CHAPTER 46 Nursing Care of Clients with Neurologic Disorders

LEARNING OUTCOMES

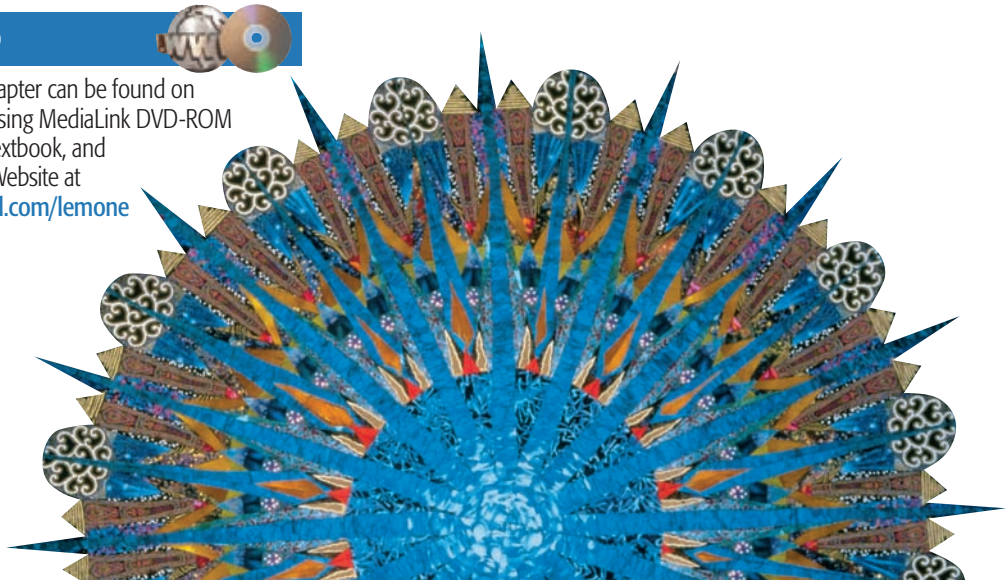
- Identify prevalence, incidence, and risk factors for degenerative neurologic, peripheral nervous system, cranial nerve, and infection- and neurotoxin-caused neurologic disorders.
- Explain the pathophysiology, manifestations, complications, interdisciplinary care, and nursing care of clients with neurologic disorders.
- Compare and contrast the manifestations of the progressive stages of Alzheimer's disease.
- Discuss the purposes, nursing implications, and health education for the client and family for medications used to treat Alzheimer's disease, multiple sclerosis, Parkinson's disease, and myasthenia gravis.
- Describe the procedures (thymectomy, percutaneous rhizotomy, plasmapheresis) used to treat selected neurologic disorders.

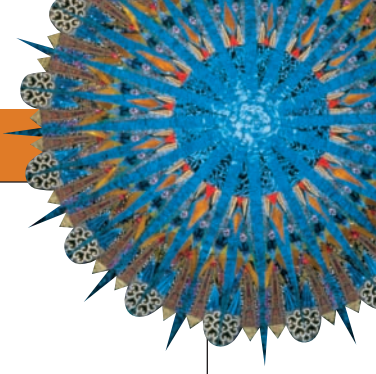
CLINICAL COMPETENCIES

- Assess functional status of clients with neurologic disorders, and monitor, document, and report abnormal manifestations.
- Use evidence-based research to design nursing interventions specific to the needs of aging clients with multiple sclerosis.
- Determine priority nursing diagnoses, based on assessed data, to select and implement individualized nursing interventions for clients with neurologic disorders.
- Administer oral and injectable medications used to treat neurologic disorders knowledgeably and safely.
- Provide skilled care to clients having a thymectomy, percutaneous rhizotomy, or plasmapheresis.
- Integrate interdisciplinary care into care of clients with neurologic disorders.
- Provide appropriate teaching to facilitate safety and communication, prevent neurologic infections and toxins (rabies, tetanus, and botulism), and facilitate community-based acute and chronic self-care for healthcare needs resulting from neurologic disorders.
- Revise plan of care as needed to provide effective interventions to promote, maintain, or restore functional health status to clients with neurologic disorders.

MEDIALINK

Resources for this chapter can be found on the Prentice Hall Nursing MediaLink DVD-ROM accompanying this textbook, and on the Companion Website at http://www.prenhall.com/lemone





KEY TERMS

Alzheimer's disease (AD), *1617*
amyotrophic lateral sclerosis (ALS), *1645*
Bell's palsy, *1657*
botulism, *1661*
Creutzfeldt-Jakob disease (CJD), *1658*
dementia, *1617*
Guillain-Barré syndrome (GBS), *1653*
Huntington's disease, *1642*
multiple sclerosis (MS), *1626*
myasthenia gravis, *1647*
Parkinson's disease (PD), *1635*
postpoliomyelitis syndrome, *1659*
rabies, *1660*
sundowning, *1620*
tetanus, *1661*
trigeminal neuralgia, *1655*

This chapter discusses a variety of neurologic disorders. Included are degenerative disorders, peripheral nervous system disorders, cranial nerve disorders, and disorders caused by neurotoxins and viruses. For many of the disorders, nursing care is based on similar nursing diagnoses. To avoid repeating those diagnoses and interventions for each disorder, they have been divided among the nursing care discussions as appropriate.

DEGENERATIVE NEUROLOGIC DISORDERS

Degenerative neurologic disorders affect the central nervous system and the peripheral nerves. By progressively disrupting cognitive processes or motor functions, disorders such as Alzheimer's disease, Parkinson's disease, and multiple sclerosis strike at the core of an individual's sense of personal autonomy and well-being and can be psychologically and emotionally devastating to family members and caregivers.

Ongoing medical research into degenerative neurologic disorders offers an increasing measure of hope to clients and their families. The discovery of genetic or biochemical markers associated with some of these disorders is leading to the development of effective screening and diagnostic methods. In addition, new drugs may make it possible to halt the progression of the disorders in some clients, transforming the disorders into manageable conditions. This chapter begins with a discussion of dementia, which is not a specific disease, but rather a collection of manifestations that are caused by a variety of disorders that affect the brain.

DEMENTIA

Dementia affects multiple cortical functions, calculation, learning capacity, language, and judgment. Impairments of cognitive function are usually accompanied by deterioration in emotional control, social behavior, and motivation. People with dementia lose their ability to solve problems, and may also have personality changes such as agitation and hallucinations. All forms of dementia result from death of neurons and/or the loss of communication among the cells. Although the exact cause is not always known, many forms of dementia are characterized by abnormal structures in the brain called inclusions, and there is clearly a genetic component in the development of some kinds of dementia.

It is estimated that as many as 6.8 million people in the United States have dementia, and at least 1.8 million of those are severely affected. Studies of some communities have found that almost half of all people age 85 and older have some form of dementia. However, despite increased incidence in older people, dementia is not a normal part of aging (National Institute of Neurological Disorders and Stroke [NINDS], 2005i).

Many different diseases and conditions may cause dementia, including Alzheimer's disease, vascular dementia, Huntington's disease, Creutzfeldt-Jakob disease, medications, metabolic disorders, poisoning, and anoxia. Table 46–1 provides an overview of the most common causes of dementia. Doctors do not diagnose dementia unless two or more brain functions (such as memory, language skills, perception, reasoning, or judgment) are significantly impaired without loss of consciousness.

Even though the actual cause of all dementias may not be known, factors that increase the risk of developing one or more kinds of dementia have been identified. These risk factors include advancing age, a family history of a disease that causes dementia, smoking and alcohol use, atherosclerosis, high cholesterol and plasma homocysteine levels, diabetes, mild cognitive impairment, and Down syndrome.

Although sometimes confused with dementia, people often experience other conditions that may mimic dementia. These include the following:

- Age-related cognitive decline, resulting from slower information processing and mild memory impairment. With aging, the brain often decreases in volume and some neurons are lost. These changes are normal and are not considered a part of dementia.
- Mild cognitive impairment, which may progress to dementia, but is not severe enough to be initially diagnosed as such.
- Depression or other emotional problems, causing people to be passive, slow, confused, or forgetful.
- Delirium, characterized by confusion, rapidly altering mental states, disorientation, and possible personality changes. Delirium is usually caused by a treatable physical or mental health illness and, when treated, results in a full recovery.

THE CLIENT WITH ALZHEIMER'S DISEASE

Alzheimer's disease (AD) (also called *dementia of Alzheimer type [DAT]* or *senile disease complex*) is a form of dementia characterized by progressive, irreversible deterioration of general intellectual functioning. Clients with AD live about 8 to 10 years following diagnosis, although some live as long as

TABLE 46–1 **Common Causes of Dementia**

NAME	CAUSE AND PRIMARY PATHOPHYSIOLOGY
Alzheimer's disease (the most common cause of dementia in people age 65 and older)	Unknown cause; characterized by two abnormalities in the brain: amyloid plaques and neurofibrillary tangles.
Vascular dementia (the second most common cause of dementia)	Caused by brain damage from cerebrovascular and cardiovascular problems (usually strokes). May also be caused by cerebral blood vessel damage from genetic disorders, endocarditis, myeloid angiopathy, vasculitis, and profound hypotension.
Lewy body dementia	Cause usually unknown, although familial cases have been reported. Cells die, and remaining cells in the in substantia nigra contain abnormal structures called Lewy bodies.
Frontotemporal dementia	Nerve cells, especially in the frontal and temporal lobes, degenerate. In many people, abnormal tau protein accumulates in neurofibrillary tangles.

20 years. The cause of death is often aspiration pneumonia because of the loss of the ability to swallow late in the disease (NINDS, 2005i).

Memory loss is usually the first sign of Alzheimer's disease. Memory deficits are initially subtle and family members and friends may not suspect a problem until the disease progresses and manifestations become more noticeable. Family members and clients with AD may also deny the manifestations and hide deficits until the person exhibits unsafe or extremely unusual behavior. Progression of the disease varies, but the course is one of deteriorating cognition and judgment with eventual physical decline and total inability to perform activities of daily living (ADLs). With the loss of the ability to perform even the most basic ADLs, the burden of meeting the client's needs shifts to the caregiver.

Incidence and Prevalence

Alzheimer's disease is the most common degenerative neurologic disorder and the most common cause of cognitive impairment in older adults (Porth, 2005). It accounts for about two-thirds of cases of dementia in America, affecting adults in middle to late life.

Two types of AD exist: *Familial AD* follows an inheritance pattern and *sporadic AD* has no obvious inheritance pattern. AD is further described as early onset (occurring in people younger than 65) and late onset (occurring in people age 65 and older). Early onset AD affects people ages 30 to 60, is relatively rare, and often progresses more rapidly than late-onset AD.

FAST FACTS

Alzheimer's Disease

- Scientists estimate that more than 4.5 million people have AD, usually beginning after age 60, with the risk increasing with age (Alzheimer's Foundation, 2005).
- One in 10 people over the age of 65 and almost one-half of all people over age 85 have AD.
- Each year, at least 360,000 people are diagnosed with AD, and about 50,000 are reported to die from it.
- Early onset forms of AD, which are usually genetic, may appear as early as age 30.

Source: NINDS, 2005i.

Risk Factors and Warning Signs

As one ages, the risk of developing AD increases. With numbers of older people increasing, the incidence of AD is predicted to also increase. The risk factors for AD are older age, family history, and female gender. Warning signs are:

- Memory loss that affects job skills
- Difficulty performing familiar tasks
- Problems with language
- Disorientation to time and place
- Poor or decreased judgment
- Problems with abstract thinking
- Misplacing things
- Changes in mood or behavior
- Changes in personality
- Loss of initiative.

Recognizing early manifestations is important, because the cause of dementia (such as from depression or hypothyroidism) may be reversible. Further information can be obtained from the Alzheimer's Association. Dementia from AD is not reversible. Treatment, however, can maximize quality of life and allow the affected person to plan for the future.

Pathophysiology

Characteristic findings in the brains of AD clients are loss of nerve cells and the presence of *neurofibrillary tangles* and *amyloid plaques* (Figure 46–1 ■). Neurofibrillary tangles result when *tau*, a kind of protein in the neurons, becomes distorted and twisted. Tau normally holds together the microtubules, which guide nutrients and molecules to the end of the axon. In AD, tau changes and twists into pairs of filaments, which then join to form tangles. Because tau no longer maintains the transport system, communication is lost between neurons. Death of neurons may follow, contributing to the development of dementia.

Groups of nerve cells (and especially the terminal axons) degenerate and clump around an amyloid core as plaques, found in the spaces between the neurons of the brain. These plaques, which develop first in areas used for memory and cognition, disrupt transmission of nerve impulses. The plaques consist primarily of insoluble deposits of beta-amyloid, a protein fragment from a larger protein called amyloid precursor protein, mixed with other neurons and nonnerve cells. It is not

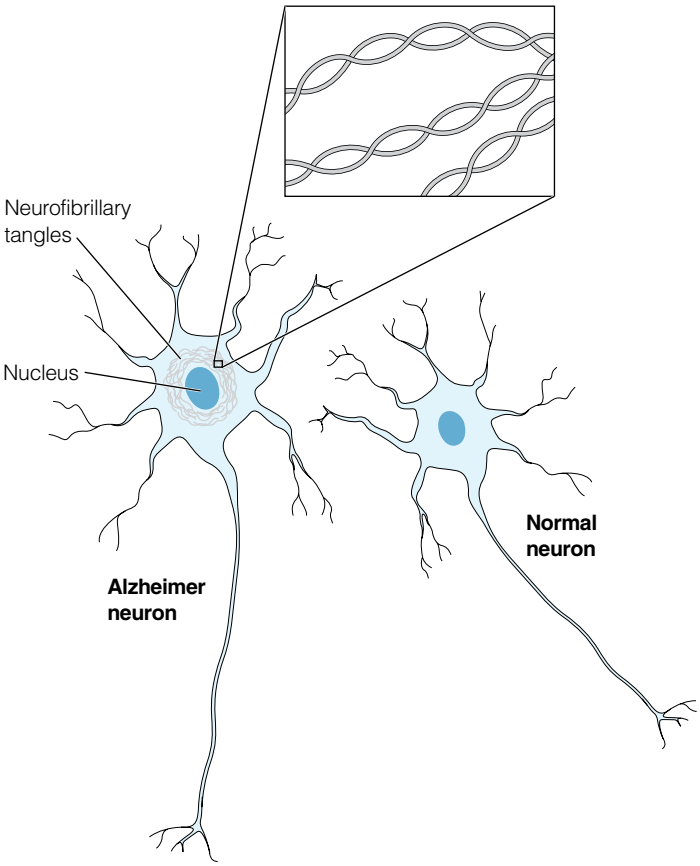


Figure 46–1 ■ Neuron with neurofibrillary tangles seen in Alzheimer's disease.

yet known if plaque formation causes AD or if plaques are a by-product of the AD process.

Blood flow to the affected areas of the brain is decreased. The brain atrophies, and corresponding enlargement of ventricles and sulci is evident (Figure 46–2 ■). As AD progresses, more areas of the brain are affected, with manifestations correlating to those affected areas of the brain. For example, neuronal and neurotransmitter losses in the parietal lobe result in problems with perception and interpretation of environmental stimuli; deficits in the frontal lobe cause changes in personality and emotional lability.

AD is characterized by atrophy of the cortical area of the brain and loss of neurons, especially in the parietal and temporal lobes. With significant atrophy and loss of brain tissue, the ventricles enlarge (a form of hydrocephalus) (Porth, 2005). Several structural and chemical changes in the brain occur with AD, especially in the hippocampus and the frontal and temporal lobes of the cerebral cortex. As AD destroys neurons in the hippocampus and related structures, short-term memory fails and the ability to perform easy and familiar tasks declines. The effect of AD on neurons in the cerebral cortex is loss of language skills and judgment. Emotional outbursts and behavior changes (such as wandering and agitation) begin to occur and become more frequent as the disease progresses. Eventually, other areas of the brain are affected; all affected areas begin to atrophy, and the person becomes totally helpless and unresponsive.

