Evidence-Based Strategies for Care of the Patient with Movement Disorders and Deep Brain Stimulation

Literature Review

Cynthia Bautista, Joan Miravite, Helen Coronel, Donna Hunt, and Ellen Dallow

AANN
American Association of Neuroscience Nurses

8735 W. Higgins Road, Suite 300
Chicago, IL 60631
888.557.2266
International phone: 847.375.4733
Fax: 847.375.6430
info@aann.org • www.AANN.org

Publisher's Note
The authors, editors, and publisher of this document neither represent nor guarantee that the practices described herein will, if followed, ensure safe and effective patient care. The authors, editors, and publisher further assume no liability or responsibility in connection with any information or recommendations contained in this document. These recommendations reflect the judgment from the American Association of Neuroscience Nurses regarding the state of general knowledge and practice in our field as of the date of publication and are subject to change based on the availability of new scientific information.

Abstract

Purpose: The purpose of this review of the literature is to provide nurses with evidence-based strategies to care for adult patients with Parkinson's disease, essential tremor, and dystonia and for the patient with a deep brain stimulator.

Methods: Neuroscience nurse experts performed a formal literature search of Cochrane, Guidelines.gov, PubMed, and CINAHL. They reviewed the published literature from 2010 to August 2018.

Results: The formal literature search yielded 53 articles that were reviewed. Evidence was used to develop a summary of the literature addressing key nursing management topics in caring for the patient with Parkinson's disease, essential tremor, or dystonia. Evidence was also used to develop a summary of the literature addressing key nursing management topics for the patient with a deep brain stimulator.

Conclusions: This review of the literature identified the evidence for best practices in caring for patients with movement disorders and the care of the patient with a deep brain stimulator.

Keywords: Parkinson's disease, essential tremor, dystonia, deep brain stimulator

Introduction

The purpose of this review of the literature is to provide nurses with evidence-based strategies to care for adult patients with Parkinson's disease, essential tremor, dystonia, and movement disorders requiring deep brain stimulation. The target population for this review is the adult client with Parkinson's disease, essential tremor, or dystonia. Although this review was not created for the pediatric client, many of the recommendations may apply to the pediatric client with movement disorders.

This review of the literature may be used in a variety of healthcare settings. It can be used in the acute, subacute, long-term, and community care settings. Practitioners in both inpatient and outpatient healthcare settings may find this evidence-based review useful. Key stakeholders and end users of this review should include advanced practice and staff nurses caring for clients with movement disorders in the acute, subacute, chronic, and community care areas. Both inpatient and outpatient practitioners should refer to this guideline to establish their evidence-based practice. This review of the literature can be used to guide the evidence-based care of clients with movement disorders in a variety of settings. It may be used to inform clinical decisions, inform policy, and provide standards of care.

Methods

This review of the literature was conducted by a group selected by the American Association of Neuroscience Nurses (AANN) that included a doctorally prepared neuroscience clinical nurse specialist, a neurology nurse practitioner, an assistant professor at a college of nursing, a pediatric neurology nurse coordinator, and a neuroscience clinical education specialist. This group represented the southern, eastern, and western United States of America. Before the literature searches, clinical questions were developed for the diseases included. Clinical questions were formulated using the Population, Issue/Intervention, Comparison, Outcome, and Time frame (PICOT) format to provide a structured mechanism for evidence-based literature search strategies. A search for the best evidence, reflecting the highest level of evidence available, was performed for each of the PICOT clinical questions. A formal literature search of the Cochrane, Guidelines.gov, PubMed, and CINAHL databases from 2010 to August 2018 was conducted.

The following search terms were used: Parkinson's disease, mobility, pain, fatigue, cognitive impairment, depression, cervical dystonia, generalized dystonia, movement disorders, activities of daily living, neuropsychiatric manifestations, nursing, essential tremor, and deep brain stimulation. Evidence was used to develop a summary of the literature addressing key nursing management topics for Parkinson's disease, dystonia, essential tremor, and care of the patient with a movement disorder with a deep brain stimulator.

Parkinson's Disease

Background

Parkinson's disease (PD) is a chronic, progressive neurodegenerative disease that causes characteristic motor symptoms of tremor, bradykinesia, and postural instability. PD affects 1% to 2% of adults over age 65 and 4% of adults over age 80. This number is expected to grow by approximately 50% by 2030 because of rising life expectancy and an aging population (Parkinson's Foundation, 2018). Males are affected more often than females at a 3:2 ratio. PD is caused by deterioration of the dopaminergic neurons in the extrapyramidal tract of the midbrain. The extrapyramidal nerve tract modulates voluntary movements and controls maintenance of posture and coordination of gait. The tract also influences autonomic activity, sequencing of movements, and habitual activities. It is unknown what triggers the initiation of PD, but it is thought to be a combination of genetic and environmental factors. As the disease progresses to late-stage PD, medication resistance is a major problem. Patients can suffer from freezing of gait with risk of falls and dysphagia. Dementia is a late manifestation, and along with falls it is a common reason for long-term care (Parkinson's Foundation, 2018).

Cardinal primary motor symptoms of PD are tremor at rest, rigidity, bradykinesia, and postural instability (Nolden, Tartavoulle, & Porche, 2014). Gait and balance impairment are common and disabling manifestations of PD. Postural stability and walking capacity decline with disease progression (Cavanaugh et al., 2016; Curtze, Nutt, Carlson-Kuhta, Mancini, & Horak, 2016; Kang & Ellis-Hill, 2015; Nakae & Tsushima, 2014; Rinalduzzi et al., 2015). Nonmotor symptoms of PD include anxiety, depression, cognitive impairment, pain, and fatigue. The revised Movement Disorder Society Unified Parkinson's Disease Rating Scale (MDS-UPDRS) is the most common assessment tool to measure PD progression. It is administered in four parts. It can be administered in whole or part. Estimated time to administer the full scale is 30 minutes (Goetz et al., 2008). Each part has a separate focus, and, when taken together, they create a whole patient picture of motor, nonmotor, and treatment complications. Motor symptoms most associated with decreased quality of life (QOL) are fear of falling and ability to maintain usual activities (Curtze et al., 2016; Kang & Ellis-Hill, 2015; Nakae & Tsushima, 2014). Assessing the mobility of patients with PD when they are off their medications

corresponds better with patient perceptions of mobility disability and balance confidence (Curtze et al., 2016). A descriptive study of 104 patients found that turning speed, gait speed, and stride length corresponded most with both disease severity and patient perceptions of disability (Curtze et al., 2016). A longitudinal study of 17 patients over 2 years reported that higher baseline activity levels correlated with higher mean daily steps. Gait and balance impairment may be delayed or slowed in patients who are physically active before these symptoms develop. Baseline activity level at the time of diagnosis may be predictive of activity levels as the disease progresses (Cavanaugh et al., 2016). A cross-sectional study with 10 participants showed that once motor symptoms were present, overall activity level was not increased by home exercises (Nakae & Tsushima, 2014).

Mild cognitive impairment (MCI) is a common finding early in the disease process (Adwani, Keshav, Chandra, & Pal, 2016; Foster, 2014; Hobson & Meara, 2015; Koster, Higginson, & MacDougall, 2015; Pistacchi, Gioulis, Contin, Sanson, & Marsala, 2015). One longitudinal cohort study demonstrated an 11% annual conversion rate of those with MCI to Parkinson's disease dementia (PDD) (Hobson & Meara, 2015). Early cognitive impairment is less amnestic than with non–PD-related cognitive impairment. Three separate case-controlled studies found that patients with PD suffer from greater attention, computation, praxis, and conceptualization deficits than memory deficits. Objective tools such as the Montreal Cognitive Assessment, Mini–Mental State Examination, and Cambridge Cognition Examination can be used to assess cognitive status (Adwani et al., 2016; Foster, 2014; Pistacchi et al., 2015). The impact of these executive impairments leads to deficits in the performance of cognitively demanding activities of daily living (ADLs) such as shopping, medication management, and meal preparation (Foster, 2014).

Depression, anxiety, suicidal risk, and psychosis are a significant comorbidity in nondemented patients with PD. Two large observational studies found that two-thirds of patients with PD have neuropsychiatric comorbidities (Fan, Chang, & Wu, 2016; Rai et al., 2015). Depression was the most common neuropsychiatric comorbidity, followed by anxiety. Psychosis was present in approximately 24%. The primary psychosis symptoms were visual and somatic hallucinations (Rai et al., 2015). Depression was more common in women and patients with higher disability. Anxiety was more prevalent in young-onset PD, but depression and psychosis were less common (Rai et al., 2015).

A systematic review of 18 studies and 15,636 patients found that 56% of patients with PD experience pain associated with PD (Ranaa, Kabirb, Jesudasana, Siddiquia, & Khondkerb, 2013). Pain reduces QOL and increases depression. The researchers agreed that the most common pain is musculoskeletal pain. Other types of pain include neuropathy, central pain, and dystonic pain. Treatment of pain related to motor symptoms or dystonia is often insufficient (Ranaa et al., 2013; Valkovi et al., 2015). One systematic review found that pain improves when the patient is taking medications or after deep brain stimulation (DBS) but that not all PD-related sensory abnormalities are motor related. This suggests that there might be different mechanisms for motor and nonmotor symptoms in PD (Cury et al., 2016).

An integrative review examining 10 years of research on fatigue and PD states that 58% of patients with PD experience fatigue (Bruno & Sethares, 2015). This examination of 19 studies found that fatigue is difficult to measure because of challenges defining fatigue. As with pain, fatigue becomes greater as the disease progresses. Fatigue in PD can be divided into two types: central and peripheral. Several fatigue assessment scales have shown reliability and validity with the PD population. According to Bruno and Sethares (2015), none have proven to be superior to assess both mental and physical fatigue in PD.

Treatment Overview

Medical Management

PD is not curable. Currently, there is no therapy that can reverse, slow, or stop disease progression. Treatment goals are aimed at maintaining functional status and QOL (Morgan & Fox, 2016). Therapy can be divided into pharmacological management (e.g., medications) and surgical management (e.g., DBS) of motor symptoms.

Medical management for the initial treatment of PD motor symptoms includes dopamine replacement with carbidopa/levodopa, dopamine agonists, and monoamine oxidase B (MAO-B) inhibitors (Nolden et al., 2014). Drug selection is influenced by disease severity, patient age, and adverse effect profile. Treatment of PD is multifaceted and requires an individually tailored approach using pharmacological and nonpharmacological treatments (Morgan & Fox, 2016). Dopamine replacement remains the most effective treatment for the cardinal motor symptoms of tremor, rigidity, and bradykinesia. Benefits of levodopa treatment include a smooth and even improvement in motor symptoms. With disease progression, the effects of levodopa weaken and leave the patient vulnerable to motor fluctuations. Motor fluctuations are characterized by wearing off of medications earlier than anticipated or a sudden switch from an "on" medication state, where symptoms are controlled, to an "off" state, where symptoms return (Khan, 2012). With progression of disease, over time the effect of dopamine weakens, creating the

need for dosage adjustments. Delayed gastric emptying can cause delays in the "on" (medication) state or "no-ons" or dosage failures (Bhidayasiri et al., 2015). Other adverse effects of dopamine replacement are somnolence, confusion, hallucinations, orthostatic hypotension, and dyskinesias.

Levodopa-induced dyskinesias (LIDs) are excessive muscular activity that may interfere with normal movement control. LIDs include chorea–ballism, dystonia, myoclonus, tics, and tremors. Approximately 80% of patients treated with levodopa experience motor complications in the first 5 years of therapy. The incidence of motor complications is more prevalent in young-onset PD (Guridi, Gonzalez-Redondo, & Obeso, 2012). LIDs can be defined by timing of the presentation in relation to levodopa activity. "Peak dose" or "on" period dyskinesia is related to high levels of levodopa and parallel maximal therapeutic benefit. This dyskinesia is typically characterized by choreic movements of the neck, arms, and trunk. Diphasic dyskinesia appears at the onset and offset of the levodopa effect. This dyskinesia is characterized by slow, repetitive tremor at 4 Hz in the upper limbs and legs. In severe cases, the tremors of the legs can be replaced with ballism that severely interferes with gait. "Off" period dyskinesia is characterized by fixed and painful dystonia that typically affects the feet (Guridi et al., 2012). Once LIDs occur, amantadine, an NMDA inhibitor, is usually added (Guridi et al., 2012, Kahn, 2012, Nolden et al., 2014). Amantadine has been demonstrated to reduce LID severity by 60% without exacerbation of motor dysfunction (Kahn, 2012). For patients in late disease with "off" period dyskinesias, difficulty swallowing, or delayed gastric emptying, levodopa/carbidopa intestinal gel (LCIG) delivered by external pump is a viable option. Studies have found positive reduction in "off" time and significant improvement of dyskinesia. A major drawback of LCIG is that it requires placement of a percutaneous endoscopic jejunostomy tube (Lopiano et al., 2016).

The use of dopamine agonists as a monotherapy is less effective in treating the motor symptoms of PD but delays the development of LID. Ergot-derived dopamine agonists have the additional risk of developing pulmonary fibrosis and have been withdrawn from the market. Non-ergot dopamine agonists, including Pramipexole, Ropinirole, Rotigotine, and Apomorphine, have a similar adverse-event profile similar to that of levodopa: nausea, vomiting, daytime sleepiness, hallucinations, and orthostatic hypotension (Morgan & Fox, 2016; Nolden et al., 2014). MAO-B inhibitors such as Selegiline, Rasagiline, or Safinamide are used as combination therapy in later disease and monotherapy in early disease. These drugs increase the risk of serotonin syndrome, especially in combination with tricyclic antidepressants (Khan, 2012; Nolden et al., 2014). Combination therapy of levodopa with MAO-B inhibitors is useful in reducing "off" state symptoms or wearing off of medications (Kahn, 2012).

The limitations of dopamine replacement in late disease can be managed with intermittent or continuous subcutaneous apomorphine injection. Intermittent administration is best achieved with a dose-dialed pen. Continuous Apomorphine infusion via pump is not currently available in the United States (Bhidayasiri et al., 2015). Candidates for pen administration are patients needing rescues during "off" periods or treatment for early morning motor problems and patients with gastroparesis resulting in delayed "on." A current clinical trial using continuous delivery of apomorphine via pump has shown promise for patients who need frequent rescue doses, for patients whose carbidopa/levodopa therapy is limited by LIDs, to improve compliance and convenience for complex medication regimes, and as an alternative to LCIG or DBS (Trenkwalder et al., 2015). Exclusion criteria for continuous subcutaneous infusion of apomorphine are patients with severe or complex patterns of dyskinesias, patients with dementia, or severe psychiatric adverse reactions associated with dopamine agonists. Mild dementia and hallucinations are not contraindications if apomorphine is controlled. Apomorphine has shown improved neuropsychiatric symptoms and verbal fluency over DBS.

