

Alzheimer's disease and Lewy body dementia: Discerning the differences

**Assessment
is key for
optimal
management.**

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LEARNING OBJECTIVES

1. Compare Alzheimer's disease and Lewy body dementia as to pathophysiology, signs, symptoms, and management.
2. Describe how to screen for memory loss.

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As the U.S. population ages, the risk for age-related illnesses such as Alzheimer's disease and other types of dementia increases. According to Lang and colleagues, about 50% of individuals with dementia are undiagnosed. When dementia is diagnosed early, opportunities to address it increase. These opportunities include pursuing health behaviors that reduce progression, initiating medications earlier to help moderate symptoms and to potentially slow progression, as well as participating in research and advanced care planning. Nurses in all settings must increase their knowledge and understanding of dementia to ensure early detection.

What is dementia?

Dementia is any syndrome that manifests in three main sets of symptoms: debilitating cognitive decline, decreased independence in activities of daily living, and behavioral changes. Alzheimer's disease (AD) is the most common type of dementia and is sometimes used incorrectly to include all forms of dementia. Another common type of neurodegenerative dementia is Lewy body dementia (LBD), which consists of two diseases: dementia with Lewy bodies and Parkinson's disease dementia. Other causes of dementia include cerebrovascular disease and frontotemporal degeneration. In many cases, the presentation of dementia is characterized as "mixed," meaning that more than one pathological process is at play. Frequently, mixed dementias involve both vascular dementia and a neurodegenerative disease. In this article, we review the core features and current management approaches for AD and LBD.

Alzheimer's disease

According to the Alzheimer's Association's 2020 *Alzheimer's Disease Facts and Figures Report*, more than 5 million Americans are living with AD, and that number is projected to reach 7 million by 2025. Traditionally viewed as a memory plus syndrome, AD dementia typically presents as a progressive episodic memory decline and impairment in a second cognitive domain such as executive function or language. Such decline may involve the inability to recall recently learned information, create new memories, or recognize familiar faces or objects. For example, someone with AD may repeat a question within minutes, or tell the same story they told earlier the same day. Other common features include forgetting impor-

tant dates or events, misplacing items, word-finding difficulty, impaired judgment and decision making, and needing more help from family members to perform daily tasks.

As the disease progresses throughout the brain, symptoms become more severe and eventually encompass all major aspects of cognition and function. In addition, patients may exhibit significant behavioral changes, including wandering, irritability, agitation, aggression, paranoid delusions, and depression.

Pathophysiology

Alois Alzheimer is credited with the 1906 discovery of the neuropathological changes associated with AD. Specifically, abnormalities in two proteins, beta amyloid and tau, represent the neuropathological hallmarks of AD. Excess beta-amyloid proteins form plaques in the extracellular space and are thought to impair neuron-to-neuron communication. Tau neurofibrillary protein tangles cluster within neurons, which affects neuron health and causes cell death. The beta amyloid and tau changes lead to inflammation and brain atrophy. Recent evidence highlighted by the Alzheimer's Association points to amyloid plaques and tau tangles depositing 20 years or more before the first AD symptoms appear and formal diagnosis.

Together, these cellular alterations lead to cognitive changes that manifest as memory loss, functional impairment, and behavioral symptoms. Previously, amyloid and tau accumulations were detectable only at autopsy, but they're now detectable in positron emission tomography (PET) scans and their presence can be inferred from abnormal findings in cerebrospinal fluid (CSF).

The first recognizable symptomatic stage of dementia is called mild cognitive impairment (MCI), which is characterized by subjective complaints of cognitive changes and objective deficits that are detectable during cognitive testing; everyday functioning remains mostly normal. MCI was operationalized in sentinel articles by Petersen and is now widely regarded as a risk state for AD. In recently published practice guidelines, Peterson and colleagues note that people over age 65 with a diagnosis of MCI are three times more likely to develop AD. Up to 55% of those with MCI may revert to normal cognitive function over time. MCI also can be a precursor to other forms of dementia.

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LBD vs. AD symptoms

The table compares selected Lewy body dementia (LBD) symptoms and common features of Alzheimer's disease (AD) in four domains.

