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Common neurodegenerative disorders in the perioperative setting: Recommendations for screening from the Society for Perioperative Assessment and Quality Improvement (SPAQI)

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Abstract

Aging is associated with normal and abnormal brain and cognitive changes. Due to the expected increase in older adults requiring surgery, perioperative clinicians will be increasingly encountering patients with neurodegenerative disease. To help perioperative clinicians understand signs of abnormal behaviors that may mark an undiagnosed neurodegenerative disorder and alert additional patient monitoring, The Society for Perioperative Assessment and Quality Improvement (SPAQI) worked with experts in dementia, neuropsychology, geriatric medicine, neurology, and anesthesiology to provide a summary of cognitive and behavioral considerations for patients with common neurodegenerative disorders being evaluated at preoperative centers. Patients with neurodegenerative disorders are at high risk for delirium due to known neurochemical disruptions, medication interactions, associated frailty, or vascular risk profiles presenting risk for repeat strokes. We provide basic information on the expected cognitive changes with aging, most common neurodegenerative disorders, a list of behavioral features and considerations to help differentiate neurodegenerative disorders. Finally, we propose screening recommendations intended for a multidisciplinary team in the perioperative setting.

Keywords

neurodegenerative syndromes; dementias; perioperative care

Introduction

Individuals aged 80 years and older are expected to triple in number between 2015 and 2050, growing from neurodegenerative disorders specifically Alzheimer’s Disease (AD), Vascular Dementia (VaD), and Parkinson’s Disease (PD). These disorders are predicted to pose significant strains on economies, health systems, and social structures worldwide.

Coupled with the exponential growth of the aging population is an increased need or desire for surgical interventions. Up to 20% of those who present for elective surgery show early signs of cognitive impairment [2, 3]. Despite this, healthcare professionals know very little about how neurodegenerative type, severity (measured by pathology, neuroimaging, or cognition), and neurodegenerative pharmaceuticals interact with anesthesia agents or respond to different intraoperative monitoring approaches [4]. Currently, patients with AD and other neurodegenerative disorders receive *comparable* perioperative care to that of their cognitively-intact counterparts [5]. Patient-centric needs are not often proactively considered.

A first step towards addressing this healthcare disparity is improved awareness of common neurodegenerative profiles and appreciation of behavioral or cognitive nuances during the preoperative encounter. We target perioperative clinicains because these professionals have unique opporutnities to observe perioperative behavioral changes. During the preoperative interview, perioperative clinicians converse with patients prior to surgery, observe brain and bodily responses throughout intraoperative events, and assess arousal and behavior during the postoperative period. Behavior predicts regions of brain pathology [6]. By paying attention to behavioral cues, anesthesia practitioners will develop insight into intraoperative monitor markers of disease [7] and appreciate subtle nuances of pre- to postoperative behavioral changes suggesting altered brain recovery. Indeed, preoperative cognition correlates with intraoperative electroencephalogram metrics [8]. Preoperative neuroimaging markers of abnormal aging including increased white matter abnormalities, ventricle size, and thinner entorhinal cortices associate with increased intraoperative variability on routine two-channel derived electroencephalographic (EEG) monitors [9]. Early detection of neurodegenerative disorders (either diagnosed or undiagnosed) can facilitate informed decision-making [10] and assist with future planning. For example, choosing treatment as usual, consultation for prehabilitation, alterations in intraoperative care [11], or early postoperative interventions.

To help perioperative clinicians understand behavioral signs of neurodegenerative disorders and prompt additional monitoring, the Society for Perioperative Assessment and Quality Improvement (SPAQI) worked with experts in dementia, neuropsychology, geriatric medicine, neurology, and anesthesiology to provide a summary of cognitive and behavioral considerations for patients with select neurodegenerative disorders. Patients with any of the following are high risk for delirium due to known neurochemical disruptions, medication interactions (e.g., levodopa), associated frailty, or vascular risk profiles suggesting high risk for repeat stroke. A summary of “normal” cognitive changes with aging is provided followed by brief synopses of neurodegenerative disorders.