The exact cause of AD is unknown. Theories include a decrease in choline acetyltransferase activity in the cortex and hippocampus. This enzyme is necessary for the synthesis of acetylcholine, a neurotransmitter associated with memory. The decrease in choline acetyltransferase is about equal to the severity of AD. Other theories include a mutation for encoding amyloid precursor protein, and alteration in apolipoprotein E. Other possible causes are gene defects on chromosomes 14, 19, or 21, which may lead to clumping and precipitation of insoluble amyloid as plaques. The role of protein kinase C, the link between AD and aluminum, a viral cause, an autoimmune cause, and mitochondrial defects that alter cell metabolism and protein processing are being studied.

Manifestations

Alzheimer's disease is classified into three stages based on the client's manifestations and abilities, as outlined in the box on page 1620. It is important to note that the progression of AD varies for each individual and may not precisely follow the model.

Stage 1 AD

In stage 1, a client typically appears physically healthy and alert, and cognitive deficits can go undetected unless thorough and periodic evaluations are performed. Clients may seem restless, forgetful, or uncoordinated; they may lack spontaneity, and be disoriented as to time and date. Usually, family members are the first to notice lapses in memory, subtle changes in personality, or problems in doing simple calculations. AD clients and families may consciously or unconsciously compensate for cognitive deficits by adjusting schedules and routines.

Stage 2 AD

In stage 2, memory deficits are more apparent, and the client is less able to behave spontaneously. Clients may wander and get

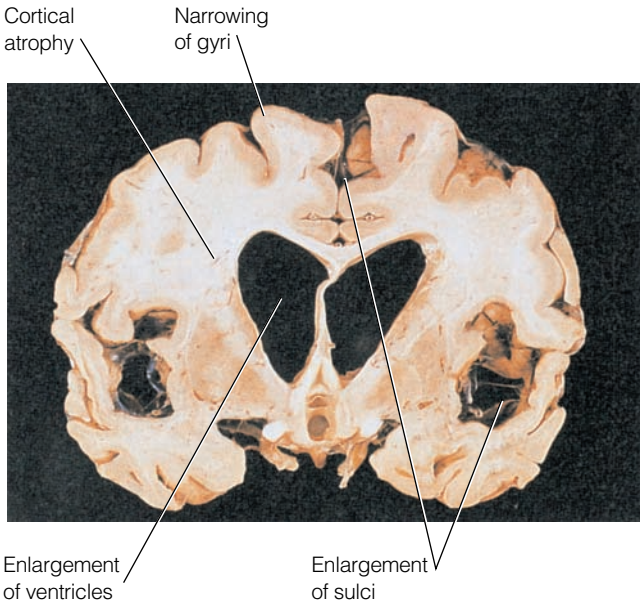


Figure 46–2 ■ Changes in neuroanatomy associated with Alzheimer's disease. Note areas of cortical atrophy, narrowing of the gyri, enlargement of sulci, and ventricular dilation.

MANIFESTATIONS of Alzheimer's Disease

STAGE 1: APPROXIMATELY 2 TO 4 YEARS

- Short-term memory loss: Forgets location and names of objects and has difficulty learning new information; long-term memory is unaffected.
- Decreased attention span.
- Subtle personality changes: Lacks spontaneity; denial, irritability, and depression are possible.
- Mild cognitive deficits: Attempts to adjust to and cover up memory loss.
- Visuospatial deficits: Some problems with depth perception.

STAGE 2: APPROXIMATELY 2 TO 12 YEARS

- Impaired cognition: Obvious memory deficits and confusion; loss of abstract thinking; astereognosis and agraphia; inability to do math calculations; loss of ability to tell time and time disorientation, manifested as "sundowning"; wandering behavior.
- Personality changes: Becomes easily agitated and irritable; may have delusions or hallucinations.
- Visuospatial deficits: Is unable to dress self; has poor spatial orientation.
- Impaired motor skills: Paces and is restless at times; motor apraxia is evident when using familiar objects.
- Impaired judgment: Diminished social skills; inability to drive a car; inability to make decisions (e.g., choose clothing).

STAGE 3: APPROXIMATELY 2 TO 4 YEARS OR LONGER

- Cognitive abilities grossly decreased or absent: Is usually disoriented to time, place, and person.
- Communication skills usually absent: Is frequently mute.
- Motor skills grossly impaired or absent: Limb rigidity and posture flexion; bowel and bladder incontinence.

lost, even in their own homes. Although progression of manifestations continues and orientation to place and time deteriorates, AD clients may still have periods of mental lucidity and engage in time-oriented conversations. Generally, however, clients become more confused and lose their sense of time, leading to changes in sleeping patterns, agitation, and stress. They may demonstrate repetitive behavior and eat ravenously. AD clients are less able to make even simple decisions and to adapt to environmental changes, and are often unable to carry out ADLs. **Sundowning** is another behavioral change, characterized by increased agitation, time disorientation, and wandering behaviors during afternoon and evening hours; it is accelerated on overcast days.

Language deficits are common in stage 2. They include *paraphasia* (using the wrong word), *echolalia* (repetition of words or phrases), and *scanning speech,* in which the client appears to search for words. Eventually, total *aphasia* (absence of speech) may occur. Frustration and depression are common among AD clients as the full extent and implications of the deficits become obvious.

The AD client slowly loses the ability to perform simple tasks required for hygiene or eating because sequencing of tasks is lost. For example, the client may open a can of soup but not remember to pour it into a pan to heat it. Instead, the client might place the can directly on the burner and leave the heat on high even after a smoke alarm sounds. The AD client may falsely interpret the smoke alarm as a telephone ringing, a tornado warning siren, or an ambulance siren. Thus, safety is a high priority for the client in stage 2.

Sensorimotor deficits in stage 2 include *apraxia,* the inability to perform purposeful movements and use objects correctly; *astereognosis,* the inability to identify objects by touch; and *agraphia,* the inability to write properly. Problems related to malnutrition and decreased fluid intake, such as anemia and constipation, may be evident. Sleep pattern disturbances are also common and are related to the loss of time orientation, sundowning phenomenon, and depression.

Stage 3 AD

Stage 3 brings increasing dependence, with inability to communicate, loss of urinary and fecal continence, and progressive loss of cognitive abilities. Common complications include pneumonia, dehydration, malnutrition, falls, depression, delusions, seizures, and paranoid reactions. AD clients are indifferent to food and lose weight. They are unable to recognize family or friends, or even themselves. The average life expectancy is 1 to 2 years from the onset of stage 3, although the individual may live as long as 10 years. Most people with AD are institutionalized during this final stage of the disease. Death frequently occurs from pneumonia secondary to aspiration.

INTERDISCIPLINARY CARE

There is no cure for AD, and the main objective of care is to provide an environment that matches the client's functional abilities. Nurses, physicians, physical therapists, and social workers collaborate with the client's family to provide the least restrictive environment in which the client can safely function.

Diagnosis

Alzheimer's disease is diagnosed by ruling out causes for the client's manifestations. The only definitive method of diagnosis is postmortem examination of brain tissue. An extensive workup is especially important, because the dementia may be due to a reversible or treatable condition. For example, an older client's misuse of medications can lead to overdosing and resulting confusion. Other categories of conditions that may be considered and ruled out include depression, infection, hypothyroidism, dehydration, heart disease, stroke, and chronic obstructive respiratory disease. Mental status is assessed with tests such as the Folstein Mini Mental Status Examination. This examination assesses areas of function such as the client's orientation to time, ability to repeat back a series of words, ability to name objects, and ability to follow written instructions.

MEDICATION ADMINISTRATION **The Client with Alzheimer's Disease**

CHOLINERGIC (PARASYMPATHOMIMETICS); CHOLINESTERASE INHIBITORS

Tacrine hydrochloride (Cognex)
Donepezil hydrochloride (Aricept)
Rivastigmine tartrate (Exelon)

In the early stages of AD, the pathologic changes in neurons result in a deficiency of acetylcholine (a key neurotransmitter involved in cognitive functioning). Cholinesterase inhibitors slow the breakdown of acetylcholine release by the remaining intact neurons. In addition, rivastigmine tartrate inhibits the G_1 form of acetylcholinesterase (found in higher levels in the brain of clients with AD), so less acetylcholine is degraded. The drugs are used to improve memory in mild to moderate AD dementia.

Nursing Responsibilities

- Administer tacrine hydrochloride 1 hour before meals, if possible.
- Administer donepezil hydrochloride at bedtime.
- Administer rivastigmine tartrate (both capsules and liquid) with food. Liquid form may be administered undiluted or mixed with water, juice, or soda. Stir to completely dissolve.
- Monitor for jaundice, increased bilirubin levels, and other signs of liver involvement, such as rising serum aminotransferase (AST, ALT) levels. Therapy is usually decreased when the enzyme level exceeds four times normal limits and discontinued when the level reaches five times normal.
- Observe for gastrointestinal bleeding and gastric ulcer pain.
- Monitor for cholinergic-related problems: bladder outlet obstruction, seizures, and slowed cardiac rate.
- Assist with ambulation because dizziness is a common side effect.
- Monitor glycemic control in clients with diabetes.
- Assess for improvement in AD symptoms, especially in reasoning, memory, and ADLs.

Health Education for the Client and Family

- Notify the physician promptly if jaundice, seizures, slowed heart rate, GI bleeding, or difficulty urinating occurs.
- Follow directions for times and instructions about administration of specific medication.
- Follow your healthcare provider's recommendation for periodic EEG, blood tests, and urine tests.
- These medications do not cure AD, and will at some point become ineffective as the disease progresses.

Guidelines for the early recognition and assessment of AD have been established by the Agency for Healthcare Research and Quality. A diagnosis of Alzheimer's disease requires the documented presence of dementia, onset between age 40 and 90 years (most often after age 65), no loss of consciousness, and absence of systemic or brain disorders that could cause mental changes.

Medications

There is no cure for AD, but some medications are effective in slowing the progression of the disease. Tacrine hydrochloride (Cognex) was the first medication specifically approved for the treatment of AD. Donepezil hydrochloride (Aricept) is used to treat mild to moderate AD dementia with some success. Rivastigmine tartrate (Exelon) is also used to treat mild to moderate AD manifestations. It improves the ability to carry out ADLs, decreases agitation and delusions, and improves cognitive function. Galantamine hydrobromide (Reminyl) is believed to increase the concentration of acetylcholine in the central nervous system (CNS) as treatment of mild to moderate AD. Memantine (Namenda) improves cognitive function in moderate-to-severe AD and mild-to-moderate vascular dementia. Memantine acts by blocking receptors for glutamate, resulting in decreased calcium accumulation into neurons (increased calcium accumulation damages neurons). See the box above for information about selected medications used to treat AD.

Depression often accompanies AD and is treated with the appropriate medication. Antihistamines and tricyclic antidepressants that have high anticholinergic activity are usually avoided because they can increase AD manifestations. Occasionally clients with AD require tranquilizers such as thioridazine (Mellaril) or haloperidol (Haldol) to manage severe agitation. Other therapies under study to prevent or delay the onset of AD include antioxidants such as vitamin E, anti-inflammatory agents, and antihypertensive drugs to lower hypertension.

Alternative and Complementary Therapy

The following types of alternative and complementary therapies may be used in treating the manifestations of AD.

- Massage, which decreases agitation
- Herbs:
  - Ginkgo biloba and vitamin E, which are thought to improve cognition
  - Huperzine A, a traditional Chinese medicine, which acts as an acetylcholinesterase inhibitor
- Coenzyme Q10, an antioxidant that naturally occurs in the body
- Supplements, such as zinc, selenium, and evening primrose oil
- Therapies involving art, music, sound, and dance.

NURSING CARE

Clients with AD often require intensive, supportive nursing interventions directed at the physical and psychosocial responses to illness. Equally important, the nurse can facilitate the long-term support of these clients by providing teaching and referrals to follow-up care in the community. A Nursing Care Plan for the client with AD is found on the next page.

Health Promotion

Health promotion for the client with AD focuses on maintaining functional abilities and safety. If the client will be cared for at home, address safety considerations (see the box

NURSING CARE PLAN **A Client with AD**

Arthur and Ruth Joste, both age 73, have been married for 47 years; he is a retired history teacher, and she has been a homemaker. They have four children; two live in the same town, and two live out of state. Arthur has noticed that he is having problems remembering friends' names and phone numbers; his wife has been asking him if he is driving in the correct direction when they go shopping.

Mrs. Joste has severe osteoarthritis and is unable to lift heavy objects or perform all but light housekeeping tasks. For about 18 months, Mrs. Joste has been aware of her husband's progressive cognitive decline, including forgetting current news from last night's newspaper; miscalculating checkbook balances; neglecting his hygiene needs; and confusing their children's and grandchildren's names. The Jostes are referred to a neurologist for evaluation.

ASSESSMENT

Martha Spital, RN, assesses Mr. Joste at the neurologist's office. She notes that he is unable to recall his home address without prompting, to name the correct date (although he does know the day of the week), to subtract serial 7s more than twice, and to recall two of three objects. He is alert to his surroundings. Mrs. Joste states that the problems seem to be getting worse with time and that she has had to "cover up" mistakes for her husband. Mr. Joste seems easily agitated, and his wife reports that his sleep habits are "jumbled"; he has long periods of wakefulness in the nighttime hours.

Following a thorough evaluation and diagnostic testing to rule out other possible disorders, the neurologist tells the couple that Mr. Joste has probable dementia of the Alzheimer's type. Both have feared this diagnosis; they want to know how they can be sure that Mr. Joste has this disease and what they can do to prevent further decline. Both are obviously much saddened, and they verbalize their feelings of being overwhelmed. The Jostes intend to remain in their home "for as long as we can."

DIAGNOSES

- *Chronic Confusion* related to deterioration of brain function and dementia
- *Self-Care Deficits* related to forgetfulness and declining physical abilities
- *Risk for Injury* related to decreased orientation
- *Disturbed Sleep Pattern* related to time disorientation
- *Caregiver Role Strain* (wife) related to need to care for self and husband

EXPECTED OUTCOMES

- Remain free of injury.
- Navigate home environment with modifications as needed.
- Participate in grooming and hygiene activities with prompting and supervision.
- Obtain a minimum of 7 uninterrupted hours of sleep a night.
- Mrs. Joste will participate in a minimum of two out-of-home activities a week.

PLANNING AND IMPLEMENTATION

The home health nurse, Erick Montane, RN, makes a home visit to evaluate the environment, assess available support, and determine needs. He meets two of the Jostes' children, Dawn and Jay, who live in the same community and are willing to participate as much as possible in providing care and modifying the home.

Mr. Montane discusses the importance of establishing and maintaining a consistent daily routine. He emphasizes the importance of matching activities to Mr. Joste's mental abilities to avoid frustration and increased agitation. Mr. Montane recommends labeling drawers with their contents, such as Mr. Joste's sock drawer. Labeling rooms may eventually be necessary.

Because his inability to comprehend and process information distresses and agitates Mr. Joste, Mr. Montane teaches the family to modify their communications to fit Mr. Joste's cognitive ability, such as using simple, direct statements and directions.

Mr. Montane recommends that family members keep background noise to a minimum because this may be a source of confusion.

