three adequate trials of first and/or second line medications with augmentation
- patients who have not attempted to complete an adequate trial of cognitive behavior therapy (CBT)
- patients with a previous surgical ablation procedure (e.g., capsulotomy)
- patients who are pregnant
- patients who are under the age of 18 years
- patients with dementia
- patients with coagulopathies or who are on anticoagulant therapy
- patients without comorbid depression and anxiety
- patients with neurological disorders
- patients with other serious medical illness including cardiovascular disease, renal or hepatic failure, and diabetes mellitus"

The labeling also stated that **"**Physicians should carefully consider the potential risks of implanting the brain stimulation system in patients with comorbid psychiatric disorders, including:

- bipolar disorder
- body dysmorphic disorder
- expanded personality impulse-control disorders or paraphilias
- psychotic disorder
- severe personality disorders
- substance abuse
- the inability to control suicidal impulses or a history of suicide attempts

The brain stimulation system may aggravate the symptoms of comorbid psychiatric disorders" (FDA, 2009).

Dystonia
Dystonia refers to a diverse group of movement disorders characterized by involuntary muscle contractions that may cause twisting and repetitive movements or abnormal postures. Primary dystonia often begins focally in the legs and progresses to a generalized (i.e., involving all of the body) syndrome. Secondary dystonias are induced by a disease or ingested substance. Dystonias may also be categorized as focal (i.e., one area of the body is involved, such as hemidystonia, cervical dystonia or torticollis), or segmental involving two or more areas.

Treatment options for dystonia include oral medications and chemodenervation (e.g., botulinum toxin [BTX], type A or type B injection therapy). Invasive interventions and surgery for dystonia are generally reserved for those patients who have significant disabilities and are refractory to aggressive medication therapy and BTX. Deep brain stimulation (DBS) is a reversible, surgical option used for the treatment of primary dystonia including generalized and/or segmental dystonia, hemidystonia, and cervical dystonia or torticollis. DBS is indicated for individuals age seven years and older who do not respond to pharmacotherapy (i.e., medically intractable).

Literature Review: Randomized controlled trials and case series have reported improvement in tremor, speech, walking, performance of activities of daily living, reduction in medication usage, mood and quality of life following DBS in patients with dystonia (Sarubbo, et al., 2012; Cif, et al., 2010; Mueller, et. al., 2008; Vidailhet, et al., 2007; Hung, et al., 2007; Kiss, et al., 2007; Grips, et al., 2007; Tisch, et al., 2006; Kupsch, et al., 2006; Diamond, et al., 2006; Vidaihet, et al., 2005; Zorzi, et al., 2005; Halbig, et al., 2005; Starr, et al., 2004; Kupsch, et al., 2003).

Professional Societies/Organizations: The European Federation of Neurological Society and the Movement Disorder Society-European Section (EFNS/MDS-ES) (Albanese, et al., 2011) task force conducted a systematic literature review and published evidence-based recommendations for the diagnosis and treatment of dystonia. The recommendations stated that pallidal DBS is a good option for primary generalized or segmental dystonia and cervical dystonia following failure of medication or botulinum toxin. In general, pallidal DBS is less effective in secondary dystonia with the exception of tardive dystonia.

In a guidance document for DBS for dystonia, the National Institute for Health and Clinical Excellence (NICE) (United Kingdom) (2006a) stated that the current evidence supports the safety and efficacy of DBS as a treatment modality for dystonia. Dystonia may be treated conservatively or surgically. Conservative treatment

only treats the symptoms, and surgical intervention (i.e., thalamotomy and pallidotomy) may not render long-term benefits.

Essential Tremor (ET) and Parkinson Disease (PD)
Essential tremor (ET) is a common movement disorder characterized by postural tremor of the outstretched upper limbs that is absent at rest, not worsened by movement, and not associated with extrapyramidal or cerebellar signs. For most individuals with ET, symptoms can be managed with propanolol and primidone. Alcohol ingestion temporarily reduces ET symptoms, an effect that may last from 30 minutes to several hours. If medications and alcohol ingestion fail to provide adequate relief, patients with severe, chronic and medically intractable ET become candidates for surgical interventions (e.g., thalamotomy and pallidotomy).

Parkinson disease (PD) is a slowly progressive, chronic neurodegenerative disorder resulting from the death of the cells of the substantial nigra which contain dopamine. Eventually, lack of dopamine leads to hyperactivity in the internal globus pallidus (GPi) resulting in direct over stimulation of the GPi and over stimulation of the subthalamic nucleus (STN) which contributes to the existing over stimulation of the GPi.

Levodopa therapy effectively relieves symptoms in approximately 95% of PD patients. However, over the course of 5–10 years, most levodopa-responsive patients manifest increasingly severe and frequent motor fluctuations. When levodopa therapy fails, propanolol can be administered as an adjuvant treatment and anticholinergic medications can counteract symptoms in some patients.

