The many faces

Dementia is an umbrella term used to describe a number of diseases caused by the destruction of brain tissue. Once considered an inevitability of aging, dementia can affect adults of any age. We give you the information you need to recognize dementia so you can provide the best possible care for your patients.

By Nancy B. Lerner, DNP, RN
Assistant Professor • University of Maryland • Baltimore, Md.

CE
2.3
ANCC
CONTACT HOURS

Nurses who work in acute care settings frequently encounter individuals experiencing dementia. Because these patients are hospitalized for other acute or chronic conditions, understanding the difference between delirium and the different types of dementia is difficult. As a result, nurses may experience stress when caring for these patients because treatment approaches differ for delirium and dementia. Occurring in adults of all ages, dementia—a global loss of cognitive ability that affects physical, social, and occupational functioning—encompasses many diseases and conditions, including Alzheimer disease (AD), vascular dementia, Parkinson disease (PD), and the less commonly known frontotemporal dementia and Lewy body dementia. Learning how to distinguish between the different types of dementia and recognizing the difference between delirium and various dementias can help reduce this stress.

Four characteristics

Dementia has four key characteristics to its definition:

- ***global impairment***—the patient experiences deficits in more areas of brain function than memory alone; dementia patients have deficits in language, planning, decision making, and coordination of movement
- ***decline in functioning over time***—knowledge of the patient's previous performance level is crucial to determine this characteristic
- ***severity of impairment***—normal functioning in daily life is limited; patients with dementia frequently can no longer live alone, manage their finances, or even find their way home when driving
- ***normal consciousness***—the patient is awake and alert, even if confused; this characteristic is often the most difficult to identify.

Patients who are drowsy or lethargic are most likely delirious, a temporary condition caused by illness, medication reactions, or anesthesia. When the underlying condition causing delirium is resolved, it disappears. Patients with dementia can experience delirium and become even more impaired until the cause of the delirium is identified and eliminated, but the underlying dementia won't go away. Delirium can be identified by using the Confusion Assessment Method (CAM), a tool available at http://consultgerirn.org/uploads/File/trythis/try_this_13.pdf. Unfortunately, recent research indicates that even with the CAM, delirium often isn't identified correctly, resulting in difficulties in care for both patients and staff.

The lobes of the brain

Dementia is actually a number of brain disorders with different presentations and symptoms and different disease courses. The symptoms are related to what part of the brain is initially affected. Reviewing the functions of the lobes of the brain can provide a key to understanding the symptoms of the different brain disorders that cause dementia (see *Picturing the cerebral lobes*).

The ***frontal lobe*** controls the ability to concentrate and focus, as well as reasoning. The frontal lobe is also the seat of our inhibitions, so individuals with frontal lobe deficits may

of dementia

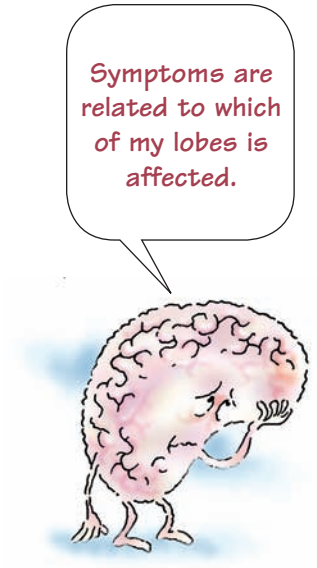


exhibit inappropriate social and sexual behaviors. This lobe also controls motor speech; deficits can cause expressive aphasia.

The ***temporal lobe*** controls auditory reception and association, receptive speech, expressed behavior, and information retrieval. Individuals with damage in the temporal lobe may have receptive aphasia, agitated and childish behavior, and hearing deficits.

The ***parietal lobe*** regulates sensory discrimination and body orientation. Individuals with a parietal lobe deficit may not be able to locate parts of their body or feel heat or cold. They may not be able to understand the position of their body, which makes them prone to falls and injuries.

The ***occipital lobe*** controls visual reception and association. Although individuals with occipital lobe deficits can see an object, they may not be able to identify the object or know what the object is used for.

Most dementias aren't treatable and are progressive. There are a few types of dementias, however, that are treatable or partially treatable by managing the underlying cause of the disorder. Treatable dementias include untreated hypothyroidism, normal pressure hydrocephalus, chronic drug abuse, and vitamin B_{12} deficiency. Patients suspected of having dementia should always have a thorough medical workup to identify and treat the underlying conditions that can cause or complicate dementias.