Catechol-o-methyl transferase (COM-T) inhibitors such as Entacapone and Tolcapone allow more levodopa to reach the central nervous system to be converted into dopamine after crossing the blood brain barrier. Anticholingergics such as Trihexyphenidyl and Benztropine can be used to treat tremor (Nolden et al., 2014) (see **Table 1**).

Functional disability including cognitive dysfunction can be addressed through occupational therapy (OT). Screenings should be performed by therapists to determine which patients are most likely to benefit and from which therapy. Home-based occupational therapy can lead to improvement in ADL performance and improved QOL. When delivered in the home, OT is more able to individualize care in context (Sturkenboom et al., 2014). Cognitive training in addition to traditional OT has demonstrated significant improvement in processing, visual memory, and functional disability (Pena et al., 2014). Patients unable to participate in outpatient cognitive training might benefit from nonspecific cognitive training using motion-controlled computer games in their home (Zimmerman et al., 2014). Entertaining activities such as group exercise may perform equal to structured outpatient rehabilitation (Foster, 2014; Foster, Golden, Duncan, & Earhart, 2013; Lee, Lee, & Song, 2015; Li et al., 2013; Uc et al., 2014; Zimmerman et al., 2014).

Table 1. Parkinson's Disease Medications

Therapeutic Class and Medication	Daily dose	Side Effect Profile
Dopamine Precursor		
Carbidopa/Levodopa (Sinemet) ODT (orally dissolving tablet) CR or ER (extended release) Rytary (long acting) Duopa (intestinal gel) Carbidopa/Levodopa/Entacapone (Stalevo)	Individualize dose, starting with tid-qid Max dose: 200/2,000 mg/day Max dose: 200/2,000 mg/day CR or ER max dose: 600/2,400 mg/day Rytary max dose: 612.5/2450 mg Duopa max dose: 2,000 mg/day via peg Stalevo Max dose: 8 tablets/day	Nausea, dizziness, dyskinesia, orthostatic hypotension, hallucinations, compulsive behavior, vivid dreams
Dopamine Agonists		
Pramipexole (Mirapex) Pramipexole (Mirapex ER) Ropinirole (Requip) Ropinirole (Requip XL) Apomorphine (Apokyn SC inj) Bromocriptine (Parlodel) Rotigotine (Neupro transdermal)	tid, Max dose: 4.5 mg/day qd, Max dose: 2.25 mg/day bid-tid, Max dose: 24 mg/day qd, Max dose: 24 mg/day Max dose: 0.6 mL/dose, up to 5x/day tid, Max dose: 100 mg/day qd, Max dose: 8 mg/24h	Somnolence, hypotension, dizziness, hallucinations, peripheral edema, compulsive behavior, vivid dreams
NMDA Receptor Antagonist		
Amantadine (Symmetrel) Amantadine ER (Gocovri) (Osmolex ER)	bid-qid, Max dose: 400 mg/day qhs, Max dose: 274 mg qd, Max dose: 322 mg/day	Hallucinations, dizziness, peripheral edema, vivid dreams, confusion, fatigue, hypotension, livedo reticularis
MAO-B Inhibitors		
Rasagiline (Azilect) Selegiline (Eldepryl) Selegiline ODT (Zelapar) Safinamide (Xadago)	qd, Max dose: 1 mg/day bid, Max dose: 10 mg qam, Max dose: 2.5 mg/day qd, Max dose: 100 mg/day	Hypotension, headache, compulsive behaviors, dizziness, dyskinesia, hallucinations
COMT Inhibitors		
Tolcapone (Tasmar) Entacapone (Comtan, Stalevo)	Give with each Carbidopa/Levodopa dose Max dose: 600 mg/day Max dose: 1600 mg/day	(Tolcapone—hepatotoxicity) Dyskinesia, nausea, dystonia, vivid dreams, hypotension somnolence, diarrhea, confusion, dizziness, hallucinations, compulsive behaviors
Anticholinergics		
Trihexyphenidyl (Artane) Benztropine (Cogentin)	tid, Max dose: 15 mg/day PO/IM/IV qhs, bid-tid, Max dose: 6 mg/day	Dry mouth, blurry vision, dizziness, nausea, confusion, urinary retention, drowsiness, constipation

Adapted from: Nolden et al., 2014; Sharma et al., 2018;. Kriebel-Gasparro, 2016; Bette et al., 2018.

Speech therapy plays an integral role in treating patients with PD. Aspiration pneumonia due to dysphagia is a significant cause of morbidity and mortality for patients with PD. Conventional swallowing therapy can aid a patient with movement disorders in improving their swallowing, but it may not help them clear food residue from their mouth completely. Patients use repeated forceful swallows as a compensatory technique after training by a speech therapist. Video-assisted swallow therapy (VAST) provides visual feedback on the repeated forceful swallow technique. It is believed the feedback motivates patients to practice the swallow techniques and improves performance of the technique itself. Feeling safe eating and drinking has been shown to improve QOL and provide a sense of control for patients trained with VAST (Manor, Mootanah, Freud, Giladi, & Cohen, 2013). Patients with PD also seek speech therapy for the treatment of dysarthria. Dysarthria includes a combination of reduced loudness; breathy, hoarse, or harsh quality; and reduced articulation, particularly toward the end of a sentence. Speech may sound monotone, rushed, dysfluent, or hesitant (Sapir, Ramig, & Fox, 2011). The Lee Silverman voice treatment (LSVT) has demonstrated significant improvement in clinical outcomes over traditional speech therapy. LSVT was developed in late 1980s and evaluated with numerous randomized controlled trials. LSVT is PD-specific, standardized, and research based. LSVT specifically trains voice amplitude using an intense high-effort mode and recalibration of vocal effort that generalizes to outside the laboratory (Sapir et al., 2011).

Clinical Pearls

- Dopamine replacement remains the most effective medication for treating the cardinal motor symptoms of tremor, rigidity, and bradykinesia.
- Functional disability including cognitive dysfunction can be addressed through OT.
- Speech therapy plays an integral role in treating patients with PD.

Nursing Management

The goals of nursing care are to promote compliance with treatment regimens, prevent hospital readmissions and unplanned hospitalizations, and address important caregiver considerations. Because much of care provided to patients with PD is provided in the home by care partners, nursing interventions should focus on patients and their care partners.

A systematic review of nine descriptive studies found that the prevalence of significant medication noncompliance in PD varied between 10% and 67%. This variation is partly reflective of differences between the studies in defining medication adherence and noncompliance. Predictors of noncompliance are more advanced disease, poor knowledge about PD, engagement in risky behaviors such as alcohol abuse, and complex drug regimens (Malek & Grosset, 2014). Malek and Grosset found that patients had better medication adherence and understanding of medication side effects if they had simpler medication regimens or used compliance aids. A randomized controlled trial of 76 patients and 46 spouses used adherence therapy (AT) to improve medication adherence. AT incorporates keeping patients engaged, minimizing resistance, exchanging information, using dialogue to generate belief discrepancies, and identifying personally relevant benefits to treatment. Patients undergoing AT demonstrated a 60% increase in adherence, compared with 16% in the control group. QOL significantly improved for the intervention group and decreased slightly in the control group (Daley et al., 2014). Semistructured interviews conducted after the study with 10 patients and six spouses in the intervention group revealed that AT enabled an appreciation of dose timing and self-awareness of symptoms (Daley, O'Leary, Gray, Hill, & Myint, 2015).

Reducing unplanned hospitalization is a goal for patients with PD. A systematic review of 10 descriptive studies resulted in only low-level evidence of interventions that can reduce unplanned hospitalizations (Muzerengi, Herd, Rick, & Clarke, 2016). Management by a neurologist, specialty nursing care, and home care reduced total hospitalization. Care by a neurologist reduced the number of admissions for traumatic events. Patients who had access to specialty care without appointment had a 50% reduction in hospitalizations over a 2-year period (Muzerengi et al., 2016). Easy access to specialty care can be provided by a PD specialty nurse in the home or clinic. Qualitative evidence suggests that a PD specialist nurse provides individualized care and education to patients and care partners (Hellqvist & Bertero, 2015). Connecting rural patients or patients unable to travel with specialty care can be difficult. A small longitudinal observational study found that improved medication management reduced emergency room use by incorporating home visits from neurology fellows and specialty nurses (Hack et al., 2014).

A survey of 66 caregivers found that the highest caregiver needs were related to symptom management, coping with changes in lifestyle, future planning, relationships, cognition, and wellness strategies. Twenty-four percent of this group reported availability and cost as barriers to supportive care (Lageman, Mickens, & Cash,

2015). One unique intervention was a nurse-led 8-week education and telesupport group conducted by the Virginia Veterans Administration. The support model focused on education about caregiving, skill training, problem solving, and support. After intervention, interviews were conducted with the seven female participants in the program. Caregivers who participated stated that they found it helpful to talk to those in similar situations and felt less guilt about taking time to care for themselves. Success of this program demonstrates a cost-effective and patient-centered intervention to support patients with PD and caregivers in their home (Shah et al., 2015).

Patients may be admitted to the hospital with PD complications such as falls, fractures, and neuropsychiatric conditions (Muzerengi et al., 2016). When the patient is in the hospital, the focus of care is the reason for admission, not PD. Medication regimens are difficult to adjust to hospital routines, leading to suboptimal symptom control (Muzerengi et al., 2016). A retrospective study of 89 surgeries for patients with PD undergoing non–PD-related surgery showed that carbidopa/levodopa was withheld an average of 12.35 hours (Fagerlund, Anderson, & Gurvich, 2013). The symptoms most commonly seen were agitation and confusion, but tremor and increased pain also were noted. Symptoms became severe if several doses were missed and included difficulty swallowing and hallucinations (Fagerlund et al., 2013). A qualitative study of patients who underwent non–PD-related surgery found that hospital routine and medication timing did not often correspond. Some nurses were better than others in allowing patients to keep to their regimen and even advocated for medications to be taken with a sip of water if NPO. Patients with PD consider themselves the experts in their own disease and know when they need medications. Patients interviewed in this study thought that hospital nurses lacked knowledge about PD and PD medications. Patients thought they needed an advocate at the bedside to ensure adequate PD care while receiving surgical care (Anderson & Fagerlund, 2013).

Clinical Pearls

- Encourage patients to engage in medication adherence.
- Management by a neurologist, specialty nursing care, and home care can reduce the number of unplanned hospitalizations.
- Highest caregiver needs are related to symptom management, coping with changes in lifestyle, future planning, relationships, cognition, and wellness strategies.
- Patients need an advocate at the bedside to ensure adequate PD care while hospitalized.

Challenges and Controversies

Patients with PD have very high fall rates in the home and the hospital (Kalilani, Asgarnejad, Palokangas, & Durgin, 2016). The literature review of nursing care related to PD revealed a lack of evidence related to fall prevention strategies specific to patients with PD. Until disease-specific evidence is available, it is reasonable to follow standardized fall prevention precautions used for any high-risk patient and focus on medication timing to improve mobility. Despite optimal medical management of motor symptoms, patients with PD still experience gait and balance disturbances, leading to declines in activity, decreased QOL, fear of falling, and actual falls. Rehabilitation strategies have shown positive effects on gait, balance, and mobility (Canning et al., 2014; Conradsson et al., 2015; Corcos et al., 2013; Ganesan, Sathyaprabha, Gupta, & Pal, 2014; Nadeau, Pourcher, & Corbeil, 2014; Picelli et al., 2013; Uc et al., 2014; Volpe, Giantin, Maestri, & Frazzitta, 2014). None of these studies demonstrated a significant difference in frequency of falls.

PD is a progressive disease that can lead to end-of-life suffering. The slow trajectory of the illness has led to uncertainty related to advance care planning and palliative care. A survey of 64 designated proxies to patients with advanced PD demonstrated that although 94% of patients completed an advance directive (AD) or living will, only 38% shared this information with providers. When provided mock scenarios, 30% of decision makers choose end-of-life care inconsistent with the patient's choices. Seventy-two percent did not know the patient's preference for hospice care. A concerning number of patients designated in their advance care plan the desire for CPR despite not wanting ventilator support or feeding tubes. If the patient spoke with the provider when completing an AD, they opted less for CPR and more often chose hospice. Most of the proxies wanted some form of shared decision making with family members and physicians (Kwak, Wallendal, Fritsch, Leo, & Hyde, 2014).

Although the need for advance care planning and end-of-life care options is established among patients and caregivers, timing of the discussion with healthcare providers is less established. A survey of 267 patients with PD found that 94% wanted information about prognosis and treatment early in the disease course (Tuck, Brod, Nutt, & Fromme, 2015). Half wanted advance care planning early in the course of illness, and 27% wanted end-of-life options such as hospice discussed early in the course of illness. Most responders thought responsibility for the discussion was shared between patients and providers. Findings of this study are similar to surveys

of patients with advanced cancer. Nurses can approach advance care planning by asking simple questions about ADs and requesting copies of documents. Nurses can facilitate end-of-life discussions by asking the patients when they would like information about end-of-life options, in a manner that allows the patient to decide. End-of-life discussions are difficult but necessary to comply with the patient's wishes. It is appropriate to schedule an appointment just for this discussion (Tuck, Brod, Nutt, & Fromme, 2015).

Future Considerations

As our population ages and technology improves, nursing research related to PD should focus on improving QOL for patients with PD and their care partners. Access to specialty care has shown promising results, but not enough research has been done to make recommendations.

Fall prevention studies thus far have not shown success for patients with PD. More research is needed in this area to reduce falls and complications related to falls. There is a large end-of-life burden related to PD on patients, caregivers, and healthcare providers. Review articles suggest palliative care is a valid option for patients with advanced disease. However, there are no studies related to the benefits of palliative care for PD (Ghoche, 2012; Miyasaki & Kluger, 2015).