After assessing the home, Mr. Montane makes the following recommendations about safety:

- Remove throw rugs from hallways, and tack down any remaining carpets.
- Secure the kitchen, bathroom, and workshop cabinets as well as the controls on the oven and stove.
- Modify the doors so that negotiating locks requires a two-step system of unlocking, such as with a deadbolt and a key.
- Provide extra lighting in dark areas, especially a night-light in the bathroom.

Mr. Montane explains that Mrs. Joste will need assistance with housekeeping as Mr. Joste continues to decline. Mr. Montane provides referrals to community services, including Meals-on-Wheels, which can supply a daily meal. He also suggests that the Jostes obtain the services of a home health aide to provide daily hygiene care. Most of the remaining home maintenance needs can be met with the children's help.

Mr. and Mrs. Joste and the two children attend the weekly local support group meetings for Alzheimer's disease and related disorders for approximately 3 months; thereafter, Mrs. Joste attends with her daughter.

EVALUATION

Six months after the initial home visit and family planning session, Mr. Joste:

- Has not had a fall, burn, or other injury.
- Has periods of confusion when outside his home, but 90% of the time is oriented to place when at home.
- Has attended several support group meetings until 3 months ago. Currently, his wife attends weekly, and a daughter occasionally accompanies her. She has continued to participate in their church and maintains contact with a few friends. She is finding it harder to leave her husband unattended for even a few minutes.
- Is able to clean and dress himself with prompting; he is not able to choose his own clothing. If hygiene articles are "set up" (e.g., if the toothpaste is placed on the toothbrush), he remembers to perform the hygiene activity. The children have been replacing buttons and zippers with Velcro closures on his clothing.
- Sleeps an average of 6 hours a night with a 30-minute nap in the afternoon; this pattern is consistent with his previous sleep pattern.

NURSING CARE PLAN A Client with AD (continued)

- Has seemed to be more easily agitated for the past month. He wanders from room to room, apparently looking for something. These behaviors are worse in the evening and on cloudy days. Mrs. Joste acknowledges her progressive inability to care for her husband.

CRITICAL THINKING IN THE NURSING PROCESS

1. Develop a tool to teach safety needs for the client and family with Alzheimer's disease.
2. List five interventions to decrease agitation in cognitively impaired older adults; give three additional examples of activities suited to an older adult with AD who has osteoarthritis.
3. You are caring for a client in stage 2 Alzheimer's disease. She is 65 inches (165 cm) tall and weighs 132 lb (59.9 kg); she has lost 3 lb within the past month. The client has difficulty focusing on eating and is easily agitated. Describe your plan for ensuring that she has adequate nutrition.

See Evaluating Your Response in Appendix C.

below) as well as the caregivers' abilities to meet the client's basic needs, such as maintaining hygiene and other ADLs. Adapt nursing interventions and teaching to the client's stage of Alzheimer's disease. Nurses also promote health in the caregiver; information about caregiver support systems and respite care should be provided.

Assessment

Collect the following data through the health history and physical examination (see Chapter 43 ∞). Further focused assessments are described with nursing interventions below.

- *Health history:* Family member/caregiver support, living arrangements, ability to carry out ADLs, drug use, work history (e.g., exposure to metals), previous history of multiple strokes, brain injury or brain infection, family history of dementia, sleep pattern, changes in cognition and memory, ability to communicate, changes in behavior.
- *Physical assessment:* Height/weight, orientation, abstract reasoning, mental status.

Nursing Diagnoses and Interventions

During the early stage of AD, nursing care focuses on helping the client make minor adaptations to his or her environment. As the client becomes progressively unable to manage self-care tasks, more adaptations are required. Equally important, the caregiver needs much support—both physical and psychosocial—as the client becomes increasingly dependent.

Impaired Memory

Impaired memory is an appropriate nursing diagnosis in stage 1 AD. At this stage, techniques to help with the memory loss should be included in teaching for both the client and the caregiver.

- Suggest complementary therapies, such as meditation, massage, or exercise. *These activities can help reduce stress; stress can aggravate memory loss.*
- Suggest using a calendar, keeping lists of reminders, or asking someone else to remind of appointments and events. *Written or verbal reminders are helpful if memory is impaired.*
- Recommend using a medication box labeled with days and times. *A medication box is a good way to remember to take medications.*

PRACTICE ALERT

It may be necessary to teach the caregiver how to refill the medication box, or to stress the importance of spot-checking if the client fills it.

MEETING INDIVIDUALIZED NEEDS Safety Interventions for the Client with AD

DECREASING THE RISK OF FALLS

- Assess usual environment for hazards, such as throw rugs, electrical cords, and slick floors.
- Observe areas of special concern, such as the bathroom, kitchen, and stairs, and modify as needed; for example, provide skidproof surfaces, and mark stairs to show depth.
- Evaluate muscle strength and gait; consult a physical therapist to plan exercises to increase strength and balance.
- Check shoes for fit and support.
- Inquire about alcohol use and medications that affect balance or cause mobility problems; for example, antihypertensive agents can cause dizziness with position changes.
- Use night-lights and increase daytime lighting in dark areas; such as hallways.
- Keep traffic areas free from clutter.

DECREASING THE INJURIES RELATED TO COGNITIVE IMPAIRMENTS

- Secure items that may be mistakenly ingested, such as cleaning preparations and house plants.
- Modify potentially unsafe areas, such as unenclosed porches.
- Provide double lock systems to outside doors and doors to rooms that are off-limits.
- Protect from fire hazards; for example, make matches and cigarettes inaccessible.
- Fence the yard with a locked gate to prevent wandering.
- Modify the controls on the oven and stove.
- Adjust the water heater to a safe temperature.

GENERAL SAFETY CONSIDERATIONS

- Plan a calling system for emergencies; have children call at about the same time every day as a check.
- Ensure that the cognitively impaired family member has no access to objects in the home such as knives and guns.

- If safety is a concern (such as turning on the stove and forgetting it), suggest using alternatives such as a microwave. Program emergency numbers into the telephone. Ask client to consider a Life-Line telephone program. *These measures can increase safety.*
- Suggest using cues, such as an alarm on a watch or a pocket computer, to trigger actions at designated times. *Cues are often helpful when memory loss is a problem.*

Chronic Confusion

Clients with AD often have memory deficits that make functioning in a nonstructured environment difficult. Many of the nursing interventions for this diagnosis need to be modified over time as the client continues to lose cognitive function.

- Label rooms, drawers, and other items as needed. *Visual cues promote the highest possible degree of independence for the client.*
- Remove potential hazards (such as sharp knives or potentially harmful liquids or chemicals) from the environment. *Ensuring safety is a critical factor in providing care.*
- Keep environmental stimuli to a minimum: Decrease noise levels; speak in a calm, low voice; and take an unhurried approach. *Minimizing sensory input and maintaining a calm manner may decrease anxiety.*
- Begin each interaction by identifying self and calling client by name. See Box 46–1 for other communication techniques. *These techniques provide information for the client with memory loss.*
- Limit questions to those that require a simple yes or no response. *Questions need to be appropriate to the client's ability as decision making and verbal skills decline.*
- Orient to the environment, person, and time as able; place large, easy-to-read calendars and clocks in the client's line of vision. Make references to the season or day of the week when conversing with the client. *Orient the client according to his or her level of ability; orienting to precise time may not be possible in the later stages of AD.*
- Provide boundaries by placing red or yellow tape on the floor. *Boundaries help the client stay within safe areas.*

PRACTICE ALERT

Red and yellow are more easily seen by older adults.

- Provide continuity in nursing staff. *This not only promotes consistency of care for the client but also allows the nurse to determine more accurately changes in the client's condition.*
- Repeat explanations simply and as needed to decrease anxiety. *Loss of short-term memory leads to loss of a point of reference; eventually, AD clients think they are experiencing everything for the first time.*

Anxiety

Managing the AD client's behaviors associated with anxiety, restlessness, and confusion is a major challenge confronting nurses and caregivers. Frequently, clients are relatively calm in the morning hours, only to experience increasing periods of agitation in the afternoon and evening hours. The AD client may even waken from the night's sleep with confusion, fearfulness, or panic attacks.

- Monitor for early behaviors of fatigue and agitation. *Early assessment of problems results in prompt intervention to promote rest or to remove the client from the situation causing anxiety.*
- Remove from situations that are causing increased anxiety, such as noisy activities involving large groups. *High-stimulus situations may increase anxious feelings and agitation.*
- Keep daily routine as consistent as possible. *Providing a structured day enhances feelings of familiarity and decreases stress.*
- Schedule rest periods or quiet times throughout the day. *Fatigue contributes to anxiety and lowers the ability to tolerate stress.*
- Provide quiet activities, such as listening to favorite music, in the afternoon or early evening. *Quiet activities may help decrease sundowning.*
- If confusion and agitation persist or escalate, assess for physical causes such as decreased oxygenation, infections, fatigue, constipation, and electrolyte imbalance. *Physical factors can increase agitation in clients with AD.*
- Use therapeutic touch or gentle hand massage. *These activities induce relaxation and have a calming effect.*

Hopelessness

As the client and family recognize the effect of AD on their lives, they may feel a sense of hopelessness. They may not have the coping skills to deal effectively with the diagnosis and anticipated problems. The increasingly degenerative, irreversible nature of the disorder tends to diminish hope; only the ability to adapt to the many problems can restore it.

- Assess the client's and family's response to the diagnosis and understanding of AD; encourage expression of feelings. *Understanding the client/family's perspective enables the nurse to dispel myths about AD.*

BOX 46–1 Communicating with the Client with AD

- Face the client and talk directly to him or her; call the client by name.
- When first approaching the client, identify yourself.
- Use simple sentences and words with few syllables.
- Speak in a calm, low voice.
- Ask one question at a time. Use questions that require only a yes or no response.
- Keep nonverbal communication relaxed and parallel to the verbal communication.
- Avoid giving the impression of being in a hurry; try to have a relaxed approach.
- Observe for anxiety—wringing hands, pacing, darting eye movements—and alter your approach to decrease anxiety.
- Avoid arguing with clients; do not insist on orienting client to reality; the client's point of reference may not be based in reality.
- Give plenty of time for the client with AD to process what you are trying to say; do not expect clients to perform skills beyond their abilities.
- Repeat explanations in simple terms.

- Provide realistic information about the disorder; provide information at the client/family's level of understanding. *Client and family may need to have separate sessions. Factual information provides a foundation for decision making.*
- Avoid criticizing or judging expressed feelings. *An environment accepting of the expression of real feelings promotes both further expression of feelings and willingness to discuss other issues.*
- Support positive family bonds and enhance communication among family members; promote mutual positive regard. *Strong family relationships can provide direction for living and convey a willingness to share the burden.*
- Encourage the client to make as many decisions as possible. *Self-determination enhances a feeling of control over a situation and may give a sense of hope.*
- Encourage the client and family to seek spiritual guidance that previously inspired hope. *The client's church is a legitimate support system. Belief in God can inspire hope beyond present circumstances.*

Caregiver Role Strain

Most caregivers of clients with AD are spouses or other family members. Because AD is a chronic and eventually debilitating disorder, caregivers may feel overwhelmed by their responsibilities. The caregiving spouse faces not only the responsibility for the client's multiple physical demands but also economic and psychosocial stressors. An area that must be discussed is the ability and safety of the client in driving an automobile. Although it may be necessary, the loss of independence represented by the loss of the ability to drive may further trigger anxiety and anger. Fear of the future, loss of income, loss of companionship and a mate—combined with fatigue—make the caregiver vulnerable. Caregivers may become physically and mentally exhausted and socially isolated because of the overwhelming responsibilities of providing total care to the incapacitated family member.

- Teach the caregivers self-care techniques, such as taking rest periods and avoiding fatigue. *Fatigue adds to stress and potentially leads to poor decision making.*
- Have the caregivers list and regularly take part in physical activities they enjoy, such as walking or swimming. *Regular physical exercise decreases stress.*
- Refer the caregivers to local AD support groups. Suggest books pertinent to the subject. *Explicit suggestions in locating support systems and providing specific information promotes coping.*
- Refer the caregivers to Meals-on-Wheels, home health, respite care, and other community services. *Community agencies can relieve some of the daily care burdens, thus providing time for other activities. Programs that support caregivers have been shown to delay nursing home placement.*
- Ensure the family knows that hospice care is available during the end stages of AD. *Hospice services can support the family during this difficult time.*

Using NANDA, NIC, and NOC

Chart 46–1 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with AD.

NANDA, NIC, AND NOC LINKAGES

CHART 46–1 **The Client with Alzheimer's Disease**

NANDA

Chronic Confusion

↓

NIC

Calming Technique

Dementia Management

Environmental Management: Safety

Family Support

↓

NOC

Cognition

Safe Home Environment

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2003), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

Community-Based Care

Teaching for clients and families centers initially on explaining the disorder and exploring available support systems. Anticipate the need to reexplain the disorder and its consequences, because clients and families may be in shock or denial during the initial period of the disease.

In addition to explaining the anticipated changes with AD, suggest practical solutions to identified problems. It is important to evaluate both the client and caregivers; interventions must be appropriate for the family's situation and resources. Maintaining the least restrictive environment that promotes safety for the client is a major goal of teaching. Using memory cues, such as labeling drawers to indicate the specific types of clothing and labeling rooms, can help orient the client and foster independence. Consistency in the environment and daily routine is an essential part of care. Emphasizing realistic expectations means adjusting care and communication techniques to the client's level of ability.

Address the following topics for home care of the client and for the caregiver:

- Support groups and peer counseling are helpful in handling caregiver stress.
- A person with AD who is confused or agitated is not comfortable and is usually frightened.
- Plan care that matches the person's level of coping, using a consistent routine.
- Provide regular rest periods to decrease the client's stress and fatigue (these do not increase nighttime wandering).

MEDIALINK AD Resources

- Plan care for the caregiver. Periodic adult day care or respite care during the initial stages, with plans for increasing assistance to meet the client's daily needs as the disease progresses, may be sufficient. Referrals to the appropriate agency for long-term care, including skilled nursing facilities, may be indicated. Family members may need help adjusting to the idea of extended care but may be relieved to relinquish the physical care needs.
- Suggest the following resources:
  - Alzheimer's Association
  - Alzheimer's Disease and Related Disorders Association
  - Alzheimer's Disease Education and Referral Center
  - National Institute of Neurological Disorders and Stroke.

THE CLIENT WITH MULTIPLE SCLEROSIS

Multiple sclerosis (MS) is a chronic demyelinating neurologic disease of the CNS (brain, optic nerves, and spinal cord), associated with an abnormal immune response to an environmental factor. The manifestations of MS vary according to the area of the nervous system affected. The initial onset may be followed by a total remission, making diagnosis difficult. In about 60% of clients, MS is characterized by periods of exacerbation, when manifestations are highly pronounced, followed by periods of remission, when manifestations are not obvious. The end result, however, is progression of the disease with increasing loss of function.

Incidence and Prevalence

The onset of MS is usually between 20 and 50 years of age, with a peak at age 30. MS is the most prevalent CNS demyelinating disorder, and is a leading cause of neurologic disability in young adults. Although all races are affected, MS is primarily a disease of people of northern European ancestry; however, MS does occur in people of African, Asian, and Hispanic descent. A definite genetic factor has not been established but studies suggest that genetic factors may make some individuals more susceptible than others (National MS Society, 2005).

FAST FACTS

Multiple Sclerosis

- Approximately 400,000 people in the United States have MS; the incidence is 2.5 million worldwide.
- Females are affected two times more often than males, and the incidence is highest in young adults.
- The disease occurs more commonly in temperate climates, including the northern United States. This association is established by approximately age 15, and moving to or from a temperate climate after that age does not change it.

Source: National MS Society, 2005; Porth, 2005.

