



Deep Brain Stimulation Clinical Coverage Criteria

Overview

Deep brain stimulation (DBS) consists of electrical stimulation of specific sites in the brain with implanted electrodes to reduce the symptoms of movement disorders such as Parkinson's disease and Essential Tremor. Targeted areas include the ventral intermediate nucleus of the thalamus, the internal globus pallidus and the subthalamic nucleus. Each of these brain regions has two halves which control movement on opposite sides of the body. Unilateral DBS has been proposed for use in patients when the symptoms are more severe on one side. Bilateral DBS has been proposed for the treatment of bilateral symptoms.

DBS is currently FDA-approved for treatment of Parkinson Disease, essential tremor, and epilepsy, with humanitarian device exemptions in dystonia and obsessive-compulsive disorder.

Policy

This Policy applies to the following Fallon Health products:

- ☒ Fallon Medicare Plus
- ☒ MassHealth ACO
- ☒ NaviCare HMO SNP
- ☒ PACE (Summit Eldercare PACE, Fallon Health Weinberg PACE)
- ☒ Community Care

Prior authorization is required.

Fallon Health Clinical Coverage Criteria

Fallon Health Clinical Coverage Criteria apply to MassHealth ACO and Community Care members.

Effective for dates of service on or after August 1, 2024, Fallon Health will use InterQual® Criteria when making medical necessity determinations for deep brain stimulation.

For coverage criteria, refer to the InterQual® Criteria in effect on the date of service:

- InterQual® CP:Procedures, Stereotactic Introduction Subcortical Electrodes

Fallon Health makes InterQual criteria available to the public through the transparency tool on our website, effective January 1, 2024.

Note: InterQual® Criteria address adult (18 years of age and older) indications. Medtronic Activa™ Deep Brain Stimulation (DBS) System has Humanitarian Device Approval (H020007) for unilateral or bilateral stimulation of the internal globus pallidus (GPi) or the subthalamic nucleus (STN) to aid in the management of chronic, intractable (drug refractory) primary dystonia, including generalized and/or segmental dystonia, hemidystonia, and cervical dystonia (torticollis) in patients seven years of age or above. Requests for deep brain stimulation for primary dystonia for plan members ≥ 7 years <18 years of age will be reviewed on an individual case-by-case basis by a Plan Medical Director.

Medicare Variation

Medicare statutes and regulations do not have coverage criteria for deep brain stimulation. Medicare has an NCD for Deep Brain Stimulation for Essential Tremor and Parkinson's Disease (160.24). Wellpoint Federal (National Government Services, Inc. began operating as Wellpoint Federal on April 1, 2026), the

Part A/B Medicare Administrative Contractor with jurisdiction in the Plan's service area, does not have an LCD for deep brain stimulation (Medicare Coverage Database search 07/26/2026). Coverage criteria for deep brain stimulation are fully established by Medicare; therefore, the Plan's coverage criteria are not applicable.

Link: [NCD Deep Brain Stimulation for Essential Tremor and Parkinson's Disease \(160.24\)](#)

Indications and Limitations of Coverage

Effective for services furnished on or after April 1, 2003, Medicare will cover unilateral or bilateral thalamic ventralis intermedius nucleus (VIM) deep brain stimulation (DBS) for the treatment of essential tremor (ET) and/or Parkinsonian tremor and unilateral or bilateral subthalamic nucleus (STN) or globus pallidus interna (GPi) DBS for the treatment of Parkinson's disease (PD) only under the following conditions:

1. Medicare will only consider DBS devices to be reasonable and necessary if they are Food and Drug Administration (FDA) approved devices for DBS or devices used in accordance with FDA approved protocols governing Category B Investigational Device Exemption (IDE) DBS clinical trials.
2. For thalamic VIM DBS to be considered reasonable and necessary, patients must meet all of the following criteria:
 - a. Diagnosis of ET based on postural or kinetic tremors of hand(s) without other neurologic signs, or diagnosis of idiopathic PD (presence of at least 2 cardinal PD features (tremor, rigidity or bradykinesia) which is of a tremor-dominant form.
 - b. Marked disabling tremor of at least level 3 or 4 on the Fahn-Tolosa-Marin Clinical Tremor Rating Scale (or equivalent scale) in the extremity intended for treatment, causing significant limitation in daily activities despite optimal medical therapy.
 - c. Willingness and ability to cooperate during conscious operative procedure, as well as during post-surgical evaluations, adjustments of medications and stimulator settings.
3. For STN or GPi DBS to be considered reasonable and necessary, patients must meet all of the following criteria:
 - a. Diagnosis of PD based on the presence of at least 2 cardinal PD features (tremor, rigidity or bradykinesia).
 - b. Advanced idiopathic PD as determined by the use of Hoehn and Yahr stage or Unified Parkinson's Disease Rating Scale (UPDRS) part III motor subscale.
 - c. L-dopa responsive with clearly defined "on" periods.
 - d. Persistent disabling Parkinson's symptoms or drug side effects (e.g., dyskinesias, motor fluctuations, or disabling "off" periods) despite optimal medical therapy.
 - e. Willingness and ability to cooperate during conscious operative procedure, as well as during post-surgical evaluations, adjustments of medications and stimulator settings.

