

# Deep Brain and Cortical Stimulation

**Policy Number:** 2026T0321LL  
**Effective Date:** February 1, 2026

[➔ Instructions for Use](#)

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<b>Related Commercial/Individual Exchange Policy</b>
• <a href="#">Vagus and External Trigeminal Nerve Stimulation</a>
<b>Community Plan Policy</b>
• <a href="#">Deep Brain and Cortical Stimulation</a>

## Application

### UnitedHealthcare Commercial

This Medical Policy applies to UnitedHealthcare Commercial benefit plans.

### UnitedHealthcare Individual Exchange

This Medical Policy applies to Individual Exchange benefit plans.

## Coverage Rationale

[➔ See Benefit Considerations](#)

**Deep brain stimulation is proven and medically necessary for treating the following indications:**

- Dystonia
- Essential tremor
- Parkinson disease
- Refractory epilepsy for a partial or focal seizure disorder

**Responsive cortical stimulation is proven and medically necessary for treating refractory partial or focal seizure disorder.**

For medical necessity clinical coverage criteria for deep brain and responsive cortical stimulation, refer to the InterQual® CP: Procedures, Stereotactic Introduction, Subcortical or Cortical Electrodes.

[Click here to view the InterQual® criteria.](#)

**The following are unproven and not medically necessary due to insufficient evidence of efficacy:**

- Deep brain stimulation and/or cortical stimulation for treating obsessive-compulsive disorder and all other indications
- Responsive cortical stimulation for treating all other indications

## Medical Records Documentation Used for Reviews

Benefit coverage for health services is determined by the member specific benefit plan document and applicable laws that may require coverage for a specific service. Medical records documentation may be required to assess whether the member meets the clinical criteria for coverage but does not guarantee coverage of the service requested; refer to the guidelines titled [Medical Records Documentation Used for Reviews](#).

### Applicable Codes

The following list(s) of procedure and/or diagnosis codes is provided for reference purposes only and may not be all inclusive. Listing of a code in this policy does not imply that the service described by the code is a covered or non-covered health service. Benefit coverage for health services is determined by the member specific benefit plan document and applicable laws that may require coverage for a specific service. The inclusion of a code does not imply any right to reimbursement or guarantee claim payment. Other Policies and Guidelines may apply.

CPT Code	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
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 (e.g., 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 (e.g., thalamus, globus pallidus, subthalamic nucleus, periventricular, periaqueductal gray), with use of intraoperative microelectrode recording; each additional array (List separately in addition to primary procedure)
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 2 or more electrode arrays
61889	Insertion of skull-mounted cranial neurostimulator pulse generator or receiver, including craniectomy or craniotomy, when performed, with direct or inductive coupling, with connection to depth and/or cortical strip electrode array(s)
61891	Revision or replacement of skull-mounted cranial neurostimulator pulse generator or receiver with connection to depth and/or cortical strip electrode array(s)
64999	Unlisted procedure, nervous system

*CPT® is a registered trademark of the American Medical Association*

HCPCS Code	Description
L8679	Implantable neurostimulator, pulse generator, any type
L8680	Implantable neurostimulator electrode, each
L8682	Implantable neurostimulator radiofrequency receiver
L8685	Implantable neurostimulator pulse generator, single array, rechargeable, includes extension
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, nonrechargeable, includes extension

## Description of Services

### Deep Brain Stimulation

Deep brain stimulation (DBS) delivers electrical pulses to select areas of the brain (e.g., internal globus pallidus interna, subthalamic nucleus, ventral intermediate nucleus of the thalamus) via surgically implanted electrodes. The mechanism of action is not completely understood, but the goal of DBS is to interrupt the pathways responsible for the abnormal movements that are associated with movement disorders such as Parkinson disease and essential tremor. The exact location of electrodes depends on the type of disorder being treated, and unlike standard surgical ablation, which causes permanent destruction of the targeted area, DBS is reversible and adjustable. The DBS device consists of an implantable pulse generator or neurostimulator, implantable lead with electrodes, and connecting wire. The neurostimulator is approximately the size of a stopwatch and is similar to a cardiac pacemaker. Subcutaneous extension wires connect the lead(s) to the neurostimulator, which is implanted near the clavicle or, in the case of younger individuals with primary dystonia, in the abdomen.