Alzheimer dementia

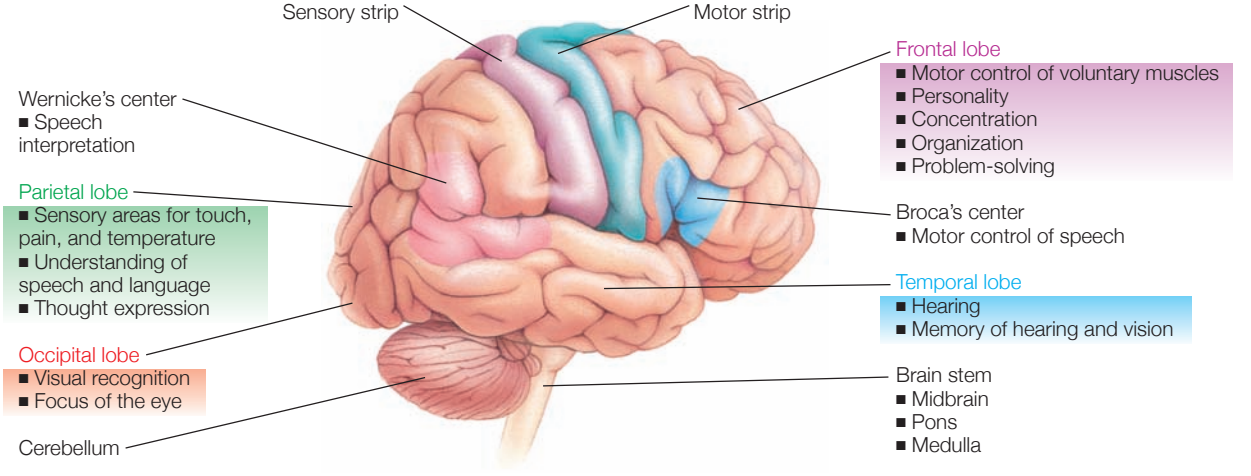
The most common type of dementia is AD, which accounts for 50% of all patients with dementia. Associated with the presence of amyloid plaques and neurofibrillary tangles in the brain, AD is incurable and progressive. The plaques and tangles cause an overall shrinking of the brain and a gradual decrease in the number of functioning neurons (see *Tissue changes in AD*). Patients with AD have reduced levels of neurotransmitters, especially acetylcholine, but serotonin, somatostatin, norepinephrine, glutamate, and corticotrophin-releasing factor levels are also deficient. The most common risk factor for AD is advancing age.

Although individuals younger than age 65 may develop the disease, the incidence and prevalence of AD increase dramatically after age 65, with prevalence doubling every 5 years between ages 65 and 95. From less than 2% of 65-year-old individuals having the disease, the number increases to almost 50% in those older than age 85. Other risk factors include family history, especially in early-onset individuals (ages 40 to 60); head trauma with loss of consciousness; and female sex. Three different genes are responsible for 90% of early-onset AD cases. One of these genes is on chromosome 21, the same chromosome affected in individuals with Down syndrome. It isn't surprising, therefore, that almost all Down syndrome patients have signs of AD by age 40.

The onset of AD is insidious, so the date of onset is impossible to pinpoint. However, after AD is identified, progression is gradual and unavoidable. Symptoms of AD develop in three distinct stages. The first stage and the most frequently recognized symptom is amnesia or memory loss, especially short-term memory. Patients have difficulty recalling new information and often ask the same question repeatedly. Patients can forget not only what they had for breakfast, but whether they had breakfast at all. Along with short-term memory loss, patients experience disorientation to time and place. Long-term memory is spared until much later in the disease process.

In the second stage, aphasia also becomes a symptom. Characteristics of aphasia include word finding difficulty, reduced ability to participate in conversations, and reduced vocabulary. (Aphasia progresses to global aphasia by the third stage of AD.) Symptoms of apraxia and agnosia also appear during the second stage of the disease. Apraxia is the inability to perform movements or tasks when asked, although the directive is understood. Individuals with apraxia may have difficulty using familiar appliances or diffi-

Picturing the cerebral lobes



culty with bathing or dressing. Agnosia is the loss of ability to recognize objects, which causes difficulty recognizing what common objects such as spoons and forks are used for.

By the third stage of AD, patients are incontinent and unable to perform self-care, ambulate, or recognize loved ones.