Patients with PD who are considered candidates for DBS include those who have been successfully treated with levodopa, but have become nonresponsive to the medication (i.e., levodopa-resistant). In general, patients who have a significant mental impairment (e.g., dementia, severe depression, affective disorders, psychosis, cognitive deficit) are not considered candidates for DBS. The presence of a significant mental impairment may preclude the ability of the patient to respond to stimulation testing during insertion of the device to assist in proper lead placement and to properly operate the stimulator following insertion. In some cases, it has been reported that DBS may worsen pre-existing mental conditions (e.g., dementia, cognitive deficits/impairment). Co-morbidities and medical contraindications (e.g., cardiovascular disease, stroke) to implantation are taken into consideration. Surgical contraindications include patients with previous ablative surgery (e.g., thalamotomy, pallidotomy) or conditions that may increase the risk of intracranial hemorrhage (Medtronic, 2013; Benabid, 2009; Olanow, et al., 2009; Pahwa, et al., 2006).

Literature Review: Systematic reviews, meta-analysis, randomized controlled trials and case series support the safety and efficacy of DBS for the treatment of ET and PD (Weaver, et al., 2012; Flora , et al., 2010; Zhang, et al., 2010; Folett, et al., 2010; Williams, et al., 2010; Weaver, et al., 2009; Tir, et al., 2007; Deuschl, et al., 2006; Kleiner-Fisman, et al., 2006; Temel, et al., 2006; Visser-Vandewalle, et al., 2005; Rodriguez-Oroz, et al., 2005; Kraus, et al., 2004; Puzke, et al., 2004; Renhcrona, et al., 2003; Deep-Brain Stimulation for Parkinson's Disease Study Group, 2001; Koller, et al., 2001; Obwegeser, et al., 2001; Ondo, et al., 2001b; Vesper, et al., 2002; Schuurman, et al., 2000).

Following a review of the evidence, the Ontario Health Technology Advisory Committee (OHTAC) (2005) concluded that the evidence from large randomized controlled trials supports bilateral DBS of the STN which is effective in the short-term control of advanced Parkinsonian symptoms. Evidence from nonrandomized controlled studies suggested that this effect in Parkinson disease is sustained for at least five years, based on measures of motor function, activities of daily living, percentage of waking day spent in good function without dyskinesia and reduction in daily drug intake. The evidence also indicated that DBS of the thalamus is effective in the control of tremor in patients with ET and PD for at least six years.

Professional Societies/Organizations: In their practice parameters on DBS, the American Society for Stereotactic and Functional Neurosurgery (2010) stated that DBS has been shown to be a safe and effective procedure for medically intractable Parkinson disease and other movement disorders (e.g., moderate to severe medically intractable primary dystonia, tardive dystonia from psychotropic medications) when performed with FDA-approved devices in appropriate medical centers.

In practice parameters for the treatment of PD nonmotor symptoms, the American Academy of Neurology (Zesiewicz, et al., 2010) stated that there was insufficient evidence to support DBS for the treatment of urinary incontinence in PD.

In April 2006, the Quality Standards Subcommittee of the American Academy of Neurology issued an evidence-based practice parameter for the treatment of PD. The report stated that although criteria are evolving, currently patients with PD who are considered candidates for DBS include levodopa-responsive, non-demented and neuro-psychiatrically intact patients who have intractable motor fluctuations, dyskinesia or tremor. According to the subcommittee, DBS of the STN may be considered as a treatment option in PD patients to improve motor function and to decrease motor fluctuations, duskiness and medication usage. Patients should be counseled regarding the risks and benefits of this procedure. Insufficient evidence was found to make any recommendations about the effectiveness of DBS of the GPi or Vim nucleus of the thalamus in reducing motor complications or medication usage, or in improving motor function in patients with PD (Pahwa, et al., 2006).

In an interventional procedure guidance on DBS for PD, NICE (2006b) concluded that the current evidence on safety and efficacy appears adequate to support the use of the procedure. NICE also issued a guidance on DBS for tremor and dystonia and found the available evidence adequate to support the use of DBS for tremor that is disabling, interferes with activities of daily living, and is refractory to the highest tolerated doses of medication.

The Quality Standards Subcommittee of the American Academy of Neurology (Zesiewicz, et al., 2011) practice parameter on therapies for ET stated that DBS of the Vim thalamic nucleus is effective in reducing contralateral limb tremor in medically refractory ET. Bilateral DBS is necessary to suppress tremor in both upper extremities, but there are insufficient data regarding the risk-benefit ratio of bilateral versus unilateral DBS in the treatment of ET. Both DBS and thalamotomy are effective in suppressing tremor in ET; however, DBS is associated with fewer adverse events. The decision to use either procedure should be based on each individual's circumstances and risk for intraoperative complications.