### Responsive Cortical Stimulation (Closed-Loop Implantable Neurostimulator)

The RNS<sup>®</sup> System (NeuroPace, Inc.) is intended to detect abnormal electrical brain signals that precede seizures and deliver electrical stimulation in response to try to normalize electrical brain activity and prevent seizures before they fully develop. The device includes a neurostimulator that is placed in the skull and leads that are placed in the seizure-originating areas of the brain. The system's intended benefits include seizure prevention, fewer adverse events than other neurostimulation methods, and data transmission from the individual's home to clinicians.

## Clinical Evidence

### Deep Brain Stimulation

#### *Obsessive-Compulsive Disorder*

There is insufficient evidence to support the use of deep brain and cortical stimulation for obsessive-compulsive disorder (OCD) due to study limitations. Larger studies are needed to establish safety, efficacy, and long-term outcomes.

Cohen et al. (2025) conducted a systematic review and meta-analysis to evaluate the efficacy and safety of deep brain stimulation (DBS) for treatment-refractory OCD using individual participant data from nine randomized, sham-controlled trials involving 91 patients. The analysis revealed that DBS significantly reduced OCD symptoms, with an average decrease of 5.1 points on the Yale-Brown Obsessive-Compulsive Scale (YBOCS) compared to sham treatment, and an odds ratio of 4.7 for clinical response. Trials that used gradual, patient-specific optimization of stimulation parameters showed greater efficacy. However, the overall quality of evidence was rated low due to study limitations that included small sample sizes and methodological variability. Adverse events, including hypomania and cognitive issues, were reported during both active and sham phases, highlighting the need for careful monitoring. The authors concluded that while DBS shows promise for severe OCD, further high-quality, sham-controlled studies are necessary to strengthen the evidence base.

Mazzoleni et al. (2023) performed a systematic review that aimed to identify relevant guidelines and assess their recommendations for the use of DBS in OCD. The second aim was to determine whether treatment recommendations were adapted to individual traits, such as age, gender, and other comorbidities. Of 532 papers, nine guidelines were identified. Three guidelines scored > 80% on AGREE II. "Scope and purpose" and "editorial independence" were the highest-scoring domains, but "applicability" scores were low. Eight guidelines recommended that DBS be used after all other treatment options have failed to alleviate OCD symptoms. One guideline did not recommend DBS beyond a research setting. The other eight did not provide details on safe or effective DBS protocols. The authors noted that while the articles supported the use of DBS for OCD as a last line of therapy, there was a lack of information on many aspects of treating DBS. They indicated that further high-quality studies are needed before DBS can be a generalized treatment for OCD.

In a recent systematic review and meta-analysis, Gadot et al. (2022) assessed the efficacy of DBS in alleviating OCD and comorbid depressive symptoms across targets in individuals with treatment-resistant obsessive-compulsive disorder (TROCD). Authors included studies that reported primary data on multiple individuals who received DBS therapy, with outcomes reported through the Y-BOCS. Primary effect measures included Y-BOCS mean difference and percent reduction as well as responder rate ( $\geq 35\%$  Y-BOCS reduction) at the last follow-up. Secondary effect measures included standardized depression scale reduction. Overall, 34 studies from 2005 to 2021, nine randomized controlled trials (RCTs;  $n = 97$ ) and 25 non-RCTs ( $n = 255$ ), were included in the systematic review and meta-analysis based on available outcome data. A random-effects model indicated a meta-analytical average 14.3-point or 47% reduction ( $p < 0.01$ ) in Y-

BOCS scores, without a significant difference between RCTs and non-RCTs. At the last follow-up, 66% of individuals were full responders to DBS therapy. Sensitivity analyses indicated a low likelihood of a small study effect bias in reported outcomes. Secondary analysis revealed a 1 standardized effect size (Hedges *g*) reduction in depressive scale symptoms. While these results are encouraging, it is important to remember that DBS does not go without limitations. The main limitation is that DBS requires chronic implantation of hardware and carries the risk of complications. The authors noted that the discoveries support DBS as an effective treatment for TROCD, and the average appropriately selected individual who experiences OCD has a 50% decrease in symptoms. Two-thirds of individuals will achieve at least a full response to DBS therapy with continued follow-up. Stimulation of current limbic and nonlimbic targets can provide considerable relief of comorbid depressive symptoms in TROCD. The rising evidence base that reports DBS for OCD outcomes reveals a predominantly low risk of bias across studies. Upcoming crossover RCTs should aim to consistently include washout periods between active and sham stimulation periods, while observational and open-label clinical studies should aim to minimize potential confounders of treatment response and maintain longer follow-up protocols.