The treatment of PD involves many specialties. Multidisciplinary care is the optimal model for PD care. The PD specialist nurse is in a position to facilitate this multidisciplinary approach to care. Care that includes a PD specialist nurse was more individualized and better able to improve QOL for patients with PD compared with care delivered by a general neurologist (van der Marck, Bloem, et al., 2013). Unlike the evaluation of individual interventions, the merits of a multidisciplinary approach are seen as the sum of the whole. Causal relationships to outcomes are difficult to make. A multidisciplinary approach is beneficial to the care of the patient with PD, and nurses remain pivotal members of the team to ensure continuity of care for both patients and caregivers (van der Marck, Munneke, et al., 2013).

Essential Tremor

Background

Essential tremor (ET), once considered a benign condition, is now considered a progressive neurodegenerative disease affecting the extremities, head, voice, and cognition (Carranza, Snyder, Elble, Boutzoukas, & Zesiewicz, 2012). Although the cause remains unknown, there is a strong genetic link. A family history of ET is found in 30%–80% of affected patients (Hedera, Cibulcik, & Davis, 2013). Tremors begin in the upper extremities, and over time many patients develop cerebellar dysfunction leading to gait abnormalities and experience cognitive deficits (Hedera, 2017). More than 90% of patients affected by ET reported deficits with their ADLs (Hedera, 2017). ET is the most common tremor disorder in United States, affecting 10 million people (Revell, 2015). The incidence of ET increases with age. Life expectancy is not affected by this disease (Zesiewicz et al., 2011).

Clinical manifestations include involuntary, rhythmic muscle contractions of an affected body part. The tremors are kinetic and postural and maintain an amplitude of 8–12 Hz. The frequency of essential tremor increases with age. Prevalence is 4.6% in people older than 65 years and is estimated to be 20% in those older than 95 years (Julius & Longfellow, 2016). Diagnosis is often difficult because of a lack of biomarkers or genetic markers and variances between patient clinical manifestations (Revell, 2015). Substances that stimulate the sympathetic nervous system, such as caffeine, nicotine, tricyclic antidepressants, valproic acid, lithium, selective serotonin reuptake inhibitors, steroids, and bronchodilators, should be avoided when possible because of the potential for increase in tremor activity (Julius & Longfellow, 2016). Some patients self-medicate with alcohol, which temporarily decreases tremor activity, but the use of ethanol to treat ET is not endorsed.

Patients with ET can present with:

- Tremors that occur with action
- Intermittent and asymmetric tremors
- Tremors affecting the extremities, head, jaw, lips, face, and voice
- Improvement of tremors during sleep
- Exacerbation by emotion, hunger, temperature extremes, or fatigue
- Mood disorders

Treatment Overview

Medical Management

ET is managed by pharmacological and surgical means. Medications are estimated to be effective in only 30%–50% of patients (Zesiewicz et al., 2011). This poor efficacy rate may be attributed to the fact that the exact cause of ET remains unidentified. As a result, current pharmacological options are limited (see **Table 2**). The current drugs in use for ET have been developed for other conditions. To date, no pharmacological agents have been developed specifically for this condition. Because of these poor outcomes, surgical options such as focused ultrasound or DBS are often necessary.

Table 2. Medications for Essential Tremor

Name	Class	Dosage	Side Effects
Propranolol hydrochloride (first line)	Beta blocker	60–160 mg twice daily or 60–320 mg daily for long-acting dosing; 20–80 mg 2 hours before event for situational dosing.	Decreased heart rate, dizziness, somnolence, fatigue, erectile dysfunction, and bronchial constriction
Primidone (first line)	Benzodiazepine	50–750 mg at bedtime. Split doses greater than 500 mg. Start older adults at 25 mg at bedtime.	Somnolence, dizziness, gastrointestinal upset, and ataxia
Gabapentin (second line)	Antiepileptic drug (AED)	900–3,600 mg daily divided into three doses. Start older adults at 300 mg daily.	Dizziness, weight gain, and sedation
Topiramate (second line)	AED	50–400 mg daily in divided doses.	Peripheral neuropathy, weight loss, change in taste, renal calculi, birth defects, and acute angle closure glaucoma
Alprazolam (second line)	Benzodiazepine	0.125–3 mg daily.	Tolerance, dependence, abuse, withdrawal, sedation, and cognitive impairment
Nimodipine (third line)	Calcium channel blocker	30 mg four times daily.	Hypotension, edema, and headache

Adapted from Hedera, Cibulc ík, & Davis, 2013; Julius & Longfellow, 2016; Albanese, Bhatia, et al., 2013

Propranolol

Currently, propranolol is the only drug approved by the U.S. Food and Drug Administration (FDA) for the treatment of ET. Propranolol hydrochloride is a nonselective beta blocker. Because of its use by patients with comorbid hypertension, it is often used as a first-line agent in this patient population. The side-effect profile is considered mild and includes decreased heart rate, dizziness, somnolence, fatigue, and erectile dysfunction. Other medications in this class include nadolol, sotalol, and atenolol. Propranolol also can be used by patients who develop tremor in certain situations, not necessitating daily treatment. Short-acting dosage is 30–160 mg twice daily; long-acting dosage is 60–320 mg daily. Special consideration should be given to older adults, who may require lower dosing due to their decreased metabolism and renal function.

Contraindications include cardiogenic shock, unstable congestive heart failure, sinus bradycardia, greater than first-degree atrioventricular block, asthma, a history of reactive airway disease (because of the risk of bronchospasm), and a known hypersensitivity to this class of medications. Because some patients may have an exacerbation of angina pectoris or myocardial infarction upon abrupt discontinuation, patients must be slowly titrated off propranolol (Hedera et al., 2013; Julius & Longfellow, 2016).

Primidone

Primidone is the other first-line agent for the treatment of ET. It is a benzodiazepine developed for use as an anticonvulsant. It is FDA approved for partial complex and generalized tonic–clonic seizures. Incidental findings of tremor reduction for patients with epilepsy led to its current use for ET (Hedera et al., 2013). Side effects include somnolence, dizziness, gastrointestinal upset, and ataxia. It can be used concurrently with a beta blocker and has a synergistic effect, allowing for lower dosages of primidone.

Primidone must be used with caution by patients with a history of suicidality because of the possibility of worsening depression. As with all anticonvulsant medication, primidone cannot be given to women who are pregnant because of possible teratogenic effects. Primidone should be avoided in women using birth control pills, because it will decrease the efficacy of birth control (Julius & Longfellow, 2016). Primidone is recommended to be given at bedtime because of its side effect of somnolence. Recommended dosing is 50 mg at bedtime, slowly titrating up to 250–750 mg. Doses above 500 mg should be divided into twice-daily amounts. Special consideration should be given to older adults because of their decreased ability to metabolize and excrete drugs. This patient population should begin at 25 mg daily (Hedera et al., 2013).

Gabapentin

Gabapentin is a second-line medication for ET. It is used for patients who cannot tolerate or do not respond to therapy with propranolol or primidone. Patients with comorbidities such as peripheral neuropathy, radiculopathy, or anxiety may have improvement in these conditions as well as their tremor. Common side effects include dizziness, weight gain, and sedation (Julius & Longfellow, 2016). Gabapentin is typically dosed three times a day, starting at 300 mg per dose. The maximum daily dose is 3,600 mg. As with propranolol and primidone, older adults should be started at 100 mg per dose to avoid toxicity (Hedera et al., 2013).

Topiramate

Topiramate is an antiepileptic drug (AED) that can also be used to treat ET. Though not as effective as the first-line agents, it can be useful for patients who are unable to tolerate those medications. Topiramate has the added benefit of being a mood stabilizing agent for patients with a comorbid mood disorder. Side effects include peripheral neuropathy, weight loss, cognitive deficits, somnolence, confusion, and change in taste. Topiramate is teratogenic, and women of childbearing age should be on a reliable form of birth control. Topiramate can also cause renal calculi and acute angle closure glaucoma (Julius & Longfellow, 2016). Because of it side effects, dosing is usually begun at bedtime, starting at 25 mg daily and gradually increasing to no more than 400 mg daily in divided doses.

Alprazolam

Alprazolam is a benzodiazepine used as second-line therapy for patients with ET. When prescribing this class of medication, providers should be concerned about dependence, tolerance, withdrawal if stopped abruptly, cognitive impairment, increased risk of falls, and sedation. The typical dosing range is from 0.125 to 3 mg daily (Hedera et al., 2013).

Nimodipine

Nimodipine is a calcium channel blocker used as a third-line therapy for patients with ET. Common side effects include hypotension, edema, and headache. Dosing is usually 30 mg four times a day (Hedera et al., 2013).

Surgical Treatment for Essential Tremor

Patients whose tremors are refractory to pharmacological management may benefit from surgical intervention. Surgical options include thalamotomy and DBS. Both treatments involve the thalamus.

During a stereotactic radiofrequency thalamotomy procedure, the nucleus ventralis intermedius of the thalamus is targeted. The cells involved in the circuitry that connects the cerebellum with cortical motor pathways are destroyed (lesion). Magnetic resonance–guided focused ultrasound is a novel form of thalamotomy or ablation that is delivered by ultrasound to create a lesion (Chang et al., 2017). Focused ultrasound is an incision-free lesioning technique that can be monitored in the operating room with magnetic resonance imaging (MRI) (Shaw, Johnston, Rush-Evans, Prather, & Maynard, 2017). The creation of this lesion deep in the thalamus aims to reduce tremor and improve QOL in this patient population, but it results in permanent damage to the lesioned brain tissue (Elias, 2016). DBS is currently the preferred surgical method for the treatment of ET, because of the reversibility and adjustability of the therapy.

Clinical Pearls

- Propranolol is the only drug approved by the FDA for the treatment of ET.
- DBS is currently the preferred surgical method for the treatment of ET.

Nursing Management

Nursing management of ET is multifocal. Whereas medical management focuses on the elimination or reduction of tremor, nurses also must address the psychosocial aspects of the disease. It is difficult for healthcare providers not to focus solely on the physical manifestations of ET, often overlooking the social and psychological aspects of the disease. A study by Louis, Rohl, and Rice (2015) found that only 10% of patients with ET were satisfied with their care. Patients identified many concerns that were not being addressed by their healthcare providers, including mental, social, and emotional needs. Because of deficits performing ADLs, many patients find themselves embarrassed by their condition, leading to social isolation, depression, and anxiety. Although patients perceived that their physical needs were assessed and treatments were planned to improve the tremors, little or nothing was done for their more invisible symptoms and concerns. The nurse should screen for and provide support for the social, mental, and emotional feelings of essential tremor and evaluate and assist the patient in enhancing self-care (Lageman, Cash, & Mickens, 2014; Louis et al., 2015).

Nurses also must be familiar with the medications used in the treatment of ET. Women of childbearing age must be educated about the teratogenic effects of AEDs and about effective birth control methods. Patients who are

taking benzodiazepines need to be evaluated frequently because of the high risk of dependency, tolerance, abuse, withdrawal, and increased risk of fall.

Clinical Pearls

- Review medications used in the treatment of ET.
- Screen and provide support for the social, mental, and emotional needs of the patient.
- Evaluate and assist the patient in enhancing self-care.

Challenges and Controversies

Because the exact cause of ET is unknown, treatment options remain elusive. Although front-line management remains pharmacological, it is effective only 30%–50% of the time (Zesiewicz et al., 2011). Given the large psychosocial overlap, few patients think these needs are being addressed, leading to depression, anxiety, suicidal ideation, and sleep deficits. These nonmotor manifestations must be given as much consideration as medication management (Jhunjhunwala & Pal, 2014).

Future Considerations

Additional research is needed on screening tools for depression, anxiety, suicidal ideation, and sleep deficits in ET. Screening for depression, anxiety, suicidal ideation, and sleep deficits should be performed at each clinic appointment, with treatments put into place to better manage nonmotor manifestations of ET.

Dystonia

Background

According to the Consensus Committee (Albanese, Bhatia, et al., 2013), dystonia can be defined as a movement disorder characterized by involuntary sustained or intermittent muscle contractions. These dystonic muscle contractions can cause abnormal, repetitive movements or postures, which are typically patterned, causing twisting movement or tremor. Dystonia is associated with overflow muscle activation and often is initiated or worsened by voluntary action.

The classification of a dystonia is important to consider because it is related to the types of motor and nonmotor disease manifestations nurses may encounter. Additionally, dystonia classification can determine the need for further investigation or consideration of alternative therapies. The latest dystonia classification system has two main axes, clinical features and etiology, as described in **Table 3** (Albanese et al. 2013).

Dystonia affects approximately 0.6 to 732 per 100,000 people, depending on the populations assessed and the methods used to determine dystonia (Comella, 2018). Dystonia is thought to result from abnormal signals coming from the basal ganglia in the brain, which are not appropriately inhibiting specific muscle groups during actions. This produces contraction of muscles that flex and extend a joint at the same time. For example, when a person with dystonia attempts to straighten the arm at the elbow, instead of the triceps contracting and biceps relaxing, both the triceps and biceps contract. The result is an unusual posture or position of the arm and slow movements as both muscle groups contract at the same time.

Treatment Overview

Medical Management

Dystonia treatment options range from least invasive (i.e., physical therapies and counseling) to most invasive (i.e., surgical interventions) depending on disease manifestation. As discussed, the classification of dystonia directs the practitioner toward what disease manifestations they may encounter and aids in treatment decisions. Therefore, correctly identifying and classifying the dystonia is of vital importance because the diagnosis serves as a guide for the most appropriate and effective treatments, as depicted in the methodological strategy in **Figure 1** (Jinnah & Factor, 2015).