Other Conditions

DBS has been proposed for the treatment of multiple other disorders including: addictions (e.g., smoking, alcohol); aggressive behavior; Alzheimer disease; anorexia nervosa; camptocormia; cerebral palsy; cluster headache; chronic pain; central pain from spinal cord injury; depression; epilepsy; Huntington's disease; Lesch-Nyhan syndrome; movement disorders secondary to structural lesions (e.g., basal ganglionic stroke, tumor or vascular malformation); multiple sclerosis tremor; non-idiopathic Parkinson disease ("Parkinson Plus"); obesity; obsessive-compulsive disorder; restless leg syndrome; short-lasting, unilateral, neuralgiform headache (SUNCT); tardive dyskinesia; tremors of the head and voice; trigeminal neuralgia; trigeminal neuropathy; Tourette syndrome (i.e., Tics); secondary tremors from birth injury, trauma, toxins and stroke; and disorders of consciousness (e.g., minimally conscious, vegetative state) (Taghva, et al., 2012; Lyons, 2011; Nyhan, et al., 2010; Prévinaire, et al., 2009; Larson, 2008; Damier, et al., 2007; Kern and Kumar 2007; Mink, et al., 2006; Skidmore, et al., 2006; Anderson and Arciniegas, 2004; Centers for Medicare and Medicaid [CMS], 2003).

There is insufficient evidence in the published peer-reviewed scientific literature to support DBS for the treatment of any of these conditions. Studies are primarily in the form of case reports and case series with small patient populations (n=2–10) and short-term follow-ups. In some studies, various areas of the brain are used for stimulation and there is a lack of consensus as to which area/areas should be targeted for each condition. Definitive patient selection criteria have not been established. Comparison of DBS to established pharmacotherapy and surgical interventions is lacking. DBS devices are not FDA approved for treatment of these conditions.

Lakhan and Callaway conducted a systematic review of clinical trials (n=17), including case series (some with randomization of on/off sessions) and case reports to evaluate outcomes of DBS for the treatment of obsessive-compulsive disorder (OCD) and treatment resistant depression (TRD). Nine OCD studies (n=42 total patients; range, 1–18 per study), seven TRD studies (n=67 total patients; range 1–21 per study), and one study with one patient with both disorders met inclusion criteria. Follow-up ranged from 3–39 months. Due to the sparse data, meta-analysis could not be conducted. The authors noted that the reports of suicide and psychoses following DBS were "disturbing," and criteria for patient selection and electrode placement need to be established.

Appleby et al. (2007) conducted a meta-analysis of 546 relevant articles (i.e., 303 clinical trials, 72 case series, 130 case reports) to "characterize the risks and benefits of DBS and to assess its possible use within the psychiatric setting." Three percent of the studies included patients with headaches, chronic pain, epilepsy, OCD and depression. Improvements in mentation, mood and behavior were reported in ten studies, six studies reported worsening and two reported no change. An improvement in chronic pain was reported in 26 studies,

with no improvement in three studies and worsening of pain in two studies. Improvements in OCD scores were reported in eight studies with one study reporting no differences. Six studies indicated an improvement in anxiety, seven studies included improved cognition, three reported worsening of cognition and in 13 studies cognition was unchanged. Three studies used depression as the primary indicator of treatment outcomes and 34 used depression as a secondary measure. Of the studies evaluating depressive symptoms, an improvement was reported in 83.3% of the studies, 2.7 % reported worsening and 14% reported no change. The authors stated that because of the number of studies that did not report on post-DBS mood, the findings of improvement in depressive symptoms should be treated with caution. Following implantation, suicide/attempted suicide and episodes of depression, hypomania and anxiety were reported. Limitations of the studies included: the heterogeneity of the studies, categorical variables (i.e., improvement, no improvement) in outcome measures, lack of outcomes separated by lead placement site, inclusion of case reports and the lack of studies that reported side effects.

Chronic Pain: DBS has been proposed for the treatment of various types of chronic, intractable pain. However, because of surgical and nonsurgical treatment interventions, its use has substantially decreased (Kern and Kumar, 2007). Two studies were initiated in the 1980s seeking FDA approval but were prematurely concluded; thus, DBS for the treatment of chronic pain has not received FDA approval (Owen, et al., 2007). Studies are primarily in the form of case series with small patient populations (n=34–56) and short term follow-ups (Owen et al., 2007; Rasche, et al., 2006).

In a meta-analysis, Bittar et al. (2005) found six studies, case series and retrospective reviews, which met inclusion criteria. Follow-up ranged from one month to 15 years. A variety of stimulation sites and methods were utilized. Patients selected for DBS included individuals with pain of known organic origin who failed or poorly tolerated conventional therapies and did not have neuroses/psychoses or severe depression. Twenty-four different pain etiologies (n=1–103) were included (e.g., phantom limb and stump pain, spinal cord pain and/or injury, peripheral neuropathy/radiculopathy, cancer pain and anesthesia dolorosa). The authors reported that DBS was more effective for nociceptive pain than for deafferentation pain ($p<0.01$). Success rates of up to 80% were reported in patients with low back pain (n=103) and failed back surgery syndrome (n=59).