In a randomized, double-blinded, sham-controlled trial, Mosley et al. (2021) investigated the effects of DBS at the bed nucleus of the stria terminalis in a sample of nine Australian participants (mean age, 47.9 ±10.7 years) with severe, treatment-resistant OCD. After a 1-month postoperative recovery phase, participants entered a 3-month, randomized phase during which their stimulators were either turned on or remained switched off. After this, participants entered a 12-month, open-label stimulation phase that incorporated a course of cognitive behavior therapy. The primary outcome measure was OCD symptom severity, as assessed by Y-BOCS score. In the blinded phase, there was a significant benefit of active stimulation over sham ( $p = 0.025$ ; mean difference, 4.9 points). One participant developed an acute implantation effect that was assessed by a reduction in the intensity of obsessive thoughts for 72 hours post operation before returning to baseline. One participant did not reach the target amplitude of 4.5 volts during the blinded phase due to mild agitation at higher amplitudes, but due to a robust observed symptom reduction, a lower amplitude was selected for chronic stimulation. One participant had a placebo response to sham stimulation, with a 20% reduction in Y-BOCS. After the open phase, the mean reduction in Y-BOCS was 17.4 ±2.0 points [ $\chi^2(11) = 39.9$ ;  $p = 3.7 \times 10^{-5}$ ], with seven participants classified as responders. The addition of cognitive behavior therapy resulted in a further Y-BOCS reduction of 4.8 ±3.9 points ( $p = 0.011$ ). Nine serious adverse effects occurred and affected four participants. Five of these nine were from one participant who was a non-responder and required hospitalization for persistent psychiatric symptoms. Two serious adverse events occurred that were related to the DBS device, the most severe of which was an infection during the open phase, necessitating device removal. The other device-related serious adverse event required resisting of a DBS electrode that migrated from target implantation. No serious psychiatric adverse events related to stimulation were observed. All participants required replacement of the implantable generator due to battery depletion during the study. The authors noted that while this is a promising treatment for severe, resistant OCD, the small sample size was a limitation of the trial, although it is consistent with other clinical trials of DBS for treatment-resistant psychiatric indications. The study is also limited by the short duration of its blinded phase and lack of long-term follow-up.

Mar-Barrutia et al. (2021) conducted a systematic review to summarize the existing knowledge on the efficacy and tolerability of DBS in treatment-resistant OCD and to compare the short-term and long-term results. A comprehensive search was conducted in the PubMed, Cochrane, Scopus, and ClinicalTrials.gov databases from start to December 31, 2020. Inclusion criteria included a main diagnosis of OCD, DBS conducted for therapeutic purposes, and variation in symptoms of OCD measured by the Y-BOCS as a primary outcome. Overall, 40 articles identified by the search strategy met the eligibility criteria to include 344 individuals. Applying a follow-up threshold of 36 months, 29 studies (with 230 individuals) provided information on short-term response to DBS, while 11 (with 155 individuals) reported results on long-term response. The mean follow-up period was 18.5 ±8.0 months in the short-term studies and 63.7 ±20.7 months in the long-term studies. Overall, the percentage of reduction in Y-BOCS scores was similar in short-term (47.4%) and long-term responses (47.2%) to DBS, but more individuals in the long-term reports met the criteria for response (defined as a reduction in Y-BOCS scores of > 35%: short term, 60.6% vs long term, 70.7%). According to the results, the first year predicted the extent to which an individual with OCD benefited from DBS since the maximum symptom reduction was achieved in most responders in the first 12 to 14 months after implantation. Reports indicated a consistent tendency for this early improvement to be maintained to the mid-term for most individuals, but it is still debatable whether this improvement continues, increases, or decreases in the long term. Three different patterns of long-term response occurred from the analysis: 49.5% of individuals had good and sustained response to DBS, 26.6% were nonresponders, and 22.5% were partial responders, who might improve at some point but experience relapses during follow-up. There was an improvement in depressive symptoms, and global functionality was observed in most studies, usually corresponding with an improvement in obsessive symptoms. Most adverse effects with DBS were mild and transient and improved after adjusting stimulation parameters; however, some severe adverse events occurred, including intracranial hemorrhages and infections. Hypomania was the most frequently reported psychiatric side effect. The relationship between DBS and suicide risk remains controversial and requires further study. There are no clear clinical or biological predictors of response that can be recognized, likely due to the differences between studies related to neuroanatomical targets and the stimulation protocols assessed. In conclusion, the authors indicated that DBS is a promising therapy for individuals with