Pathophysiology

MS is believed to occur as a result of an autoimmune response to a prior viral infection in a genetically susceptible person. The infection, which is thought to occur early in life, activates T cells. T cells usually move in and out of the CNS across the blood–brain barrier, but for an unknown reason, they remain in the CNS in people with MS. The T cells facilitate infiltration by other leukocytes, and an inflammatory process follows. Inflammation destroys myelin and oligodendrocytes (myelin-producing cells), leading to axon dysfunction.

Myelin sheaths are fatty, segmented wrappings that normally protect and insulate nerve fibers and increase the speed of transmission of nerve impulses. In multiple sclerosis, these myelin sheaths of the white matter of the spinal cord, brain, and optic nerve are destroyed in patches, called *plaques,* along the axon (see *Pathophysiology Illustrated* on pages 1628–1629). The demyelination of nerve fibers slows and distorts the conduction of nerve impulses and sometimes results in the total absence of impulse transmission. The neurons usually affected by MS are located in the spinal cord, brainstem, cerebral and cerebellar areas, and the optic nerve.

Both plaques and diffuse lesions form as demyelinating lesions. Plaques typically are scattered through the white matter of the CNS, although they may extend into adjacent gray matter. Early manifestations are the result of inflammatory edema in and around the plaque and partial demyelination. These manifestations typically disappear within weeks after the initial episode. With progression of the disease, the demyelination and plaque formation result in scarring of glia (*gliosis*) and degeneration of axons. Continued loss of function leads to permanent disability, usually over about 20 years.

There are four classifications of MS: relapsing-remitting, primary progressive, secondary progressive, and progressive-relapsing (Box 46–2). Most individuals with MS present with the relapsing-remitting type.

Various stressors have been suggested as triggers for MS. These stressors include febrile states, pregnancy, extreme physical exertion, and fatigue. These precipitating factors can also cause a relapse of the manifestations during the course of the disease.

Manifestations

The manifestations of MS vary according to the areas destroyed by demyelination and the affected body system (see *Multisystems Effects of MS* on page 1630). Fatigue is one of the most disabling manifestations, and affects almost all clients

BOX 46–2 Classifications of Multiple Sclerosis

Relapsing-remitting: The most common clinical course of MS, characterized by exacerbations (acute attacks) with either full recovery or partial recovery with disability.

Primary progressive: Steady worsening of disease from the onset with occasional minor recovery.

Secondary progressive: Begins as with relapsing-remitting, but the disease steadily becomes worse between exacerbations.

Progressive-relapsing: This rare form continues to progress from the onset but also has exacerbations.

with MS. The manifestations, categorized by the established syndromes of MS, are listed in the box below.

Brief attacks of manifestations are described as short lived or paroxysmal. Short-lived attacks of neurologic deficits indicate the appearance or worsening of manifestations. Conditions that cause short-lived attacks include (1) minor increases in body temperature or serum calcium concentrations (both increase the leakage of current through demyelinated neurons) and (2) functional demands that exceed conduction capacity. Paroxysmal attacks are sensory or motor manifestations that occur abruptly and last for only seconds or minutes; the manifestations are paresthesias, dysarthria and ataxia, and tonic head turning. Paroxysmal attacks, which may occur many times a day, result from the direct transmission of nerve impulses between adjacent demyelinated axons.

INTERDISCIPLINARY CARE

Management of the client with MS varies according to the severity of the manifestations. The focus is on retaining the optimal level of functioning possible, given the degree of disability. Rehabilitation—physical, occupational/vocational, and psychosocial—is a cornerstone of an interdisciplinary approach to treatment. During exacerbations, the focus of interventions shifts to controlling manifestations and quickly returning to remission.

Diagnosis

Diagnosis of MS is challenging because the disease does not present uniformly. A diagnosis requires that the client have one of the following: (1) two or more exacerbations separated by 1 month or more and lasting more than 24 hours, followed by recovery; (2) a history of repeated exacerbations and remissions with or without complete recovery, followed by progressively more severe manifestations lasting for 6 months or more; or (3) slowly increasing manifestations for at least 6 months.

Diagnostic tests vary with the presenting complaints. Magnetic resonance imaging (MRI) with findings of lesions is the most definitive test available; however, it is only one of several laboratory and diagnostic tests that may be performed when establishing the diagnosis. Other tests (described in Chapter 43 ∞) include:

- Cerebrospinal fluid (CSF) analysis reveals an increased number of T lymphocytes that are reactive with antigens, indicating the presence of an immune response in the client (but is not specific to MS). Of MS patients, 80% have elevated levels of immunoglobulin G (IgG) in the CSF.
- CT scan of the brain shows atrophy and white matter lesions. In about 25% of clients with MS, enlarged ventricles are visible on CT.
- Positron emission tomography (PET) scan measures brain activity. In MS clients, the scan reveals areas with changes in glucose metabolism.
- Evoked response testing of visual, auditory, or somatosensory impulses may show delayed conduction.

MEDIALINK Multiple Sclerosis Animation

Medications

Medications slow the progression of MS and decrease the number of attacks. (See the Medication Administration box on page 1631.) Medications are used for a variety of reasons, including to treat manifestations, to modify the course of the disease, or to interrupt the progression of the disease.

The medications used during an exacerbation are aimed at decreasing inflammation to inhibit manifestations and induce remission. Frequently, a combination of adrenal corticosteroid hormone (ACTH) and glucocorticoids is used to decrease inflammation and suppress the immune system. Immunosuppressive agents, including azathioprine (Imuran) and cyclophosphamide (Cytoxan), are also used. Interferon and glatiramer acetate are used to reduce exacerbations in clients with relapsing-remitting MS. Interferon alpha, beta, and gamma (Roferon-A, Intron A, Wellferon, Infergen, Avonex or Rebif, Betaseron, Actimmune) enhance immune function, while glatiramer acetate (Copaxone) stimulates parts of the myelin basic protein to reduce the relapse rate of MS. Both drugs are given by injection and are usually well tolerated.

Other medications treat the manifestations of MS. Anticholinergics are administered for bladder spasticity; cholinergics

MANIFESTATIONS of Multiple Sclerosis

MIXED OR GENERALIZED TYPE (50% OF CASES)

- Visual deficits, with visual blurring, fogginess, or haziness; impaired color perception, decreased central visual acuity, area of diminished vision in the visual fields, acquired color vision deficit (especially to red and green), and an altered pupillary reaction to light.
- Brainstem lesions (cranial nerves III to XII) with nystagmus, dysarthria, deafness, vertigo, vomiting, tinnitus, facial weakness, decreased sensation, diplopia and eye pain; and cognitive dysfunctions involving concentration, short-term memory, word finding, and planning.
- Mood alterations are manifested as depression more often than euphoria.

SPINAL TYPE (25% OF CASES)

- Weakness and/or numbness in one or both extremities (most often the legs).
- Upper motor neuron involvement is manifested by stiffness, slowness, weakness (spastic paresis).
- Bladder dysfunctions include urgency, hesitancy, and incontinence.
- Bowel dysfunction is most often seen as constipation.
- Neurogenic impotence is noted.

CEREBELLAR TYPE (5% OF CASES)

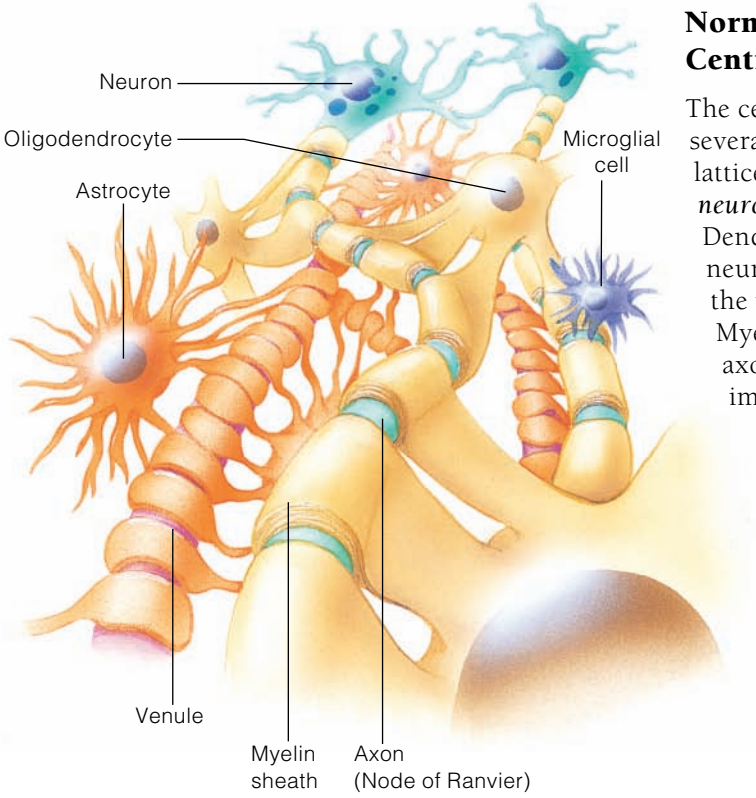
- Manifestations of nystagmus, ataxia, and hyptonia.

AMAUROTIC FORM (5% OF CASES)

- Blindness

PATHOPHYSIOLOGY ILLUSTRATED

Multiple Sclerosis



Normal Anatomy of the Central Nervous System

The central nervous system (CNS) is composed of several cell types arranged in a dense, interconnected lattice. The basic functional cell of the CNS is the *neuron*, which transmits electrochemical impulses. Dendrites, thin projections extending from the neuron body, receive impulses that are passed down the neuronal axon for transmission to other cells. Myelin, a lipid-protein substance, surrounds the axons, insulating them and speeding nerve impulse transmission.

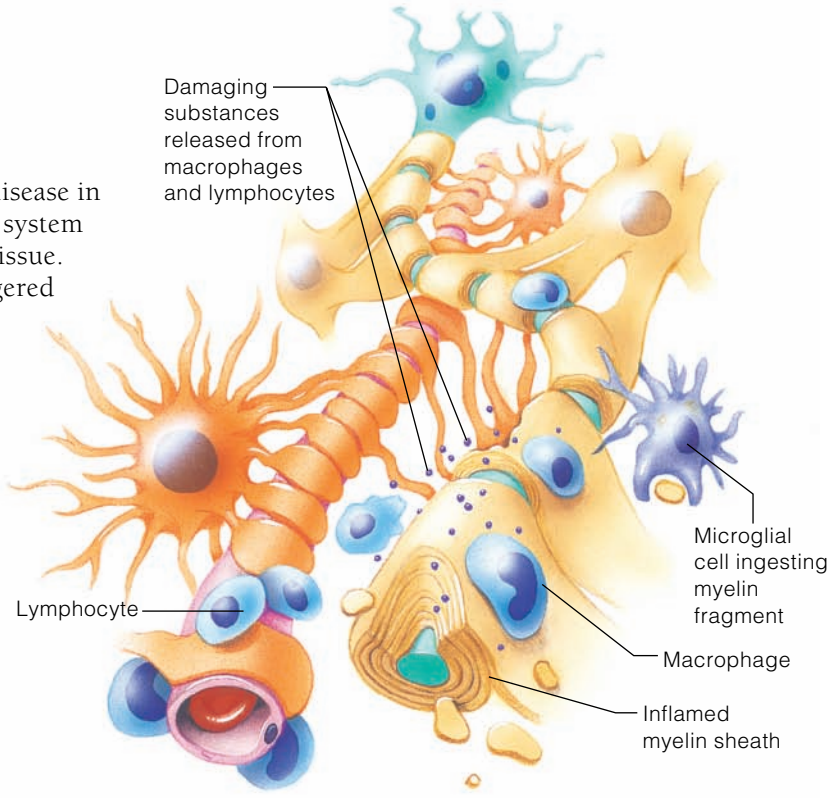
Neurons are surrounded by a network of cells:

- *Astrocytes* support neurons and connnect them to surrounding capillaries and venules.
- *Microglia* are motile phagocytic cells.
- *Oligodendrocytes* wrap concentric layers of myelin around nearby axons.

Acute Attack

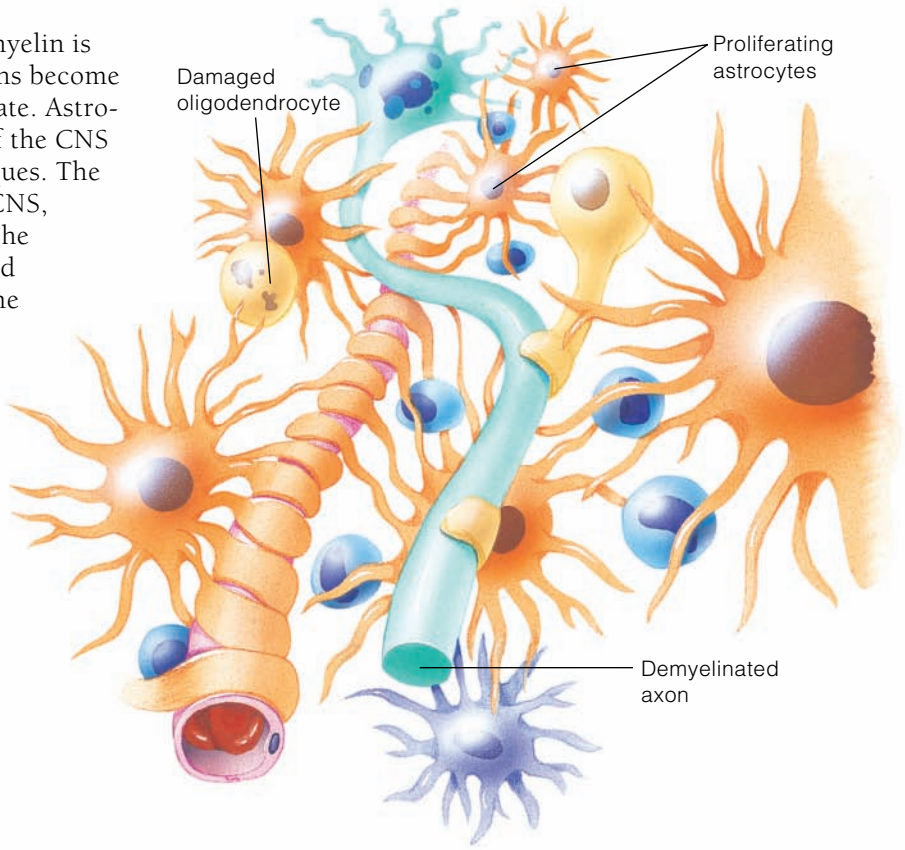
Multiple sclerosis (MS) is a demyelinating disease in which axonal myelin in the central nervous system is eroded, destroyed, and replaced by scar tissue.

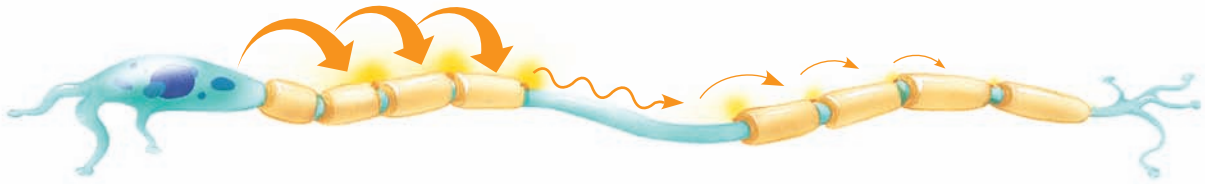
An autoimmune process apparently triggered by genetic and environmental factors is believed to cause inflammation of venules in the CNS. This disrupts the blood–brain barrier, allowing lymphocytes to enter CNS tissue. These lymphocytes proliferate and produce IgG, an antibody that attacks and damages myelin and causes the release of inflammatory chemicals and edema. As the inflammation subsides, the myelin regenerates and manifestations of the disease subside.