Cluster Headache: A cluster headache is a severe, chronic headache that typically occurs in cyclical patterns (i.e., clusters) on one side of the face at the same time of the day for several weeks. Due to the severity of pain, cluster headaches are often referred to as “suicide headaches.” Treatment options include pharmacotherapy and oxygen administration. DBS has been proposed for the treatment of severe cluster headaches that are refractory to medical management, but there is insufficient evidence in the published peer-reviewed literature to supports its effectiveness in this patient population.

Fontaine et al. (2010) conducted a randomized, crossover, double-blind, multicenter study including 11 patients with refractory chronic cluster headaches. Patients were randomized to two, one-month periods of active stimulation vs. sham stimulation separated by a one-week wash-out period. Thereafter, a 10-month open phase was conducted. At the end of the crossover period, there was no significant difference in the frequency of weekly attacks in either group. Following the 10-month open phase, the frequency of the attacks significantly decreased ($p=0.08$) and patients reported reduced emotional impact. Three “serious” adverse events included an infection requiring removal of the device, loss of conscious with hemiparesia, and severe micturition syncope episodes with hypotension. The study is limited by the small patient population and the short-term follow-up. As the authors noted, due to the conflicting results in the blinded phase and the open phase additional randomized controlled trials are needed to determine the clinical utility of DBS for cluster headaches.

Depression: Depression is an illness characterized by persistent sadness, anxiety, hopelessness, helplessness, pessimism, and a loss of energy and interest in activities. It typically interferes with the activities of daily living and normal functioning (Hauptman, et al., 2008). DBS has been proposed for the treatment of chronic depression nonresponsive to conventional therapies (e.g., behavioural therapies, pharmacotherapy). There is insufficient evidence in the peer-reviewed literature to support DBS for depression. Studies are primarily in the form of small case series (n=15–20) with short-term follow-ups (Lozano et al., 2012; Kennedy, et al., 2011; Malone, et al., 2009; Lozano et al., 2008).

The Canadian Network for Mood and Anxiety Treatments (CANMAT) (Kennedy, et al., 2009) conducted a systematic review to update 2001 guidelines developed by CANMAT and the Canadian Psychiatric Association for the treatment of adults with major depressive disorders and concluded that DBS was still considered

investigational. Seven studies met inclusion criteria. According to the authors, there were no large randomized controlled trials to judge efficacy and consensus on the most effective target brain region for implantation and patient selection criteria have not been established. There was also “no published evidence” on the relative effectiveness of DBS with or without concurrent antidepressant medication in the treatment of this patient population.

Epilepsy: Epilepsy is a common condition with repeated seizures caused by abnormal bursts of electrical activity in the brain. Seizures may cause problems with muscle control, movement, speech, vision and/or awareness. DBS of the thalamus, STN, cerebellum, hippocampus, caudate nucleus and mammillary nuclei has been proposed for the treatment of drug-refractory epilepsy. It is also been proposed that individuals who do not respond to vagal nerve stimulation or surgical resection may be DBS candidates. Although improvements in seizures of 70%–80% have been reported, the results have rarely been reproducible. Therefore, DBS for epilepsy is still considered in the experimental stage (Halpern, et al., 2008; Villanueva, et al., 2007).

Fisher et al. (2010) conducted a multicenter, double-blind, randomized controlled trial (n=110) to evaluate DBS for the treatment of epilepsy. Patients underwent bilateral stimulation of the anterior nuclei of the thalamus for epilepsy (SANTE). Diagnosis included medically refractory partial seizures including secondarily generalized seizures. Postoperatively, patients kept a daily diary of seizure activity for primary analysis. One month following implantation, patients were randomized to stimulation (n=54) or no stimulation (n=55) (one outlier patient was excluded due to lack of diary information). The intent-to-treat (ITT) analysis, with exclusion of the one patient, across the three-month blinded phase favored the stimulation group (p=0.039) and showed a significant decrease in seizure activity (p=0.039). Complex partial seizures significantly improved more in the stimulation group compared to the no stimulation group (p=0.041, outlier removed). The severity of seizures decreased more in the stimulated group (p=0.047) and injuries from seizures was greater in the control group (p=0.01). Effectiveness of therapy was dependant on the region of the seizure origin. Patients with seizure origin in frontal, parietal or occipital regions did not demonstrate significant differences in seizure reduction between the two groups. Following completion of the blinded period, 108 patients received stimulation for an additional 1–10 months. ITT analysis of patients who elected to continue stimulation following the blinded period revealed a 50% responder rate in 81 patients (54%) at 25 months, and 42 patients (67%) at 37 months. Three patients had > 50% worsening of seizures. At the two-year follow-up, 13 of 81 patients (16%) had a $\geq$ 90% median seizure frequency reduction compared to baseline. Fourteen patients (13%) were seizure-free for at least six months, eight patients (7.3%) for at least one year, four patients (3.6%) for at least two years, and one patient (0.9%) was seizure-free for more than four years. Adverse events included paresthesia, infection, implant site pain, depression, asymptomatic hemorrhages and new seizure events. Eighteen patients withdrew due to adverse events. Limitations of the study include the small, heterogeneous patient population and the short-term follow-ups.