severe, resistant OCD, providing both short-term and long-term evidence of efficacy. Many unknowns remain, including the optimal anatomical targets, criteria for standardized stimulation protocols, and identification of biomarkers or factors that predict outcomes and allow treatment individualization. Larger, more robust studies are needed to evaluate this technology to better determine the unknowns presented in this review.

Hageman et al. (2021) performed a meta-analysis that compared the clinical outcomes of the ablative procedures capsulotomy and cingulotomy and DBS. Ablative surgery (ABL) and DBS are last-resort treatment options for individuals with treatment-refractory OCD. A PubMed search was used to identify all clinical trials on capsulotomy, cingulotomy, and DBS. Random-effects meta-analyses were performed on 38 articles, with a primary focus on efficacy in reducing OCD symptoms, as measured by a reduction in Y-BOCS score, and the responder rate ( $\geq 35\%$  reduction in Y-BOCS score). With responder rates of 48% and 53% after 12 to 16 months and 56% and 57% at the last follow-up for ABL and DBS, respectively, and large effect sizes in the reduction in YBOCS scores, both surgical modalities showed effectiveness in treating refractory OCD. Meta-regression did not show a statistically significant difference between ABL and DBS regarding these outcomes. Regarding adverse events, a statistically significant higher rate of impulsivity was reported in studies on DBS. This meta-analysis shows equal efficacy of ABL and DBS in the treatment of refractory OCD. For now, the choice of intervention should, therefore, rely on factors such as risk of developing impulsivity, individuals' preferences, and the experience of the psychiatrist and neurosurgeon. Additional research is needed to provide a better understanding of the differences between ABL and DBS and response prediction following direct comparisons between the surgical modalities to enable personalized and valid choices between ABL and DBS. The safety and efficacy of these techniques must be studied more thoroughly before wider clinical application.

In a 2021 (updated 2022) report, Hayes evaluated the use of DBS for the treatment of refractory OCD. An overall low-quality body of evidence suggests that the effectiveness of DBS for the treatment of highly refractory OCD remains uncertain, despite several double-blinded, crossover trials. Despite its favorable results, the sample sizes were very low; there were no studies that compared DBS with an alternate intervention; and treatment planning was highly individualized with trial phases, with included considerable heterogeneity. Additional studies that are sufficiently driven, with consistent reporting of nonprimary outcome measures and long-term follow-up, would help to inform whether DBS offers any sustained benefit to individuals with refractory OCD. Specifically, studies that compare DBS with clinical alternatives in a non-crossover design would help to inform whether DBS is a viable treatment option (Hamani et al., 2014, included in this report).

Vázquez-Bourgon et al. (2019) systematically reviewed the literature to identify the main characteristics of DBS, its use, and applicability as treatment for OCD. According to the authors, the critical analysis of the evidence showed that the use of DBS in treatment-resistant OCD is providing satisfactory results regarding efficacy, with assumable side effects. However, there is insufficient evidence to support the use of any single brain target over another. Selection of individuals has to be completed following analyses of risks/benefits, being advisable to individualize the decision of continuing with concomitant psychopharmacological and psychological treatments. The authors concluded that the use of DBS is still considered to be in the field of research, although it is increasingly used in refractory OCD, producing in the majority of studies significant improvements in symptomatology and in functionality and quality of life. Random and controlled studies need to be done to determine its long-term efficacy.