The DBS is not reasonable and necessary and is not covered for ET or PD patients with any of the following:

1. Non-idiopathic Parkinson's disease or "Parkinson's Plus" syndromes.
2. Cognitive impairment, dementia or depression, which would be worsened by or would interfere with the patient's ability to benefit from DBS.
3. Current psychosis, alcohol abuse or other drug abuse.
4. Structural lesions such as basal ganglionic stroke, tumor or vascular malformation as etiology of the movement disorder.
5. Previous movement disorder surgery within the affected basal ganglion.
6. Significant medical, surgical, neurologic or orthopedic co-morbidities contraindicating DBS surgery or stimulation.

Patients who undergo DBS implantation should not be exposed to diathermy (deep heat treatment including shortwave diathermy, microwave diathermy and ultrasound diathermy) or any type of MRI, which may adversely affect the DBS system or adversely affect the brain around the implanted electrodes.

The DBS should be performed with extreme caution in patients with cardiac pacemakers or other electronically controlled implants, which may adversely affect or be affected by the DBS system.

For DBS lead implantation to be considered reasonable and necessary, providers and facilities must meet all of the following criteria:

Neurosurgeons must:

- a. Be properly trained in the procedure;
- b. Have experience with the surgical management of movement disorders, including DBS therapy; and
- c. Have experience performing stereotactic neurosurgical procedures.

Operative teams must have training and experience with DBS systems, including knowledge of anatomical and neurophysiological characteristics for localizing the targeted nucleus, surgical and/or implantation techniques for the DBS system, and operational and functional characteristics of the device.

Physicians specializing in movement disorders must be involved in both patient selection and post-procedure care.

Hospital medical centers must have:

- a. Brain imaging equipment (MRI and/or CT) for pre-operative stereotactic localization and targeting of the surgical site(s);
- b. Operating rooms with all necessary equipment for stereotactic surgery; and
- c. Support services necessary for care of patients undergoing this procedure and any potential complications arising intraoperatively or postoperatively.

MassHealth Variation

MassHealth does not have Guidelines for Medical Necessity Determination for deep brain stimulation, therefore, Fallon Health Clinical Coverage Criteria are applicable (MassHealth website search 07/26/2026).

Exclusions

- The Plan considers deep brain stimulation experimental/investigational and therefore not medically necessary for any indication not meeting coverage criteria described herein.

Summary of Evidence

Background Dystonia

Dystonia is a general term for a large group of movement disorders that vary in their symptoms, causes, progression and treatments. This group of neurological conditions is generally characterized by involuntary muscle contractions that force the body into abnormal, sometimes painful, movements and positions (postures). The muscular contractions may be sustained or come and go (intermittent). Movements may be patterned and twisting and/or in some cases shaking or quivering (tremulous) resembling a tremor. Dystonia may occur or be worsened when an individual attempts a voluntary action. There are many different causes for dystonia. Genetic as well as non-genetic factors can contribute to the development of these disorders. In some cases, the exact, underlying cause is unknown (idiopathic). The onset of dystonia can be very early in life or during adulthood, depending on the cause. Treatment for dystonia depends upon several factors including the specific subtype present and can include medications, botulinum toxin injections, physical therapy and surgery (NORD, 2024).

A basis for classifying the dystonias has been proposed based on a consensus achieved by an international expert group of physicians (Albanese A, et al. 2013). This group has proposed to classify along two axes: clinical characteristics, including age at onset, body distribution, temporal pattern and associated features (additional movement disorders or neurological features), and etiology, which includes nervous system pathology and inheritance.

Albanese et al. proposed the following definition for dystonia: Dystonia is a movement disorder characterized by sustained or intermittent muscle contractions causing abnormal, often repetitive, movements, postures, or both. Dystonic movements are typically patterned, twisting, and may be

tremulous. Dystonia is often initiated or worsened by voluntary action and associated with overflow muscle activation.

Classifying dystonia by clinical features includes age of onset, body distribution, temporal pattern and associated features.

Age of onset is broken down into infancy (birth to 2 years), childhood (3-12 years), adolescence (13-20), early adulthood (21-40) and late adulthood (greater than 40 years).

Dystonia that begins in childhood is more likely to have a discoverable cause, and more likely to progress from focal to generalized (Albanese et al., 2013).

Classification by body region affected is clinically important because of implications for diagnosis and therapy. Generally, dystonia may be focal (affecting an isolated body part), segmental (affecting adjacent body areas), multifocal (two or more noncontiguous areas), generalized (affecting the trunk and two other body regions) and affecting one side of the body (hemidystonia).

The diagnostic considerations in adult-onset focal dystonia are very different from those in young-onset generalized dystonia. The treatment of choice for focal and segmental dystonias involves botulinum neurotoxins, while for generalized dystonias more often involves medications or surgery.

The temporal pattern is an important clinical characteristic that facilitates diagnosis and treatment choices. Important temporal characteristics are related to disease course and distinguish static from progressive forms. This terminology is particularly used by pediatric neurologists, but it also suits adult cases. Disease course can be either static or progressive. The variability can have four different patterns:

- Persistent. Dystonia that persists to approximately the same extent throughout the day.
- Action-specific. Dystonia that occurs only during a particular activity or task.
- Diurnal fluctuations. Dystonia fluctuates during the day, with recognizable circadian variations in occurrence, severity and phenomenology.
- Paroxysmal. Sudden self-limited episodes of dystonia usually induced by a trigger with return to pre-existing neurological state.