In addition to the four A's—amnesia, aphasia, apraxia, and agnosia—patients will frequently experience behavioral or psychiatric symptoms. About 50% of AD patients have depression, even early in the illness, and many patients suffer from paranoia and delusions later in the disease process. Hallucinations are rare but do occasionally occur.

No current treatments are available to cure or stop the progression of AD. Cholinesterase inhibitors do provide a short-term increase in functionality, but the disease will continue to progress. Available cholinesterase inhibitors include donepezil, rivastigmine, and galantamine. These drugs work by increasing the levels of acetylcholine in the brain, slowing the symptoms for a limited period (generally 6 months to 1 year). Donepezil is approved for all stages of AD; the other two medications are approved for mild-to-moderate disease only. Common adverse reactions, which are usually mild, include diarrhea, vomiting, nausea, fatigue, insomnia, loss of appetite, and weight loss. These drugs should be avoided in patients taking cholinergic agents.

The *N*-methyl-D-aspartate receptor antagonist memantine may also produce improvements in cognition. Approved for moderate-to-severe dementia, memantine works by regulating the amount of glutamate in the brain. Glutamate is increased in patients with AD. Adverse reactions include fatigue, dizziness, confusion, constipation, and headache. Memantine has been shown to delay institutionalization in patients with AD.

Additionally, antidepressants are sometimes helpful in improving mood, and antipsychotics can be used for behavioral disturbances, although these drugs are associated with higher mortality in AD patients. Adverse reactions of antipsychotics need to be monitored carefully in patients with AD. The risk of delirium, which worsens symptoms, should always be considered because this complication can frequently occur. Patients may also experience orthostatic hypotension and dizziness. Longer-term adverse reactions, such as the parkinsonian symptoms of stiffness, shuffling gait, and masked facies, may also occur.

Caring for patients with AD can be challenging because of their cognitive losses.

Patients sometimes react with hostility and agitation when they're unable to perform tasks or communicate effectively. Limiting challenges to decrease frustration can help prevent these reactions. Establishing and following a set routine is very helpful and reduces anxiety, as does allowing additional time for communication and task performance. Communication can be improved by limiting choices and keeping instructions simple by breaking them into single steps.

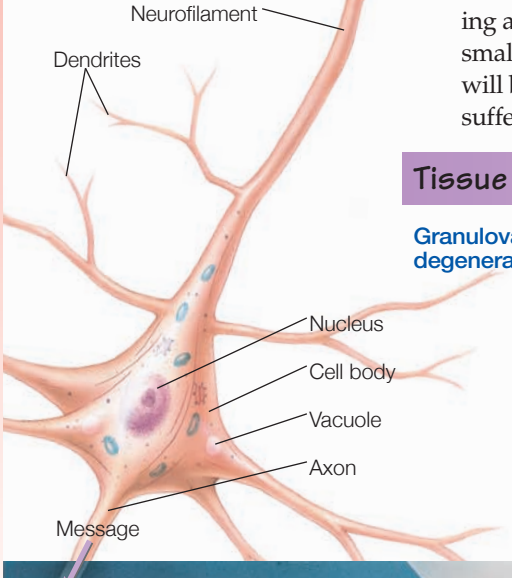
Reducing distractions during communication so patients can focus their attention is also helpful.

Nurses often have difficulty with the delusions that are a characteristic of some AD patients. Attempting to reason with these patients or arguing with them to correct delusional thinking may produce agitation and sometimes aggression. Examples of delusions would include a patient who feels that a personal item he or she can't find is stolen or a patient who wants to go home to be there when his or her spouse or children come home. Instead of arguing with or correcting patients, try to distract them by offering a snack or changing the subject. Telling a small lie such as "your husband called and will be late" may help reduce the patient's suffering and frustration. Most importantly,