Rapinesi et al. (2019) conducted a systematic review to assess the effect of brain stimulation techniques in OCD. DBS showed best results when targeting the crossroad between the nucleus accumbens and the ventral capsule or the subthalamic nucleus. The authors concluded that different brain stimulation techniques are promising as an add-on treatment for refractory OCD, although studies frequently reported inconsistent results. DBS could possibly find some use with adequate testing, but its standard methodology still needs to be established. The authors indicated that the review was limited because of the inclusion of methodologically inconsistent, underpowered studies.

In a systematic review, Naesström et al. (2016) reviewed the current studies on psychiatric indications for DBS, with a focus on OCD and major depressive disorder (MDD). A total of 52 studies met the inclusion criteria, with a total of 286 unique individuals treated with DBS for psychiatric indications; 18 studies described 112 individuals treated with DBS for OCD in six different anatomical targets, while nine studies included 100 individuals with DBS for MDD in five different targets. The authors concluded that DBS may show promise for treatment-resistant OCD and MDD, but the results are limited by a small sample size and insufficient randomized controlled data. According to the authors, other psychiatric indications are currently of a purely experimental nature.

Hamani et al. (2014) conducted a systematic review of the literature and developed evidence-based guidelines on DBS for OCD that was sponsored by the American Society for Stereotactic and Functional Neurosurgery and the Congress of Neurological Surgeons and endorsed by the Congress of Neurological Surgeons and American Association of Neurological Surgeons. Of the 353 articles identified, seven were retrieved for full-text review and analysis. The quality of

the articles was assigned to each study, and the strength of recommendation was graded according to the guideline's development methodology of the American Association of Neurological Surgeons/Congress of Neurological Surgeons Joint Guidelines Committee. Of the seven studies, one class I and two class II double-blinded RCTs reported that bilateral DBS is more effective in improving OCD symptoms than sham treatment. The authors concluded that based on the data published in the literature, the following recommendations can be made: (1) there is level I evidence, based on a single class I study, for the use of bilateral subthalamic nucleus DBS for the treatment of medically refractory OCD; (2) there is level II evidence, based on a single class II study, for the use of bilateral nucleus accumbens DBS for the treatment of medically refractory OCD; and (3) there is insufficient evidence to make a recommendation for the use of unilateral DBS for the treatment of medically refractory OCD. The authors noted that additional research is needed to determine which patients respond to deep brain stimulation and if specific targets may be more suitable to treat a specific set of symptoms.

## Clinical Practice Guidelines

### *National Institute for Health and Care Excellence (NICE)*

- Evidence on the safety and efficacy of DBS for chronic, severe, treatment-resistant OCD in adults is inadequate in quality and quantity. Therefore, this procedure should only be used in the context of research.
- Patient selection should be done by a multidisciplinary team that is experienced in managing OCD. It should include experts in psychiatry, neuropsychiatry, clinical psychology, neurology, neurosurgery, and DBS.
- The procedure should only be done in centers with expertise in DBS and experience in managing OCD.
- Further research should primarily be RCTs. It should clearly define the area of the brain that should be targeted in this procedure. It should also describe details of patient selection, comorbidities, and use of adjunctive therapies. Outcomes should include reduction in OCD symptoms, improvement in quality of life, and any neuropsychiatric and cognitive effect.  
(NICE, April 28, 2021)

## Responsive Cortical Stimulation

There is insufficient evidence to support responsive cortical stimulation for treating indications other than partial or focal seizure disorders due to the lack of clinical studies. Large, well-designed studies are needed to establish safety, efficacy, and long-term outcomes.