Chronic Lesion

After repeated inflammatory attacks, myelin is irreparably damaged. Segments of axons become totally demyelinated and may degenerate. Astrocytes proliferate in damaged regions of the CNS (a process called *gliosis*), forming plaques. The plaques are scattered throughout the CNS, appearing as gray or pinkish lesions. The relapsing-remitting character of MS and the scattered areas of damage within the CNS account for the variable nature of MS manifestations.

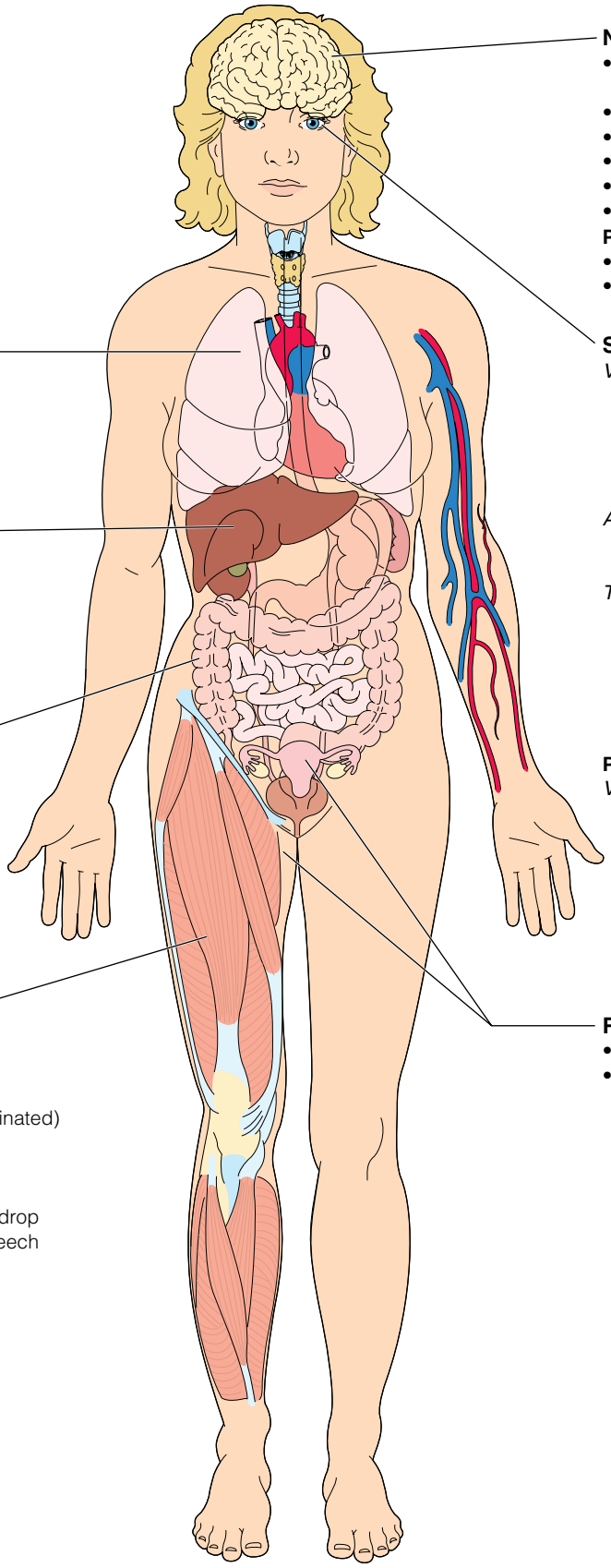




Abnormal Nerve Impulse Transmission

In an undamaged neuron, nerve impulses travel down the axon by "leaping" from one node of Ranvier to the next, thus greatly increasing the speed of impulse transmission. When nerve impulses travel down an axon damaged by MS, they are significantly slowed and weakened as they pass across the surface of demyelinated areas. Impulses may be blocked entirely when axons degenerate. The weakening or interruption of the transmission of nerve impulses and plaque formation within the CNS cause the manifestations of MS, including extremity weakness, paresthesias, visual disturbances, bladder dysfunction, and vertigo.

MULTISYSTEM EFFECTS OF **Multiple Sclerosis**



Neurologic
- Emotional lability (euphoria or depression)
- Forgetfulness
- Apathy
- Scanning speech
- Impaired judgment
- Irritability

Potential Complications
- Convulsive seizures
- Dementia

Sensory

Visual
- Blurred vision
- Diplopia
- Nystagmus
- Visual field defects (blind spots)
- Eye pain

Auditory
- Vertigo
- Nausea

Tactile (especially hands or legs)
- Numbness
- Paresthesias (tingling, burning sensation)
- Diminished sense of temperature
- Pain with spasms
- Loss of proprioception

Potential Complication

Visual
- Blindness

Respiratory
- Diminished cough reflex

Potential Complication
- Respiratory infections

Urinary
- Hesitancy
- Frequency
- Retention
- Reflex bladder emptying

Potential Complications
- Recurring UTIs
- Incontinence

Gastrointestinal

Oral/esophageal
- Difficulty chewing
- Dysphagia

Upper/lower GI
- Decreased or absent sphincter control
- Bowel incontinence
- Constipation

Musculoskeletal
- Fatigue
- Limb weakness
- Ataxic movements (shaky, irregular, uncoordinated)
- Intention tremors
- Spasticity
- Muscular atrophy
- Dragging of foot and foot drop
- Dysarthria with slurred speech

Reproductive
- Impotence (male)
- Loss of genital sensation

MEDICATION ADMINISTRATION The Client with Multiple Sclerosis

IMMUNOMODULATORS

Interferon beta-1a (Avonex)
Interferon beta-1b (Betaseron)
Glatiramer acetate (Copaxone, Copolymer-1)

Interferon beta-la, interferon beta-1b, and glatiramer acetate are administered to clients with relapsing-remitting MS to prolong the time of onset to disability. Their use is based on the assumption that MS is an immunologically mediated disease. Interferon beta-1b produces a decrease in the MS lesions in some clients. Some clients, however, develop a decrease in the absolute neutrophil count and increases in the levels of liver enzymes. Anxiety, confusion, and depression with suicidal tendencies also have been reported. Other adverse reactions include pain, inflammation, hypersensitivity at the injection site, and generalized flulike manifestations. Some women experience menstrual disorders. Pregnant women should not take these medications.

Nursing Responsibilities

- Assess baseline parameters to evaluate drug side effects: psychologic profile, liver function tests, and CBC with differential.
- Monitor CBC and liver function tests every 3 months or as prescribed.
- Assess injection site and report ulceration promptly (pain and redness are common reactions).
- Evaluate client's baseline neurologic, sensory, and motor function. Monitor changes in condition and function.
- Report if client is pregnant or breast-feeding.

Health Education for the Client and Family

- These drugs may cause depression and thoughts of suicide; report these feelings immediately to the physician.
- Administer medication within 3 hours of reconstitution. Rotate injection sites, and avoid any areas that are red or show other skin reactions.
- Seek follow-up care to monitor neurologic changes, CBC, and liver function.
- Avoid prolonged exposure to sunlight.

ADRENALCORTICOSTEROID THERAPY

Adrenocorticotropic hormone (ACTH) (Acthar)
Prednisone (Deltasone, Meticorten, Orasone)
Methylprednisolone (Medrol, Solu-Medrol)

Adrenalcorticosteroids are used both to sustain a remission and to treat exacerbations of MS. ACTH is usually given to induce a remission; It is administered intravenously for 1 week and may be followed by oral prednisone therapy. Another protocol involves administering ACTH intravenously for 3 days followed by intramuscular injections every 12 hours for 1 week (Hickey, 2003). The drugs are given to suppress the immune system, implicated in the etiology of MS. If the drug is used long term the usual steroid precautions are indicated, such as monitoring for glucose intolerance, osteoporosis, and cataract formation. The drugs are used with caution in pregnant and lactating women.

MUSCLE RELAXANTS

Baclofen (Lioresal)
Dantrolene (Dantrium)
Diazepam (Valium)

Muscle relaxants are given to clients with MS to relieve muscle spasms. Baclofen and diazepam act by suppressing CNS reflexes that regulate muscle activity; neither drug affects muscle strength. Baclofen therapy should be discontinued over 1 to 2 weeks; sudden withdrawal may cause seizures and paranoid ideation. In contrast to diazepam and baclofen, dantrolene acts directly on skeletal muscles, and it may affect muscle strength. Dantrolene may cause hepatotoxicity and should not be administered when hepatitis or cirrhosis is present.

Nursing Responsibilities

- Evaluate baseline muscle strength and spasticity, ROM, and dexterity.
- Maintain safety or fall precautions; dizziness and drowsiness are common side effects.
- For the client taking dantrolene, monitor liver function tests (enzymes and bilirubin) for signs of hepatotoxicity.

Health Education for the Client and Family

- These drugs may cause sedative effects. Take appropriate safety measures (e.g., avoid driving).
- Avoid CNS depressants (antihistamines, alcohol); they can increase the sedative effects of the medication.
- Continue follow-up care; if you are taking dantrolene, for example, liver function will need to be monitored.
- If you are taking baclofen, do not suddenly stop the medication.
- Increase fiber and fluids in the diet to prevent constipation.
- Change positions slowly to minimize dizziness and other effects of orthostatic hypotension.

IMMUNOSUPPRESSANTS

Azathioprine (Imuran)
Cyclophosphamide (Cytoxan)

Immunosuppressants are given to clients with MS because of the autoimmune component of the disease. Both medications can cause bone marrow suppression and increase the risk of cancer. Azathioprine may produce hepatitis. Toxic effects of cyclophosphamide include hemorrhagic cystitis, sterility, and stomatitis.

Nursing Responsibilities

- Monitor baseline parameters: CBC with platelet count and differential, urinalysis, liver function tests, hepatitis profile.
- Assess for anemia: fatigue, lethargy, pallor.
- Watch for bleeding.
- Protect against and observe for subtle signs of infection.

Health Education for the Client and Family

- Report infection, bleeding, and anemia immediately.
- Drink at least 2 L (2 quarts) of fluid a day, and observe urine for blood.
- Report jaundice immediately.
- Check oral cavity daily for changes or ulcers.
- Avoid becoming pregnant while taking these drugs.
- Obtain follow-up care, including frequent blood tests.

are given if the client has a problem with urinary retention related to flaccid bladder. Depression is treated with antidepressant drugs.

Surgery

Surgery may be indicated for clients who experience severe spasticity and deformity. However, physical therapy can prevent most severe problems. Foot drop from severe plantar flexion can be relieved with an Achilles tenotomy, a surgical procedure in which the Achilles tendon is transected.

Nutrition and Fluids

Several diets involving manipulation of fats are currently under investigation. Clients with MS may be overweight because of their inability to ambulate; depression may contribute to the problem because people who are depressed tend to eat more. Ideally, the client should maintain a weight as close as possible to that recommended for the client's height and body type.

As MS progresses, the client's ability to prepare food and eat is compromised. Changes in muscle tone, tremor, weakness, and ataxia all contribute to nutritional problems. Dysphagia also is a common problem. The diet must be adapted to accommodate changes in the client's ability to chew and swallow.

Rehabilitation

Physical and rehabilitative therapies are tailored to the client's level of functioning. The long-term goal is to enable the client to retain as much independence as possible. One major intervention is to maintain and increase existing muscle strength.

Spasticity is managed with stretching exercises, gait training, and braces, splints, or other assistive devices. To maintain balance, the client is encouraged to widen the base of support by standing with the feet slightly further apart. Walkers and canes may be weighted to provide support and balance for the ataxic client.

An interdisciplinary approach to rehabilitation will provide supportive services: speech therapy for problems with phonation, occupational therapy to maintain strength in the upper extremities and carry out ADLs, and occupational counseling. Referrals to a urologist are indicated for problems with urinary incontinence, urinary tract infections, retention, and impotence. Consultation with a respiratory therapist may be needed if the client develops chronic respiratory infections from inability to cough, move secretions, or breathe deeply, especially with increased debilitation.

NURSING CARE

Because the disease most often affects young adults in the prime of life, the psychosocial and economic effect can be devastating. People with MS have to make adjustments to the body image changes while simultaneously adapting to the altered relationships and decreased earnings usually encountered with the disease. A once-healthy spouse becomes wheelchair bound; a person once independent may eventually become dependent for even the most basic ADLs. The unpredictable course of MS is a challenge for long-term planning. A Nursing Care Plan for the client with MS is given on the next page.

Health Promotion

Following an overview of the disorder, the client needs to understand how to prevent fatigue and exacerbations. Teach the client to avoid stress, extremes of cold and heat, high humidity, physical overexertion, and infections. Because pregnancy can exacerbate manifestations, counseling about this risk is indicated. Also, address preventive measures to avoid risk of respiratory and urinary tract infections.

Assessment

Collect the following data through the health history and physical examination (see Chapter 43 ∞):

- *Health history:* History of childhood viral illnesses, geographical residence when a child, exposure to physical or emotional stressors (pregnancy/delivery, extremes of heat), medications, symptom onset, severity of manifestations.
- *Physical assessment:* Affect, mood, speech, eye movements, gait, tremors, vision and hearing, reflexes, muscle strength and movement, sensation.

Nursing Diagnoses and Interventions

Interventions for the client with MS vary with the acuity of exacerbations and the presenting problems. Many nursing diagnoses relate to the inability to perform ADLs, for example, *Self-Care Deficit* and *Impaired Home Maintenance.* Others reflect problems with musculoskeletal changes or altered nerve conduction, for example, *Impaired Physical Mobility*, *Ineffective Breathing Pattern*, *Constipation*, and *Functional Urinary Incontinence*. The nursing diagnoses discussed in this section are *Fatigue* and *Self-Care Deficit*.

Fatigue

Fatigue is defined by NANDA as an overwhelming sustained sense of exhaustion and decreased capacity for physical and mental work at the usual level. Fatigue affects every aspect of the MS client's life: the ability to remain independent and perform self-care, sexual function, mobility, airway clearance, and ultimately self-concept and coping. A great deal of teaching is needed to help the client and family understand fatigue and how to adapt. Clients and families need assistance managing fatigue in a society in which energy is highly valued.

- Assess degree of fatigue and identify contributing factors. *Fatigue is a subjective experience that needs to be evaluated thoroughly before planning can begin.*
- Arrange daily activities to include rest periods. *Rest is essential to manage feelings of fatigue; periods of relaxation may help replenish energy reserves.*

PRACTICE ALERT

It is important to remember that the fatigue from chronic illnesses such as MS is very different from being "tired," and that rest and sleep may not result in improvement.

- Ask the client to consider which activities are really necessary and to set priorities. *Prioritizing activities promotes independence and self-control.*

NURSING CARE PLAN A Client with MS

George McMurphy, a 45-year-old from northern Minnesota, was diagnosed with MS approximately 5 years ago. He states that he probably had mild symptoms as long ago as 10 years. He works as a manager for a large grocery store chain near his home. He lives at home with his wife and two children, ages 12 and 15. Recently, Mr. McMurphy has had increasing problems with urinary incontinence, lack of energy, weakness, extreme fatigue, and altered mobility from spasticity in his leg muscles. He also has a fever, chest congestion, and a cough productive of green sputum. He is admitted to the hospital for evaluation and treatment of pneumonia and exacerbation of his MS.

ASSESSMENT

Denise Miller, RN, primary care nurse, is assigned to care for Mr. McMurphy. His major complaint is the inability to "bring up all this sputum; I feel rotten from being so congested. I hate not being able to get to work and for my wife having to tend to my personal needs." Vital signs are as follows: BP 134/84, P 94, R 30, T 102°F (38.8°C). Mr. McMurphy is admitted for an acute exacerbation of the disorder, probably triggered by pneumonia. He will be treated with ACTH and intravenous antibiotics during this admission.