Multiple Sclerosis (MS): Multiple sclerosis (MS) is a disease of the central nervous system (CNS) that is characterized by areas of demyelination in the white matter of the brain and by recurrent exacerbations of neurologic dysfunction. It is estimated that approximately 10% of MS patients have disabling tremors. Although DBS has been proposed as a treatment option for MS, there is insufficient evidence to support the safety and efficacy of DBS for this condition.

A review by Hayes (2006) of clinical trials that investigated the use of DBS for the treatment of MS tremors included four case series and one nonrandomized controlled trial that compared DBS with thalamotomy. The results suggested that tremor reduction resulted in little or no functional improvement. Only one study reported a statistically significant improvement in one functional outcome. A 2007 update search included a case series with six patients which reported significant improvement in performance of alternating forearm movements during VIM stimulation.

Obsessive-Compulsive Disorder (OCD): OCD is a type of anxiety disorder in which individuals have unwanted thoughts (obsessions) and repeated behaviors (compulsions) over and over again. Severe cases of OCD can be disabling and interfere with activities of daily living and relationships. Treatment for OCD may include pharmacotherapy (e.g., selective serotonin reuptake inhibitors [SSRIs] and/or antipsychotic medications) and/or psychotherapy. DBS has been proposed as a treatment option for chronic, severe OCD in individuals who are unresponsive to adequate medical and behavioral therapy including, but not limited to failure of at least three SSRIs (Kuhn, et al., 2010; FDA, 2009; Mallet, et al., 2008).

There is insufficient evidence in the published peer-reviewed literature to support DBS for the treatment of OCD. Studies are primarily in the form of small case series (n=2–16) (Denys, et al., 2010; Okun, et al., 2007; Greenberg, et al., 2006; Rauch, et al., 2006) and case reports.

A multicenter randomized controlled trial by Mallet et al. (2008) compared stimulation of the subthalamic nucleus to sham stimulation in 16 patients, age range 18–60 years, with a primary diagnosis of OCD. Patients were unresponsive to pharmacotherapy (e.g., at least three serotonin-reuptake inhibitors) and cognitive behavioral therapy. The on-off group underwent DBS stimulation followed by sham stimulation and the off-on group underwent sham stimulation followed by DBS stimulation. The stimulation periods involved two 3-month phases (i.e., month 3 to month 6 and month 7 to month 10) separated by a 1-month washout phase. Patients received medications during the trial. Following DBS, a significant decrease was seen in the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) score (p=0.01). The on-off group had a significantly larger treatment effect than the off-on group (p=0.06). The Global Assessment of Functioning (GAF) score and the Clinical Global Impression (CGI) score were significantly improved after DBS compared to sham (p=0.005, p=0.008, respectively). At the end of the first three months, six (75%) patients were responders based on the Y-BOCS score and eight (100%) were responders based on the GAF scores compared to three (38%) responders following sham. No significant differences following DBS or sham were seen in the scores on the Montgomery and Asberg Depression Scale (MADRS), the Brief Scale for Anxiety, and the Sheehan Disability Scale. Due to the adverse events (i.e., intracerebral hemorrhage, infections requiring removal of the electrode) the authors stated that the benefits should be weighed carefully against the risks. Author-noted limitations included the variable deep brain stimulation settings used, small patient population and short duration of the study.

In an Emerging Technology Evidence Report, ECRI Institute (2010) evaluated DBS for treatment-resistant OCD using the Reclaim DBS system. Three non-randomized studies (n=46) which included one comparative study (n=16) met inclusion criteria. The available data were insufficient to calculate statistically and clinically significant differences in outcomes and adverse events and evaluate consistency across studies for symptom control and function. The studies assessed different DBS targets and a “high number” of adverse events were reported.

In their practice guideline for the treatment of OCD, the American Psychiatric Association (2007; reaffirmed 2013) described DBS as a “less-well-supported” monotherapy that may be considered after first and second-line therapies have been exhausted but clarify that there is little supporting evidence (e.g., “few small trials or case reports or uncontrolled case series”). DBS has been reported to show efficacy in individuals with severe, highly treatment-resistant OCD, but the procedure is not without its risk.

Tardive Dyskinesia: Tardive dyskinesia is a neurological syndrome characterized by repetitive, involuntary, purposeless movements and caused by the long-term use of neuroleptic drugs. Additional features may include grimacing; tongue protrusion; lip smacking, puckering and pursing; rapid eye blinking; and rapid movements of the arms, legs, and trunk (National Institute of Neurological Disorders and Stroke [NINDS], 2011).