Bronte-Stewart et al. (2025) evaluated the long-term safety, tolerability, and efficacy of personalized adaptive deep brain stimulation (aDBS) in individuals with Parkinson's disease who were previously stable on continuous DBS (cDBS). The study enrolled 68 participants across multiple international sites and compared two closed-loop aDBS modes—single-threshold and dual-threshold—using a crossover design. Both modes adjusted stimulation based on neural activity in the 8–30 Hz range. Results showed that aDBS was well tolerated and comparably effective to cDBS in maintaining “on-time” without troublesome dyskinesia. Notably, 91% of participants using dual-threshold aDBS and 79% using single-threshold aDBS met the revised performance goal. Additionally, aDBS demonstrated energy efficiency and was preferred by most participants, with nearly all opting to continue aDBS during the 10-month follow-up. While the ADAPT-PD trial demonstrated promising results for personalized adaptive deep brain stimulation (aDBS) in Parkinson's disease, several limitations should be considered. First, the study was nonrandomized and open label, which may introduce bias and limit the strength of causal inferences. The trial also enrolled participants who were already stable on continuous DBS (cDBS), potentially excluding individuals with more variable or refractory symptoms, thereby limiting generalizability. Additionally, while the study used a crossover design for comparing single- and dual-threshold aDBS modes, not all participants were able to tolerate both modes, resulting in uneven group sizes and potential selection bias. The reliance on self-reported outcomes and clinician assessments without blinding may also affect objectivity. Finally, although the trial showed good tolerability and efficacy over 10 months, longer-term data are needed to fully understand durability, safety, and potential adverse effects of aDBS over years of use.

Sasaki et al. (2021) explored a closed-loop programming approach for deep brain stimulation (DBS) in Parkinson's disease using external motion sensor data to guide stimulation settings. In a randomized, double-blind, crossover trial involving 12 patients with bilateral subthalamic nucleus implants, the closed-loop algorithm (CLA) was compared to standard-of-care programming. Both methods improved motor symptoms, but CLA required fewer programming steps, indicating greater efficiency. However, the study's small sample size, short duration, and lack of long-term follow-up limit its generalizability and ability to assess sustained benefits. Additionally, CLA did not outperform standard programming in symptom control, suggesting further research is needed to validate its clinical advantage.

## U.S. Food and Drug Administration (FDA)

This section is to be used for informational purposes only. FDA approval alone is not a basis for coverage.

For information on deep brain stimulation devices, refer to the following website (use product codes MHY, NHL, OLM, and OLX): <http://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMN/pmn.cfm>. (Accessed August 18, 2025)

For information on responsive cortical devices, refer to the following website (use product code PFN): <https://www.accessdata.fda.gov/scripts/cdrh/cfdocs/cfPMA/pma.cfm>. (Accessed August 18, 2025)

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## Policy History/Revision Information

Date	Summary of Changes
02/01/2026	<p data-bbox="337 201 613 233"><b>Coverage Rationale</b></p> <ul data-bbox="337 233 1482 327" style="list-style-type: none"><li data-bbox="337 233 1482 327">• Added language to clarify deep brain stimulation and/or cortical stimulation are unproven and not medically necessary for treating obsessive-compulsive disorder (OCD) and all other indications [not listed in the policy as proven and medically necessary]</li></ul> <p data-bbox="337 327 662 359"><b>Supporting Information</b></p> <ul data-bbox="337 359 1382 449" style="list-style-type: none"><li data-bbox="337 359 1382 422">• Updated <i>Clinical Evidence</i>, <i>FDA</i>, and <i>References</i> sections to reflect the most current information</li><li data-bbox="337 422 943 449">• Archived previous policy version 2026T0321KK</li></ul>

## Instructions for Use

This Medical Policy provides assistance in interpreting UnitedHealthcare standard benefit plans. When deciding coverage, the member specific benefit plan document must be referenced as the terms of the member specific benefit plan may differ from the standard plan. In the event of a conflict, the member specific benefit plan document governs. Before using this policy, check the member specific benefit plan document and any applicable federal or state mandates. UnitedHealthcare reserves the right to modify its Policies and Guidelines as necessary. This Medical Policy is provided for informational purposes. It does not constitute medical advice.

This Medical Policy may also be applied to Medicare Advantage plans in certain instances. In the absence of a Medicare National Coverage Determination (NCD), Local Coverage Determination (LCD), or other Medicare coverage guidance, CMS allows a Medicare Advantage Organization (MAO) to create its own coverage determinations, using objective evidence-based rationale relying on authoritative evidence ([Medicare IOM Pub. No. 100-16, Ch. 4, §90.5](#)).

UnitedHealthcare may also use tools developed by third parties, such as the InterQual® criteria, to assist us in administering health benefits. UnitedHealthcare Medical Policies are intended to be used in connection with the independent professional medical judgment of a qualified health care provider and do not constitute the practice of medicine or medical advice.