DIAGNOSES

- *Ineffective Airway Clearance* related to lung infection and thick mucus
- *Activity Intolerance* related to fatigue and spasticity
- *Self-Care Deficit: Toileting, Feeding, and Grooming* related to muscle weakness

EXPECTED OUTCOMES

- Be able to clear airway.
- Have breath sounds clear to auscultation and pulse oximetry readings above 95%.
- Be able to ambulate using assistive devices, if needed.
- Perform self-care activities without becoming overly fatigued and tired.
- Verbalize methods to adapt daily routine to his level of tolerance.

PLANNING AND IMPLEMENTATION

- Initiate pulmonary hygiene measures (e.g., incentive spirometry, turning, deep breathing and coughing, breathing exercises, and postural drainage) at least every 2 hours. Assess lung sounds, oxygen saturation, and ability to clear airway.
- Teach the importance of maintaining an oral fluid intake of at least 2000 mL per day to prevent tenacious sputum and urinary tract infections. Teach signs and symptoms of urinary and respiratory infections.
- Encourage participation in decision making about care.
- Assist with ADLs only as needed, based on level of fatigue and muscle weakness.
- Plan self-care activities so that they are performed during periods of peak level of energy; intersperse rest periods throughout the day.
- Refer to an MS support group.
- Refer to physical and occupational therapists for counseling regarding control of spasticity and possible splinting of spastic muscles.
- Consult a urologist for assessment of bladder incontinence; teach intermittent catheterization. Alternatively, the use of an external condom catheter may be indicated.

EVALUATION

Mr. McMurphy is discharged 3 days following admission. He states that he feels stronger; on discharge, he has no problem clearing his airway. Although he continues to pace his activities to avoid fatigue, his muscle strength and "tiredness" have improved. He is able to complete ADLs unassisted.

Pulmonary function has returned to normal prehospitalization levels: ABGs and pulse oximetry are within normal limits. Both Mr. McMurphy and his wife have listed several ways to modify their daily routine to allow more rest and decreased stress. Follow-up visits to his primary care physician have been arranged, and they have been provided with information about the local MS support group.

CRITICAL THINKING IN THE NURSING PROCESS

1. Describe approaches the nurse could take to ensure that Mr. McMurphy does not exceed his activity tolerance.
2. Develop a teaching plan for Mr. McMurphy to help prevent future respiratory infections.
3. Develop a care plan for Mr. McMurphy for the nursing diagnosis *Risk for Injury* related to fatigue, muscle weakness, and spasticity.

See Evaluating Your Response in Appendix C.

- Suggest performing tasks in the morning hours. *Biorhythm studies indicate that people usually have greater energy reserves in the morning hours and diminished reserves in the afternoon.*
- Advise to avoid temperature extremes, such as hot showers or exposure to cold. *Maintaining a relatively constant body temperature may avoid exacerbation of the disorder. Heat can delay impulse transmission across demyelinated nerves, which contributes to fatigue.*
- Refer to the appropriate professionals to manage fatigue: stress management groups, support groups, occupational or physical therapist, as indicated. *Support groups and therapy can facilitate self-management and improve coping.*

Self-Care Deficit

Clients with MS may need assistance with bathing, toileting, dressing, grooming, and feeding. The help needed can range from minimal guidance to total dependence. The client's ability to perform self-care activities is the gauge by which family members and caregivers need to adjust assistance. Self-care encompasses both the decisions about care and the provision of care; most clients are capable of making decisions even after physical limitations prevent physical self-care. The need to maintain self-determination cannot be overemphasized and must be incorporated into each intervention. As the client with MS ages, there may be even more need for teaching to provide self-care, as described in the Nursing Research box on the next page.

NURSING RESEARCH Evidence-Based Practice for Aging Clients with MS

MS is a chronic debilitating disease, but it does not reduce life expectancy. Most clients are diagnosed with the disease as young to middle adults, and they must learn how to live with the disease for the rest of their lives. However, little research has been published regarding the experiences and health-related concerns of people aging with MS. This study by Finlayson and colleagues (2004) was conducted to initiate dialogue about the role of nurses in addressing those concerns. The study subjects perceived two major differences between their experience of aging with MS and that of people aging without MS: having less freedom (not being able to travel, restriction of social activities, physical accessibility of buildings, and financial limitations) and needing more assistance (with housework, shopping, hygiene, cooking, getting to places).

IMPLICATIONS FOR NURSING

The findings of this study support the need for nurses to include consideration of issues of daily living for clients with chronic disabilities, including MS. Nurses have the knowledge and ability to include information in client teaching that facilitate quality of life, coping with disability, and symptom management. By working closely with members of the interdisciplinary health team, nurses select and implement appropriate interventions to enable clients with MS to age in place, and to continue to be active and involved members of the family and community as they age.

CRITICAL THINKING IN CLIENT CARE

1. How would your teaching differ for the following clients with MS?
   a. A 45-year-old woman, married, with family who lives nearby
   b. A 70-year-old woman, widowed, no family living
   c. A man who has been a farmer all his life and lives in an isolated rural area
   d. A man who has always lived in an urban area with access to public transportation.
2. You are making a home visit to an older woman who has had MS for 30 years. She tells you, "I never leave my condo anymore because I just can't control my urine and I'm so afraid I will have an accident." What can you do to help her resolve this problem?
3. Design a plan of care focused on the nursing diagnosis *Fatigue* for a 73-year-old man who is living independently but says, "Some days I am just too tired to even cook anything to eat."

- Assess the extent of the client's self-care deficit; refer to other health team members for assessment as appropriate. For example, refer to a speech pathologist to assess swallowing and gag reflex, if indicated. *An accurate assessment is crucial to individualizing interventions.*
- Suggest adaptive devices, such as arm or wrist braces, as needed. *Meeting hygiene needs and feeding self are essential for positive self-concept, self-esteem, and socialization.*
- Teach to use assistive devices, such as plate guards; to modify consistency of foods; and to eat when energy level is better. If unable to buy and prepare meals, provide referral to Meals-on-Wheels. *Proper nutrition is basic to health; adapting utensils and foods can facilitate meeting nutritional needs.*
- Teach interventions related to altered bowel and bladder function: fluid intake of at least 2000 mL daily, bowel routine as indicated to prevent constipation, self-catheterization skills as necessary. *Maintaining optimal bowel and bladder function decreases the risk of urinary tract infection and bowel impaction.*

Using NANDA, NIC, and NOC

Chart 46–2 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with MS.

NANDA, NIC, AND NOC LINKAGES
CHART 46–2 **The Client with Multiple Sclerosis**

NANDA

Disturbed Sensory Perception: Visual

↓

NIC

Communication Enhancement: Visual Deficit

Emotional Support

Environmental Management

↓

NOC

Sensory Function: Vision

Neurological Status: Cranial Sensory/Motor Function

Risk Control: Visual Impairment

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2003), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

Community-Based Care

The inconsistent and erratic nature of MS can make teaching for self-care difficult. Initial teaching focuses on a realistic explanation of MS. Referral to a support group early in the course of the disease is indicated. Social support can make a positive difference in a client's ability to cope with MS. Address the following topics in preparing the client for home care:

- Various treatment options and their side effects
- Information about medications, particularly steroid use, and about possible interactions with prescription or over-the-counter (OTC) medications

- Ongoing care from nurses, counselors, and physical, occupational, and speech therapists, as well as the physician and community health nurse
- Helpful resources:
  - National MS Society
  - National Institute of Neurological Disorders and Stroke.

THE CLIENT WITH PARKINSON'S DISEASE

Parkinson's disease (PD) is a progressive, degenerative neurologic disease characterized by tremor (shaking), muscle rigidity, and bradykinesia (slowness of movement) (Porth, 2005). People with PD are faced with multiple problems involving independence in ADLs, emotional well-being, financial security, and relationships with caregivers.

Incidence and Prevalence

Parkinson's disease is one of the most common neurologic disorders affecting older adults. The National Parkinson Foundation (2005a) estimates that as many as 60,000 new cases are diagnosed each year, joining the 1.5 million people in the United States who currently have PD. The disorder usually develops after the age of 65 years, but 15% of those diagnosed are under 40 years of age. Men and women are affected equally. Recent discovery of inherited forms of PD suggest a genetic role in the development of this disease.

Parkinson's-like manifestations, called *secondary parkinsonism,* may result from other disorders such as trauma, encephalitis, tumors, toxins, and drugs. Drug-induced parkinsonism, which is usually reversible, may occur in people taking neuroleptics, antiemetics, antihypertensives, and illegal designer drugs containing MPTP, a toxic chemical (Porth, 2005). Carbon monoxide or cyanide poisoning can also cause secondary parkinsonism. This discussion focuses on primary Parkinson's disease, the cause of which is unknown.

Pathophysiology

Coordinated, voluntary body movement is achieved through the actions of neurotransmitters in the basal ganglia of the brain. Some neurotransmitters facilitate the transmission of excitatory nerve impulses, while other neurotransmitters inhibit their transmission. Together, this system allows control of movement. A disturbed balance between excitatory and inhibitory neurotransmitters causes disorders of voluntary motor function, such as PD.

In PD, neurons in the cerebral cortex atrophy and are lost, the dopaminergic nigrostriatal (pigmented) pathway degenerates, and the number of specific dopamine receptors in the basal ganglia decreases. These pathologic processes cause a decrease in the production of dopamine (a neurotransmitter that helps regulate nerve impulses involved in motor function) from the substantia nigra. The usual balance of dopamine (an inhibitory neurotransmitter) and acetylcholine (an excitatory neurotransmitter) in the brain is disrupted, and dopamine no longer inhibits acetylcholine. The failure to inhibit acetylcholine is the underlying basis for the manifestations of the disorder. Parkinson's disease has five stages, outlined in Box 46–3.

BOX 46–3 Stages of Parkinson's Disease

I	Unilateral involvement only, usually with minimal or no functional impairment.
II	Bilateral or midline involvement, without impairment of balance.
III	First sign of impaired righting reflexes, evidenced as unsteadiness as the client turns or demonstrated when the client is pushed from standing equilibrium with the feet together and eyes closed. Functionally, the client is somewhat restricted in activities but may have some employment potential, depending on the type of employment. Clients are physically capable of leading independent lives, and their disability is mild to moderate.
IV	Fully developed, severely disabling disease; the client is still able to walk and stand unassisted but is markedly incapacitated.
V	Client is confined to bed or wheelchair unless aided.

Manifestations

Parkinson's disease begins with subtle manifestations. Clients complain of feeling tired and seem to move more slowly; a slight tremor may accompany the fatigue. Over time, the manifestations progressively increase in severity. The manifestations and complications of PD are presented in the box on the next page.

Tremor

Tremor at rest is usually the first manifestation experienced, with one of the upper extremities more often affected. Resting tremors of the hand show a "pill-rolling" motion of the thumb and fingers. (This name reflects the way in which medicinal pills were formed in the early days of medicine.) The tremor may be controlled with purposeful, voluntary movement, and is worsened by stress and anxiety. Clients have progressive impairment in performing skills that require dexterity and fine muscle control, such as writing and eating.

Rigidity and Bradykinesia

Manifestations related to motor and postural effects include rigidity, bradykinesia, and uncoordinated movements. Rigidity (resulting from involuntary contraction of all skeletal muscles) makes both active and passive movement difficult. It is manifested as increased resistance to passive range of motion (ROM). Although the extremity moves, it does so in a jerky motion, called *cogwheel rigidity.* The first manifestation of rigidity may be muscle cramps in the toes or hands, but most often the client describes stiffness, heaviness, or aching in muscles.

Bradykinesia, experienced as difficulty in starting, continuing, or coordinating movements, is the most common and crippling manifestation. All striated muscles are affected, including those that involve chewing, swallowing, and speaking. Slowed or delayed movements affect the eyes, mouth, and voice, causing a masklike face and softened or muffled voice. Disorders of swallowing result in problems with eating and with drooling. Clients have a staring gaze with minimal

MEDIALINK Case Study: Parkinson's Disease

MEDIALINK Akinesia/Bradykinesia Animations

MANIFESTATIONS AND COMPLICATIONS of Parkinson's Disease

RELATED TO MOTOR DYSFUNCTION

- Nonintention tremor
- Bradykinesia or akinesia
  a. Slowed movements; inability to initiate voluntary movements
  b. Slowed speech, low amplitude
  c. Poor articulation
  d. Decreased eye movements (i.e., blinking)
  e. Masklike, expressionless face
- Rigidity
- Posture and gait disturbances
  a. Trunk tilted forward
  b. Shuffling gait, propulsive at times
  c. Retropulsion
- Complications: falls, fractures, impaired communication, social isolation

RELATED TO AUTONOMIC SYSTEM DYSFUNCTION

- Skin problems
  a. Seborrhea
  b. Excess sweating on face and neck, absence of sweating of trunk and extremities
  c. Mottled skin
- Heat intolerance
- Postural hypotension
- Constipation
- Complications: skin breakdown, dizziness, falls, constipation

RELATED TO COGNITIVE AND PSYCHOLOGIC DYSFUNCTION

- Dementia
  a. Memory loss
  b. Lack of insight and problem-solving ability
  c. Declining intellectual abilities
- Anxiety
- Depression
- Complications: loss of ability to function, social isolation

change in expression (Figure 46–3 ■). Clients describe being "frozen" in place as voluntary movement is lost, and they sit or lie in one position without movement for long periods of time. Movement is interspersed with freezing, brought about by turning, increasing the effort to move, or making visual or touch contacts.

Abnormal Posture

The loss of normal postural reflexes results in postural abnormalities, including disorders of postural fixation, equilibrium, and righting. Involuntary flexion of the head and shoulders means the person with PD cannot maintain an upright position of the trunk when sitting or standing. This problem of postural fixation results in the characteristic stooped, leaning forward position. Disorders of equilibrium follow loss of postural fixation with an inability to make adjustments when leaning or falling, increasing the risk of injury from falls (the person usually falls backward). The client takes short, accelerated steps to try to maintain an upright position when walking.

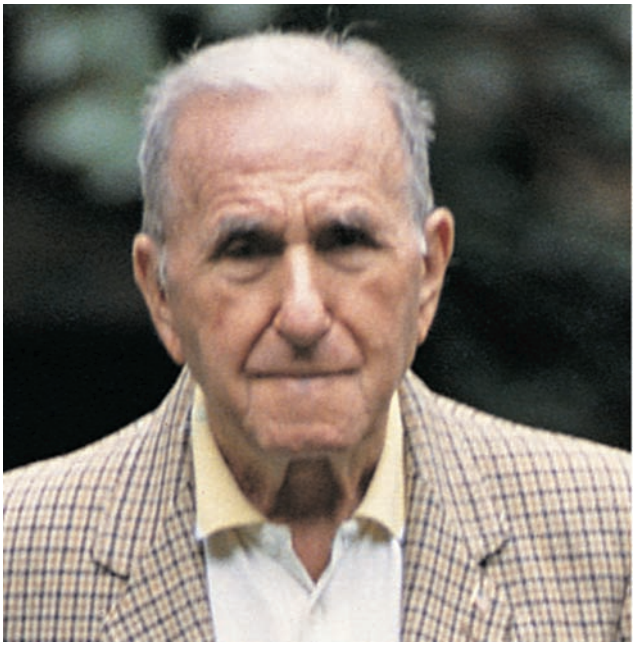


Figure 46–3 ■ In Parkinson's disease, the client's face lacks expression or animation.

Source: Yoav Levy/Phototake NYC.