Domain	LBD	AD*
Cognitive	• Episodic memory deficits that improve with cued recall	• Episodic memory deficits that don't improve with cued recall
	• Difficulty retrieving information	• Difficulty learning and encoding information
	• Early alteration in visuospatial perception	• Visuospatial perception changes not as prevalent
	• Early poor performance in sustained and divided attention tasks	• Attention impairment early is possible
	• Fluctuating cognition, attention, and alertness	• Fluctuating cognition, attention, and alertness uncommon
Behavioral	• Vivid visual hallucinations (e.g., little people, furry animals) occur early	• Hallucinations less common, not as well-formed; tend to occur late in disease course
	• Misidentification delusions (e.g., Capgras)	• Paranoid delusions
	• Sleep difficulty	• Fragmented sleep
	• REM sleep behavior disorder	• Naps during the day and awake periods at night
	• Depression	• Depression
	• Anxiety	• Anxiety possible
	• Apathy	• Apathy possible
Movement	• Bradykinesia	• Uncommon
	• Rigidity	• Uncommon
	• Unsteady gait	• Uncommon
	• Rest, postural, or action tremor	• Uncommon
	• Postural instability with falls	• Slow imbalance progression
Autonomic	• Orthostatic hypotension	• Not as common
	• Loss of smell	• Not as common
	• Constipation	• Not as common
	• Sialorrhea/rhinorrhea	• Not as common
	• Sexual dysfunction	• Not as common
	• Urinary incontinence	• Can occur early in disease
	• Hyperhidrosis	• Can occur early in disease
	• Seborrheic dermatitis	• Can occur early in disease

*Without influence of medication effects or other underlying illness

AD stages

AD stages are classified as mild (early stage), moderate (middle stage), and severe (late stage). The *mild stage* is characterized by readily detectable cognitive ability impairment with some impairment in daily function. Affected individuals may or may not be aware of the cognitive and functional deficits. Behavioral symptoms such as mood changes

and apathy may begin in the mild stage.

In the *moderate stage*, cognitive and functional symptoms progress and individuals struggle to complete instrumental and basic activities of daily living (such as dressing, bathing, and sleeping) on their own. Behavioral symptoms may be more pronounced and can include delusions, agitation, hallucinations, and disinhibition. Ornstein and Gaugler found that these behavioral symptoms are associated with family caregiver depression and burden.

In the *severe stage*, people with AD may lose the ability to communicate, become unable to attend to activities of daily living (such as eating and bathing), and experience loss of awareness of their current surroundings. These individuals frequently require 24-hour care until death.

AD medications

Although current medications won't slow or prevent AD onset and progression, two categories of medications have received Food and Drug Administration approval for treating symptoms. Information about management is presented by Austrom and other experts in the Dementia Care Practice Recommendations from the Alzheimer's Association.

Cholinesterase inhibitors. Donepezil, rivastigmine, and galantamine are cholinesterase inhibitors approved to treat mild-to-moderate AD symptoms. Donepezil also is approved to treat severe AD symptoms. Cholinesterase inhibitors interfere with the breakdown of the neurotransmitter acetylcholine (which plays a key role in attention and memory) and help increase the brain's circulating supply.

These medications are available as pills and liquids; donepezil is available as an orally dissolving tablet, and rivastigmine can be administered via a transdermal patch.

The most common side effects of cholinesterase inhibitors—diarrhea, nausea, vomiting, fatigue, loss of appetite and weight, and insomnia—are transient and subside within the first month of treatment. However, bradycardia and syncope may develop later in the course of treatment, and decisions about benefit vs. harm must be made in collaboration with the patient's care team.

N-methyl-D-aspartate (NMDA) receptor antagonists. Memantine is the only drug in this class indicated for moderate or severe AD, either alone or in combination with a cholinesterase inhibitor. Memantine is neuroprotective but also

LBD pharmacologic treatment options

No pharmacologic treatments have been approved to treat Lewy body dementia (LBD) in the United States. However, the following medications are used off-label to treat LBD symptoms.

Domain	Possible treatment options
Cognitive symptoms	<ul style="list-style-type: none">• Cholinesterase inhibitors*• N-methyl-D-aspartate receptor antagonists
Motor symptoms	<ul style="list-style-type: none">• Carbidopa/levodopa
Behavioral symptoms	<ul style="list-style-type: none">• Antidepressants• Atypical antipsychotics
Sleep symptoms	<ul style="list-style-type: none">• Melatonin• Clonazepam
Orthostatic hypotension	<ul style="list-style-type: none">• Fludrocortisone• Midodrine

*Donepezil has been approved to treat LBD in Japan and the Philippines, and rivastigmine has been approved to treat Parkinson's disease dementia in the United States and Europe.

may help diminish symptoms of agitation, aggression, and delusion. It works by blocking the binding of glutamate (a neurotransmitter that's harmful when present in excess) to its receptor. A fixed combination of donepezil and memantine also is available but is used less frequently due to its high cost and inability to adjust the doses of its individual components.