Dystonia can also be classified by whether it occurs along with another movement disorder. Isolated dystonia is when dystonia is the only motor feature except for tremor. Combined dystonia is used when another movement disorder such as Parkinsonism or myoclonus is also present.

The etiology axis refers to whether degenerative changes or structural damage is present in the nervous system (nervous system pathology) and whether the disorder is inherited or acquired, or whether the underlying cause is unknown or unproven (idiopathic) (Albanese et al., 2013).

Previous terminology used to categorize dystonia includes primary dystonia, secondary dystonia, dystonia plus syndromes and hereditodegenerative dystonia. Primary dystonia referred to cases in which dystonia was the only clinical feature (isolated dystonia), there was no evidence of brain degeneration and without an acquired cause. Primary dystonia may be inherited or occur for unknown reasons (idiopathic). Secondary dystonia referred to cases in which dystonia resulted from a broad range of causes including genetic variants, birth injury, stroke, brain tumors, certain infections and as a reaction to certain drugs. Dystonia plus syndromes referred to disorders in which dystonia occurred in conjunction with another neurological disorder such as myoclonus or Parkinsonism. Hereditodegenerative dystonia referred to hereditary cases that were associated with neurodegeneration and occur with other neurological symptoms (NORD, 2024).

Until a consistent, straightforward classification system is adopted by the medical community confusion regarding terminology in describing dystonia will persist. In addition, dystonia is a rapidly growing disease family and information about these disorders is constantly changing.

The diagnosis of dystonia is based upon identification of characteristic symptoms, a detailed patient and family history and a thorough clinical evaluation. Evaluation by a movement disorder specialist may help to confirm a diagnosis of dystonia. Various, specialized tests may be recommended to rule out other conditions. Laboratory testing is essential in acquired dystonia to determine the underlying cause.

Molecular genetic testing can confirm a diagnosis of certain inherited forms of dystonia. Molecular genetic testing can detect variants in the specific genes known to cause inherited dystonia.

There is no cure for dystonia and treatment is therefore directed at relieving symptoms. Current treatments target specific symptoms and are intended to relieve muscle spasms, pain and discomfort, and unnatural postures. No single treatment program is appropriate for every patient.

There are three treatment options: oral medications, botulinum toxin injections and surgery. These treatments may be used alone or in combination. In addition, physical and speech therapy may provide a helpful complement to medical treatment in some patients.

There are no oral medications approved by the U.S. Food and Drug Administration (FDA) for dystonia. Sometimes oral medications that affect the activity of neurotransmitters are prescribed. Anticholinergic agents such as benztropine and trihexyphenidyl block the neurotransmitter acetylcholine, and benzodiazepines such as clonazepam, diazepam or lorazepam block the neurotransmitter gamma-aminobutyric acid (GABA). These drugs are most effective in children with generalized dystonia.

Some individuals with dystonia, particularly those with dopa-responsive dystonia respond to treatment with very low doses of levodopa, a synthetic version of the neurotransmitter dopamine. Levodopa increases dopamine levels. People with certain different forms of dystonia may respond to medications that block the activity of dopamine (antidopaminergic agents).

A muscle relaxant known as baclofen, which may help periodically to reduce muscle spasms, may be prescribed and delivered by means of an implantable pump that releases the drug directly into the area around the spinal cord. Baclofen can stimulate the body's ability to process the neurotransmitter GABA.

There is no standard treatment for rapid-onset dystonia-parkinsonism (RDP), although levodopa/carbidopa medications and dopamine agonists (drugs that stimulate dopamine receptors in the absence of dopamine) may provide mild improvement for some affected individuals.

In complex syndromes caused by metabolic conditions where dystonia represents one of their manifestations, such as Wilson's disease, different treatments specific for each condition can be attempted.

Botulinum toxin therapy is often used for certain forms of dystonia, particularly certain focal dystonias such as cervical dystonia and laryngeal dystonia. Botulinum toxin is a neurotoxin that is injected into muscles in very small doses. After injection into a muscle, the action of botulinum toxin is to interrupt nerve messages to the muscle, preventing the release of the neurotransmitter acetylcholine, which stimulates muscular contractions, and causes weakness of that muscle. The effect of botulinum toxin on the muscle begins approximately 2-3 days following injection, peaks at around 4 weeks and provides relief for approximately 3-6 months. When the effect of botulinum toxin wears off, the symptoms of dystonia recur. The degree of effectiveness of botulinum toxin will differ in each individual. Using ultrasound to guide the injection in the target muscles can significantly increase the benefit of the treatment for certain types of dystonia (such as task specific dystonias or truncal dystonia).

Botulinum toxin is approved by the FDA for cervical dystonia and blepharospasm and is widely used off label to treat all forms of dystonia. The FDA has a black box warning concerning the use of botulinum toxins. The black box warning denotes that a drug known to be effective for some individuals may cause serious side effects in others.