Autonomic and Neuroendocrine Effects

Many manifestations result from the loss of functions controlled by the autonomic nervous system. Elimination problems include constipation and urinary hesitation or frequency. Clients may experience problems related to orthostatic hypotension, including dizziness with position change. Eczematous skin changes and seborrhea are related to the increase in sweat gland activity secondary to increased sebotrophic hormone production.

Mood and Cognition

Both depression and dementia are pathologies associated with PD. Depression occurs in half of all clients and a third have dementia. Dementia, resulting from loss of cholinergic cells, loss of neurons, senile plaques, neurofibrillary tangles, and amyloid changes in small blood vessels, occurs in 20% of clients with PD and develops later in the disease (Porth, 2005). The client has manifestations similar to the person with Alzheimer's disease, including confusion, disorientation, memory loss, distractibility, and changes in abstraction and judgment. *Bradyphrenia* may also occur, resulting in slow thinking and a decreased ability to form thoughts, to plan, or to make decisions.

Sleep Disturbances

Clients with PD commonly have sleep disturbances, although they may experience decreased manifestations during sleep in the early stages. The ability to fall and stay asleep is affected by acetylcholine. Muscle rigidity may compromise sleep because of the inability to change position. This lack of muscle movement causes the client to awaken and consciously shift position.

Interrelated Effects

Some of the manifestations that clients with PD experience have multiple contributing factors. For example, constipation is common because of decreased peristalsis. However, decreased peri-

stalsis is not the only cause: immobility, tremors (resulting in being unable to drink from a glass easily), and dietary changes from dysphagia all contribute to the problem of constipation.

Complications

The following complications are associated with Parkinson's disease:

- Oculogyric crisis, in which the eyes become fixed with a lateral and upward gaze
- Paranoia and hallucinations, which may accompany dementia
- Impaired communication due to changes in speech, handwriting, and expressiveness
- Falls from balance, posture, and motor changes
- Infections, such as pneumonia, related to immobility
- Malnutrition related to dysphagia and inability to prepare meals
- Altered sleep patterns due to loss of dopamine, L-dopa side effects (nightmares, dreams), or side effects of anticholinergics (hyperreflexia, muscle twitching), and depression
- Skin breakdown and pressure ulcers associated with urinary incontinence, malnutrition, and sweat reflex changes
- Depression and social isolation.

INTERDISCIPLINARY CARE

Prognosis is poor, owing to the progressive degeneration that ultimately affects multiple physiologic systems and their function. Psychosocial effects are equally devastating, and the family needs more support as the client's debilitation increases. Total disability is usually seen 10 to 20 years after diagnosis. The leading cause of death is pneumonia.

Diagnosis is based primarily on a thorough history and physical examination, and is made based on having two of the following manifestations: tremor at rest, bradykinesia, rigidity, and postural instability. Interventions vary with the clinical stage of the disorder and include medication, surgery, and rehabilitation to retain the optimal level of functioning possible. An interdisciplinary approach is essential for these clients.

Diagnosis

Diagnostic studies may support a potential diagnosis of Parkinson's disease; no test clearly differentiates Parkinson's disease from other neurologic disorders (Hickey, 2003). However, PET scan will show decreased uptake of 6-[18F]-fluoro-dopa.

Medications

The goal of drug therapy is to control manifestations to the extent possible. Generally, medications vary with the stage of the disease; however, response is individualized and guides the selection of medications. Types of drugs used include monoamine oxidase (MAO) inhibitors, dopaminergics, dopamine agonists, and anticholinergics. Information about these drugs is presented in the Medication Administration box below.

MEDICATION ADMINISTRATION The Client with Parkinson's Disease

DOPAMINERGICS

Levodopa (Larodopa, Dopar)
Carbidopa-levodopa (Sinemet)
Amantadine (Symmetrel)

These drugs have their major effect on the akinesia of Parkinson's disease, improving mobility while decreasing muscle rigidity and tremor. Levodopa is a metabolic precursor of dopamine, but unlike dopamine, it can cross the blood–brain barrier. Levodopa is converted to dopamine in the brain by decarboxylase, a catalytic enzyme, and stimulates dopamine receptors to balance the dopamine/acetylcholine concentrations. Carbidopa prevents decarboxylase from converting levodopa to dopamine in the peripheral tissues; therefore, carbidopa is frequently given in combination with levodopa. Amantadine is used to treat dyskinesia and also elevates mood.

Levodopa is avoided in clients with narrow-angle glaucoma, severe angina pectoris, transient ischemic attacks, or melanoma. The "on–off" phenomenon occurs after the client takes levodopa for several years; this phenomenon is characterized by unexpected dyskinesias and lack of symptom control.

Common side effects are nausea and vomiting; darkening of urine and sweat; dyskinesias, especially in the first few months of therapy; dysrhythmias; orthostatic hypotension; and psychologic reactions, such as hallucinations and vivid dreams. Older adults are particularly susceptible to psychologic disturbances.

Nursing Responsibilities

- Establish the client's baseline functional abilities in performing ADLs and administering the medication; assess motor control and coordination.
- To avoid adverse reactions, assess the client's overall health status before initiating therapy.
- Monitor medications known to cause adverse drug interactions: anticholinergics, pyridoxine, and antipsychotic agents alter the effectiveness of levodopa; MAO-B inhibitors can cause severe hypertension because of their vasoconstrictive effects.
- Withhold levodopa for 8 hours prior to administering Sinemet to avoid potentiating the effects of the circulating levodopa.

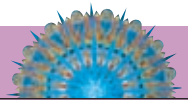
Health Education for the Client and Family

- Levodopa may not take effect for several weeks to months.
- Do not alter dosages of medications; taking more of a medication may not result in better symptom control and can cause severe side effects.
- Your protein intake should be divided into equal amounts for the day's meals. Avoid foods high in pyridoxine, such as pork, beef, ham, avocado, beans, and oatmeal.
- Levodopa may cause a darker color of urine; this is harmless, however.
- To prevent side effects:
  - Prevent nausea by taking medication with food.
  - Change position slowly to avoid a drop in blood pressure and risk of falling.
  - Prevent constipation by increasing fluid intake and exercising regularly.
- Notify practitioner if you begin to have difficulty making voluntary movements or cardiac or psychologic symptoms develop.

MEDIALINK Levodopa Animation

(continued)

MEDICATION ADMINISTRATION **The Client with Parkinson's Disease (continued)**



- Watch for the "on–off" phenomenon, in which periods of symptom control alternate with periods when the drug fails to control symptoms.

MONOAMINE OXIDASE INHIBITORS

Selegiline (Eldepryl, Carbex)

Selegiline works by selectively inhibiting the enzyme that inactivates dopamine in the brain. It may be administered alone or as an adjunct therapy with levodopa: Selegiline inhibits the enzyme system that would otherwise break down and destroy dopamine. This synergistic effect lasts approximately 1 to 2 years. The combination of selegiline and levodopa increases the adverse reactions of dopamine; nurses must be alert for orthostatic hypotension, changes in movement, hallucinations, and confusion. These responses can be modified by lowering the dose of levodopa. Because it is highly selective for the MAO-A enzyme, selegiline does not have antidepressant effects like the MAO-B inhibitors. The risk of severe hypertension is low.

Nursing Responsibilities

- Establish baseline functional abilities: motor control and movements, position changes, mental status.
- Monitor problems with insomnia.
- Assess for orthostatic hypotension; look for unsteadiness with position change and complaints of dizziness.
- Assess for hypertension, which can occur with higher than usual doses.

Health Education for the Client and Family

- It is very important to take the medication as directed, especially dose and time of administration.
- Notify the practitioner if insomnia occurs.
- Report signs of dizziness when changing positions or standing, changes in ability to move, or psychologic changes.
- Change positions slowly, especially when moving from a sitting to standing position.
- Keep follow-up appointments for evaluation of the medication's effectiveness.

DOPAMINE AGONISTS

Bromocriptine (Parlodel)
Pergolide (Permax)
Pramipexole (Mirapex)
Ropinirole (Requip)

Dopamine agonists act by directly activating dopamine receptors in the brain. They are frequently used in combination with levodopa therapy: When dopamine agonists are given with levodopa, they increase the therapeutic effects of levodopa and reduce fluctuations in motor symptoms. Adverse reactions are similar to those of levodopa: nausea, orthostatic hypotension, and psychologic disturbances are common. Nursing responsibilities and client and family teaching information are similar to those that apply to the dopaminergics.

COMT INHIBITORS

Tolcapone (Tasmar)
Entacapone (Comtan)

COMT inhibitors inhibit catechol-*O*-methyltransferase (COMT), which is responsible for metabolizing dopamine. The concurrent administration of a COMT inhibitor with levodopa increases the amount of levodopa available to the brain to control Parkinson's disease.

Nursing Responsibilities

- Monitor liver function test results and manifestations of liver impairment (dark urine, jaundice).
- Administer with food.
- If given concurrently with warfarin, monitor PT and INR.

Health Education for the Client and Family

- Avoid using alcohol and sedatives.
- Rise slowly from a sitting or lying position to avoid falling.
- Nausea is common at the beginning of therapy.
- Do not abruptly stop taking the medication.
- Report increased loss of muscle control, yellow skin or eyes, dark urine, hallucinations, severe diarrhea.

ANTICHOLINERGICS

Trihexyphenidyl (Artane)
Benztropine (Cogentin)
Biperiden (Akineton)
Cycrimine (Pagitane)
Procyclidine (Kemadrin)
Chlorphenoxamine (Phenoxene)

Anticholinergics are effective in Parkinson's disease because they block the excitatory action of the neurotransmitter acetylcholine. They are frequently used during the early stages of the disease or when the client can no longer take levodopa. They may be given in combination with carbidopa-levodopa therapy. These medications ease drooling, tremors, and rigidity; however, side effects are common and may include blurred vision, dry mouth, constipation, delayed gastric emptying, urinary retention, photophobia, and tachycardia. Older adults are especially susceptible to heat stroke and psychologic side effects, including confusion, depression, delusions, and hallucinations. Anticholinergics should be tapered slowly when discontinued to avoid enhancing parkinsonian symptoms.

Nursing Responsibilities

- Perform baseline assessment for presence of glaucoma, cardiac dysfunction, and prostatic hypertrophy.
- Note other medications, including OTC medications that have anticholinergic effects, such as antihistamines and tricyclic antidepressants.
- Monitor for side effects, especially changes in vision, elimination, gastric emptying, and mentation.

HEALTH EDUCATION FOR THE CLIENT AND FAMILY

- Inform your practitioner if you begin taking any new medications or notice any new symptoms.
- Avoid overexposure to heat, and take precautions to avoid heat stroke: Drink fluids, keep cool, and avoid strenuous activity on hot days.
- Drink adequate amounts of fluid to minimize constipation.
- Practice home safety to prevent falls associated with blurred vision.
- Avoid taking OTC antihistamines or sleeping aids; these have anticholinergic activity.
- Have the eyes examined annually to check for glaucoma; wear dark glasses if photophobia develops.
- Do not suddenly stop taking anticholinergics.

Initially clients are treated with selegiline (Carbex, Eldepryl), amantadine (Symmetrel), or anticholinergics. As the disease progresses, levodopa (Dopar, Larodopa) in combination with carbidopa (Lodosyn) is used in a medication named carbidopa-levodopa (Sinemet). Because levodopa eventually loses its effectiveness, dopamine agonists are added to increase the effectiveness of levodopa. Eventually, pharmacotherapeutic agents lose their efficacy, and the disease continues to progress despite treatment. Response to the drugs fluctuates; this phenomenon is called the "on–off" response.

Bromocriptine (Parlodel) and pergolide (Permax), agents that inhibit the breakdown of dopamine, are used to delay progression of the disease. Catecholamine-*O*-methyl transferase (COMT) inhibitors (tolcapone [Tasmar] and entacapone [Comtan]) are used in conjunction with carbidopa-levodopa therapy to reduce the metabolism of levodopa, leading to more sustained dopaminergic stimulation of the brain. Selegiline (Deprenyl) increases dopaminergic activity, and is used as an adjunctive therapy for clients who have fluctuations in response, or become unresponsive, to levodopa.

Other medications may be used to treat problems related to Parkinson's disease. Antidepressants may be prescribed. Propranolol (Inderal) may be used to treat tremors; it should be used cautiously when clients have orthostatic hypotension. Botulism toxin injections may be given to treat eyelid spasms and abnormal posturing (dystonia) involving the extremities.

Deep Brain Stimulation

Activa TM tremor control therapy uses an implanted pacemaker-like device to deliver mild electrical stimulation to block the brain impulses that cause tremor, rigidity, stiffness, slowed movement, and problems with walking. In this procedure, an insulated wire is surgically placed in the thalamus and connected to an implanted pulse generator (similar to an advanced cardiac pacemaker) near the clavicle. It is used only for clients who cannot adequately control manifestations with medications (NINDS, 2005c).

Surgery

Pallidotomy is a surgical technique for Parkinson's disease, and its results have been helpful for many clients. In this procedure, the neurosurgeon locates the affected areas of the globus pallidus and destroys the involved tissue. As a result, clients who could not previously ambulate are able to walk, and tremors cease. The long-term effects are still being evaluated.

Stereotaxic thalamotomy (an x-ray is taken during neurosurgery to guide the insertion of a needle into a specific area of the brain) has been used only for clients who do not respond to medications—generally, younger people with extreme unilateral tremor. The surgeon destroys a small amount of tissue by creating a lesion in the ventrolateral nucleus of the thalamus. This surgery decreases tremors and rigidity in the contralateral extremity.

Fetal tissue transplantation is a controversial surgical procedure limited to a few medical centers. In this procedure, brain cells from aborted fetuses are implanted into the brain in the hopes that the new cells will grow and produce enough dopamine to restore some lost mobility.

Rehabilitation

Depending on their individual needs, clients frequently benefit from rehabilitation therapy with a physical therapist, social worker, psychologist, and/or speech therapist.

Physical therapists (PT) can implement an individual exercise program to improve coordination, balance, gait, and transfers. Preventing contractures is an important goal of exercise therapy. It is crucial that family and healthcare personnel permit the client adequate time to perform not only exercise regimens but also ADLs. Activities should not be rushed.

An occupational therapist (OT) helps the client adapt to changing abilities pertinent to work, self-care, and recreational activities. Some rehabilitation centers assign OT personnel the responsibility of addressing the client's upper extremity functions while assigning PT personnel to manage lower extremity problems. For example, skills related to cooking and grooming would be supervised by the OT, whereas mobility and posture skills would be supervised by the PT.

Speech therapists frequently address not only the client's speech but also chewing and swallowing. These therapists evaluate clients and plan treatment regimens. The challenge with clients who have PD is that they not only have vocalization problems, but also dexterity deficits; speech therapists therefore must evaluate the potential benefits of assistive devices, such as a magic slate, voice synthesizer, or computer, for each client.

Nursing Care

The chronic and eventually debilitating nature of PD poses many challenges to clients, families, and healthcare professionals. Dependence due to declining physical and mental abilities is of major concern. In the early stages, most clients are able to remain at home, with the family assisting with or providing many of the client's ADL needs. As the disease progresses and the burden of care increases, the client and family may prefer placement in a long-term care facility. A Nursing Care Plan for the client with PD is given on the next page.

Health Promotion

Teaching preventive measures is extremely important when caring for clients who have Parkinson's disease. Preventing malnutrition, falls and other environmental accidents, constipation, skin breakdown from incontinence or immobility, and joint contracture requires teaching and reinforcement.

In addition to incorporating information about safety needs, teach ways to prevent orthostatic hypotension when the client changes positions; some clients may also benefit from wearing individually fitted compression hose. In addition, address safety considerations about proper administration of medications.