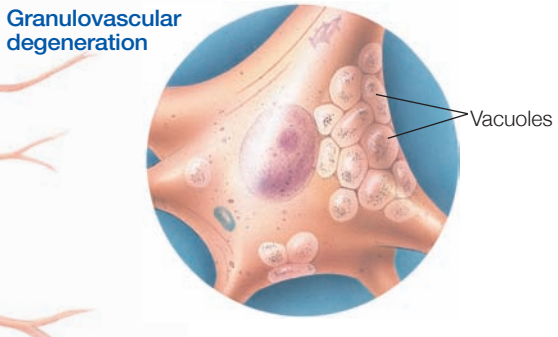
How it happens

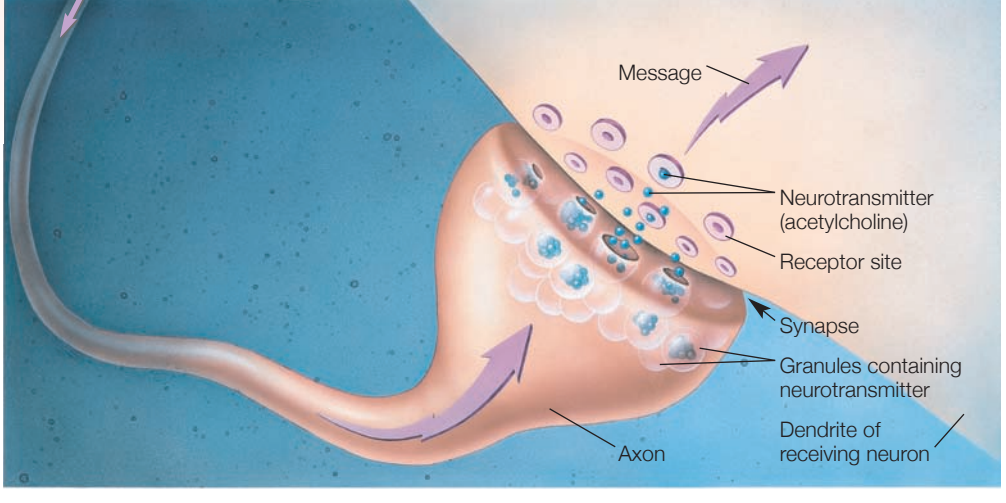
The cause of AD is unknown, but there are thought to be four contributing factors:

1. neurochemical factors, such as deficiencies in the neurotransmitters acetylcholine, somatostatin, substance P, and norepinephrine
2. viral factors such as slow-growing central nervous system viruses
3. trauma
4. genetic factors.



Tissue changes in AD





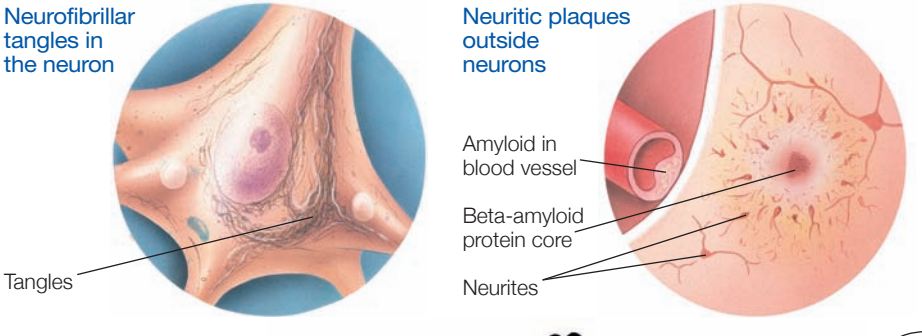
when caring for patients with AD, be flexible and individualize care to the patient's unique needs and concerns.

Vascular dementia

Vascular dementia is generally considered to be the second most common type of dementia. Sometimes vascular dementia and Alzheimer dementia occur in the same individual, a combination that can modify the course and symptomatology of the disease; this is known as mixed dementia.

Vascular dementia is caused by reduced blood flow to the brain, usually due to small, often undetectable "mini" strokes. Although this type of dementia can accompany a typical stroke, the vascular damage that characterizes it is usually more insidious. Unlike Alzheimer dementia, which is incurable and uniformly progressive, lifestyle changes and medications to control hypertension, hypercholesterolemia, and cardiac disease can occasionally alter the course of vascular dementia.

Typically, vascular dementia's development is insidious as more of the brain is damaged by small ischemic episodes. As opposed to the gradual progression of AD, the progression of vascular dementia is often stepwise, with periods of stability in which no new events occur. The symptoms of vascular dementia are dependent on the area of the brain affected by the vascular events and can include focal neurologic signs such as arm or leg weakness, gait disturbance, mood swings, and difficulty with executive functioning such as decision making and planning. There may also be early incontinence,



dizziness, and balance problems—symptoms that appear later in AD. The memory changes in vascular dementia can affect both short- and long-term memory early in the disease process.