Surgery is generally reserved for those patients with severe dystonia who do not respond to drug therapy or cannot tolerate side effects as well as those with severe dystonia who become non-responsive to drug treatment. Deep brain stimulation (DBS) with an implantable pulse generator may be performed for some types of dystonia. DBS involves the surgical placement of very thin electrodes into certain areas of the brain such as the globus pallidus. The leads from these electrodes are then connected to a small device called a neurostimulator that is surgically implanted usually near the collarbone. These stimulators send small electrical pulses to the brain. After the DBS is placed, the stimulators are programmed for the optimal outcome. The electrical pulses block or interfere with the nerve signals that cause the symptoms

of dystonia. DBS has replaced other surgical techniques such as stereotactic thalamotomy, pallidotomy, and cervical rhizotomy because of its success and lower risk for side effects (NORD, 2024).

Deep Brain Stimulation

Deep brain stimulation (DBS) is currently FDA-approved for treatment of Parkinson Disease, essential tremor, and epilepsy, with humanitarian device exemptions in dystonia and obsessive-compulsive disorder.

This evidence review focuses on pediatric primary dystonia.

The Medtronic Activa™ Deep Brain Stimulation (DBS) System received Humanitarian Device Approval (HDE) (H020007) from the FDA on April 15, 2003, for the treatment of patients with primary dystonia seven years of age and older. Pediatric DBS use has been increasing, especially for treatment of dystonia when pharmacologic treatment is ineffective or poorly tolerated. There are no evidence-based guidelines or consensus statements for pediatric DBS. Generally accepted criteria for globus pallidus internus (GPi) DBS implantation include age 7 years or older, dystonia refractory to medical treatment, and significant disability (Mills et al., 2014).

The success of DBS is dependent on many factors including selection of appropriate patients, accurate placement of DBS lead, and a thorough programming process to identify the optimal stimulation parameters. Inefficient programming can result in suboptimal clinical outcomes and lead to side effects. A focus group consisting of 13 pediatric movement disorder specialists and 1 neurosurgeon experienced in deep brain stimulation from 9 academic institutions in the United States published recommendations for DBS programming in children with dystonia (Gelineau-Morel et al., 2023). Pre-operative evaluation and post-operative management by an experienced, multidisciplinary team comprised of a pediatric movement disorder specialist, functional neurosurgeon, neuropsychologist, pediatric neuroradiologist, physiatrist, physical and occupational therapists, nurses, and social workers are recommended. A suggested algorithm for GPi DBS programming in pediatric dystonia is presented, including pre-programming, initial, and follow up sessions.

The first session of DBS programming often requires two hours or more and is critical for successful subsequent programming sessions. Standardized dystonia rating scales (such as the Barry-Albright Dystonia Rating Scale, Burke Fahn Marsden Dystonia Rating Scale) and video documentation should be completed prior to programming at the first session and periodically thereafter to document response. These may be most effective when combined with additional metrics assessing patient-specific goals, function, and quality of life. Key parameters in DBS programming include frequency, pulse width, amplitude (voltage or current), and contact selection. The frequency is the number of electrical pulses delivered per second (Hz) while the pulse width is the duration of each electrical pulse (μ s). The amplitude is the magnitude of the electrical pulse, measured either as voltage (V) or current (mA). The goal in the first DBS programming session should be to complete monopolar review, a systematic exploration of each available contact to determine the therapeutic window, or range of stimulation settings which produce benefit without intolerable side effects. In monopolar review, the user first selects and sets the frequency and pulse width which are kept constant throughout the programming session while current/voltage settings are adjusted.

Follow-up, 1-hour programming sessions are usually scheduled every 2–4 weeks for the first 6–12 months, or until reaching stable stimulation settings. Patients may appear to achieve significant reduction in symptoms during a programming session, but the effect wears off within the first 1–2 weeks. Some programming strategies can help to mitigate this.

Systematic Reviews

For the treatment of pediatric primary dystonia, two systematic reviews were identified.

Hale et al., 2020 performed a systematic review of the literature including studies of DBS for pediatric (age < 21) dystonia. Nineteen studies reporting outcomes including in 76 children (58% male) were identified in peer-reviewed publications from 1980 to March 2018. All studies were retrospective, and no prospective studies were reported. The mean age at surgery was 13.8 ± 3.9 (mean $\pm$ SD) years. Duration of time between onset of symptoms and surgery was 6.4 ± 3.5 years, and post-operative follow-up was

2.8 ± 2.8 years, with 78% of patients having more than 1 year of follow-up. Primary generalized dystonia (PD) was reported in 52 patients (68%), of whom 29 patients (56%) had a pathological mutation in DYT1 (DYT1+), while other causes of dystonia—including secondary generalized and focal dystonia were reported in 24 children (32%). Causes of secondary dystonia varied. Because there was such heterogeneity in secondary causes and because outcomes of DBS in secondary dystonia have been sparsely reported, all secondary causes were treated as one category. All patients were described as poorly responsive or refractory to medical therapy for dystonia and failed medication trials of anticholinergic drugs, benzodiazepine derivatives, botulinum toxin injections, neuroleptics, oral baclofen, and/or intrathecal baclofen. Ninety-one percent of individuals were implanted with a bilateral globus pallidus interna (GPi) target, one (1.3%) patient received a unilateral GPi implant, one (1.3%) individual received bilateral GPi plus subthalamic nucleus (STN), and five (6.6%) children received a unilateral GPi implant and a contralateral GPi lesion. Thus, only surgical interventions targeting the GPi were considered.