Cognitive Changes Related to Normal Aging

Normal aging is associated with subtle decrements in cognition, including slower processing speed, reduced mental flexibility, and diminished fluid reasoning relative to younger age groups [12, 13]. Crystallized knowledge such as single word reading and reasoning remain relatively consistent with premorbid demographic, educational, and occupational status. Additionally, day-to-day functions remains relatively preserved. Medication management, financial planning, home maintenance, shopping, meal preparation, and ability to engage in social activities remain unchanged relative to younger years [14].

Normal aging is associated with mild reductions in brain integrity. Cross sectional studies show that aging involves reductions in whole brain volume and increases in cerebrospinal fluid [15]. Within the brain, white matter volume declines during the middle-age years while cortical gray matter loss occurs later [16]. The most common white matter changes observed in normal aging involve leukoaraiosis (LA) [17, 18], such that cognitively-well older adults have LA within one percent or less of their white matter fibers [19] . LA is most commonly located around the lateral ventricles (called periventricular LA) [17]. Age-related brain

changes also occur at the neurotransmitter level. Acetylcholine (Ach) is an important neurotransmitter involved in cortical activation, arousal, and cognitive functioning [20, 21]. Cell bodies of neurons that provide acetylcholinergic innervation to the cortex reside within the basal nucleus of Meynert (BNM), which is anterior to the thalamus and basal ganglia [21, 22]. Cholinergic axons linking the BNM and the cortex are mostly unmyelinated [23] and thus are more vulnerable to aging-related vascular changes in the brain as well as pathologies of AD and small vessel VaD.

Abnormal Cognitive Aging

Abnormal cognitive aging is intrinsically different from the cognitive changes of normal aging. Symptoms of abnormal aging include problems around learning and memory, as well as the cognitive domains of executive function, language, visuospatial function, and emotional stability. Neurodegeneration is typically insidious, progresses over time, and associates with a reduction in day to day functional abilities [24].

In the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-V American Psychiatric Association [APA]) [25], neurodegenerative syndromes are divided into Mild Neurocognitive Disorder and Major Neurocognitive Disorder (also known as dementia). It should be noted that “Mild Neurocognitive Disorder” and “Major Neurocognitive Disorder” are relatively recent diagnoses that were introduced in the DSM-V published in 2013. As such, most health care providers continue to use the terms “Mild Cognitive Impairment” (MCI) and “Dementia” to refer to neurodegenerative disorders. The relationships between Mild Neurocognitive Disorder, Major Neurocognitive Disorder, and other contemporary terms (i.e., Mild Cognitive Impairment and Dementia) are described in more detail below.

i. *Mild Neurocognitive Disorder* is a clinical diagnosis based on evidence (self-reported or obtained via objective measures) of mild cognitive decline from a previous level of performance [25]. Changes occur within domains of processing speed, attention, working memory, learning and memory, language, perceptual-spatial, executive function, or social cognition. Change in cognitive function is based on one of the following: 1. A concern based on self-report of the individual or a knowledgeable informant; 2. A modest impairment in cognitive performance preferably documented by neuropsychological testing which does not require a certain standard deviation of decline. The cognitive changes do not interfere with capacity to function independently, cannot occur exclusively in the context of delirium, and cannot be explained by another mental disorder [25]. Classification of Mild Neurocognitive Disorder may include information about disease etiology (i.e., AD, VaD). Finally, clinicians specify whether or not the patient has active behavioral disturbances such as psychotic symptoms, mood disturbance, agitation, apathy, and other behavioral symptoms. There is no formal staging of Mild Neurocognitive Disorder in the DSM-V in regard to disease severity. The term Mild Cognitive Impairment (MCI) is subsumed under the Mild Neurocognitive Disorder umbrella. MCI was initially used to specifically describe the prodromal phase of AD [26]. More recently, MCI represents the prodromal phase of any form of dementia including AD, VaD, PD Dementia (PDD), and Frontotemporal Dementia (FTD).