In a prospective phase two multicenter study, Damier et al. (2007) investigated DBS in patients with severe TD refractory to medical management (n=10). Patients had been treated with antipsychotic medication for depression, schizophrenia or childhood disintegrative disorder. At the six-month follow-up, a double-blind evaluation resulted in successful outcomes by a decrease in the Extrapyramidal Symptoms Rating Scale, including choreic movements and dystonia score, by more than 40% (p=0.05). A significant decrease in the Abnormal Involuntary Movement Scale score (p=0.006) was also reported.

Tourette Syndrome (Tics): Tourette syndrome (TS), also known as chronic motor tic, chronic multiple tics, Gilles de la Tourette's disease or syndrome (GTS), habit spasms, maladie de tics, and paulitis tics, is a chronic neuropsychiatric disordered characterized by motor (e.g., repetitive involuntary movements of the face, head, upper body) and phonic, or vocal (e.g., sniffing, grunting, barking) tics. TS is often associated with behavioral abnormalities such as attention-deficit hyperactivity disorder and OCD. The waxing and waning characteristics of tics makes it difficult to investigate the safety and efficacy of DBS. It has been proposed for patients who have not received adequate benefit from behavioral therapy and pharmacotherapy (NORD, 2007; Mink, et al., 2006). Current studies include small patient populations, and the optimal DBS target for these individuals has not been defined (Cannon, et al., 2012; Kuhn, et al., 2010; Ackermans, et al., 2008).

Piedad et al. (2012) conducted a systematic review to determine which patients with Tourette syndrome (TS) should be treated with DBS and the best target areas for electrode placement. Thirty-six studies met inclusion

criteria including case reports, three case series and three randomized controlled trial. Based on the available data, the authors noted that it was “suggested” that the best candidates are patients with significant functional impairment due to tic symptoms and are nonresponsive to conventional pharmacotherapy and behavioral interventions. The globus pallidus internus and thalamus appeared to be the safest and most effective targets, especially for patients with “pure” TS and patients with comorbid obsessive-compulsive symptoms, anxiety and depression. There is a lack of consensus on treatment-refactoriness and large randomized controlled trials are needed to establish patient selection criteria and the appropriate target areas for placement.

The European Society for the Study of Tourette Syndrome (ESSTS) (Muller-Vahl, et al, 2011) conducted a systematic review of the literature to evaluate DBS for the treatment of Tourette syndrome (TS). Twenty four studies (n=63) including three randomized controlled trials, 18 case reports and three case series were reviewed. ESSTS concluded that DBS should only be used in “treatment resistant and severely affected adults” and “highly” recommended that it be in the context of controlled clinical trials.

The Tourette Syndrome Association (Mink, et al., 2007) convened a group of TS and DBS experts to develop guidelines for the early use and potential clinical trials of DBS for the treatment of TS believing that investigation of DBS for TS was justified due to the success of DBS with other disorders. The subgroup stated that although DBS has the potential to be an effective therapy for a carefully selected subgroup of TS patient’s “there are many unknowns about the potential applications” of DBS and investigation is warranted.

Motor Cortex Stimulation

Motor cortex stimulation (MCS), also referred to as cerebral cortex stimulation or extradural motor cortex stimulation (EMCS), is primarily proposed for relief of refractory neuropathic pain and involves implantation of epidural electrodes in the cerebral cortex. Although the exact mechanism of MCS is unknown, it has been hypothesized that it may induce the release of endogenous opioids in various brain structures, resulting in pain relief (Cheng and Eskandar, 2010; Maarrawi, et al., 2007).

Typically, temporary placement of a MCS device is performed to determine if the device will relieve the pain. If the patient consistently (e.g., 3–14 days) experiences at least a 50% reduction in pain, a second surgery is performed to permanently connect the electrodes and implant the programmable device under the skin near the collarbone. Image-guided localization (e.g., magnetic resonance imaging [MRI], functional MRI [fMRI], computerized tomography) and intraoperative mapping using somatosensory evoked potential (SSEP), intraoperative stimulation of the cortex, and/or neuronavigation are used to locate the precise placement of the electrodes, which is critical for successful pain relief. Electrodes are introduced through a burr hole or frontoparietal craniotomy into the protective layer covering the motor cortex area (epidural) of the brain, placed over the targeted area and connected to a programmable pulse generator. The lead wire from the programmable device goes up the back of the neck under the scalp to the electrodes (Cheng and Eskandar, 2010; Levy, et al., 2010; Arle and Shils, 2008).

Because MCS is a less invasive procedure than other invasive surgical procedures such as DBS, it is proposed to be a safer procedure with less serious complications. MCS has been proposed for treatment when invasive procedures have failed or when patients are not appropriate candidates for an invasive procedure. Some proponents of MCS report that MCS is less harmful than long-term opioid use. However, serious complication including intracranial bleeding; infection; permanent neurological deficits; and seizure activity, especially during programming and reprogramming of the MCS device, have been reported (Cheng and Eskandar, 2010; Levy, et al., 2010; Maarrawi, et al., 2007).