Rehabilitation including physical, occupational, and speech therapies are all forms of nonpharmacological treatments used to help compensate for the abnormal postures and movements of dystonia. Although rehabilitation is widely used as an adjunct therapy to medications, systematic reviews have shown that there is a lack of evidence supporting any specific type of rehabilitation regimen over another for the patient with dystonia (Jinnah & Factor, 2015). In particular, a systematic review conducted in 2015 showed low to moderate evidence regarding the use of rehabilitation for patients with cervical dystonia (Callahan, Shrotri, Raje, & Beninato, 2015). However, it is important for the practitioner to keep in mind that referral to rehabilitation for children and adolescents with generalized dystonia may be beneficial. The Dystonia Society (2014) has recognized that physiotherapists can offer support with the following:

- Identifying handling, positioning, and adaptive techniques for patients with dystonia and their caregivers
- Recommending patient-specific exercises and techniques to help decrease nonmotor manifestations of dystonia, including pain and discomfort
- Promoting functional mobility and advise on adaptive equipment such as wheelchairs and adaptive communication devices

Table 3. Proposed Classification of Dystonia

Axis I. Clinical Characteristics

Clinical characteristics of dystonia

Age at onset
- Infancy (birth to 2 years)
- Childhood (3–12 years)
- Adolescence (13–20 years)
- Early adulthood (21–40 years)
- Late adulthood (>40 years)

Body distribution
- Focal
- Segmental
- Multifocal
- Generalized (with or without leg involvement)
- Hemidystonia

Temporal pattern
- Disease course
  - Static
  - Progressive
- Variability
  - Persistent
  - Action-specific
  - Diurnal
  - Paroxysmal

Associated features

Isolated dystonia or combined with another movement disorder
- Isolated dystonia
- Combined dystonia

Occurrence of other neurological or systemic manifestations
- List of co-occurring neurological manifestations

Axis II. Etiology

Nervous system pathology

Evidence of degeneration

Evidence of structural (often static) lesions

No evidence of degeneration or structural lesion

Inherited or acquired

Inherited
- Autosomal dominant
- Autosomal recessive
- X-linked recessive
- Mitochondrial

Acquired
- Perinatal brain injury
- Infection
- Drug
- Toxic
- Vascular
- Neoplastic
- Brain injury
- Psychogenic

Idiopathic
- Sporadic
- Familial

- Assisting in recognition of contractures and deformities that may warrant referral to additional services, including orthopedics.

The treatment goal of oral pharmaceutical agents is to reduce or eliminate the dystonia while taking the lowest possible dosage with the least amount of side effects. Oral medication decisions depend on the classification of dystonia. In general, dystonias are treated with the following classes of medications: carbidopa/levodopa, anticholinergics (e.g., trihexyphenidyl), benzodiazepines, baclofen, and dopamine-depleting agents (Shanker & Bressman, 2016).

Anticholinergic medications are one of the most commonly prescribed oral medications because of several studies demonstrating their effectiveness. Trihexyphenidyl is the only oral medication that has been proven effective for the treatment of torsion dystonia, according to a prospective double-blind study (Burke, Fahn, & Marsden, 1986). This makes it the preferred anticholinergic medication for most dystonias among practitioners.

Carbidopa/levodopa is the standard treatment for patients with dopa-responsive dystonia, because evidence has shown significant and ongoing improvements with low-dose therapy (Shanker & Bressman, 2016). The oral medication therapies, typical dosing schedules, and common side effects are summarized in **Table 4**.

It is important for the neuroscience nurse to keep in mind that some oral medications, including dopamine-blocking medications, antiemetics (prochlorperazine and metoclopramide), and antipsychotics (risperidone,

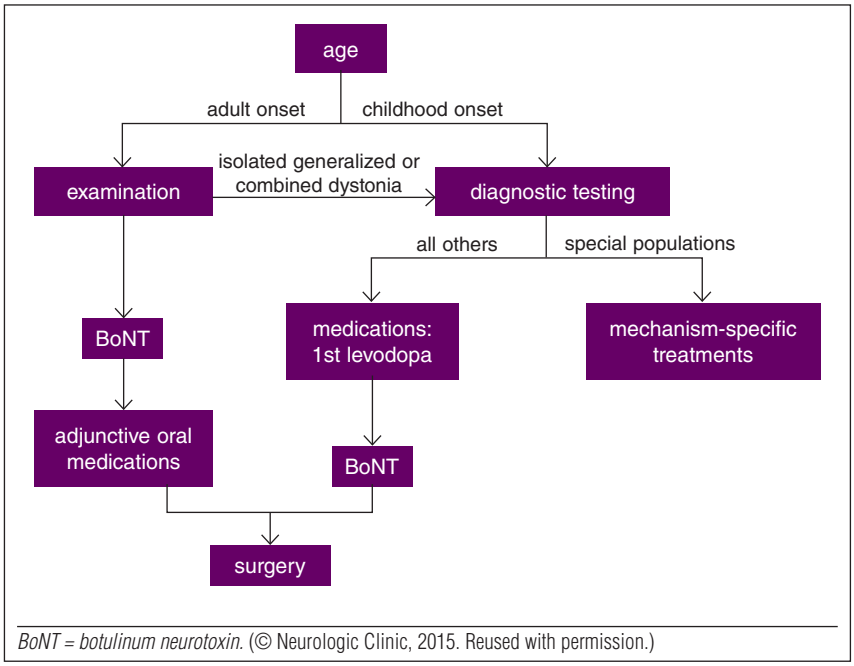
olanzapine, ziprasidone, and aripiprazole) (Shanker & Bressman, 2016), may cause or exacerbate dystonia symptoms. Additionally, there are no FDA-approved oral pharmaceutical agents for the dystonias at this time. Because of the lack of strong evidence, recommendations developed from anecdotal experience, nonblinded trials, small controlled trials, and retrospective reviews, it is important for the practitioner to use good clinical judgment when selecting oral medications and monitor for side effects (Jinnah & Factor, 2015).

Botulinum neurotoxin (BoNT) is a neurotoxic protein that is produced by the bacterium *Clostridium botulinum*. In general, the toxin takes effect by being injected into the dystonic muscles and is received by the motor neurons. This allows the toxin to inhibit the release of acetylcholine into the neuromuscular junction, which blocks or reduces the unwanted muscle activity (Shanker & Bressman, 2016).

Medical-grade BoNTs have been studied on many occasions, and systematic reviews have concluded their effectiveness for various classifications of dystonia (Albanese et al., 2006; Hallett, Benecke, Blitzer, & Comella, 2009; Marques et al., 2016; Simpson et al., 2008, 2016). FDA-approved serotypes include type A, onabotulinumtoxinA (Botox™), abobotulinumtoxinA (Dysport™), incobotulinumtoxinA (Xeomin™); and type B, rimabotulinumtoxinB (Myobloc™). There are multiple types of medical-grade BoNTs. The selection BoNT type depends on the location of the dystonia. Please refer to Simpson et al. (2016) and Hallett et al. (2013) for detailed information on BoNT injectables.

BoNT is the treatment of choice for most focal and segmental dystonias and can be used to target specific areas for patients with generalized dystonia. A systematic review concluded that the desired effects from BoNT typically take place in about 2–7 days and last about 3–4 months (Marsh, Monroe, Brin, & Gallagher, 2014).

Figure 1. Methodological strategy for diagnosis of dystonia



BoNT = botulinum neurotoxin. (© Neurologic Clinic, 2015. Reused with permission.)

Table 4. Common Oral Medications Used to Treat Dystonia

Therapeutic Class and Medication	Therapeutic Daily Dose (Typically Divided Into 2- to 4-Times Daily Dosing)	Common Side Effect Profile
Anticholinergic		
Trihexyphenidyl	6–40 mg	Blurry vision, confusion, constipation, urinary retention, xerostomia
Benzodiazepines		
Clonazepam	1–4 mg	Drowsiness, fatigue, and aspartate transaminase and alanine transaminase elevation
Diazepam	10–40 mg	Drowsiness, fatigue
Dopamine precursor		
Carbidopa/levodopa	75 mg/300 mg–500 mg/2,000 mg	Nausea
-Aminobutyric acid B (GABA-B) agonist		
Baclofen	40–120 mg	Drowsiness, fatigue, nausea, muscle weakness

© Continuum, 2016. Reused with permission.

Clinical Pearls

- A practitioner should use the patient's classification of dystonia as a guide for the most appropriate and effective treatment options.
- Rehabilitation including physical, occupational, and speech therapy can be recommended by the practitioner as an adjunctive therapy to medical and surgical treatment options for dystonia.
- Carbidopa/levodopa is the standard treatment for patients with dopa-responsive dystonia.
- The use of good clinical judgement is imperative when selecting oral medications for the patient with dystonia and monitoring for side effects.
- Medical grade BoNTs are an effective treatment option for most focal and segmental dystonias.
- The practitioner is to recommend a 3- to 4-month follow-up evaluation when treating patients with BoNT because the desired effects typically take place in 2–7 days and last about 3–4 months.

Nursing Management

It is crucial for the practitioner suspecting dystonia to complete a full movement disorder examination. In addition, a comprehensive history and neurological examination can help reveal certain dystonia phenomenology and disease manifestations that may not be found in the typical neurological examination (Shanker & Bressman, 2016). The information found in the history and exams serves as the practitioner's guide toward developing a plan of care and appropriate treatment options. Furthermore, it can lead to an understanding of the severity of the patient's dystonic symptoms and their impact on QOL. If not personally conducting the exam, the nurse should be aware of abnormal exam findings when caring for the patient with dystonia.

Counseling and education about dystonia and its disease manifestations are important for patients with suspected or diagnosed dystonia and their caregivers. Research has shown that diagnosing dystonia can be delayed, with evidence concluding an average timeframe of 4–6 years in even the most common dystonia classifications (e.g., cervical dystonia) (Jinnah & Factor, 2015). Additionally, treatment options for dystonia are not curative, and it is important to counsel the patient with dystonia regarding realistic goals of treatment to avoid frustration and potential noncompliance.

Nurses should use a clinical scoring system to monitor worsening dystonia and prevent status dystonicus. In 2013, a group of movement disorder specialists (Lumsden, Lundy, Fairhurst, & Lin, 2013) developed a clinical scoring system. The specialists understood that the scoring system had to be straightforward enough for any healthcare provider involved in the patient's care to use, not just movement disorder specialists.

Patients with dystonia can suffer from social anxiety, generalized anxiety, and depression, which can affect their QOL (Ward, 2009). Nurses should evaluate and recognize the mood of the patient with dystonia to improve QOL and overall treatment success. Using a holistic approach with a psychiatric, physical, and neurological evaluation in a multidisciplinary team setting can optimize the care of patients with dystonia (Ward, 2009).

Clinical Pearls

- A patient with suspected dystonia should undergo a comprehensive movement disorder examination and a full neurological examination.
- Providing counseling and education to patients with suspected or diagnosed dystonia is important to promote a timely diagnosis, the nurse–patient relationship, and realistic treatment goals.
- The practitioner may use the DSAP for patients with worsening dystonia to assist in early recognition of potential status dystonicus.
- The nurse should evaluate psychosocial issues and QOL for the patient with dystonia.

Challenges and Controversies

The challenges and controversies include medical management and the appropriate management of DBS to treat symptoms of dystonia. Patient selection, genetics, and proper programming settings play a role in optimal outcomes for patients with dystonia treated with DBS. The neuroscience nurse can play an active role in support and education of patients with dystonia and their families, from diagnosis to treatment.

Future Considerations

Future considerations for dystonia include elucidating genetic mutations, targeted therapy for patients with genetic forms of dystonia, new medical therapies, and a better understanding of appropriate DBS management.

Deep Brain Stimulation

Background

Deep brain stimulation (DBS) is an effective and established therapy for appropriately chosen patients with disabling movement disorders that are refractory to medications (Shukla & Okun, 2016). DBS has been approved by the FDA to treat PD; ET; and primary generalized, segmental, or cervical dystonias or hemidystonias (Martinez-Ramirez & Okun, 2014). DBS electrodes or leads are implanted by stereotactic functional neurosurgeons

in specific areas of the brain within the basal ganglia (Fasano & Lozano, 2015). These electrodes are fixed in place with a burr hole cap and are connected by extension wire to implantable pulse generators (IPGs) in the chest (Shukla & Okun, 2016). IPGs are also known as batteries, neurostimulators, or "brain pacemakers" (Gardner, 2013). Once implanted, the IPGs are programmed by clinicians to send electrical impulses back to the brain to treat the abnormal involuntary movements of the patient (Fasano & Lozano, 2015).

Programming settings are adjustable and are individualized to each patient (Grill, 2015). DBS can be programmed to help treat the motor symptoms of tremor, rigidity, bradykinesia, dystonia, myoclonus, and dyskinesia (Martinez-Ramirez & Okun, 2014). Optimal outcomes from DBS programming result from multiple and simultaneous modifications of stimulation and medications (Morita, Susatia, Foote, & Okun, 2015). As of 2016, more than 140,000 patients worldwide had undergone DBS surgery (Shukla & Okun, 2016).

The exact mechanism of action of DBS remains unknown (Martinez-Ramirez & Okun, 2014). Researchers believe that the effects of DBS are multifactorial, occurring at multiple levels of neuronal pathways with chemical and biological effects (Shukla & Okun, 2016). It is believed that within the field of electrical stimulation, neurons are inhibited and axonal fibers are excited, stimulating a release of neurotransmitters (Shukla & Okun, 2016). An increase in blood flow also has been observed within the DBS field of stimulation. By modulating the neuronal firing pattern, DBS improves symptoms (Shukla & Okun, 2016).

Three DBS device manufacturers have FDA approval for systems that are currently available in the United States: Medtronic, Abbott (formerly St. Jude Medical), and Boston Scientific. A prospective, multicenter, nonrandomized, open-label intervention study in Europe found the Boston Scientific directional DBS electrode to be effective in reduction of overall Unified Parkinson Disease Rating Scale (UPDRS) scores. These PD patients also had reduction in PD medications, improvement in ADLs, quality of life and "on medication" time (Timmermann et al., 2015).

Medtronic

The Medtronic device uses radiofrequency to communicate between the clinician programmer controller or patient programmer and an IPG to program or change DBS settings through the skin. The programming head of the clinician programmer and the patient programmer both need to be held directly over the IPG in order to connect and adjust stimulation settings (*Medtronic Clinician Manual*, 2017).

The Medtronic Activa™ DBS system has two leads. Each lead has four titanium contacts that are each 1.5 mm in length. Stimulation emanates from the contacts chosen by the clinician. The Medtronic device is MRI compatible under certain conditions (*Medtronic Clinician Manual*, 2017).

Abbott

The Abbott device uses Bluetooth technology to access and program DBS settings. Bluetooth technology allows wireless connection between a variety of different electronic devices to a system for data transfer. Bluetooth technology uses radio waves to communicate between devices. The pairing process identifies and connects any two devices exclusively to each other. Nonpaired Bluetooth devices in the vicinity are not able to interfere with paired devices. Once the clinician programmer and battery have been paired, the clinician can program the IPG wirelessly by using the St. Jude Medical Clinician Programmer application on the iPad Clinician Programmer (St. Jude Medical, 2015).

The Abbott Infinity™ DBS system has directional capabilities and has two leads with four 1.5-mm contact points. The second and third contacts are each segmented further into three discrete contacts. Each segmented contact stimulates 120° of area. These segmented contacts allow precise direction of current to maximize symptom reduction and avoid side effects (Fasano & Lozano, 2015; St. Jude Medical, 2015). The Abbott device is MRI compatible under certain conditions (St. Jude Medical, St. Paul, MN).