Memantine is available in pill as both immediate and extended-release forms. It's also available as a liquid.

The most common side effects of memantine—headache, confusion, dizziness, and constipation—are transient and occur during the initial titration.

Lewy body dementia

LBD affects over 1.4 million Americans. It's more common in men than women and progresses more quickly than AD. LBD typically begins at age 50 and older.

LBD is an umbrella term that encompasses two related conditions: Parkinson's disease dementia (which occurs when cognitive decline develops after the motor symptoms of Parkinson's disease are established) and dementia with Lewy bodies (which occurs when cognitive symptoms begin before or concurrently with motor symptoms). According to Galvin, no unique sign or symptom distinguishes dementia with Lewy body from Parkinson's dementia; however, time of onset frequently is used to determine which LBD subtype is present. If dementia occurs *before or with* the onset of motor symptoms associated with Parkinson's disease, then dementia with Lewy body is suspected. If the dementia occurs *12 or more months after* Parkinsonism, then Parkinson's disease dementia is diagnosed. Although this 1-year rule continues to be debated, discerning if AD or LBD is present will help direct treatment. Additional neurologic testing can help to determine if dementia with Lewy body or Parkinson's disease dementia is responsible for most of the symptoms. For example, the Lewy Body composite risk score can help detect LBD by focusing the clinical interview on common LBD symptoms (psychomotor slowing, hallucinations, rigidity).

Pathophysiology

About 80% of patients with LBD will have co-existing AD pathology (amyloid plaques and tau tangles). However, the signature pathology is a

Lewy body composed of abnormal intraneuronal aggregations of the presynaptic protein alpha-synuclein. Friederich Lewy first described Lewy bodies in 1912, but alpha synuclein wasn't described until the late 1990s.

Symptoms

How Lewy bodies accumulate in the brain may predict its clinical presentation. If they begin accumulating in the brain stem, the result may be autonomic and motor symptoms; Lewy bodies beginning in the neocortex may lead to cognitive or behavioral symptoms. Symptom presentation is classified into four domains: cognition, behavior, movement, and autonomic function. (See *LBD vs. AD symptoms*.) A consortium of experts led by McKeith established that the diagnosis of LBD is made in the presence of dementia combined with at least two core criteria: parkinsonism, cognitive fluctuations, visual hallucinations, and rapid eye movement sleep behavior disorder (RBD).

Cognitive symptoms. Fluctuating cognition includes seemingly spontaneous changes in attention and alertness, staring spells, on/off periods of lethargy, and incoherent thought that can change from minute to minute, hour to hour, or day to day. Cognition fluctuations can be assessed using tools such as the Mayo Fluctuations Questionnaire, the Clinical Assessment of Fluctuation, and the One-day Fluctuation Assessment Scale.

Similar to people with AD, those with LBD

Because of the importance of early detection, cognitive screening is now a required component of the Medicare/Medicaid annual wellness visit.

may have memory impairment. However, according to Galvin, one difference is that patients with LBD sometimes benefit from cued recall (using cues to jog memory when free recall fails). In addition to memory, prominent symptoms in attention, executive function, and visual perception occur early in the course of LBD. Although no single clinical indicator of LBD exists, careful early assessment for visuospatial impairment can help differentiate LBD from other dementias.

Behavioral symptoms. Prominent visual hallucinations are a key distinguishing hallmark of LBD. When present, they typically occur early in the course of the disease. The hallucinations are well formed and typically of small or dysmorphic people, children, or animals. Auditory or olfactory hallucinations also may occur but are less common. Delusions frequently occur in LBD. These delusions include Capgras syndrome, where the person believes that a family member or friend has been replaced by an identical imposter.

One of the most overlooked but earliest symptoms of LBD is sleep difficulty and RBD, which is characterized by acting out dreams during sleep through violent movements, talking, and falling out of bed. Other common symptoms are excessive daytime sleepiness (2 or more hours of sleepiness daily), restless leg syndrome (walking or movement is needed to help relieve uncomfortable leg sensations), and increased discomfort at rest.

People with LBD, similar to those with AD, frequently experience depression, exhibited by feelings of worthlessness, sadness, and trouble eating or sleeping. Other behavior and mood changes include apathy or lack of desire to participate in previously enjoyed activities, agitation (which manifests as restlessness and unexplained irritation), intense levels of anxiety (which manifests as anger or fear when a loved one isn't present), and fear about a future event.