Across all patients with data available, Burke-Fahn-Marsden Dystonia Rating Scale-Motor (BFMDRS-M) scores improved by 43.8 ± 36% (mean ± SD) after surgery, with 45% of individuals achieving ≥ 50% improvement, while Burke-Fahn-Marsden Dystonia Rating Scale-Disability (BFMDRS-D) scores improved by 43.7 ± 31% post-operatively, with 47% of children achieving ≥ 50% improvement.

Patients with PD were more likely to experience ≥ 50% improvement (56%) in BFMDRS-M scores compared to patients with secondary causes of dystonia (21%, $p = 0.004$). DYT1+ patients were more likely to achieve ≥ 50% improvement (65%) in BFMDRS-D than DYT1- individuals (29%, $p = 0.02$), although there was no difference in BFMDRS-M ≥ 50% improvement rates between DYT1+ (66%) or DYT1- (43%) children ($p = 0.11$). Age, gender, duration of symptoms, and length of follow-up were not found to be predictive of BFMDRS-M or BFMDRS-D outcomes ($p > 0.05$ for each, logistic regression).

Elkhaïm et al., 2019, performed a systematic review and meta-analysis for deep brain stimulation in pediatric patients (age ≤ 21 years) with dystonia. The primary outcomes were changes in Burke-Fahn-Marsden Dystonia Rating Scale (BFMDRS) or Barry-Albright Dystonia Scale scores. Seventy-two (321 children) articles published from January 1999 to August 2017 were included. Of these, 111 children and young people were treated for inherited dystonia without evidence of degeneration or structural lesions, 50 for inherited dystonia with degeneration, 76 for acquired dystonia with static lesions, and 72 for idiopathic dystonia. The remaining 12 patients were included in an alternate group (other or unknown) when the diagnosis stated by the authors was not recognized as having an etiological link to dystonia (i.e. Crigler-Najjar syndrome) or when a diagnosis of secondary dystonia was given without mention of the underlying cause. Eighteen children were treated for status dystonicus.

The DBS target was the globus pallidus interna (GPi) in 309 patients, the subthalamic nucleus alone in three patients, a combination of the subthalamic nucleus and GPi in three patients, and the thalamus with or without GPi in three patients.

Overall median improvement (interquartile range) in the motor subscore of the BFMDRS was 42.1 percent (12%–80%) with 86.3 percent ($n=277$) of patients showing any improvement at last follow-up (median 12mo). Significant improvement (>20%) were reported in 66.1 percent ($n=203$) of patients. Overall median improvement in the disability subscore of the BFMDRS was 27.7 percent (reported in 218 patients). In general, the best responders to DBS were patients with inherited dystonia without degeneration (76.5% median BFMDRS-motor [BFMDRS-M] change) or idiopathic dystonia (50.5% mean BFMDRS-M change). Comparatively, patients with inherited dystonia with nervous system pathology showed less, although still clinically significant (median 26.8%) improvement. Patients with acquired dystonia were the worst responders with a median (interquartile range) change of 10.5 percent (6%–23.1%) in BFMDRS-M score after DBS.

Subgroup analysis:

- Inherited dystonia without degeneration or structural lesions - Subgroup analysis was performed on the 111 patients with inherited dystonia without nervous system pathology treated with DBS. Children and young people included in this group were those with confirmed DYT1 or DYT6 mutations (n=102) or patients with myoclonus-dystonia (n=9), seven of whom had confirmed SGCE (DYT11) mutations. Most (n=107) patients were treated with bilateral GPi stimulation.

The median change in dystonia scores were 76.5 percent (motor) and 70 percent (disability, n=77), with 93.3 percent of children and young people (n=105) demonstrating some improvement and 88.2 percent (n=98) showing clinically significant (>20%) improvement in motor scores at last follow-up (median 13.5 month). A median improvement of 78.1 percent (BFMDRS-M) was reported after DBS at last follow-up (median 15 month) in patients with DYT1/DYT6 dystonia.

Findings from this meta-analysis found that children and young people with DYT1 dystonia respond well to DBS. When compared with patients with idiopathic dystonia (most of whom had been previously diagnosed with primary dystonia), patients with positive DYT1 genetic status responded significantly better, although patients with idiopathic dystonia still responded well.

Patients with myoclonus-dystonia responded similarly well, with a median improvement of 68.3 percent (BFMDRS-M) at last follow-up (mean 10.5 months). All nine patients with myoclonus-dystonia showed clinically significant improvement after DBS. Myoclonic movements improved by 83.3 percent on the Unified Myoclonus Rating Scale rest/action subscore in five patients and by 89.1 percent in Unified Myoclonus Rating Scale total score in one patient. DBS seems to be effective in treating both dystonic and myoclonic symptoms in patients with myoclonus-dystonia.

- Inherited dystonia with degeneration or structural lesions - For the 50 children and young people in this group treated with DBS, the median change in motor and disability subscores were 26.8 percent and 0 percent respectively at last follow-up (median 12 months). The most common etiology was pantothenate kinase-associated neurodegeneration (PKAN, n=36). In general, patients with PKAN dystonia showed clinically significant (median 27.7%, BFMDRS-M) improvement after DBS. Patients with Lesch-Nyhan disease responded similarly well. The worst responders were those with glutaric aciduria type 1 who showed a median change of 6.4 percent on BFMDRS-M after DBS. Except for PKAN dystonia, these findings are limited by small sample size (n=5 for glutaric aciduria type 1, and n=4 for Lesch-Nyhan disease).