MCS was initially used for the treatment of medically refractory central pain syndrome following ischemic or hemorrhagic stroke and facial neuralgias (e.g., trigeminal neuralgia, postsurgical trigeminal deafferentation such as anesthesia dolorosa, postherpetic neuralgia). However, its use has been proposed for the treatment of other conditions including: neuropathic pain following spinal cord injuries (e.g., supraspinal pain after hemorrhage and infarction), post-stroke pain, chronic pain, amyotrophic lateral sclerosis (ALS), thalamic pain syndrome, plexus avulsion, dysphagia, Parkinson disease, dystonia, spasticity, multiple sclerosis, chronic regional pain syndrome (CRPS), phantom limb pain, epilepsy and peripheral nervous system lesions. MCS has also been used for intraoperative monitoring (Tanei, et al., 2011; Cheng and Eskandar, 2010; Levy, et al., 2010; Fontaine, et al., 2009; Prévinaire, et al., 2009; Arle and Shils, 2008).

U.S. Food and Drug Administration (FDA)

There are no devices approved by the FDA for motor cortex stimulation.

Literature Review
There is insufficient evidence in the published peer-reviewed literature to support the safety and efficacy of MCS for any indication. Studies are primarily in the form of case reports and case series with small heterogeneous patient populations (n=3–10) and short-term follow-ups. Outcomes regarding the benefits of MCS are conflicting. Some studies reported that the initial pain relief following MCS was not sustained over time and in some cases, worsening of pain followed MCS. Surgical techniques, electrode placement, device programming, outcome measures and patient selection criteria have not been established.

Fontaine et al. (2009) conducted a systematic review to evaluate the safety and efficacy of MCS for the treatment of chronic neuropathic pain. Fourteen studies (n=210), case series and retrospective reviews, met inclusion criteria. Reported mean follow-up was 30.5 months (range, several weeks to ten years). Overall, 56.7% of patients reported a 40%–50% (good) improvement in pain. Sixty-nine patients with ≥ 1 year follow-up reported a good response, and in two studies with 49-month follow-up, 47% and 22.6% of patients reported good results. The reported Visual Analog Scale scores for 76 patients reflected an average 56.6% improvement in postoperative scores. The most common adverse events were intraoperative or trial stimulation period seizures, infections and hardware-related problems. The authors stated that these results should be viewed with caution due to the limited number of studies that were primarily retrospective study designs with heterogeneous small patient populations (n=3–31). Short-term follow-up, loss of efficacy and the variable surgical techniques, stimulation settings and electrode placement were other noted limitations.

A limited number of randomized controlled trials have evaluated the use of MCS for the treatment of neuropathic pain comparing outcomes of on/off stimulation. In a crossover trial, Lefaucheur et al. (2009) reported that patients with trigeminal neuralgia (n=4), brachial plexus lesion (n=4), neurofibromatosis type-1 (n=3), upper limb amputation (n=2), herpes zoster ophthalmicus (n=1), atypical orofacial pain secondary to dental extraction (n=1), and traumatic nerve trunk transection in a lower limb (n=1) did not experience sustained pain relief during the crossover phase of the trial. Of the 12 patients who participated in the open study phase, 60% reported a mean pain relief of 48% on Visual Analog Scale scores at 12 months follow-up. In a study involving 11 patients (Velasco, et al., 2008) with chronic deafferentation pain syndromes (n=11), three patients reported no improvement following a temporary trial of MCS. The remaining patients who underwent permanent implantation reported a significant reduction in pain ($p<0.01$) during the one-year follow-up. The authors stated that “given the heterogeneous information that one gathers from the literature on MCS, it is impossible at present to draw a conclusion concerning candidates for this treatment.”

Professional Societies/Organizations
In their guidelines on neurostimulation for neuropathic pain, the European Federation of Neurological Societies (EFNS) (Cruccu, et al., 2007) stated that the literature primarily consisted of case series including patients with central post-stroke pain (CPSP) (n=20 case series with much overlap; 143 non-overlapping patients) and facial neuropathic pain (n=8 case series; 60 patients). Success rates ranged from 0%–100% for CPSP and 43%–100% for facial pain. Most studies did not have comparators, and outcome and treatment assessments were dissociated. Only case reports were found on patients with phantom pain, brachial plexus, nerve trunk lesion, spinal cord lesions and complex regional pain syndrome (CRPS). Based on these studies, EFNS stated that “MCS is useful in 50–60% of patients with CPSP and central or peripheral facial neuropathic pain, with small risk of medical complications,” but the evidence was insufficient to support MCS for any other condition.

Summary
Professional societies and evidence in the published peer-reviewed scientific literature support DBS for the treatment of a carefully selected subset of individuals with chronic, medically intractable primary dystonia, essential tremor (ET), and Parkinson disease (PD).