Treatment for vascular dementia focuses on treating the underlying cause of the disease in an attempt to stop future vascular events and disease progression, but recovering function isn't usually possible. The medications used in AD may be helpful in patients with vascular dementia, as well as the care approaches to minimize frustration and communication difficulties.

Parkinson dementia

Dementia occurs in 25% to 30% of patients with PD after 2 years and up to 68% of patients who've had the disease for 15 years or more. PD is characterized by alpha-synuclein proteins in the nuclei of nerve cells. In PD, the affected cells are in the noncortical substantia nigra, a portion of the brain that's an important motor center. (See "The Puzzle of Parkinson Disease" on page 47.)

Patients with PD experience a tremor at rest, often in the thumb and forefinger, which isn't especially disabling, particularly early in the disease. They also have bradykinesia (a slowing of spontaneous and automatic movement), muscle rigidity, and impaired posture and coordination. PD is progressive, although some patients progress faster than others. Treatment to replace the lost dopamine in the brain with levodopa/carbidopa, dopamine agonists, catechol-*O*-methyltransferase inhibitors, and monoamine oxidase B inhibitors significantly reduces symptoms, but the dosage of these medications must be increased over time. Patients with PD are very sensitive to neuroleptic medications.

The Lewy bodies that produce the symptoms of PD can develop in the cortical area of the brain, causing dementia. Patients with Parkinson dementia may not have an early memory decline, but may have delusions, hallucinations, and fluctuations in attention and alertness.

The existence of Parkinson dementia is controversial, with many practitioners believing that all patients with PD exhibit dementia symptoms, whereas others believe it's relatively rarer. Because PD patients tend to be older, some practitioners believe that dementia symptoms may be caused by AD or vascular dementia in patients who demonstrate cognitive decline.

Frontotemporal dementia

An early-onset form of dementia, with most cases occurring before age 65, frontotemporal dementia causes about 15% of all dementia cases. Frontotemporal disease, as the name implies, begins with damage in the frontal and temporal lobes of the brain, causing early symptoms that are markedly different than AD. Memory loss isn't seen in the early stages of frontotemporal dementia, although because the disease is progressive, memory loss does appear later in the disease process.

The first symptoms of frontotemporal dementia are problems with behavior and social skills. These patients are disinhibited and seemingly unable to recognize the difference between acceptable and unacceptable behavior. They may disrobe in public or behave aggressively, and the behavior may have inappropriate sexual components. These patients also have difficulty with executive functioning, so there are often early changes in occupational performance. Aphasia and, in some instances, mutism occur when the temporal lobe is affected first. Some patients also exhibit hyperorality, characterized by overeating or eating nonfood products and putting objects in their mouths indiscriminately.

There are actually several different forms of frontotemporal dementia, including Pick disease, primary progressive aphasia, semantic dementia, and Klüver-Bucy syndrome, but all are progressive dementias and share eventual symptoms. The progressive course of frontotemporal dementia is steady and more rapid than other dementias. No treatment will halt the progressive nature or course of the disease, but psycho-

tropic medications can sometimes mitigate aggression or agitation, and antidepressants can improve apathy and mood.

Caring for a patient with frontotemporal dementia is very difficult. Because of the bizarre behaviors, family caregivers experience greater distress and strain, and are more likely to be depressed and feel less in control than family caregivers of patients with other dementias. Family caregivers need support and should be referred to support groups and, if appropriate, individual counseling. Managing the behaviors in frontotemporal dementia can help alleviate some of the caregiver's burden. The first consideration in management is whether the patient's behavior will cause harm. If the behavior isn't harmful to the patient or family, even if it's disturbing, allowing the behavior to continue may be best. When behaviors are harmful, identifying and avoiding triggers that precipitate the behavior is helpful, as is maintaining a calm environment and anticipating patient needs.

Lewy body dementia

Another form of dementia is Lewy body disease, which is estimated to cause another 15% of all dementias. Lewy bodies are accumulated clusters of alpha-synuclein proteins in the nuclei of neurons that are found in the brains of patients with Lewy body dementia and PD. In PD, these abnormal cells are found in the substantia nigra, located in the midbrain. In Lewy body dementia, the abnormal cells are found in the neocortex. Like all dementias, Lewy body disease is progressive.

This dementing illness is characterized by early executive functioning problems and some cognitive issues, but it's also typified by fluctuations in attention and alertness, recurrent visual hallucinations, and parkinsonian motor symptoms such as rigidity, shuffling walk, and propensity for balance problems and falling. Early in the disease process, patients can present with differing symptoms. Some may show cognitive problems, although many don't have early memory loss, whereas others may demonstrate parkinsonian movements, and still others may have symptoms characterized by fluctuations in alertness and attention.