Boston Scientific

The Boston Scientific device Vercise™ delivers directional stimulation, will be rechargeable, and will not be MRI compatible (Boston Scientific, n.d.).

Treatment Overview

Preoperative DBS Management

Candidates for DBS should be evaluated by a DBS team, which includes a neurosurgeon, the neurologist or programmer, a neuropsychologist, and a psychiatrist. The patient should be evaluated with disease-appropriate assessment tools or scales to document preoperative symptoms, such as the UPDRS for PD, the Burke–Fahn–Marsden Dystonia Rating Scale (BFMDRS) for dystonia, or the Tremor Rating Scale for ET (Munhoz et al., 2016). Preoperative documentation is recommended for post-stimulation comparison of symptoms (Grill, 2015). For PD DBS candidates, the Core Assessment Program for

Surgical Intervention Therapies (CAPSIT-PD) protocol is used widely to determine the steps for selection, preoperative evaluation, and postoperative follow-up of candidate patients for DBS (Pal et al., 2015). The CAPSIT is an assessment of levodopa responsiveness and represents the best predictor of DBS outcome. PD symptoms that are resistant to levodopa, such as postural instability and speech and gait disorders, will not improve with DBS. There is no consensus with regard to upper age limit and DBS (Munhoz et al., 2016). Recording video of preoperative symptoms, both off and on medications, is also recommended (de Rosa, Tessitore, Bilo, Peluso, & De Michele, 2016). This is also a useful tool for patients to assess their own progress postoperatively.

Preoperative Patient Education

The neuroscience nurse can play a major role in the success of the DBS program through effective patient education. Educational sessions for potential DBS candidates and their caregivers can be used as an open forum for patients to ask questions of the nurse, the surgeon, or patients with DBS already implanted. Knowledge about DBS components, surgery, and the programming process can allay fears and mitigate anxiety about surgery (Grill, 2015; Dinkelbach, Möller, Witt, Schnitzler, & Südmeyer, 2017). The following are teaching objectives of patient education on DBS:

- DBS is not a cure for movement disorders.
- Patients will need continued medical therapy.
- Optimal results from stimulation may take months to achieve and will be different for each patient.
- DBS settings and medications must be adjusted concurrently.
- DBS is a process and may not yield immediate results (Revell, 2015).
- Although discontinuation of medications is not a primary goal, many patients are able to reduce their medications by 25%–50%.

The primary goal of DBS is to obtain symptomatic benefit without side effects. The symptoms of tremor, bradykinesia, rigidity, motor fluctuations, improvement of off-medication periods, myoclonus, dyskinesia, and dystonia can be treated by DBS but may not be eliminated (Rabin & Kumar, 2015). PD tremor that is refractory to levodopa may still respond to DBS. Cognition, speech, gait dysfunction, depression, anxiety, and postural instability do not respond to DBS therapy (Martinez-Ramirez & Okun, 2014). Future decline in these areas with disease progression may not be prevented by DBS therapy.

The components of successful DBS therapy include an appropriately selected patient and an evaluation at a leading DBS center by an experienced DBS team (Grill, 2015). Patient and family education regarding realistic expectations, optimally placed leads, and proper DBS programming with coordinated and supportive care are also necessary for DBS to be successful (Grill, 2015).

Clinical Pearls

- Screen patients for appropriate and realistic expectations about surgery.
- Educate patients about the therapeutic goals of DBS surgery.
- Screen patients for appropriate expectations about the postoperative course.

DBS Candidates for ET

The FDA approved the Medtronic unilateral DBS for tremor refractory to medication for patients with ET and PD in 1997. The St. Jude/Abbott device has FDA approval for bilateral ventral intermediate (VIM) nucleus treatment of ET. Electrodes are typically implanted in the VIM nucleus of the thalamus, with significant reduction of tremor and disability in ET. Loss of efficacy over time is thought to be due to the progression of the disease, not tolerance to DBS (Picillo, Kou, et al., 2016). A highly controversial, single-patient controlled trial used a signal-to-noise analysis to provide class I evidence for the efficacy of DBS for ET (Hyam et al., 2015). Before this study, despite the demonstration of profound improvement on tremor for a large number of patients, no double-blind, placebo-controlled studies of DBS for ET existed.

Clinical Pearl

- A patient with ET is a DBS candidate if he or she has a severe disabling tremor that does not respond to medical therapy and affects their QOL (Hyam et al., 2015).

DBS Candidate for PD

Bilateral DBS of the subthalamic nucleus (STN) or the globus pallidus pars interna (GPi) for PD was FDA approved in 2002 for patients with at least 4 years of idiopathic PD, without dementia, with motor symptoms that have adequate response to levodopa, but with motor fluctuations in response to medications. Proper patient selection is critical in PD, allowing for reduction in disability and improvement in QOL (Martinez-Ramirez & Okun, 2014). Multiple randomized controlled trials (class I evidence) have compared DBS for PD with medical therapy and medical therapy alone. DBS has been shown to be effective in treating PD symptoms, with improvement of function and QOL (Perestelo-Pérez et al., 2014). A 69.1% reduction of dyskinesia and 68.2% reduction of off-medication periods also were observed postoperatively across multiple studies (Kleiner-Fisman et al., 2006).

Improvement of UPDRS scores was 60.3% compared with baseline, and the average reduction in levodopa equivalent dose was 55.9% (Kleiner-Fisman et al., 2006). The average improvement in ADLs was 50% across studies.

Stimulation of either STN or GPi targets improves motor symptoms in PD. STN DBS allows greater reduction in medication compared with GPi stimulation, but GPi may have more benefit after surgery with respect to speech, swallowing, balance, and gait (Weaver et al., 2012). The Cooperative Study Program 468 study, or the Veterans Administration 24-month follow-up study of patients randomly assigned to either STN or GPi versus best medical therapy, showed that patients with GPi stimulation may have fewer cognitive problems than with STN stimulation.

Clinical Pearls

- A patient with PD is a DBS candidate if he or she has had idiopathic PD for at least 4 years and is without a cognitive deficit, untreated mood disorder, or brain abnormality.
- A good DBS candidate with PD responds well to levodopa, is experiencing motor fluctuations in response to medication, has few comorbidities that would preclude surgery, and has realistic expectations about their postoperative course.

DBS Candidate for Dystonia

The FDA approved the Medtronic Activa system in 2003, under a Humanitarian Device Exemption, for treatment of patients with medically refractory primary generalized, cervical, or segmental dystonia or hemidystonia who are at least 7 years of age (Fox & Alterman, 2015). A center that performs DBS for dystonia under the exemption must do so with oversight by that center's institutional review board (Fox & Alterman, 2015). Randomized controlled trials have reported that DBS of the GPi results in statistically significant reduction in dystonia severity scores and disability scores for primary generalized dystonia, segmental dystonia (Kupsch et al., 2006), and cervical dystonia (Volkmann et al., 2014) when compared with sham neurostimulation. Long-term results from follow-up studies show that the benefits from surgery are maintained for at least 5 years after stimulation (Volkmann et al., 2012). A long-term retrospective study reported statistically significant efficacy of DBS after 7 years (Panov et al., 2013). Current evidence suggests that genetic factors may influence outcomes, but further research is needed (Jinnah et al., 2017). Experience with DBS for dystonia suggests that patients with known genetic mutations have an optimal response to DBS (Fox & Alterman, 2015). Knowledge of genetic status can assist in setting realistic expectations and counseling about the postoperative course (Jinnah et al., 2017).

Clinical Pearls

- DBS for dystonia should be considered for patients aged 7 or older with medically refractory primary generalized, cervical, or segmental dystonia or hemidystonia.
- A clear diagnosis of dystonia, age of onset, duration of symptoms, genetic status, types of dystonic movements, and lack of comorbidities dictate response to DBS (Bronte-Stewart et al., 2011).

Risk and Potential Complications from DBS

There are three types of risks in DBS: surgical, stimulation related, and hardware related. Surgical complication risk varies between centers, with reduced risk of side effects in the hands of an experienced DBS functional neurosurgeon at a proficient DBS center. Surgical risks include intracranial hemorrhage, stroke, seizure, sterile seroma, deep cerebral venous hemorrhage, pulmonary embolism, pneumonia, perioperative confusion, and suboptimal electrode placement (Machado, 2006).

Device-related risks include infection, skin erosion, electrode or extension wire fracture, loose wire connections, IPG malfunction, and device migration. Stimulation-related risks for VIM electrode placement include paresthesia, muscle contraction, ataxia or postural instability, and dysarthria (Rabin & Kumar, 2015).

Stimulation-related risks for GPi electrode placement include muscle contractions, phosphenes, and dysarthria (Rabin & Kumar, 2015). The stimulation-related risks for STN electrode placement include dyskinesia, paresthesia, dysarthria, muscle contractions, dysphonia, gait instability, diplopia, and apraxia of eyelid opening (Morita et al., 2015). Stimulation-related side effects can be avoided by adjusting DBS programming settings.

Clinical Pearls

- Educate patients about and evaluate for risks and complications of DBS surgery.
- Evaluate patients for side effects from medications or stimulation.

DBS Surgery (Perioperative Period)

A trained stereotactic and functional neurosurgeon can use multiple techniques to implant DBS electrodes in their intended targets. There are no consensus guidelines on best practice for DBS surgery (Machado, 2006). Each surgeon will have his or her own approach, based on training; experience; and availability of resources, technology, staff, and facilities. Most surgeons use a stereotactic headframe

to perform surgery. Some surgeons use a frameless technique or perform MRI-guided DBS surgery.

In general, the patient with a movement disorder will stop taking medications the night before the surgery and will be awake for at least part of the DBS lead implantation surgery.

Perioperative Nursing Management

The patient returns to the operating room from radiology and is prepped for surgery. If used, the stereotactic frame is affixed to the operating room table, and sterile drapes are applied to the head and frame (Machado, 2006). Whether frame-based or frameless technology is used, the patient is off medications and awake for some or all of the DBS surgery. The patient should be made as comfortable as possible. At this point, a neurologist or neurophysiologist typically examines the patient for comparison between the "off" medication state and the patient's intraoperative response to stimulation of the implanted electrode (Bronte-Stewart, 2015).

Some anesthesiologists use light sedation for specific parts of surgery or if a patient is highly anxious. Patient blood pressure is monitored closely throughout surgery and kept low to reduce the risk of hemorrhage in the brain (Shukla & Okun, 2016).

Extension Wire and IPG Placement Surgery

The extension wires and IPGs typically are surgically placed under general anesthesia. The surgeon will tunnel a guide tube, as in a ventriculoperitoneal shunt placement procedure, under the skin to a subcutaneous subclavicular pocket that will hold one IPG. The extension wire will connect each brain electrode to the IPGs in the chest. The patient typically is discharged and sent home with a patient programmer or controller and a recharging device if a rechargeable battery is implanted.

Clinical Pearls

- Comfort awake patient.
- Monitor blood pressure intraoperatively.
- Monitor medications (avoid giving antinausea medications or beta blockers).
- Educate patient about postoperative wound care.

After DBS Surgery

After DBS lead placement, the patient will be transferred to a stepdown unit or critical care setting for overnight monitoring, to be discharged home the next day. After ambulatory surgery for extension wire and IPG placement, the patient will be discharged home the same day (Godden, 2014).

Clinical Pearls

- The nurse should monitor the patient for signs and symptoms of seizures or stroke. The nurse also should educate the family to watch for these signs and symptoms after discharge.
- The nurse should resume preoperative medication schedule and avoid giving antiemetics other than ondansetron (Hutchinson & Wick, 2016).
- The nurse should educate the patient and caregiver about wound care (Revell, 2015).
- The nurse should provide the patient with the appropriate DBS equipment (patient programmer or controller and recharging system with instruction manuals) that he or she will need postoperatively, if not provided before initial programming.
- The nurse should manage patient postoperative pain, at the discretion of the neurosurgeon or neurologist (Katus & Shtilbans, 2014).

DBS Programming

Overall, the success of DBS depends on multiple factors including the nature of the patient's disease, optimal placement of electrodes, optimal DBS programming settings, and medication adjustments (Shukla & Okun, 2016). It is important to note that there is no consensus on best practice for DBS programming and management (Shukla & Okun, 2016). Much of the available literature regarding DBS programming is expert opinion and empirical evidence based on extensive DBS experience from major DBS centers worldwide (Picillo, Kou, et al., 2016; Picillo, Lozano, et al., 2016).

Initial Programming

Before the initial programming visit, the programming clinician should be aware of details about the DBS surgery and the devices implanted (Rabin & Kumar, 2015). Specifically, the programmer should know which DBS system was implanted (Medtronic, Abbott, or Boston Scientific), which type of electrodes were implanted (narrow- or wide-spaced contacts), in which target the electrodes were placed (VIM, STN, or GPi), and the patient's intraoperative symptomatic response to stimulation. The programmer also should be aware of the patient's presurgical medication schedule and preoperative movement disorder symptoms (Rabin & Kumar, 2015).

Many patients experience a microlesioning effect in which they have a transient reduction in symptoms immediately after surgery because of edema around the electrode, resulting from surgical electrode placement (Rabin & Kumar, 2015). In some cases, the symptoms improve dramatically as a result of surgery, making it

difficult to assess benefit from stimulation immediately and for some time after surgery. Most centers begin programming the DBS devices 2–4 weeks after electrode implantation to allow the brain to heal and the microlesioning effect to subside (Shukla & Okun, 2016).

When presenting for initial programming, PD patients with either STN or GPi DBS are instructed to come to the office off medications from the night before and to bring their medications and any DBS patient devices (programmer, controller, or recharging device) with them to their appointment. It is helpful to have the patient hold any long-acting dopaminergic medications the day before initial programming (Rabin & Kumar, 2015).

For the programming clinician, using a systematic approach is paramount. Clear documentation of symptoms, side effects, and benefit for each programming parameter on each side can aid the programmer in planning settings and avoid retrial of settings that have caused adverse effects, such as dystonia or dysarthria (Rabin & Kumar, 2015).

Clinical Pearls

- Assess the patient's and caregiver's DBS programming expectations.
- Evaluate the patient for hardware-, stimulation-, or medication-related side effects.
- Educate the patient and caregiver about DBS wound checks.