Movement disorder. Many patients with LBD experience Parkinsonism symptoms; by definition, all patients with the Parkinson's disease dementia form of LBD experience them. Early signs may include changes in facial expression, hand posture, walking, balance, and handwriting. LBD motor symptoms include bradykinesia (slowness in initiating or carrying out a movement), rigidity (stiffness), and noticeable gait change such as shuffling

(festination). Unstable posture (such as stooping over) and balance problems that lead to increased falls also frequently are associated with LBD. Other common symptoms include diminished facial expressions, difficulty swallowing, and tremor or shaking at rest.

Autonomic/constitutional changes. Lewy body deposits affecting the autonomic nervous system produce autonomic or constitutional symptoms—changes in bodily operations that patients have no intentional control over. Consequently, those with LBD may have orthostatic hypotension, anosmia (loss of smell), and frequent constipation. Other commonly observed symptoms include hyperhidrosis (excessive sweating), sialorrhea (excessive salivation), seborrheic dermatitis (itchy rash with flaky skin), rhinorrhea (runny nose), sexual dysfunction, urinary incontinence, and heat and cold insensitivity. Consistent nursing support is essential to effectively manage these complex symptoms.

LBD medications

If a patient with LBD is misdiagnosed as having a psychiatric illness because of symptoms such as hallucinations and illogical thinking, providers may prescribe medications that are detrimental. For example, carbidopa/levodopa, although helpful for patients with Parkinson's disease, may exacerbate hallucinations in those with LBD. Antipsychotic medications may relieve hallucinations but also could exacerbate rigidity and motor slowness.

No medications have been approved in the United States to specifically treat LBD, but rivastigmine does have approval to treat symptoms of mild-to-moderate Parkinson's disease dementia. Treatment should be based on the four LBD domains; however, all treatment is off-label. (See *LBD pharmacologic treatment options*.) To treat cognitive symptoms, cholinesterase inhibitors and NDMA receptor antagonists used in AD frequently are prescribed for LBD. Cholinesterase inhibitors may show a more robust initial response in LBD compared with AD. For motor symptoms, carbidopa/levodopa may help reduce Parkinsonian symptoms such as slow gait and muscle rigidity. Unfortunately, these medications may increase confusion and hallucinations so titration should be performed slowly.

No medications have been approved to manage LBD behavioral symptoms, and many

Lewy body composite risk score assessment

The Lewy body composite risk score is an accurate, valid, and reliable tool for differentiating between Lewy body dementia and Alzheimer's disease.

Please rate the following as being present or absent at least three times over the past 6 months. Does the patient have:

	Yes	No
Slowness in initiating and maintaining movement, or frequent hesitations or pauses during movement?		
Rigidity (with or without cogwheeling) on passive range of motion in any of the four extremities?		
Loss of postural stability (balance) with or without frequent falls?		
Tremor at rest in any of the four extremities or the head?		
Excessive daytime sleepiness and/or seem drowsy and lethargic when awake?		
Episodes of illogical thinking or random, incoherent thoughts?		
Frequent staring spells or periods of blank looks?		
Visual hallucinations (see things not really there)?		
Appear to act out his/her dreams (kick, punch, thrash, shout, or scream)?		
Orthostatic hypotension or other signs of autonomic insufficiency?		

Reprinted with Permission from Dr. James E. Galvin. Galvin JE. Improving the clinical detection of Lewy body dementia with the Lewy body composite risk score. *Alzheimers Dement (Amst)*. 2015;1(3):316-24.

common psychotropic medications can have deleterious side effects in patients with LBD. In general, all classic neuroleptic medications, such as haloperidol, should be avoided. When possible, nonpharmacologic approaches should be attempted to alleviate these symptoms. (To learn more about nonpharmacologic strategies, visit myamericannurse.com/?p=69690.) If necessary, careful use of atypical antipsychotic medications (such as quetiapine or pimavanserin) can be tried but require close monitoring. Attention to physical ailments such as constipation and pain should be the first priority because they can trigger behavioral changes. Mood symptoms are common in patients with LBD. Selective serotonin reuptake inhibitors, which help increase the amount of serotonin in the brain, appear to reduce irritability, agitation, and depression. RBD frequently is treated with melatonin or clonazepam.