As opposed to other dementias in which delusions, not hallucinations, characterize the disease, visual hallucinations are frequently noted in Lewy body dementia along with delusions. Many Lewy body dementia patients will also have autonomic system dysfunction, characterized by BP fluctuations, heart rate variability, excessive sweating, sexual and urinary dysfunction, and fainting. About half of all patients with Lewy body dementia also suffer from rapid eye movement (REM) sleep disorder. REM sleep is the stage in which people dream. In normal REM sleep, body movement is suppressed so that individuals don't act out their dreams. In REM sleep disorder, movement isn't blocked, allowing these individuals to act out their dreams, sometimes vividly and violently.

Treatment for Lewy body dementia is symptomatic because the disease is progressive regardless of treatment. Cholinesterase inhibitors used in AD are temporarily effective in improving the cognitive and memory symptoms, although progression of the symptoms

General nursing interventions for dementia patients

cheat sheet

- Follow a set routine and follow the routine the patient follows at home when possible.
- Don't rush! Allow additional time for communication and task performance.
- When communicating, keep instructions simple and break complex instructions into single steps.
- Reduce distractions to help patients focus their attention.
- Don't argue or try to correct patients. If they're delusional, allow them to think they're right. Arguing or correcting may produce agitation and aggression.
- Distract patients by changing the subject or offering a snack.
- Be flexible! Individualize your care to the patient instead of having the patient follow your guidelines.
- Be careful with giving as-needed medications to reduce agitation. Many medications, especially benzodiazepines, can actually increase agitation by causing delirium. Behavioral interventions work best.

Comparing different types of dementia

Dementia type	Clinical features	Treatments
Alzheimer dementia	• Continuous gradual progression • Short-term memory loss • Difficulty speaking and communicating (aphasia) • Difficulty recognizing objects (agnosia) • Difficulty performing tasks (apraxia) • Delusions • 50% incidence of depression	• Cholinesterase inhibitors • *N*-methyl-D-aspartate receptor antagonists • Symptom management with antidepressants and sometimes antipsychotics
Vascular dementia	• Step-wise progression • Symptoms depend on location of vascular events • May include mood disorders, executive function issues, early incontinence, and balance disorders • Long-term and/or short-term memory issues	• Treatments for underlying cause of vascular disease • Symptom control
Frontotemporal dementia	• Frequently early onset • Behavior changes • Changes in social skills • Executive functioning decline • Early-onset aphasia • Hyperorality • Quicker disease progression	• Antidepressants for symptoms • Antipsychotics for behavioral changes
Lewy body dementia	• Early symptoms differ for each patient • Severe sensitivity to neuroleptics • Executive functioning decline • Autonomic system fluctuations • Fluctuations in attention and alertness • Visual hallucinations • Parkinson-like motor problems	• Cholinesterase inhibitors • Levodopa/carbidopa • Neuroleptic sensitivity makes antipsychotic use problematic

will continue. For patients with significant movement disorders, levodopa/carbidopa will provide improvement, although many patients have movement disorders that are mild and don't require treatment.

Individuals with Lewy body disease are usually severely sensitive to neuroleptic medications. Even very small doses of neuroleptics cause worsening cognition, severe sedation, and worsening or sometimes permanent parkinsonian symptoms. Up to half of Lewy body dementia patients are also susceptible to malignant neuroleptic syndrome when antipsychotic medications are taken, which is characterized by high fever, severe muscle rigidity, and rhabdomyolysis leading to kidney failure and death in 10% to 20% of cases. Although the newer atypical antipsychotics are less likely to cause the syndrome than traditional antipsychotics, the risk of reaction is still high.

The hallucinations and delusions that accompany Lewy body dementia, as well as aggression and agitation, are more of a problem for these patients and their families than patients with other dementing illnesses. Borrowing caregiving techniques from other dementing illnesses with similar problems is helpful (see *Comparing different types of dementia*). For example, delusions can be handled in the same manner as patients with AD, and behavioral manifestations can be handled in the same way as frontotemporal dementia. The hallucinations seen in Lewy body dementia respond to environmental manipulation. Removing clutter, closing blinds to reduce glare and eliminate shadows, and removing mirrors

can reduce the environmental triggers for hallucinations.