The literature pertaining to DBS in patients with inherited dystonia with nervous system pathology is comprised of subcohorts. This, combined with highly variable responses has led to debate about the use of DBS as a treatment for these patients. This meta-analysis found highly variable, but generally inferior outcomes.

Acquired dystonia - The median changes in BFMDRS-M and disability score for 59 children and young people with cerebral palsy were 11.1 percent and 3.5 percent respectively (median follow-up 12 months). Patients with a history of kernicterus responded negatively, with a median change of 10.5 percent BFMDRS-M postoperatively. Three patients with poststroke dystonia did not show clinically significant improvement after DBS (mean 11.2%) at last follow-up. This meta-analysis found poor response (median 11.1% [0%–21.4%], BFMDRS-M) to DBS in patients with one of the most frequent causes of acquired dystonia, cerebral palsy.

- Idiopathic dystonia - Patients with idiopathic dystonia improved by 50.5 percent and 39.2 percent on BFMDRS motor and disability scores after DBS respectively. Of 70 patients with BFMDRS data, 80 percent (n=56) showed clinically significant improvement at last follow-up. When compared with the best responders on univariate analysis (inherited dystonia without nervous system pathology), children and young people with idiopathic dystonia responded worse; however, the difference was not significant on subsequent multivariate analysis.

Children and young people included in the idiopathic dystonia group generally responded well, with 80 percent of patients showing clinically significant improvement after DBS. The positive results are comparable with those seen in patients with DYT1 or myoclonus-dystonia, as no significant difference between the groups was noted on multivariate analysis. Before the introduction of the Albanese

classification, most of the patients with idiopathic dystonia had been previously diagnosed with primary dystonia. As such, it may be unsurprising that they respond so favorably. As more genetic mutations with links to dystonia become recognized, patients in this group may potentially be reclassified as having inherited dystonia without nervous system pathology.

- **Status dystonicus** - Eighteen patients were treated for status dystonicus with DBS. Among these patients, six had DYT1 dystonia, five had idiopathic dystonia, three had PKAN, two had Batten disease, 15 and two were of unknown etiology. Median improvement (interquartile range) in motor score was 54 percent (17.7%–88.5%) at last follow-up (median 11 mo), with six patients achieving over 85 percent improvement. Resolution of the crisis was observed in 16 out of 18 patients in the postoperative course. One patient did not have information pertaining to resolution of crisis postoperatively but maintained marked improvement at last follow up. Perioperative heart failure and death occurred in one patient treated for status dystonicus.

Status dystonicus is a potentially life-threatening condition requiring early diagnosis and treatment. DBS for status dystonicus has been described as an effective method of treating most patients with status dystonicus, although with potentially higher incidence of complications. The results of this meta-analysis found resolution of status dystonicus post-DBS in 16 out of 18 patients and 8 complications among 16 reported. In a recent literature review of DBS for status dystonicus (patients of all ages), cessation of dystonic storm was seen in 26 out of 28 patients, with best outcomes amongst DYT1-positive patients (Ben-Haim et al., 2016).

In summary, the most consistent positive response to DBS is among patients with inherited dystonia without nervous system pathology (DYT1, DYT6, and myoclonus-dystonia). Generally positive response is also seen in patients with idiopathic dystonia. Poor treatment response is associated with inherited dystonia with nervous system pathology, acquired dystonia, younger ages at dystonia onset, and lack of truncal involvement. Importantly, the current report also highlights shortcomings in the scope and quality of literature evaluating childhood dystonia treated with DBS.

The effect of patient age and timing of intervention on DBS outcomes in children remains unclear. Some studies have suggested that younger age is associated with improved response to DBS. In the current analysis, representing the largest synthesis of data on DBS in dystonic children, age at onset of dystonia, and not age at surgery is associated with treatment response.

Ethical Challenges

Kostick-Quenet and colleagues (2023), conducted a survey of 56 clinicians working with pediatric patients with movement disorders and who either specialize in pediatrics or have substantial experience with pediatric patients; 29 clinicians participated (response rate = 52%). Analysis revealed four pressing ethical concerns related to pediatric deep brain stimulation:

- (1) Uncertainty about risks and benefits of pediatric DBS (73%) that pose a challenge to informed decision-making;
- (2) Ethically navigating decision-making roles (50%), including how best to integrate perspectives from diverse stakeholders (patient, caregiver, clinician) and how to manage surrogate decisions on behalf of pediatric patients with limited capacity to make autonomous decisions;
- (3) Effects of information scarcity on informed consent and decision quality (46%) in the context of patient and caregivers' expectations for treatment; and
- (4) Narrow regulatory status and access (7/29; 24%) such as the lack of FDA-approved indications that contribute to decision-making uncertainty and liability and potentially limit access to DBS among patients who may benefit from it.

Clinicians said that because DBS is a novel treatment not yet widely used among children and adolescents, there is a consequent lack of definitive evidence concerning outcomes or insights into ideal approaches to program stimulation parameters in pediatric population. The high degree of variation within the small population sample of pediatric dystonia patients receiving DBS exacerbates these uncertainties.

Clinicians said they find it challenging to mitigate conversations with families about risks versus benefits in the absence of extensive outcome data.