Treating autonomic symptoms is challenging and requires a holistic nursing approach, including identifying therapies targeted to each symptom. For example, constipation and diaphoresis in the context of orthostatic hypotension require comfort measures combined with safety strategies. Effective nursing care will make a critical difference in preventing or mitigating the physical health challenges associated with dementia syndromes.

Screening for memory loss

Because of the importance of early detection, cognitive screening is now a required component of the Medicare/Medicaid annual wellness visit. Several measures are available that nurses can use to briefly assess for risk of cognitive decline. For example, the Quick Dementia Rating Scale (QDRS) is a 5-minute informant interview that addresses ten cognitive domains: memory and recall, orientation, decision-making and problem-solving abilities, activities outside the home, function at home and hobbies, toileting and personal hygiene, behavior and personality changes, language and communication abilities, mood, and attention and concentration. The QDRS format helps to avoid problems such as patient anxiety with cognitive testing, which can lead to refusing to be tested. The scale also can be completed by family members. Scores of the 30-item scale range from 0 (no impairment in any domain) to 30 (indicating severe cognitive impairment). The QDRS has strong reliability and validity when correlated with other common cognitive assessment tools, such as the Clinical Dementia Rating scale ($r=0.80$, $P<.001$) and the Mini-Mental State Exam ($r=-.599$, $P<.001$); the Mini-Mental State Exam requires payment for use. In addition, the QDRS provides enough information to stage dementia severity, and it can be used by clinicians to

Modifying behaviors to decrease dementia risk

In the past, it was believed that nothing could be done to delay the onset of dementia or slow its progress. However, research on lifestyle modifications that may diminish risk factors for dementia is growing. Recommended strategies that may reduce Lewy body dementia risk include

- refraining from smoking
- maintaining a healthy diet
- getting adequate sleep
- participating in cognitively stimulating activities
- interacting with others socially
- exercising.

Edwards and colleagues identified several medical (obesity, diabetes, hypertension, elevated cholesterol, depression) and lifestyle (dietary fat and cholesterol consumption, smoking, lack of physical activity) Alzheimer's disease (AD) risk factors that may be modifiable with health-promoting interventions. In August 2020, Livingston and colleagues added other risk factors to this list—traumatic brain injury, pollution, and excessive alcohol consumption. They reported that all of the modifiable risk factors account for about 40% of worldwide dementias that could be theoretically prevented or delayed.

Recent evidence from Haslam and colleagues' work with the Framingham Heart Study about the impact of sugary drink consumption indicates that even a small but consistent change in diet, such as eliminating sugar-sweetened soft drinks, may help reduce dementia risk. Improving nutritional habits to include more fruits and vegetables, beans, nuts, whole grains, olive oil, and fish (the MIND [Mediterranean-DASH Intervention for Neurodegenerative Delay] diet) should be encouraged to promote brain health. Also, promising new biomarkers may aid in early dementia detection.

determine the need for more formal testing.

The Montreal Cognitive Assessment (MoCA), developed by Nasreddine and colleagues, is a performance-based assessment that addresses attention, memory, concentration, orientation, visuconstruction skills, and language. Julaianont and colleagues' MoCA-B is geared toward individuals with health literacy and education levels below 8th grade. The MoCA is available in multiple languages, including Spanish, Chinese, and Hindi and also as a telephone Mini-MoCA, which was designed for use during periods of quarantine. These assessments are available at MoCAtest.org. Galvin's Lewy Body Composite Risk Score assesses LBD risk and can differentiate LBD from AD with high accuracy, validity, and reliability. (See *Lewy body composite risk score assessment*.)

The science behind dementia detection continues to change rapidly. Patients identified with dementia ideally should receive brain imaging (non-contrast computed tomography or magnetic resonance imaging) to rule out other causes. PET and CSF biomarkers can be used to diagnose AD by testing for the presence of amyloid plaque and tau proteins

in the brain or CSF. These tests are expensive.

Dopamine transporter imaging using single PET scans can be used to diagnose Parkinson's disease. Blood tests aren't yet available for these dementias, but advances are being made. For example, Thijssen and colleagues tested for the presence of pTau181 protein in the blood plasma of 400 individuals and were successful in distinguishing those who were healthy from those with AD pathology. These findings were corroborated with two established biomarkers (presence of pTau181 in spinal fluid and amyloid protein in PET scans) being used in AD research.

After screening

If screening indicates that a patient is at risk for cognitive decline, they should be referred to a board-certified advanced gerontological nurse practitioner, advanced practice registered nurse gerontological specialist, neurologist, or other provider comfortable with diagnosing and treating cognitive impairment.