One term, many conditions

Regardless of the cause or type of dementia, you're likely to care for these patients. By understanding the differences in illnesses causing dementia and learning how to prevent or control the behaviors dementia patients experience, you'll become more comfortable caring for these patients. ■

Learn more about it

Alagiakrishnan K. Vascular dementia. http://emedicine.medscape.com/article/292105-overview.

Alzheimer's Research Trust. Antipsychotic drugs double risk of death among Alzheimer's patients. http://www.sciencedaily.com/releases/2009/01/090108194416.htm.

Crystal HA. Dementia with Lewy bodies. http://emedicine.medscape.com/article/1135041-overview.

Kirshner HS. Frontotemporal lobe dementia. http://emedicine.medscape.com/article/1135164-overview.

Mayo Clinic. Alzheimer's care: simple tips for daily tasks. http://www.mayoclinic.com/health/alzheimers-caregiver/HO00125.

Merino JG, Luchsinger J, Huang Z. Parkinson's disease dementia. http://www.emedicinehealth.com/parkinson_disease_dementia/article_em.htm.

Rice KL, Bennett M, Gomez M, Theall KP, Knight M, Foreman MD. Nurses' recognition of delirium in the hospitalized older adult. *Clin Nurse Spec.* 2011;25(6):299-311.

Salomone S, Caraci F, Leggio GM, Fedotova J, Drago F. New pharmacological strategies for treatment of Alzheimer's disease: focus on disease-modifying drugs. *Br J Clin Pharmacol.* 2011 Oct 28. [Epub ahead of print.]

Steele C. *Nurse to Nurse Dementia Care.* Columbus, OH: McGraw Hill; 2010.

Wilkinson D. A review of the effects of memantine on clinical progression in Alzheimer's disease. *Int J Geriatr Psychiatry.* 2011 Oct 1. [Epub ahead of print.]

Wong C, Merrilees J, Ketelle R, Barton C, Wallhagen M, Miller B. The experience of caregiving: differences between behavioral variant of frontotemporal dementia and Alzheimer disease. *Am J Geriatr Psychiatry.* 2011 Sep 21. [Epub ahead of print.]

The author and planners have disclosed that they have no financial relationships related to this article.

DOI-10.1097/01.NME.0000411089.93747.25

For more than 27 additional continuing education articles related to psychiatric/psychosocial topics, go to **Nursingcenter.com/CE.**

CE CONNECTION

Earn CE credit online:
Go to http://www.nursingcenter.com/CE/nmie and receive a certificate *within minutes*.

INSTRUCTIONS

The many faces of dementia

TEST INSTRUCTIONS

- To take the test online, go to our secure Web site at **http://www.nursingcenter.com/CE/nmie**.
- On the print form, record your answers in the test answer section of the CE enrollment form on page 55. Each question has only one correct answer. You may make copies of these forms.
- Complete the registration information and course evaluation. Mail the completed form and registration fee of $21.95 to: Lippincott Williams & Wilkins, CE Group, 2710 Yorktowne Blvd., Brick, NJ 08723. We will mail your certificate in 4 to 6 weeks. For faster service, include a fax number and we will fax your certificate within 2 business days of receiving your enrollment form.
- You will receive your CE certificate of earned contact hours and an answer key to review your results.There is no minimum passing grade.
- Registration deadline is April 30, 2014.

DISCOUNTS and CUSTOMER SERVICE

- Send two or more tests in any nursing journal published by Lippincott Williams & Wilkins together by mail and deduct $0.95 from the price of each test.
- We also offer CE accounts for hospitals and other health care facilities on nursingcenter.com. Call 1-800-787-8985 for details.

PROVIDER ACCREDITATION

Lippincott Williams & Wilkins, publisher of *Nursing made Incredibly Easy!*, will award 2.3 contact hours for this continuing nursing education activity.

Lippincott Williams & Wilkins is accredited as a provider of continuing nursing education by the American Nurses Credentialing Center's Commission on Accreditation.

This activity is also provider approved by the California Board of Registered Nursing, Provider Number CEP 11749 for 2.3 contact hours. Lippincott Williams & Wilkins is also an approved provider of continuing nursing education by the District of Columbia and Florida #FBN2454.

Your certificate is valid in all states.

The ANCC's accreditation status of Lippincott Williams & Wilkins Department of Continuing Education refers only to its continuing nursing educational activities and does not imply Commission on Accreditation approval or endorsement of any commercial product.