This inability to clearly delineate risk/benefit ratios further complicates clinicians' ability to ensure informed consent and decision quality.

Clinicians said that their potential liability is further exacerbated by a lack of guidelines and evidence-based best practices to inform their treatment decisions.

Analysis of Evidence (Rationale for Determination)

In general, studies of DBS for dystonia in children are limited by small sample size, and thus, indications and outcomes are poorly understood. The most consistent positive responses to DBS in children and young adults (≤ 21 years of age) have been found in those with inherited dystonia without nervous system pathology. The ethical challenges involved in treating children with DBS are numerous. DBS treatment guidelines for pediatric patients with dystonia are not well-established. There is no consensus on specific recommendations regarding when, if at all, DBS should be performed and for patients with various causes of dystonia.

Coding

The following codes are included below for informational purposes only; inclusion of a code does not constitute or imply coverage.

Coding for deep brain stimulation consists of a series of CPT codes describing the various steps of the procedure, i.e., implantation of the electrodes, implantation of the pulse generator, intra-operative monitoring and programming of the electrodes, and postoperative neuro-programming.

Test stimulation to confirm correct target site placement of the electrode array(s) and/or to confirm the functional status of the system is inherent to placement and is not separately reported as electronic analysis or programming of the neurostimulator system. Electronic analysis (95970) at the time of implantation is not separately reported.

When an existing lead is removed and replaced by a new lead, only the lead implantation code CPT 61863-61867 is reported. NCCI edits do not allow CPT 61880 (Revision or removal of intracranial neurostimulator electrodes) with CPT 61863-61867 (mutually exclusive procedures).

When an existing generator is removed and replaced by a new generator, only the generator replacement code CPT 61885 or 61886 may be assigned. NCCI edits do not allow removal of the existing generator to be coded separately. Also note that, according to NCCI policy, use of CPT code 61885 or 61886 for generator "insertion or replacement" requires placement of a new generator. When the same generator is removed and then re-inserted, "revision" code CPT 61888 is used ([NCCI Policy Manual 1/1/2026, Chapter VIII, C.16](#)).

For bilateral stimulation via implantation or replacement of two single array pulse generators, one on each side connected to a single lead, use CPT 61885 with modifier 50 for the generators plus CPT 61863 and 61864 or 61867 and 61868 for the leads.

For bilateral stimulation via implantation or replacement of one dual array pulse generator with connection to two leads, use 61886 for the generator plus 61863 and 61864 or 61867 and 61868 for the leads.

Patients may undergo several sessions of electronic analysis with or without programming to find the optimal programming parameters. Electronic analysis with programming, when performed, of cranial nerve and brain neurostimulator pulse generator/transmitters is billed using 96970, 95976, 95977, 95983, 95984.

CPT Codes 95983 and 95984 are defined for 15 minutes. According to CPT manual instructions, a unit of service is attained when the midpoint of time is passed, ie, 8 minutes. Initial or additional programming of less than 8 minutes is not reported.

Code	Description
Lead and Pulse Generator Implantation	
61863	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), 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 (eg, 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	Incision and subcutaneous placement of cranial neurostimulator pulse generator or receiver, direct or inductive coupling; with connection to two or more electrode arrays
Revision or Removal of Lead and Pulse Generator	
61880	Revision or removal of intracranial neurostimulator electrodes
61888	Revision or removal of cranial neurostimulator pulse generator or receiver
Functional Cortical Mapping	
95961	Functional cortical and subcortical mapping by stimulation and/or recording of electrodes on brain surface, or of depth electrodes, to provoke seizures or identify vital brain structures; initial hour of physician attendance
95962	Functional cortical and subcortical mapping by stimulation and/or recording of electrodes on brain surface, or of depth electrodes, to provoke seizures or identify vital brain structures; each additional hour of physician attendance (List separately in addition to code for primary procedure)
Analysis and programming	
95970	Electronic analysis of implanted neurostimulator pulse generator/transmitter (eg, contact group(s), interleaving, amplitude, pulse width, frequency [Hz], on/off cycling, burst, magnet mode, dose lockout, patient selectable parameters, responsive neurostimulation, detection algorithms, closed loop parameters, and passive parameters) by physician or other qualified health care professional; with brain, cranial nerve, spinal cord, peripheral nerve, or sacral nerve, neurostimulator pulse generator/transmitter, without programming
95983	Electronic analysis of implanted neurostimulator pulse generator/transmitter (eg, contact group[s], interleaving, amplitude, pulse width, frequency [Hz], on/off cycling, burst, magnet mode, dose lockout, patient selectable parameters, responsive neurostimulation, detection algorithms, closed loop parameters, and passive parameters) by physician or other qualified health care professional; with brain neurostimulator pulse generator/ transmitter programming, first 15 minutes face-to- face time with physician or other qualified health care professional

95984	Electronic analysis of implanted neurostimulator pulse generator/transmitter (eg, contact group[s], interleaving, amplitude, pulse width, frequency [Hz], on/off cycling, burst, magnet mode, dose lockout, patient selectable parameters, responsive neurostimulation, detection algorithms, closed loop parameters, and passive parameters) by physician or other qualified health care professional; with brain neurostimulator pulse generator/ transmitter programming, each additional 15 minutes face-to-face time with physician or other qualified health care professional (List separately in addition to code for primary procedure)
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HCPCS C-codes

HCPCS C-codes are used in Medicare Outpatient Prospective Payment System (OPPS) and Ambulatory Surgical Center (ASC) payment methodology only.