The definitions of Mild Neurocognitive Disorder and MCI differ in a number of ways. First, the DSM-V criteria for Mild Neurocognitive Disorder does not quantify how much change from baseline warrants a formal diagnosis [25]. In MCI, a change of at least 1 standard deviation in a given score should be present prior to establishing the diagnosis [26, 27]. A second difference revolves around disease classification. As stated earlier, Mild Neurocognitive Disorder is classified based on the hypothesized disease. For example, Mild Neurocognitive Disorder due to human immunodeficiency virus infection or due to PD. In contrast, MCI is classified based on the type and number of neurocognitive domains affected. As such, four different MCI types are currently recognized in the literature: MCI single domain amnestic, MCI single domain non-amnestic, MCI multiple domain amnestic, and MCI multiple domain non-amnestic.

ii. *Major Neurocognitive Disorder or dementia* involves evidence of significant decline from a previous level of performance in one or more cognitive domains (e.g., complex attention, language, perceptual-spatial, executive function, motor, social cognition). Change in cognitive function is based on either: 1. A concern of the individual or a knowledgeable informant 2. A substantial impairment in cognitive performance ideally based on neuropsychological testing that cannot exclusively occur in the context of delirium [25]. Contrary to Mild Neurocognitive Disorder, the degree of cognitive impairment must interfere with everyday functioning, and the stage of the disorder is informed by the level of functional limitations reported by the patient. In other words, Major Neurocognitive Disorder - mild, refers to subtle difficulties with instrumental activities of daily living (ADLs) such as housework or managing money. Major Neurocognitive Disorder - moderate, refers to difficulties with basic ADLs such as feeding, dressing, or bathing. Major Neurocognitive Disorder - severe, refers to a state of full reliance on others to complete basic and instrumental ADLs.

It is important to note that the decline in functional abilities is the differentiating factor between Mild Neurocognitive Disorder and Major Neurocognitive Disorder such that individuals with Mild Neurocognitive Disorder do not experience impairments in everyday functioning. In this context, functional status is divided into basic ADLs and instrumental ADLs. Basic ADLs are a set of activities that are necessary for independent living. Ability to maintain hygiene, consume nutrients, maintain continence, and move around safely are all considered basic ADLs. Instrumental ADLs (IADLs) refer to actions that are important, but not necessary for independent living. Transportation, meal preparation, medication dispensing, and financial management are all considered IADLs. The Physical Self Maintenance Scale [28] is a frequently used measure that assesses basic ADLs. The Lawton Instrumental Activities of Daily Living Scale [28] and the Functional Activities Questionnaire (FAQ) [29] are commonly used to measure IADLs and typically take less than five minutes to administer.

It is critical to assess for ADL/IADL functioning in the preoperative setting. Preoperative functional status can help physicians identify cases wherein caregiving assistance is needed to complete medical recommendations in preparation for a procedure. In patients undergoing high risk procedures (e.g., cardiac surgery), baseline functional status predicts postoperative cognitive recovery [30]. Dysfunction in IADL performance can also occur postoperatively

[31], and may increase caregiving demands. In addition to ADLs and IADLs, behavioral profiles, type of cognitive impairment, medical status, and information about comorbidities all provide diagnostic information on primary disease location, possible pathology, and consequently the diagnosis assigned (e.g., Major Neurocognitive Disorder due to AD).

Common Major Neurocognitive Disorders (Dementias)

The next section briefly characterizes the Major Neurocognitive Disorders clinicians are most likely to encounter in perioperative settings.

a. Alzheimer's Disease (AD)