These providers will consider other causes of cognitive decline, such as negative medication effects; trauma; depression; or vitamin B-12, folate, or thyroid deficiency. Symptom-directed treatment is more effective the earlier it's initiated in the disease process. Education to adapt healthy behaviors may potentially delay a patient's decline, and participation in research studies is an option for those in the early stages of dementia. (See *Modifying behaviors to decrease dementia risk*.)

Ease the burden

As the population ages, nurses need to learn more about AD and LBD. Distinguishing between these two conditions is critical to ensure proper treatment and symptom management. Regular screening and early dementia detection offer opportunities for nurses and families to partner in research and symptom management and ease illness burden. **AN**

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To view a list of references, visit myamericannurse.com/?p=69690.

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Please mark the correct answer online.**1. The neuropathological hallmarks of Alzheimer's disease (AD) are abnormalities in**

- a. alpha amyloid and tau proteins.
- b. alpha amyloid and gamma proteins.
- c. beta amyloid and gamma proteins.
- d. beta amyloid and tau proteins.

2. The first recognizable symptomatic stage of dementia is referred to as

- a. initial cognitive impairment.
- b. mild cognitive impairment.
- c. neurofibrillary protein tangle.
- d. neurofibrillary protein strand.

3. In which stage of AD do patients begin to experience more pronounced delusions and hallucinations?

- a. Mild
- b. Moderate
- c. Severe
- d. Advanced

4. Which type of drugs used to treat AD interfere with the breakdown of the neurotransmitter acetylcholine?

- a. Anticholinergic agents
- b. N-methyl-D-aspartate (NMDA) receptor antagonists
- c. Cholinesterase inhibitors
- d. Monoamine oxidase inhibitors

5. Which NMDA drug used to treat AD blocks the binding of glutamate to its receptor?

- a. Donepezil
- b. Rivastigmine
- c. Galantamine
- d. Memantine

6. Lewy body dementia (LBD) encompasses two related conditions: dementia with Lewy bodies and

- a. Parkinson's disease dementia.
- b. mild cognitive impairment.
- c. moderate-stage AD.
- d. generalized dementia.

7. The signature pathology of LBD is a Lewy body composed of abnormal

- a. intraneuronal aggregations of the presynaptic protein alpha-synuclein.
- b. intraneuronal aggregations of the postsynaptic protein gamma-synuclein.
- c. interneuronal aggregations of the presynaptic protein beta-synuclein.
- d. interneuronal aggregations of the postsynaptic protein alpha-synuclein.

8. A key behavioral symptom of LBD that usually occurs early in the course of disease is

- a. occasional sensory delusions.
- b. prominent olfactory hallucinations.
- c. occasional auditory delusions.
- d. prominent visual hallucinations.

9. A frequently overlooked but early symptom of LBD is

- a. hyperkinesia.
- b. reduced salivation.
- c. sleep difficulties.
- d. postural hypertension.

10. Which of the following statements comparing AD and LBD symptoms is correct?

- a. Misidentification delusions are common in LBD.
- b. Loss of smell occurs less often in LBD.
- c. Visuospatial perception changes are more prevalent in AD.
- d. Hallucinations are more common in AD.

11. Which drug is used to treat motor symptoms of LBD?

- a. Clonazepam
- b. Carbidopa/levodopa
- c. Melatonin
- d. Midodrine

12. A drug used to treat orthostatic hypotension in patients with LBD is

- a. Clonazepam
- b. Carbidopa/levodopa
- c. Melatonin
- d. Midodrine

13. Which statement about the management of behavioral symptoms related to LBD is correct?

- a. Nonpharmacologic approaches should be used to ease these symptoms.
- b. Neuroleptic medications, such as haloperidol, are effective for treatment.
- c. Pimavanserin is an effective drug that doesn't require close monitoring.
- d. Medications should be tried before nonpharmacologic approaches are used.

14. Which statement about the Quick Dementia Rating Scale (QDRS), which is used to screen for memory loss, is correct?

- a. It addresses five cognitive domains.
- b. It has strong reliability compared to other common cognitive assessment tools.
- c. It provides insufficient information to stage dementia severity.
- d. It can't be used to determine the need for more formal testing.

15. Which screening assessment tool is geared toward individuals with health literacy and education levels below 8th grade?

- a. Mini-Mental State Exam
- b. Lewy Body Composite Risk Score
- c. Montreal Cognitive Assessment-B
- d. The Quick Dementia Rating Scale