Follow-Up DBS Programming

Typically, a patient is brought back to clinic for follow-up DBS programming sessions every 2–4 weeks early on in programming, to verify symptomatic benefit from stimulation and assess for stimulation- or medication-induced side effects (Revell, 2015). Review of any side effects experienced by the patient since the last visit will inform changes to be made in that patient's care. At every follow-up visit, the clinician will assess the patient's surgical incisions, reconcile medications, evaluate the patient's movement disorder symptoms, and document the DBS settings (Rabin & Kumar, 2015).

The edema surrounding the electrode upon implantation (causing the microlesioning effect) acts as a conductor to stimulation, spreading stimulation to areas that could cause side effects. This edema subsides over time, and the stimulation will need to be increased slowly to compensate for return of symptoms as the brain heals (Rabin & Kumar, 2015).

Stimulation is slowly increased by adjusting DBS parameters to aid in the symptomatic control of movement disorders. Typically, the clinician first makes a small increase in the voltage or amplitude to treat the patient's symptoms as the microlesioning subsides. This small increase can be made in 0.05- or 0.1-V or mA increments, up to a setting that is tolerated by the patient, where symptoms abate or before side effects are seen (Picillo, Kou, et al., 2016; Picillo, Lozano, et al., 2016).

Once the stimulation settings and medication adjustments have been optimized, the patient can follow up in clinic as infrequently as every 6 or 12 months for a physical examination, medication reconciliation, and battery or IPG check. Settings are usually optimized within 3 to 6 months for PD and ET. Clinical improvement in dystonia symptoms typically takes 6 months to a year or more (Picillo, Kou, et al., 2016; Picillo, Lozano, et al., 2016).

If an increase in voltage or amplitude results in insufficient symptomatic control, an increase in pulse width or frequency can be attempted. If the increases in voltage or amplitude, pulse width, and frequency are not adequate in suppressing patient symptoms, additional contacts can be activated to increase the size of the field of stimulation or the volume of tissue activation to produce the desired clinical effect (Picillo, Kou, et al., 2016; Picillo, Lozano, et al., 2016).

If the patient experiences intolerable side effects in response to an increase in any of the DBS parameters, the clinician can switch from monopolar to bipolar settings (Picillo, Kou, et al., 2016; Picillo, Lozano, et al., 2016). If there is little or no therapeutic benefit despite switching to bipolar settings and increasing parameters, a clinician can try interleaving settings (*Medtronic Clinician Manual,* 2017) or MultiStim settings or can activate one or two of the segmented contacts on the directional leads (St. Jude Medical, 2015).

Clinical Pearls

- Assess the patient for signs and symptoms of overstimulation.
- Monitor the patient for signs and symptoms of undermedication or overmedication.
- Assess the patient's and caregiver's DBS programming expectations.
- Educate the patient and caregiver about DBS wound care, programming, use of the patient controller or programmer, and medication adjustments.
- Tell the patient to notify any healthcare providers that they have a DBS device before any procedure or diagnostic testing.
- Educate the patient and family about contraindications with DBS, MRI conditionality, caution with other devices, caution with activities that could damage the DBS device, and airport procedures (*Medtronic Clinician*

Manual, 2017; Picillo, Kou, et al., 2016; Picillo, Lozano, et al., 2016; St. Jude Medical, 2015).

Outpatient DBS Patient Education

Outpatient education for DBS patients should include content regarding:

- Signs and symptoms of infection (see below)
- Signs and symptoms of a stroke: BE-FAST (balance, eyes, face, arm, speech, time), headache, weakness, and change in speech or gait
- Abrupt return of symptoms
- Worsening of symptoms
- Burning sensation along the DBS hardware

Several postsurgical expectations should be reviewed:

- DBS is not a cure for movement disorders.
- DBS programming involves frequent office visits, communication, adherence, and patience. The results from DBS programming sessions can take weeks to months to show optimal benefit, and a given setting will affect each patient differently.
- Medication adjustments are made concurrently with DBS programming adjustments. The goal in medication adjustments is overall reduction in medications, not discontinuation of medications.
- Adjunctive therapies such as physical therapy, occupational therapy, and speech therapy continue to be used after DBS surgery to optimize the patient's physical condition.

A major postoperative concern for the DBS patient is wound care. These wound care concerns should be reviewed with the patient (Mangram, Horan, Pearson, Silver, & Jarvis, 1999).

- Wash hands frequently and any time before touching the surgical incisions while healing.
- Look for signs and symptoms of wound infection or skin erosion. Call the neurology or neurosurgery office for any erythema, bleeding, purulent discharge or drainage, edema, tenderness, warmth, delayed healing, discoloration, or fever greater than 101°F (Revell, 2015).
- Change dressings as instructed.
- Refrain from picking, scratching, or unnecessarily touching the incisions.

Challenges and Controversies

The main challenges and controversies in DBS include the current lack of a known mechanism of action and lack of consensus for the best target for electrode placements, best practice DBS surgical technique, or best practice DBS programming (Fasano & Lozano, 2015). One challenge is to make DBS surgery more efficient and tolerable for patients. The use of intraoperative MRI for direct targeting of surgical targets can reduce time in the operating room, avoid awake surgery, minimize time off medication, minimize brain shift, and reduce hemorrhage risk (Sillay, Rusy, Buyan-Dent, Ninman, & Vigen, 2014). Intraoperative computed tomography also can aid in reducing surgical time through real-time imaging (Sokal, Harat, Rusinek, Ruda, & Litwinowicz, 2015).

Controversies include the timing in which to offer surgical options to patients. Studies have shown that patients with shorter disease duration have better QOL after DBS in comparison to their counterparts who have received the best medical therapy only (Deuschl et al., 2013), whereas in the past patients and clinicians have waited for severely advanced disease before considering DBS.

Another controversy involves the treatment of freezing gait in PD. Further research is needed to study the use of low-frequency stimulation in programming or placement of the DBS electrode in the pedunculopontine nucleus as possible treatments for freezing gait.

Recent advances in imaging sequences have allowed accurate mapping of iron deposits in deep brain nuclei to aid in precise targeting of structures for placement of DBS electrodes (Rasouli et al., 2018). Additionally, MRI sequencing with tractography allows direct visualization of the fiber tracts of axons in the brain, which will facilitate electrode placement, define newer surgical targets, and improve DBS programming in the future (Rodrigues et al., 2018).

Newer DBS technology includes segmented leads to allow the ability to steer current or send stimulation in a particular direction, ability to use lower pulse width settings, and ability to program each contact independently from others to avoid side effects (Picillo, Kou, et al., 2016; Picillo, Lozano, et al., 2016). The challenge now is how best to use these features to manage symptoms and care for patients with movement disorders.

Future Considerations

Future considerations should include novel DBS surgical techniques, improved imaging, and enhanced DBS programming capabilities. Research is currently underway to find new disease indications for DBS, new DBS electrodes and stimulation configurations, smaller and longer-lasting batteries, and closed-loop or sensing technology (Fasano & Lozano, 2015).

Current research is actively seeking optimal brain targets and programming settings for Gilles de la Tourette syndrome, depression, pain, epilepsy, and tinnitus. Additionally, DBS is being studied as treatment for eating disorders, addiction, cognitive decline, autism,

posttraumatic stress disorder, and minimally conscious states, among others (Hariz, Blomstedt, & Zrinzo, 2013).

Movement Disorders Review of Literature Summary

In summary, this review of the literature was performed to identify evidence-based strategies for nursing management of patients with movement disorders. A key topic that was identified for patients with movement disorders is to assess symptoms by using standardized tools. Patients with movement disorders should be evaluated for their ability to perform ADLs and for QOL issues. Another key topic was the use of medication adherence strategies to alleviate symptoms and improve QOL for these patients. Patients with movement disorders should be screened for coping, psychosocial issues, and emotional needs, another key topic found in the literature in caring for patients with movement disorders. The needs of caregivers also should be assessed. Nurses should take advantage of opportunities to discuss advance directives and end-of-life care with their patients who have movement disorders.

This review of literature on the use of DBS to treat patients with movement disorders found it to be safe and efficacious in properly selected patient. Successful DBS therapy for movement disorders depends on the choice of an ideal surgical candidate, treatment at a leading DBS center with a multidisciplinary approach, optimal surgical placement of electrodes, proper medical management, and appropriate DBS programming. Adjunctive therapies such as physical, occupational, and speech therapies can further improve QOL and restore function, another key topic found in this review of literature.

Recommendations for future study include assessing motor ability when the patient with PD is not on medication to better reflect the patient's perception of his or her disability. The effects of anxiety, psychosis, and sleep disturbance on the QOL of patients with movement disorders also should be researched. Further investigation is needed to discover methods to assess the needs and barriers to support for caregivers, timing of end-of-life care discussions, and the effects of palliative care on patients with movement disorders and their family caregivers. Tools are needed to screen for social, psychological, and emotional needs; to evaluate patients' ability to perform ADLs; and to prevent falls for patients with PD. Improvements in DBS surgery, devices, and programming will lead the way to enhanced care for patients with movement disorders. Educational opportunities regarding movement disorders should be required in nursing schools to improve care for this population.

Using evidence found in the literature can help nurses care for patients with PD, ET, and dystonia and patients treated with DBS. Evidence-based nursing care can improve patient outcomes even in chronic illnesses. This literature review also has identified some gaps in the literature, and nurses should consider further studies in the care of patients with movement disorders.

References

Adwani, S., Keshav, R., Chandra, S., & Pal, P. (2016). Neuropsychological profile in early Parkinson's disease: Comparison between patients with right side onset versus left side onset of motor symptoms. *Annals of Indian Academy of Neurology, 19,* 74–78. doi:10.4103/0972-2327.167711

Albanese, A., Barnes, M. P., Bhatia, K. P., et al. (2006). A systematic review on the diagnosis and treatment of primary (idiopathic) dystonia and dystonia plus syndromes: report of an EFNS/MDS-ES Task Force. *European Journal of Neurology. 13*(5):433.

Albanese, A., Bhatia, K., Bressman, S. B., DeLong, M. R., Fahn, S., Fung, V. S. C., . . . Teller, J. K. (2013). Phenomenology and classification of dystonia: A consensus update: Dystonia: Phenomenology and classification. *Movement Disorders, 28*(7), 863–873. doi:10.1002/mds.25475

Albanese, A., Sorbo, F. D., Comella, C., Jinnah, H. A., Mink, J. W., Post, B., . . . Schrag, A. (2013). Dystonia rating scales: Critique and recommendations. *Movement Disorders, 28*(7), 874–883. doi:10.1002/mds.25579

Anderson, L. C., & Fagerlund, K. (2013). The perioperative experience of patients with Parkinson's disease: A qualitative study. *American Journal of Nursing, 113*(2), 26–32.

Bette, S., Shpiner, D. S., Singer, C., & Moore, H. (2018). Safinamide in the management of patients with Parkinson's disease not stabilized on levodopa: a review of the current clinical evidence. *Therapeutics & Clinical Risk Management, 14*, 1737–1745.

Bhidayasiri, R., Chaudhuri, R., LeWitt, P., Martin, A., Boonpang, K., & Van, Laar, T. (2015). Effective delivery of apomorphine in the management of Parkinson disease: Practical considerations for clinicians and Parkinson nurses. *Clinical Neuropharmacology, 38,* 89–103.

Boston Scientific. (n.d.). Vercise™ Indications, Safety, and Warnings. Deep Brain Stimulator Systems. Retrieved from http://www.bostonscientific.com/en-US/products/deep-brain-stimulation-systems/vercise-tm-dbs/vercise_indications_safety_and_warnings.html

Bronte-Stewart, H. (2015). Surgical placement of deep brain stimulating leads for the treatment of movement disorders—intraoperative aspects: Physiological mapping, test stimulation, and patient evaluation. In W. Marks Jr. (Ed.), *Deep brain stimulation management* (pp. 31–42). Cambridge, UK: Cambridge University Press. doi:10.1017/CBO9781316026625.004

Bronte-Stewart, H., Taira, T., Valldeoriola, F., Merello, M., Marks, W. J., Albanese, A., & . . . Moro, A. E. (2011). Inclusion and exclusion criteria for DBS in dystonia. *Movement Disorders, 26S5*. doi:10.1002/mds.23482

Bruno, A. E., & Sethares, K. A. (2015). Fatigue in Parkinson disease: An integrative review. *Journal of Neuroscience Nursing, 47*, 146–153. doi:10:1097/JNN.000000000000131

Burke, R. E., Fahn, S., & Marsden, C.D. Torsion dystonia. Neurology. 1986;36(2):160.

Callahan, J., Shrotri, S. S., Raje, S. N., & Beninato, M. (2015). Non-surgical and non-pharmacological interventions for cervical dystonia: A systematic review. *Physiotherapy Practice & Research*, *36*(1):1.

Canning, C. G., Sherrington, C., Lord, S. R., Close, J. C., Heritier, S., Heller, G. Z., . . . Fung, V. S. (2014). Exercise for falls prevention in Parkinson's disease: A randomized controlled trial. *Neurology, 84,* 304–312.

Carranza, M. A., Snyder, M. R., Elble, R. J., Boutzoukas, A. E., & Zesiewicz, T. A. (2012). Methodological issues in clinical drug development for essential tremor. *Tremor and Other Hyperkinetic Movements, 2012,* 2. http//tremorjournal.org/article/view/68

Cavanaugh, J., Ellis, T., Earhart, G., Ford, M., Foreman, K., & Dibble, L. (2016). Toward understanding ambulatory activity decline in Parkinson disease. *Physical Therapy, 95,* 1142–1150. doi:10.2522/ptj.20140498

Chang, J. W., Park, C. K., Lipsman, N., Schwartz, M. L., Ghanouni, P., Henderson, J. M., . . . Cosgrove, G. R. (2017). A prospective trial of magnetic resonance–guided focused ultrasound thalamotomy for essential tremor: Results at the 2-year follow-up. *Annals of Neurology, 83*(1), 107–114.

Comella, C. (2018). Classification and evaluation of dystonia. *UpToDate.* Retrieved May 8, 2018, from https://www.uptodate.com/contents/classification-and-evaluation-of-dystonia?search=dystonia&source=search_result&selectedTitle=1~150&usage_type=default&display_rank=1#H4

Conradsson, D., Löfgren, N., Nero, H., Hagströmer, M., Ståhle, A., Lökk, J., & Franzén, E. (2015). The effects of highly challenging balance training in elderly with Parkinson's disease: A randomized controlled trial. *Neurorehabilitation and Neural Repair, 29,* 827–836.