Between the years of 2010 and 2050 the number of individuals with AD is expected to triple [32]. Hallmark neuropathological features traditionally associated with AD include the presence of amyloid-beta peptide plaques (Abeta, Aß) and neurofibrillary tangles made of accumulated hyperphosphorylated tau protein (tau). Elevated levels of Aß as well as abnormal tau phosphorylation lead to cell death and disrupt the cytoarchitecture of cells [33, 34]. Measurement of Aß, total tau (t-tau), and hyperphosphorylated tau (p-tau) is possible via collection of cerebrospinal fluid (CSF) [33] and neuroimaging techniques such as positron emission tomography (PET). Imaging studies, which are particularly useful in identifying affected brain regions, suggest that the entorhinal cortex in the medial temporal lobe is one of the first regions to show AD pathology [35]. Over time, the pathology progresses in an outward fashion towards the inferior frontal regions, the lateral temporal lobe, and the parietal regions, until it arrives at motor and sensory regions. While CSF and neuroimaging techniques have improved our classification of AD in the prodromal and preclinical phases, challenges related to the invasiveness of performing CSF sampling and limited access to PET scans inhibit the utility of these assessment techniques in clinical practice [36]. Moreover, CSF and imaging biomarkers do not perfectly forecast neuropsychological decline and have limited utility in predicting functional changes.

The classic cognitive profile of AD has sometimes been described as a *cortical* dementia and is associated with degradation of the gray matter (cell bodies) and secondary changes in subcortical white matter fibers (cell axons) and subcortical gray matter nuclei [37]. AD-related brain changes first occur in the hippocampus and other surrounding structures, a region instrumental to the formation of new memories. Thus, marked deficiencies in learning and memory constituting evidence for an anterograde amnesia for new information (inability to retain new information after a short time delay in which the individual is given distraction tasks) are hallmark signs of AD and are present in even the milder stages of the disease. As the disease progresses, prominent lexical access deficits involving word finding and naming difficulties emerge.

There are subtle clinical signs that may allude to an underlying AD pathology. Patients with AD may exhibit word-finding difficulties and may be prone to using generic words such as "thing" to refer to common objects. Since AD involves a degradation of networks that support knowledge of objects and the meaning of concepts, patients with this pathology may have difficulties accessing the unique features associated with an object. For example, they may have difficulty producing terms that are related to one another and describing the

essential features of common objects. In contrast, individuals with AD may be able to maintain excellent eye contact, demonstrate appropriate attention and problem-solving skills, and engage in basic conversations. Particularly during the early stages of the disease, signs of underlying impairment may not be readily apparent and may only emerge when these patients are asked pointed questions about orientation or with detailed neuropsychological assessment.

Physically, individuals with mild AD typically show normal, preserved gait and balance. In later stages of the disease, changes in body composition (i.e., percentage of fat, muscle, and water) as well as problems with balance can be observed and are likely secondary to systemic deterioration [38]. In patients with suspected neurodegenerative processes, assessing frailty using brief available tools may assist with differential diagnosis.[39, 40].

b. Vascular Dementia (VaD)

There is considerable overlap between cerebrovascular disease and AD. This has been shown in epidemiological, clinical, and pathological data [41]. Schneider et al., (2009) observed mixed AD/vascular neuropathology in approximately 50 percent of an autopsy sample. For this reason, references to the AD/VaD spectrum disorders are becoming more common.[42] For individuals whose dementia is primarily due to cerebrovascular disease common co-morbidities include hypertension, hypercholesterolemia, diabetes, and stroke. Classically, the two major subtypes of VaD include dementia due to small-vessel disease and large-vessel disease.

i. *Small-vessel vascular disease* is the most common form of VaD and frequently occurs with AD [43]. Like AD, small vessel VaD is insidious and progressive [44]. Pathologically, small vessel vascular disease on neuroimaging will present with white matter abnormalities around the lateral ventricles and in the deep white matter [45, 46]. Clinically, there is gradual worsening of cognition and psychiatric symptoms. Deficits attributable to executive impairment pervade the overall neuropsychological presentation [19]. Individuals may exhibit slower thinking speed, impulsivity, and distractibility. These patients might struggle to sustain their attention over time and are more likely to produce repetitive errors (perseverations) [47]. In terms of learning and memory, adults with small vessel VaD exhibit inefficient learning, marginally benefit from repetition, and often demonstrate errors in source memory. In other words, the source of where information was learned is incorrectly attributed to some other experience (e.g., attributing information provided by a second provider to details provided by a first provider earlier in the current visit). Psychiatric symptoms are also present, including higher rates of depression and apathy [48].