Section 1833(t)(6)(B) of the Social Security Act requires that, under the OPPS, categories of devices be eligible for transitional pass-through payments for at least two, but not more than three years. In addition, section 1833(t)(6)(B)(ii)(IV) of the Act requires that CMS create additional categories for transitional pass-through payment of new medical devices not described by existing or previously existing categories of devices.

The pass-through payment status for all of the following codes has expired. As such, no payment will be made for any of the following codes.

Even though the pass-through status has expired, the codes remain active. For OPPS billing, because charges related to packaged services are used for outlier and future rate setting, hospitals are advised to report the device category HCPCS codes on the claim whenever they are provided in the hospital outpatient setting. However, ASCs should not separately bill for packaged codes) since they are not reportable under the ASC payment system.

Code	Description
C1767	Generator, neurostimulator (implantable), non-rechargeable
C1820	Generator, neurostimulator (implantable), with rechargeable battery and charging system
C1883	Adaptor/extension, pacing lead or neurostimulator lead (implantable)
C1787	Patient programmer, neurostimulator

Device L-Codes

Code	Description
L8679	Implantable neurostimulator pulse generator, any type
L8681	Patient programmer (external) for use with implantable programmable neurostimulator pulse generator
L8686	Implantable neurostimulator pulse generator, single array, nonrechargeable, includes extension
L8687	Implantable neurostimulator pulse generator, dual array, rechargeable, includes extension
L8688	Implantable neurostimulator pulse generator, dual array, non-rechargeable, includes extension
L8689	External recharging system for battery (internal) for use with implantable neurostimulator

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Policy history

Origination date:	11/01/2000
Approval(s):	Benefits Committee: 01/2001 Technology Assessment Committee: 11/1/2000; 01/31/2006, 09/30/2009; 2/26/2014 ICD 10 CM codes mapped; 4/23/2014 correction due to ICD 10 CM implementation delay. 03/25/2015 (updated references) 03/23/2016 (removed ICD 9 codes, updated references) 04/26/2017 (updated references), 03/28/2018 (updated references), 03/27/2019 (removed termed codes, updated references), 05/27/2020 (updated criteria), 06/15/2021 (Added clarifying language related to Medicare Advantage, NaviCare and PACE under policy section), 07/02/2024 (annual review, adopter InterQual® Criteria), 7/2/2024 (annual review, updated Medicare regulatory language in Policy section, adopted InterQual Criteria, added Summary of Evidence and Analysis of Evidence, updated References), 06/24/2025 (annual review; added new sections for Medicare and MassHealth Variation; no changes to coverage criteria), 07/28/2026 (annual review, no changes to coverage criteria). Utilization Management Committee: 07/15/2025: (annual review, approved with no changes to coverage criteria), 08/18/2026 (annual review, approved with no changes to coverage criteria).

Instructions for Use

Fallon Health complies with CMS's national coverage determinations (NCDs), local coverage determinations (LCDs) of Medicare Contractors with jurisdiction for claims in the Plan's service area, and applicable Medicare statutes and regulations when making medical necessity determinations for Medicare Advantage members. When coverage criteria are not fully established in applicable Medicare statutes, regulations, NCDs or LCDs, Fallon Health may create internal coverage criteria under specific circumstances described at § 422.101(b)(6)(i) and (ii).

Fallon Health generally follows Medical Necessity Guidelines published by MassHealth when making medical necessity determinations for MassHealth members. In the absence of Medical Necessity Guidelines published by MassHealth, Fallon Health may create clinical coverage criteria in accordance with the definition of Medical Necessity in 130 CMR 450.204.

For plan members enrolled in NaviCare, Fallon Health first follows CMS's national coverage determinations (NCDs), local coverage determinations (LCDs) of Medicare Contractors with jurisdiction for claims in the Plan's service area, and applicable Medicare statutes and regulations when making medical necessity determinations. When coverage criteria are not fully established in applicable Medicare statutes, regulations, NCDs or LCDs, or if the NaviCare member does not meet coverage criteria in applicable Medicare statutes, regulations, NCDs or LCDs, Fallon Health then follows Medical Necessity Guidelines published by MassHealth when making necessity determinations for NaviCare members.

Each PACE plan member is assigned to an Interdisciplinary Team. PACE provides participants with all the care and services covered by Medicare and Medicaid, as authorized by the interdisciplinary team, as well as additional medically necessary care and services not covered by Medicare and Medicaid. With the exception of emergency care and out-of-area urgently needed care, all care and services provided to PACE plan members must be authorized by the interdisciplinary team.

Not all services mentioned in this policy are covered for all products or employer groups. Coverage is based upon the terms of a member's particular benefit plan which may contain its own specific provisions for coverage and exclusions regardless of medical necessity. Please consult the product's Evidence of Coverage for exclusions or other benefit limitations applicable to this service or supply. If there is any discrepancy between this policy and a member's benefit plan, the provisions of the benefit plan will govern. However, applicable state mandates take precedence with respect to fully insured plans and self-funded non-ERISA (e.g., government, school boards, church) plans. Unless otherwise specifically excluded, federal mandates will apply to all plans.