Corcos, D. M., Robichaud, J. A., David, F. J., Leurgans, S. E., Vaillancourt, D. E., Poon, C., . . . Cornella, C. L. (2013). A two-year randomized control trial of progressive resistance exercise for Parkinson's disease. *Movement Disorders, 28,* 1230–1240.

Curtze, C., Nutt, J. G., Carlson-Kuhta, P., Mancini, M., & Horak, F. B. (2016). Objective gait and balance impairment related to balance confidence and perceived mobility in people with Parkinson's disease. *Physical Therapy, 96,* 1734–1743. doi:10.2522/ptj.20150662

Cury, R. G., Galhardoni, R., Fonoff, E. T., Perez Lloret, S., dos Santos Ghilardi, M. G., Barbosa, E. R., . . . Ciampi de Andrade, D. (2016). Sensory abnormalities and pain in Parkinson disease and its modulation by treatment of motor symptoms. *European Journal of Pain, 20,* 151–165.

Daley, D., Deane, K. H. O., Clark, A. B., Pfeil, M., Sabanathan, K., Worth, P. F., & Myint, P. (2014). Adherence therapy improves medication adherence and quality of life in people with Parkinson's disease: A randomized controlled trial. *International Journal of Clinical Practice, 68,* 963–971. doi:10.1111/ijcp.12439

Daley, D., O'Leary, K., Gray, D., Hill, R., & Myint, P. (2015). Qualitative evaluation of adherence therapy in Parkinson's disease: A multidirectional model. *Patient Preference and Adherence, 9,* 989–998.

De Rosa, A., Tessitore, A., Bilo, L., Peluso, S., & De Michele, G. (2016). Feature article: Infusion treatments and deep brain stimulation in Parkinson's disease: The role of nursing. *Geriatric Nursing, 37,* 434-439. doi:10.1016/j.gerinurse.2016.06.012

Deuschl, G., Schüpbach, M., Knudsen, K., Pinsker, M. O., Cornu, P., Rau, J., . . . Schade-Brittinger, C. (2013). Stimulation of the subthalamic nucleus at an earlier disease stage of Parkinson's disease: Concept and standards of the EARLYSTIM study. *Parkinsonism and Related Disorders, 19,* 56–61.

Dinkelbach, L., Möller, B., Witt, K., Schnitzler, A., & Südmeyer, M. (2017). How to improve patient education on deep brain stimulation in Parkinson's disease: The CARE Monitor study. *BMC Neurology, 17,* 36. doi:10.1186/s12883-017-0820-7

Dystonia Society. (n.d.) Physiotherapy: Generalized Dystonias. Retrieved from: https://www.dystonia.org.uk/physiotherapy

Elias, W. J. (2016). A Trial of Focused Ultrasound Thalamotomy for Essential Tremor. *The New England Journal Of Medicine*, *375*(22):2202-2203. doi:10.1056/NEJMc1612210.

Fagerlund, K., Anderson, L. C., & Gurvich, O. (2013). Perioperative medication withholding in patients with Parkinson's disease. *American Journal of Nursing, 113*(1), 26–35.

Fan, J., Chang, B., & Wu, Y. (2016). Relationships among depression, anxiety, sleep and quality of life in patients with Parkinson's disease in Taiwan. *Parkinson's Disease, 2016,* 1–8. doi:10.1155/2016/4040185

Fasano, A., & Lozano, A. M. (2015). Deep brain stimulation for movement disorders: 2015 and beyond. *Current Opinion in Neurology, 28*(4), 423. doi:10.1097/WC0.0000000000000226

Foster, E. (2014). Instrumental activities of daily living performance among people with Parkinson's disease without dementia. *American Journal of Occupational Therapy, 68,* 353–362. doi:10.5014/ajot.2014.010330

Foster, E. R., Golden, L., Duncan, R. P., & Earhart, G. M. (2013). Community-based Argentine tango dance program is associated with increased activity participation among individuals with Parkinson's disease. *Archives of Physical Medicine and Rehabilitation, 94,* 240–249.

Fox, M. D., & Alterman, R. L. (2015). Brain stimulation for torsion dystonia. *JAMA Neurology, 72*(6), 713. doi:10.1001/jamaneurol.2015.51

Ganesan, M., Sathyaprabha, T. N., Gupta, A., & Pal, P. K. (2014). Effect of partial weight-supported treadmill gait training on balance in PD patients with PD. *Physical Medicine and Rehabilitation, 6,* 22–33.

Gardner, J. (2013). A history of deep brain stimulation: Technological innovation and the role of clinical assessment tools. *Social Studies of Science, 43*(5), 707–728.

Ghoche, R. (2012). The conceptual framework of palliative applied to advanced Parkinson's disease. *Parkinsonism and Related Disorders, 18,* S2–S5.

Godden B. (2014). Deep Brain Stimulation for Parkinson's Disease. *Journal of Perianesthesia Nursing*. *29*(3):230-233. doi:10.1016/j.jopan.2014.03.006.

Goetz, C. G., Tilley, B. C., Shaftman, S. R., Stebbins, G. T., Fahn, S., Martinez-Martin, P., . . . LaPelle, N. (2008). Movement Disorder Society–sponsored revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS): Scale presentation and clinimetric testing results. *Movement Disorders, 23,* 2129–2170.

Grill, S. (2015). Implementing deep brain stimulation in practice: Models of patient care. In W. Marks Jr. (Ed.), *Deep brain stimulation management* (pp. 182–191). Cambridge, UK: Cambridge University Press. doi:10.1017/CBO9781316026625.014

Guridi, J., Gonzalez-Redondo, R., & Obeso, J. A. (2012). Clinical features, pathology and treatment of levodopa-induced dyskinesias in Parkinson's disease. *Parkinson's Disease, 2012.* 943159. doi:10.1155/2012/943159

Hack, N., Akbar, U., Monari, E. H., Eilers, A., Thompson-Avila, A., Hwynn, N. H., . . . Okun, M. S. (2014). Person-centered care in the home setting for Parkinson's disease. Operation house call quality of care pilot study. *Parkinson's Disease, 2015,* 639494. doi:10.1155/2015/639494

Hallett, M., Benecke, R., Blitzer, A., & Comella, C. L. Treatment of focal dystonias with botulinum neurotoxin. Toxicon. 2009;54:628-633. doi:10.1016/j.toxicon.2008.12.008.

Hallett, M., Albanese, A., Dressler, D., Segal, K. R., Simpson, D. M., Truong, D., & Jankovic, J. (2013). Evidence-based review and assessment of botulinum neurotoxin for the treatment of movement disorders. *Toxicon* (pp. 1060). doi:10.1016/j.toxicon.2012.12.004

Hariz, M., Blomstedt, P., & Zrinzo, L. (2013). Future of brain stimulation: New targets, new indications, new technology. *Movement Disorders, 28*(13), 1784–1792.

Hedera, P. (2017). Emerging strategies in the management of essential tremor. *Therapeutic Advances in Neurological Disorders, 10*(2), 137–148. doi:10.1177/1756285616679123

Hedera, P., Cibulcik, F., & Davis, T. L. (2013). Pharmacotherapy of essential tremor. *Journal of Central Nervous System Disease, 5,* 43–55. doi:10.4137/JCNSD.S6561

Hellqvist, C., & Bertero, C. (2015). Support supplied by Parkinson's disease specialist nurses to Parkinson's disease patients and their spouses. *Applied Nursing Research, 28,* 86–91. doi:10.1016/j.apnr.2014.12.008

Hobson, P., & Meara, J. (2015). Mild cognitive impairment in Parkinson's disease and its progression into dementia: A 16 year outcome evaluation of the Denbighshire cohort. *International Journal of Geriatric Psychiatry, 30,* 1048–1055. doi:10.1002/gps.4261

Hutchinson, K., & Wick, J. Y. (2016). Deep brain stimulation and medication management in Parkinson's disease. *The Consultant Pharmacist, 31*(2), 73–84. doi:10.4140/TCP.n.2016.73

Hyam, J. A., Pereira, E. C., McCulloch, P., Javed, S., Plaha, P., Mooney, L., . . . Green, A. L. (2015). Implementing novel trial methods to evaluate surgery for essential tremor. *British Journal of Neurosurgery, 29*(3), 334–339. doi:10.3109/02688697.2014.997670

Jhunjhunwala, K., & Pal, P. K. (2014). The non-motor features of essential tremor: A primary disease feature or just a secondary phenomenon? *Tremor and Other Hyperkinetic Movements, 2014,* 4, 255. doi:10.7916/D8D798MZ

Jinnah, H. A., Alterman, R., Klein, C., Krauss, J. K., Moro, E., Vidailhet, M., & Raike, R. (2017). Deep brain stimulation for dystonia: A novel perspective on the value of genetic testing. *Journal of Neural Transmission (Vienna, Austria: 1996), 124*(4), 417–430. doi:10.1007/s00702-016-1656-9

Jinnah, H. A., & Factor, S. A. (2015). Diagnosis and treatment of dystonia. *Neurologic Clinics, 33*(1), 77–100. doi:10.1016/j.ncl.2014.09.002

Julius, A., & Longfellow, K. (2016). Movement disorders: A brief guide in medication management. *Medical Clinics of North America, 100*(2016), 733–761. doi:10.1016/j.mcna.2016.03.002

Kalilani, L., Asgarnejad, M., Palokangas, T., & Durgin, T. (2016). Comparing the incidence of falls/fractures in Parkinson's disease patients in the US population. *PLoS One, 11*(9), e0161689.

Kang, M., & Ellis-Hill, C. (2015). How do people live life successfully with Parkinson's disease? *Journal of Clinical Nursing, 24,* 2314–2322. doi:10.1111/jocn.12819

Katus, L., & Shtilbans, A. (2014). Review: Perioperative management of patients with Parkinson's disease. *American Journal of Medicine, 127,* 275–280. doi:10.1016/j.amjmed.2013.11.014

Khan, T. S. (2012). Off spells and dyskinesias: Pharmacological management of motor complications. *Cleveland Clinic Journal of Medicine, 79,* S8–S13.

Kleiner-Fisman, G., Herzog, J., Fisman, D. N., Tamma, F., Lyons, K. E., Pahwa, R., . . . Deuschl, G. (2006). Subthalamic nucleus deep brain stimulation: Summary and meta-analysis of outcomes. *Movement Disorders, 21*(S14), S290. doi:10.1002/mds.20962

Koster, D., Higginson, C., & MacDougall, E. (2015). Subjective cognitive complaints in Parkinson disease without dementia: A preliminary study. *Applied Neuropsychology: Adult, 22,* 287–292. doi:10.1080/23279095.2014.925902

Kriebel-Gasparro, A. (2016). Featured Article: Parkinson's Disease: Update on Medication Management. *The Journal for Nurse Practitioners, 12*, e81–e89.

Kupsch, A., Benecke, R., Müller, J., Trottenberg, T., Schneider, G. H., Poewe, W., . . . Volkmann, J.; Deep-Brain Stimulation for Dystonia Study Group. (2006). Pallidal deep-brain stimulation in primary generalized or segmental dystonia. *New England Journal of Medicine, 355,* 1978–1990.

Kwak, J., Wallendal, M. S., Fritsch, T., Leo, G., & Hyde, T. (2014). Advance care planning and proxy decision making for patients with advanced Parkinson disease. *Southern Medical Journal, 107,* 178–185.

Lageman, S. K., Cash, T. V., & Mickens, M. N. (2014). Patient-reported needs, non-motor symptoms, and quality of life in essential tremor and Parkinson's disease. *Tremor and Other Hyperkinetic Movements, 4,* 240. doi:10.7916/D8RF5S4J

Lageman, S. K., Mickens, M. N., & Cash, T. V. (2015). Caregiver-identified needs and barriers to care in Parkinson's disease. *Geriatric Nursing, 36*(3), 197–201.

Lee, N., Lee, D., & Song, H. (2015). Effect of virtual reality dance exercise on the balance, activities of daily living and depressive disorder status of Parkinson's disease patients. *Journal of Physical Therapy Science, 27,* 145–147.

Li, F., Harmer, P., Liu, Y., Eckstrom, E., Fitzgerald, K., Stock, R., & Chou, L. (2013). A randomized controlled trial of patient reported outcomes with tai chi exercise in Parkinson's disease. *Movement Disorders, 29,* 539–545.

Lopiano, L., Modugno, N., Marano, P., Sensi, M., Meco, G., Cannas, A., . . . Antonini, A. (2016). Motor outcomes in patients with advanced Parkinson's disease treated with levodopa/carbidopa intestinal gel in Italy: An interim analysis from the GREENFIELD observational study. *Neurological Science, 37,* 1785–1792.

Louis, E. D., Rohl, B., & Rice, C. (2015). Defining the treatment gap: What essential tremor patients want that they are not getting. *Tremor and Other Hyperkinetic Movements, 5,* 331. doi:10.7916/D87080M9

Lumsden, D. E., Fairhurst, C., Lin, J.-P., & Lundy, C. (2013). Dystonia Severity Action Plan: A simple grading system for medical severity of status dystonicus and life-threatening dystonia. *Developmental Medicine and Child Neurology.* doi:10.1111/dmcn.12108

Machado, A. (2006). Deep brain stimulation for Parkinson's disease: Surgical technique and perioperative management. *Movement Disorders, 21*(suppl 14), S247–S258.

Malek, N., & Grosset, D. (2014). Medication adherence in patients with Parkinson's disease. *CNS Drugs, 29,* 47–53. doi:10.1007/s40263-014-0220-0

Mangram, A. J., Horan, T., Pearson, M. L., Silver, L. C., & Jarvis, W. R. (1999). Guideline for prevention of surgical site infection, 1999. *Infection Control and Hospital Epidemiology, 20*(4), 250. doi:10.1086/501620

Manor, Y., Mootanah, R., Freud, D., Giladi, N., & Cohen, J. T. (2013). Video-assisted swallowing therapy for patients with Parkinson's disease. *Parkinsonism and Related Disorders, 19,* 207–211.