ii. *Large-vessel vascular disease (Multi-Infarct Dementia)* is a condition characterized by damage to the major arteries in the brain [49, 50] and is less common than small-vessel vascular disease [51]. Individuals with large vessel VaD may show a step-wise decline in cognitive functioning. These individuals decline with the first stroke, followed by a period of cognitive stability, followed by an additional stroke and a further drop in cognitive function, followed by stability. These individuals are at a higher risk for additional strokes and subsequent decline in cognitive function. Individuals can have a wide variety of cognitive

and emotional profiles depending on the location of the strokes. Physically, there may be gait and balance impairments [51].

c. Parkinson's Disease (PD) and Parkinson's Disease Dementia (PDD)

Parkinson's Disease (PD) impacts at least 1.5 million people in the United States alone. Its prevalence increases with age, and most diagnoses occur after age 60 years [52]. Although diagnosis of PD does not, in and of itself, suggest the presence of dementia, the majority of PD patients who survive more than 10 years after the onset of PD will progress to a dementia state [53]. Additionally, 20%-40% of individuals with PD meet criteria for PD-MCI at the time of PD diagnosis and the point prevalence of PD-MCI is 25%-50% [54].

Adults with PD and with Major Neurocognitive Disorder due to PD exhibit reduced pigmentation in the substantia nigra as well as reduced volume in the putamen and the caudate nucleus [55, 56]. Pathologically, neuronal intracytoplasmic filamentous inclusions, referred to as Lewy bodies (LBs), are frequent in the substantia nigra and are considered the histological hallmark of PD. Abnormalities within the neuronal support cells, glial cells that are responsible for myelination of the white matter fibers and regulation of iron metabolisms, have been linked to a mutation of NACP/α-synuclein - a presynaptic protein whose physiological role is associated with synaptic function.[57–59] NACP/α-synuclein-positive glial inclusions and LBs have been identified even in the intact white matter of the cerebrum, cerebellum, and spinal cord of deceased PD patients. Additionally, PD staging criteria report infiltration of pathology from the nuclei brainstem to the gray matter of the frontal cortex, with suggested pathways following ventral (temporal) and medial dorsal subcortical gray matter to the frontal streams [60].

The primary cognitive deficit of PD involves a reduction in speed of information processing [61]. For example, patients may be slow to acquire new information, or slow to retrieve words during conversation. These patterns are consistent with pathological changes to the frontal white matter regions of the brain and subcortical gray matter (caudate nuclei and thalami) [56]. As the pathology progresses through the cortical regions of the brain, other cognitive difficulties emerge, and cognitive and motor symptoms result in reduced quality of life, nursing home placement, and increased mortality.

During interviews, a clinician may notice resting tremors, rigidity and reduced word retrieval during conversational speech. Even if tremors are present, motor and cognitive screening tests (e.g., grip strength or clock drawing tests) should be administered. Tremors associated with PD are typically at rest and may reduce when the patient is performing a goal-directed action. It is important to gather as much information as possible about cognitive and motor function, as patients with PD are at heightened risk of delirium and postoperative cognitive complications. Establishing a baseline is consequently important for predicting risk and recognizing changes which may develop postoperatively. Other considerations include evaluating for gait disturbances, depression, apathy, orthostatic hypotension, likelihood of falls, sleep disturbances (particularly REM sleep disorder) and constipation. Some of which could be the result of treatment medications.

d. Dementia with Lewy Bodies (DLB)

Major Neurocognitive Disorder due to DLB accounts for approximately 1%-2% of the 65 and older population [62] and approximately 5% of all dementias over the age of 75 years of age [63]. DLB is more common in men and its presentation can mimic that of individuals with PD and AD. In fact, DLB can be misdiagnosed as AD early in the disease course [64] and DLB also shares many features with PD. When presented together, symptoms of both AD and PD combine to represent the umbrella term “Lewy body dementia.” In contrast to PD, cognitively impairment is present early in the disease course, with Parkinsonism motor features occurring at the same time or after dementia onset. Visual hallucinations, cognitive fluctuations, and REM sleep behavior disorder are other core DLB features [65]. Neuropathologically, DLB is a disorder with alpha-synuclein deposition and Lewy bodies that infiltrate the subcortical and cortical regions. There is a higher rate of earlier mortality after diagnosis with the median time from estimated first cognitive symptoms to death being 7.4 years (interquartile range 5.7–10.2) [66].