Studies investigating DBS for the treatment of all other disorders are primarily in the form of case reports and case series with small, heterogeneous patient populations and short-term follow-ups. Patient selection criteria, targeted areas in the brain, and appropriate stimulation systems have not been defined, nor have studies compared DBS to established pharmacotherapy or surgical interventions. The evidence in the published peer-reviewed scientific literature does not support DBS for any of the following conditions (list may not be all inclusive):

- cerebral palsy
- central pain from spinal cord injury
- cluster headaches
- chronic pain
- depression
- epilepsy
- Huntington's disease
- Lesch-Nyhan syndrome
- movement disorders secondary to structural lesions (e.g., basal ganglionic stroke, tumor or vascular malformation)
- multiple sclerosis tremors
- non-idiopathic Parkinson disease ("Parkinson Plus")
- obsessive-compulsive disorder
- Tourette syndrome (Tics)
- tardive dyskinesia
- tremors of the head and voice
- tremors from birth injury, trauma, toxins, and stroke

There is insufficient evidence in the published peer-reviewed scientific literature to support the safety and efficacy of motor cortex stimulation (MCS) for any indication. Studies are primarily in the form of case reports and case series with heterogeneous, small patient populations and short-term follow-ups. The studies used various outcome measures and results were conflicting. Criteria for patient selection, surgical techniques, electrode placement and device programming have not been established. There is no US Food and Drug Administration approved device for MCS.

Coding/Billing Information

Note: 1) This list of codes may not be all-inclusive.
2) Deleted codes and codes which are not effective at the time the service is rendered may not be eligible for reimbursement.

Deep Brain Stimulation

Covered when medically necessary:

CPT®* Codes	Description
61863	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
61864	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)
61867	Twist drill, burr hole, craniotomy, or craniectomy with stereotactic implantation of neurostimulator electrode array in subcortical site (eg, thalamus, globus pallidus, subthalamic nucleus, periventricular, periaqueductal gray), with use of intraoperative microelectrode recording; first array
61868	Twist drill, burr hole, craniotomy, or craniectomy with stereotactic implantation of neurostimulator electrode array in subcortical site (eg, thalamus, globus pallidus, subthalamic nucleus, periventricular, periaqueductal gray), with use of intraoperative microelectrode recording; each additional array (List separately in addition to primary procedure)

61885	Insertion or replacement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to a single electrode array
61886	Insertion or replacement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to two or more electrode arrays

HCPCS Codes	Description
C1767	Generator, neurostimulator (implantable), nonrechargeable
C1778	Lead, neurostimulator (implantable)
C1787	Patient programmer, neurostimulator
C1816	Receiver and/or transmitter, neurostimulator (implantable)
C1820	Generator, neurostimulator (implantable), with rechargeable battery and charging system
C1883	Adaptor/extension, pacing lead or neurostimulator lead (implantable)
C1897	Lead, neurostimulator test kit (implantable)
L8680	Implantable neurostimulator electrode (with any number of contact point), each
L8682	Implantable neurostimulator radiofrequency receiver
L8683	Radiofrequency transmitter (external) for use with implantable neurostimulator radiofrequency receiver
L8685	Implantable neurostimulator pulse generator, single array, rechargeable, includes extension
L8686	Implantable neurostimulator pulse generator, single array, non-rechargeable, includes extension
L8687	Implantable neurostimulator pulse generator, dual array, rechargeable, includes extension
L8688	Implantable neurostimulator pulse generator, dual array, non-rechargeable, includes extension

Motor Cortex Stimulation

Experimental/Investigational/Unproven/Not Covered when used to report motor cortex stimulation:

CPT®* Codes	Description
61850	Twist drill or burr hole(s) for implantation of neurostimulator electrodes, cortical
61860	Craniectomy or craniotomy for implantation of neurostimulator electrodes, cerebral, cortical
61885	Insertion or replacement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to a single electrode array
61886	Insertion or replacement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to two or more electrode arrays

HCPCS Codes	Description
C1767	Generator, neurostimulator (implantable), nonrechargeable
C1778	Lead, neurostimulator (implantable)
C1787	Patient programmer, neurostimulator
C1816	Receiver and/or transmitter, neurostimulator (implantable)
C1820	Generator, neurostimulator (implantable), with rechargeable battery and charging system
C1883	Adaptor/extension, pacing lead or neurostimulator lead (implantable)
C1897	Lead, neurostimulator test kit (implantable)
L8680	Implantable neurostimulator electrode (with any number of contact point), each
L8682	Implantable neurostimulator radiofrequency receiver
L8683	Radiofrequency transmitter (external) for use with implantable neurostimulator radiofrequency receiver

L8685	Implantable neurostimulator pulse generator, single array, rechargeable, includes extension
L8686	Implantable neurostimulator pulse generator, single array, non-rechargeable, includes extension
L8687	Implantable neurostimulator pulse generator, dual array, rechargeable, includes extension
L8688	Implantable neurostimulator pulse generator, dual array, non-rechargeable, includes extension

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