Marsh, W. A., Monroe, D. M., Brin, M. F., & Gallagher, C. J. Systematic review and meta-analysis of the duration of clinical effect of onabotulinumtoxinA in cervical dystonia. *BMC Neurology*. 2014;14:91. doi:10.1186/1471-2377-14-91

Marques, R. E., Duarte, G. S., Rodrigues, F. B., et al. Botulinum toxin type B for cervical dystonia. *Cochrane Database of Systematic Reviews*. 2016(5). doi:10.1002/14651858.CD004315.pub3

Martinez-Ramirez, D., & Okun, M. S. (2014). Rationale and clinical pearls for primary care doctors referring patients for deep brain stimulation. *Gerontology, 60*(1), 38–48. doi:10.1159/000354880

Miyasaki, J., Kluger, B. (2015). Palliative Care for Parkinson's Disease: Has the Time Come? *Current Neurology & Neuroscience Reports*, *15*(5):1.

Medtronic Clinician Manual. (2017). Retrieved from http://manuals.medtronic.com/content/dam/emanuals/neuro/M927893A_b_056_view.pdf

Morgan, J. C., & Fox, S. H. (2016). Treating the symptoms of Parkinson disease. *Continuum, 22,* 1064–1085.

Morita, H., Susatia, F., Foote, K., & Okun, M. (2015). Assessing patient outcome and troubleshooting deep brain stimulation. In W. Marks Jr. (Ed.), *Deep brain stimulation management* (pp. 138–181). Cambridge, UK: Cambridge University Press. doi:10.1017/CBO9781316026625.004

Munhoz, R., Picillo, M., Fox, S., Bruno, V., Panisset, M., Honey, C., & Fasano, A. (2016). Eligibility Criteria for Deep Brain Stimulation in Parkinson's Disease, Tremor, and Dystonia. *Canadian Journal of Neurological Sciences/Journal Canadien Des Sciences Neurologiques*, *43*(4), 462-471. doi:10.1017/cjn.2016.35

Muzerengi, S., Herd, C., Rick, C., & Clarke, C. (2016). A systematic review of interventions to reduce hospitalization in Parkinson's disease. *Parkinsonism and Related Disorders, 24,* 3–7. doi:10.1016/j.parkreldis.2016.01.011

Nadeau, A., Pourcher, E., & Corbeil, P. (2014). Effects of 24 weeks of treadmill training on gait performance in Parkinson's disease. *Medicine & Science in Sports & Exercise, 46*(4), 645–655. doi:10.1249/MSS.0000000000000144

Nakae, H., & Tsushima, H. (2014). Effects of home exercise on physical function and activity in home care patients with Parkinson's disease. *Journal of Physical Therapy Science, 25,* 1701–1706.

Nolden, L. F., Tartavoulle, T., & Porche, D. J. (2014). Parkinson's disease: Assessment, diagnosis and management. *Journal for Nurse Practitioners, 10,* 500–506.

Pal, G. D., Persinger, V., Bernard, B., Ouyang, B., Goetz, C. G., & Verhagen Metman, L. (2015). The Core Assessment Program for Surgical Interventional Therapies in Parkinson's Disease (CAPSIT-PD): Tolerability of preoperative neuropsychological testing for deep brain stimulation in Parkinson's disease. *Movement Disorders Clinical Practice, 2*(4), 379–383.

Panov, F., Gologorsky, Y., Connors, G., Tagliati, M., Miravite, J., Alterman, R. L. (2013, July). Deep brain stimulation in DYT1 dystonia: A 10-year experience. *Neurosurgery, 73*(1), 86–93.

Parkinson's Foundation. *Understanding Parkinson's*. Retrieved from http://www.parkinson.org/understanding-parkinsons

Pena, J., Ibarretx-Bilboa, N., Garcia-Gorostiga, I., Gomez-Beldarrain, M. A., Diez-Cirarda, M., & Ojeda, N. (2014). Improving functional disability and cognition in Parkinson disease. *Neurology, 83,* 2167–2174.

Perestelo-Pérez, L., Rivero-Santana, A., Pérez-Ramos, J., Serrano-Pérez, P., Panetta, J., & Hilarion, P. (2014). Deep brain stimulation in Parkinson's disease: Meta-analysis of randomized controlled trials. *Journal of Neurology, 261*(11), 2051–2060. doi:10.1007/s00415-014-7254-6

Picelli, A., Melotti, C., Origano, F., Neri, R., Waldner, A., & Smania, N. (2013). Robot-assisted gait training versus equal intensity treadmill training in patients with mild to moderate Parkinson's disease: A randomized controlled trial. *Parkinsonism and Related Disorders, 19,* 605–610.

Picillo, M., Kou, N., Puppi Munhoz, R., Fasano, A., & Lozano, A. (2016a). Programming deep brain stimulation for tremor and dystonia: The Toronto Western Hospital Algorithms. *Brain Stimulation, 9*(3), 438–452. doi:10.1016/j.brs.2016.02.003

Picillo, M., Lozano, A. M., Kou, N., Puppi Munhoz, R., & Fasano, A. (2016b). Programming deep brain stimulation for Parkinson's disease: The Toronto Western Hospital Algorithms. *Brain Stimulation, 9,* 425–437. doi:10.1016/j.brs.2016.02.004

Pistacchi, M., Gioulis, M., Contin, F., Sanson, F., & Marsala, S. (2015). Cognitive profiles in mild cognitive impairment (MCI) patients associated with Parkinson's disease and cognitive disorders. *Annals of Indian Academy of Neurology, 18*(2), 200–205. doi:10.4103/0972-2327.150611

Rabin, S., & Kumar, R. (2015). Managing Parkinson's disease patients with deep brain stimulation. In W. Marks Jr. (Ed.), *Deep brain stimulation management* (pp. 84–107). Cambridge, UK: Cambridge University Press. doi:10.1017/CBO9781316026625.014.

Rai, N., Goyal, V., Kumar, N., Shukla, G., Srivastava, A., Singh, S., & Behari, M. (2015). Neuropsychiatric co-morbidities in non-demented Parkinson's disease. *Annals of Indian Academy of Neurology, 18*(1), 33–38.

Ranaa, A. O., Kabirb, A., Jesudasana, M., Siddiquia, I., & Khondkerb, S. (2013). Pain in Parkinson's disease: Analysis and literature review. *Clinical Neurology and Neurosurgery, 115,* 2313–2317.

Rasouli, J., Panov, F., Zhang,Y., Ramdhani, R., Cho, C., Dimov, A., . . . Kopell, B. (2018). Utilization of quantitative susceptibility mapping for direct targeting of the subthalamic nucleus during deep brain stimulation surgery. *Operative Neurosurgery, 14*(4), 412–419.

Revell, M. A. (2015). Deep brain stimulation for movement disorders. *Nurses Clinics of North America, 50*(2015), 691–701. doi:10.1016/j.cnur.2015.07.014

Rinalduzzi, S., Trompetto, C., Marinelli, L., Alibardi, A., Missori, P., Fattapposta, F., . . . Curra, A. (2015). Balance dysfunction in Parkinson's disease. *BioMed Research International, 2015,* 434683, 1–10. doi:10.1155/2015/434683

Rodrigues, N. B., Mithani, K., Meng, Y., Lipsman, N., & Hamani, C. (2018). The emerging role of tractography in deep brain stimulation: Basic principles and current applications. *Brain Sciences, 8*(2), 23.

Sapir, S., Ramig, L. O., & Fox, C. M. (2011). Intensive voice treatment in Parkinson's disease: Lee Silverman Voice Treatment. *Expert Review of Neurotherapeutics, 11,* 815–830.

Shah, S. P., Glenn, G. L., Hummel, E. M., Hamilton, J. M., Martine, R. R., & Wilkinson, J. R. (2015). Caregiver tele-support group for Parkinson's disease: A pilot study. *Geriatric Nursing, 36,* 207–211.

Shanker, V., & Bressman, S. B. (2016). Diagnosis and management of dystonia. *Continuum, 22*(4, Movement Disorders), 1227–1245. doi:10.1212/CON.0000000000000352

Sharma VD, Lyons KE, & Pahwa R. (2018). Amantadine extended-release capsules for levodopa-induced dyskinesia in patients with Parkinson's disease. Therapeutics and Clinical Risk Management, Vol Volume 14, Pp 665-673 (2018), 665.

Shaw, K. D., Johnston, A. S., Rush-Evans, S., Prather, S., & Maynard, K. (2017). Nursing management of the patient undergoing focused ultrasound: A new treatment option for essential tremor. *Journal of Neuroscience Nurses, 49*(5), 307–310.

Shukla, W. A., & Okun, M. S. (2016). State of the art for deep brain stimulation therapy in movement disorders: A clinical and technological perspective. *IEEE Reviews in Biomedical Engineering, 9,* 219–233. doi:10.1109/RBME.2016.2588399

Sillay, K. A., Rusy, D., Buyan-Dent, L., Ninman, N. L., & Vigen, K. K. (2014). Wide-bore 1.5T MRI-guided deep brain stimulation surgery: Initial experience and technique comparison. *Clinical Neurology and Neurosurgery, 127,* 79–85.

Simpson, D. M., Blitzer, A., Brashear, A., et al. (2008). Assessment: Botulinum neurotoxin for the treatment of movement disorders (an evidence-based review): report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology*. *70*(19):1699-1706. doi:10.1212/01.wnl.0000311389.26145.95.

Simpson, D. M. (2016). Evidence-based approach to use of botulinum toxin in selected disorders. *Toxicon*, *123*(Supplement), S77. https://doi.org/10.1016/j.toxicon.2016.11.213

Sokal, P., Harat, M., Rusinek, M., Rudaś, M., & Litwinowicz, A. (2015). Intraoperative CT verification of electrode localization in DBS surgery in Parkinson's disease. *Interdisciplinary Neurosurgery, 2*(1), 6–9.

St. Jude Medical. (2015). *St. Jude Medical Infinity™ IPG clinician's manual*. Plano, TX: Author. Retrieved from https://manuals.sjm.com

Sturkenboom, I. H. W. M., Graff, M. J. L., Hendriks, J. C. M., Veenhuizen, Y., Munneke, M., Bloem, B. R., . . . Nijhuis-van der Sanden, M. W. (2014). Efficacy of occupational therapy for patients with Parkinson's disease: A randomized controlled trial. *Lancet, 13,* 557–566.

Timmermann, L., Jain, R., Chen, L., Maarouf, M., Barbe, M. T., Allert, N., … Alesch, F. (2015). Articles: Multiple-source current steering in subthalamic nucleus deep brain stimulation for Parkinson's disease (the VANTAGE study): a non-randomised, prospective, multicentre, open-label study. *The Lancet Neurology, 14*, 693–701.

Trenkwalder, C., Chaudhuri, K. R., Garcia Ruiz, P. J., LeWitt, P., Katzenschlager, R., Sixel-Döring, F., . . . Wenzel, K. (2015). Expert consensus group report on the use of apomorphine in the treatment of Parkinson's disease: Clinical practice recommendations. *Parkinsonism and Related Disorders, 21,* 1023–1030.

Tuck, K. K., Brod, L., Nutt, J., & Fromme, E. K. (2015). Preferences of patients with Parkinson's disease for communicating about advanced care planning. *American Journal of Hospice,* 32(1), 68–77.

Uc, E. Y., Doerschug, K. C., Magnotta, V., Dawson, J. D., Thomsen, T. R., Kline, J. N., Darling, W. G. (2014). Phase I/II randomized trial of aerobic exercise in Parkinson disease in a community setting. *Neurology, 83,* 413–425.

Valkovi, P., Minar, M., Singliarova, H., Harsany, J., Hanakova, M., & Benetin, J. (2015). Pain in Parkinson's disease: A cross-sectional study of its prevalence, types, and relationship to depression and quality of life. *PLOS One, 10*(8), e0136541. doi:10.1371/journal.pone.0136541

van der Marck, M. A., Bloem, B. R., Borm, G. F., Overeem, S., Munneke, M., & Guttman, M. (2013). Effectiveness of multidisciplinary care for Parkinson's disease: A randomized, controlled trial. *Movement Disorders, 28,* 605–611.

van der Marck, M. A., Munneke, M., Mulleners, W., Hoogerwaard, E. M., Borm, B. R., Overeem, S., & Bloem, B. R.; IMPACT study group. (2013). Integrated multidisciplinary care in Parkinson's disease: a non-randomized, controlled trial (IMPACT). *Lancet, 12,* 947–956.

Volkmann, J., Chabardes, S., Schuurman, P. R., Blum, D., Steinke, G. K., Carcieri, S., . . . Van Dyck, N. (2016). DIRECT DBS: A prospective, multi-center clinical trial with blinding for a directional DBS lead. *Stereotactic & Functional Neurosurgery, 94,* 87.

Volkmann, J., Mueller, J., Deuschl, G., Kühn, A. A., Krauss, J. K., Poewe, W., . . . Benecke, R. (2014). Pallidal neurostimulation in patients with medication-refractory cervical dystonia: A randomised, sham-controlled trial. *Lancet Neurology, 13*(9), 875–884. doi:10.1016/S1474-4422(14)70143-7

Volkmann, J., Wolters, A., Kupsch, A., Müller, J., Kühn, A. A., Schneider, G., . . . Benecke, R. (2012). Pallidal deep brain stimulation in patients with primary generalised or segmental dystonia: 5-year follow-up of a randomised trial. *Lancet Neurology, 11*(12), 1029–1038. doi:10.1016/S1474-4422(12)70257-0

Volpe, D., Giantin, M. G., Maestri, R., & Frazzitta, G. (2014). Comparing the effects of hydrotherapy and land-based therapy on balance in patients with Parkinson's disease: A randomized controlled pilot study. *Clinical Rehabilitation, 28,* 1210–1217.

Ward, H. E. (2009). Chapter 9: Psychiatric considerations in the dystonia patient. In *Dystonia patient: A guide to practical management* (pp. 211–225). New York, NY: Demos Medical Publishing, LLC.

Weaver, F. M., Follett, K. A., Stern, M., Luo, P., Harris, C. L., Hur, K., . . . Reda, D. J. (2012). Randomized trial of deep brain stimulation for Parkinson disease: Thirty-six-month outcomes. *Neurology, 79*(1), 55–65. doi:10.1212/WNL.0b013e31825dcdc

Zesiewicz, T. A., Elble, R. J., Louis, E. D., Gronseth, G. S., Ondo, W. G., Dewey, R. B., . . . Weiner, W. J. (2011). Evidence-based guideline update: Treatment of essential tremor. *Neurology, 77,* 1752–1755.

Zimmerman, R., Gschwandtner, U., Benz, N., Hatz, F., Schindler, C., Taub, E., & Fuhr, P. (2014). Cognitive training in Parkinson disease: Cognitive-specific vs nonspecific computer training. *Neurology, 82,* 1219–1226.