For DLB, there are a number of clinical symptoms for providers to consider [65]. Cognitively, DLB is characterized by waxing and waning, visuo-perceptual distortions, and disorganizations. Balance instability is a supportive feature of DLB, and so these individuals should be considered at severe risk for falls and injury. Depression and anhedonia are commonly reported by patients with DLB. Visual hallucinations often occur in vibrant color. Individuals with DLB may experience sleep disturbance and particularly REM sleep disorder with vivid dreaming and night thrashing that precede DLB for several years. These individuals also often report autonomic symptoms including frequent constipation and orthostatic hypotension. In their interaction with clinical providers, patients with DLB may exhibit difficulties providing details about their personal histories.

e. Frontotemporal Degeneration (FTD)

Major Neurocognitive Disorder due to degeneration of areas within the frontal and temporal lobes is an umbrella term that encompasses a group of dementia syndromes presenting histopathologically with neuronal loss, prominent microvacuolar spongiform change, and gliosis within the frontal and temporal regions of the brain. This combination of features combined with the usual absence of tau-positive intraneuronal inclusions (Pick-bodies) differentiates FTD from Pick’s Disease [67]. In addition, FTD histology shows a noticeable absence of neurofibrillary tangles or Aß plaques thereby distinguishing itself, at least histopathologically, quite clearly from AD. The three most commonly discussed FTD forms are Progressive Non-Fluent Aphasia (PNFA), Semantic Dementia (SD), and FTD Dysexecutive. Due to the complexity of these FTD forms, the imaging and biomarker perspectives will not be discussed in this section.

Each FTD profile is associated with unique patterns of cortical degeneration and behavioral presentation. These include:

i. PNFA: Individuals with PNFA exhibit significant reductions in their ability to produce fluent speech that occurs in the context of intact attention, comprehension, learning, and memory. Physically, approximately 60% of patients with FTD experience dysphagia

(difficulty swallowing), though caregivers of those with FTD only recognize the dysphagia approximately a third of the time [68, 69].

ii. **SD:** These individuals will appear to not understand some words including even the simplest of nouns. They will have difficulty with object naming that can be easily confused with AD.

iii. **Dysexecutive/Behavioral:** These individuals typically present with changes in personality such as showing impulsivity and social inappropriateness. Personality changes may be reported by a family member. Staff members may observe inappropriate jocularity or aggressive tendencies during the interview.

Cognitive or Brain Reserve and Dementia with Surgery and Anesthesia

Cognitive or brain reserve is defined as a reserve supply that allows some people to cope with progressive neurodegenerative pathology or successive neuronal insults [70, 71]. Higher cognitive reserve is related to psychosocial and experiential factors (e.g., greater educational attainment) and genetic factors (e.g., childhood intelligence). It has been theorized that individuals can remain above a critical threshold due to cognitive or brain reserve [70, 71] until some combination of factors (e.g., brain damage, neuronal stress) summates to accelerate symptom manifestation (*threshold theory*). The concepts of cognitive and brain reserve have been applied to explain differences in the manifestation of neurodegenerative disorders including PD, AD, and VaD. Cognitive reserve has also been used to explain intra-individual differences in perioperative cognitive outcome [72].

Whether different anesthesia techniques and anesthetic agents interact with the surgical environment to alter cognitive trajectories remains uncertain. Studies prospectively following patients with neurodegenerative disorders are rare [73] largely due to the challenges of screening, enrolling, and retaining representative samples. Likewise, retrospective large data studies are rare largely due to the lack of a DSM diagnosis code for postoperative cognitive decline. Nevertheless, preoperative cognitive impairment and presence of a neurodegenerative disease or dementia is associated with poor postoperative outcomes [74–77] and particularly delirium [78–80]. Given the association between preoperative cognition and postoperative outcomes, clinicians in preoperative settings are uniquely posed to detect patients who are at an elevated risk for anesthesia and surgery-related complications.

Perioperative Care Team Members

Perioperative care is a team effort, with every individual maximizing positive patient outcomes. Table 2 outlines a basic summary of the roles of some key perioperative care team members for older adults with and without cognitive impairment. Anesthesiologists and surgeons cannot complete all of the perioperative care needs for older patients. We encourage clincians to consult colleagues with expertise in geriatrics and dementia for assistance with pre operative cognitive assessment and postoperative acute care agitation and discharge care planning. In a multidisciplinary team, neuropsychologists are uniquely

trained to provide differential diagnosis information based on psychometrically-grounded information, assist monitoring patients' pre- and post-operative neurocognitive function, educate the patient and caregivers about postoperative risks, hypothesize how preoperative cognition can potentially interact with anesthesia, and predict postoperative cognitive needs. Colleagues from geriatric medicine are the perfect bridge between neuropsychology and other medical teams. They are equipped to integrate findings from the preoperative neuropsychological assessment and the patient's medical history to inform postoperative care and avert potential complications. Primary care providers play a pivotal role in communicating delirium risk and discharge care follow-up needs to the patient and family. These clinicians are most accessible to the patient's family and can provide follow-up treatment and palliative care if needed. Finally, caregivers are crucial members of the patient's care team. They are responsible for helping communicate with the patient and providing postoperative and palliative care needs.

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Recommendation Statement

Aging is associated with normal and abnormal brain and cognitive changes. Due to the expected increase in older adults requiring surgery, clinicians will increasingly encounter patients with neurodegenerative disease. In the future, it can be anticipated that brain pathology markers will present on intraoperative monitoring. We encourage proactive education and awareness of neurodegenerative disorders within the preoperative anesthesia setting. At the very minimum, we recommend clinicians engage in astute behavioral observations for potential neurodegenerative diseases, interview families, and foster collaborations with multidisciplinary allied health care members.

Table 2.

Some Key Perioperative Care Team Members for Older Adult Individuals with and without Cognitive Impairment and Basic Summary of Perioperative Roles

Team Members	Roles
Anesthesiology team members	-Integrate information from geriatric medicine and neuropsychology into perioperative practice considerations -Communicate with surgery care team to ensure referral to geriatric inpatient medicine when needed -Observe behavioral changes from pre- to postoperative settings
Caregiver	-May participate in giving information to medical providers, teaching the patient, communication with patient and family, preoperative initiation, postoperative care management, and palliative care needs
Geriatric medicine	-Integrate medical history with current medical regimen to provide insight into surgical medical risk and perioperative delirium risk medication management -Use baseline cognitive testing and baseline neurobehavioral assessment from neuropsychology to assess for postoperative status change -Assist with postoperative monitoring, delirium intervention, and follow-up palliative care needs -Integrate with neurology and memory care team for patients with persistent cognitive change
Primary care	-Provide referring information to patient, family, and care teams -Integrate information from perioperative care providers and surgical providers -Provide follow-up treatment and palliative care initiation, as needed
Neuropsychology	-Integrate background history with preoperative neurobehavioral assessment to provide information regarding premorbid intellectual ability relative to type of current cognitive impairment -Provide providers with applied information on patient reading ability, comprehension, learning memory strengths, attention deficits, and methods for improving communication and learning strategies -Available for postoperative cognitive domain comparison and perioperative cognitive monitoring relative to baseline -Assist with differential diagnosis via more comprehensive evaluation -Integrate with neurology and memory care team for patients with persistent cognitive change
Surgery team members	-Integrate information from team providers for optimal surgical decision making, postoperative rehabilitation planning, referral to inpatient providers including follow-up with geriatric medicine.

Note. Suggested interdisciplinary team members and their roles identifying patients with cognitive impairment perioperatively