Assessment

Collect the following data through the health history and physical examination (see Chapter 43 ∞). Further focused assessments are described with nursing interventions. When assessing the older client, be aware of normal changes with aging, outlined in Chapter 43 ∞.

NURSING CARE PLAN A Client with PD

Walter Avneil, age 78, was diagnosed with PD at age 64. His wife died 5 years ago and he has no other family living. Mr. Avneil worked for more than 40 years as a mechanic in a large factory. He is a resident of a long-term care facility. During his last clinic visit for a review of his medications, the following assessment was made.

ASSESSMENT

White male with history of PD for the past 14 years. Skin oily and damp. Tremors in both hands and the lips. Gait is slow and shuffling, with a forward leaning posture. Speech slow and slurred. Face expressionless. Has lost 10 lb since last visit 3 months ago. Has been on levodopa with carbidopa since diagnosis. States major problems are "eating problems, bowel problems, walking problems."

DIAGNOSIS

- *Constipation* related to lack of exercise, decreased food intake, and effects of medications
- *Impaired Verbal Communication* related to lip tremors, slow/slurred speech, and facial muscle involvement of PD
- *Imbalanced Nutrition: Less than Body Requirements* related to difficulty swallowing and chewing
- *Impaired Physical Mobility* related to rigidity and bradykinesia

EXPECTED OUTCOMES

- Have a soft stool at least every other day.
- Practice exercises provided by speech therapist twice a day.
- Increase number of calories, fluids, and fiber in diet provided at long-term facility.
- Improve joint mobility and ability to ambulate.

PLANNING AND IMPLEMENTATION

- Discuss problems with bowel elimination with staff at long-term facility; suggest increasing fluids to 3000 mL per day and also increasing fiber in the diet with oatmeal for breakfast, and more fruits and vegetables at meals.
- Encourage exercises provided by speech therapist to improve speech and swallowing. If these are not effective, make a referral for another evaluation.
- Discuss diet plan with dietitian at the long-term care facility, including consistency of foods and number of calories. Suggest dietitian be a part of swallowing evaluation by the speech therapist.
- Refer for physical therapy and occupational therapy for a program to improve gait and joint mobility, and to decrease risk of falling.

EVALUATION

In a return visit 3 months later, Mr. Avneil reports that "my bowels are working better." He has gained 7 lb, and the staff report that this is related to multiple factors, including practicing his swallowing exercises, getting more exercise that stimulated his appetite, and changing his diet to six small meals a day of soft or pureed foods. The staff is offering him liquids at meals and snack times, and he usually drinks all they give him. His speech is not much improved. His posture and gait are somewhat better, and he is doing the exercises provided by the physical therapist and occupational therapist. Mr. Avneil's functional abilities have improved so much that the staff is considering training sessions specific to care of residents with PD.

CRITICAL THINKING IN THE NURSING PROCESS

1. Although Mr. Avneil did not mention it, the staff reports that he is frustrated by not being able to dress himself. What suggestions could you make to facilitate his independence?
2. Mr. Avneil spends most of his time alone, although he enjoys the company of the other residents. List assessments and interventions you might provide to increase his diversional activity.
3. The loss of his wife and the debilitating effects of his disease increase Mr. Avneil's risk for the nursing diagnosis of *Chronic Sorrow*. What might you suggest the long-term staff do to reduce this risk?

See Evaluating Your Response in Appendix C.

- *Health history:* Brain trauma, stroke, infection, exposure to heavy metals or carbon monoxide, medication and drug use, incontinence, constipation, weight loss, sweating, sleep problems, muscle pain, mood.
- *Physical assessment:* Affect; appearance; speech, scalp, eyelashes, and skin; drooling; tremor; coordination; posture; gait; muscle rigidity; mental status.

Nursing Diagnoses and Interventions

Clients with PD have complex and, ultimately, multisystem needs. Deficits in mobility and self-care are common. Psychosocial needs may include problems related to *Ineffective Coping, Powerlessness*, and *Disturbed Body Image*. Refer to the nursing care sections throughout this chapter for discussions of fatigue, self-care deficit, ineffective airway clearance, and other pertinent diagnoses. This section focuses on the nursing diagnoses related to impaired physical mobility, impaired verbal communication, imbalanced nutrition, and disturbed sleep pattern.

Impaired Physical Mobility

Clients with PD have impaired mobility for several reasons, including tremors, gait pattern disturbances, and alterations in body positioning, such as forward bending of the trunk. Poor self-esteem may contribute to the client's lack of motivation and willingness to be mobile.

- Suggest referral to a physical therapist to develop an individualized exercise program. *A program specific to the client supplies motivation as well as helping the client maintain muscle tone, flexibility, and mobility.*
- Request the physical therapist teach caregivers how to do ROM exercises at least twice a day, emphasizing the trunk, neck, arms, hips, and legs. *Maintaining joint mobility promotes better function and strength, improving gait pattern. Consistent ROM exercises can prevent contractures.*
- Ask caregivers to ambulate the client at least four times a day if possible. *Exercise fosters independence and self-esteem.*

- Recommend assistive devices, such as lift chairs, canes, splints, or braces, as indicated. *Adaptive equipment improves balance, protects joints, and promotes proper anatomic positioning.*
- To promote mobility and safety:
  - Slightly elevate the back legs of chairs and raise the toilet seat to help rise from a sitting position to a standing position.
  - Wear shoes with Velcro closures.
  - Remove potential hazards, such as unanchored throw rugs.
  - Install handrails and nonskid surfaces in bath tubs and showers.
  - Ensure adequate lighting throughout the home and in outside areas, especially in areas where transfers are common.

  Safety measures prevent potential complications that may result from falls or other accidents and promote self-esteem through self-care.

PRACTICE ALERT

Parkinson's disease is a disorder common in older adults, who are at greater risk for falls resulting from orthostatic hypotension, osteoporosis, poor vision, and other problems causing disorientation and confusion, such as Alzheimer's disease.

Impaired Verbal Communication

Diminished vocal amplitude and loss of muscular control can impair the client's ability to speak. Both caregivers and family members must remember to give clients enough time for self-expression; an unhurried approach is recommended. Seek input from family members when determining alternative methods of communicating with the client.

- Assess current communication abilities in speech, hearing, and writing. *Communication involves both sending and receiving messages.*
- Develop methods of communication appropriate to coordination abilities, such as a magic slate; flash cards with common phrases; pointing to objects. *Individualizing a method of communication decreases anxiety and isolation.*
- Suggest referral to a speech pathologist to develop oral exercises and interventions that will facilitate speaking. *The muscles of speech and swallowing are affected by the Parkinson's disease process.*
- Remind client to speak more loudly, if possible. *A low, monotonous voice is characteristic of the client with Parkinson's disease.*

Imbalanced Nutrition: Less than Body Requirements

Tremors, altered gait, and impaired chewing and swallowing can cause nutritional problems in the client with PD. As the disorder progresses, interventions for ensuring optimal nutrition need to be adapted to the client's functional abilities. Assess the client's swallow reflex before starting any feeding program. During the initial stages of the disorder, some clients may have the nursing diagnosis *Imbalanced Nutrition: More than Body Requirements* if kilocalorie intake exceeds energy expenditure.

- Assess nutritional status and self-feeding abilities; suggest referral to an occupational or speech therapist, if needed. *An initial assessment of abilities ensures that interventions are personalized to the client's current functional abilities.*
- Teach caregivers how to prepare foods of proper consistency as determined by swallowing function. *The client may aspirate food that is too liquid.*
- Weigh weekly. *Early recognition of weight loss allows for intervention.*
- Teach eating methods to decrease tremors, such as holding a piece of bread in the hand that is not holding an eating utensil. *Nonintention tremor may be reduced through purposeful activity.*
- Encourage diet that is high in bulk and fluids. *Several anti-Parkinson's medications and inactivity can cause constipation.*

Disturbed Sleep Pattern

Rigidity and weakness can cause clients with Parkinson's disease to lose the ability to move and change positions during sleep. The resulting discomfort causes periods of wakefulness. Medications to treat Parkinson's disease contribute to sleep pattern disturbance; for example, levodopa can cause vivid dreams. Nurses can help accurately assess the sleep pattern disturbance and in planning interventions to improve or increase sleep time.

- Assess sleep pattern and existing conditions that may affect sleep, such as depression or pain. *Clients experiencing anxiety, depression, and dementia have a difficult time falling asleep and may wake up more at night.*

PRACTICE ALERT

Remember to assess pain status; lack of adequate pain control may interfere with sleep.

- Explain the disease process and the effects of decreased dopamine on the sleep–wake cycle. *Depending on the dosage, levodopa causes less REM sleep and deep sleep.*
- Review the client's medication. *Bromocriptine and levodopa, especially if used with an anticholinergic, can cause vivid dreams. Other medications (diuretics, theophylline, hypnotics) also may interfere with sleep.*
- Teach how to modify lifestyle activities that affect sleep:
  - Institute a routine of activities with limited rest periods during the day; avoid napping close to bedtime. Avoid strenuous exercise in the evening. *Daytime sleeping may contribute to decreased nighttime sleeping. Vigorous exercise just before bedtime may act as a stimulant.*
  - Incorporate diet modifications, such as limiting caffeine and alcohol intake. *Caffeine is a stimulant, and alcohol may cause early morning awakenings, increased daytime sleepiness, and nightmares.*
  - Drink a glass of milk before bedtime. *Milk contains L-tryptophan, which produces sedative effects by shortening the time taken to fall asleep (sleep latency).*
  - Adapt the environment to aid in sleep (e.g., darken the room and decrease noises). *Reducing environmental stimuli decreases external sleep disturbances.*

Using NANDA, NIC, and NOC

Chart 46–3 shows links between NANDA nursing diagnoses, NIC, and NOC when caring for the client with PD.

Community-Based Care

It is important for both the client and the family to maintain independence and self-care as long as possible. To maintain function and quality of life, the following topics should be addressed:

- Realistic expectations
- Equipment suppliers
- Home environment conducive to using equipment
- Referrals to speech therapist, occupational therapist, physical therapist, and dietitian
- Gait training and exercises for improving ambulation, speech, swallowing, and self-care
- Increased fluid intake of 3000 mL/day and increased fiber in every meal
- Stool softeners or laxatives as needed for bowel elimination
- Swallowing during eating and taking medications (Have suction equipment available and know the Heimlich maneuver if choking occurs.)
- Foods that can be easily swallowed (such as pureed or soft) and feed six small meals a day if possible

NANDA, NIC, AND NOC LINKAGES

CHART 46–3 **The Client with Parkinson's Disease**

NANDA

Impaired Physical Mobility

↓

NIC

Energy Management

Fall Prevention

Surveillance: Safety

Self-Care Assistance

↓

NOC

Coordinated Movement

Balance

Fall Prevention Behavior

Self-Care: Activities of Daily Living (ADL)

Data from *NANDA's Nursing Diagnoses: Definitions & Classification 2005–2006* by NANDA International (2003), Philadelphia; *Nursing Interventions Classification (NIC)* (4th ed.) by J. M. Dochterman & G. M. Bulechek (2004), St. Louis, MO: Mosby; and *Nursing Outcomes Classification (NOC)* (3rd ed.) by S. Moorhead, M. Johnson, and M. Maas (2004), St. Louis, MO: Mosby.

- Helpful resources:
  - American Parkinson's Disease Association
  - National Parkinson Foundation, Inc.
  - Parkinson's Disease Foundation
  - The National Institute of Neurological Disorders and Stroke.

THE CLIENT WITH HUNTINGTON'S DISEASE

Huntington's disease (HD) is a progressive, degenerative, inherited neurologic disease characterized by increasing dementia and chorea (jerky, rapid, involuntary movements). It is a single-gene autosomal-dominant inherited disease that causes localized death of neurons of the basal ganglia (Porth, 2005). The exact cause is unknown, but postmortem studies have demonstrated a decrease in gamma-aminobutyric acid (GABA), an inhibitory neurotransmitter in the basal ganglia. There is also a decrease in acetylcholine levels, suggesting that the manifestations are the result of an imbalance in dopamine and acetylcholine. HD is a familial disease; each child of an HD parent has a 50% chance of inheriting the HD gene and, if so, will eventually develop the disease (NINDS, 2005f). There is no cure for the disease. Huntington's disease causes progressive chorea, speech problems, and dementia.

Because the client is usually asymptomatic until age 30 to 40, he or she may already have passed the gene to the next generation. The psychologic effect is devastating to clients and their families. The family not only experiences guilt from passing the disease from one generation to the next, but also is faced with the overwhelming long-term care needs of those affected. It is common for several family members to have the disease.

Pathophysiology

Huntington's disease causes destruction of cells in the caudate nucleus and putamen areas of the basal ganglia. Other areas of the brain, such as the frontal lobes, may selectively atrophy. Several neurotransmitters and their receptors are decreased, including GABA and acetylcholine. The neurotransmitter dopamine is not affected in Huntington's disease, but the decrease in acetylcholine results in a relative excess of dopamine in the basal ganglia. Whereas in Parkinson's disease a deficit of dopamine causes slow movement or lack of movement, in Huntington's disease the opposite occurs: There is a relative excess of dopamine, causing excessive, uncontrolled movement.

Manifestations

Manifestations and complications primarily involve abnormal movement and progressive dementia (see the box on the next page). The progression and sequence of manifestations varies somewhat; however, initially the psychologic manifestations are more debilitating than the choreiform (rapid and jerky) movements.

Early signs of personality change include severe depression, memory loss with decreased ability to concentrate, emotional lability, and impulsiveness. The client experiences frequent mood swings ranging from uncontrollable periods of anger to

MANIFESTATIONS AND COMPLICATIONS of Huntington's Disease

MOTOR EFFECTS

Early
- Restlessness
- "Fidgety" feeling
- Minor gait changes—unsteady on feet
- Posture and positioning disturbances, frequent falls
- Inability to keep the tongue from protruding
- Slurred speech with poor articulation
- Complications: increasing problem with self-care activities, such as bathing, grooming, eating

Late
- Chorea—severely altered gait with irregular, uncontrollable movement; shoulders shrug arrhythmically
- Facial grimacing—raising of eyebrows, uncontrollable protrusion of the tongue
- Dysphagia
- Unintelligible speech
- Impaired diaphragmatic movement
- Complications: immobility, aspiration, choking, and, eventually, total dependence, poor oxygenation, emaciation, and cachexia

PSYCHOSOCIAL EFFECTS

Early
- Irritability
- Outbursts of rage alternating with euphoria
- Depression
- Complication: suicide

Late
- Decreasing memory
- Loss of cognitive skills
- Eventual dementia
- Complication: total dependence

apathy. Eventually, signs of dementia, including disorientation, confusion, and lack of sense of time, become evident and interfere with self-care.

Motor manifestations usually parallel personality and mood changes. The motor manifestations worsen with environmental stimuli and emotional stress but are absent when the client is sleeping. Initially, movement problems are described as "fidgeting" or restlessness, followed by progressive worsening of abnormal movements. The choreiform movements, which begin in the face and arms and then involve the entire body, are manifested by facial grimaces, tongue protrusion, jerky movement of the distal arms or legs, and a rhythmic, lurching gait that almost resembles a dance. (The term *chorea* comes from *choreia,* the Greek word meaning "dance.") Gait changes cause uncoordinated movements and contribute to frequent falls.

The muscles of swallowing, chewing, and speaking are affected, leading to dysphagia and dysarthria and associated problems with communication and nutrition. The client's constant movement and difficulty in swallowing contribute to weight loss and eventual cachexia. Breathing is impaired because the diaphragm is unable to move effectively.

The manifestations slowly progress over approximately 15 to 20 years after initial manifestations appear. Prognosis is poor, with inevitable debilitation and total dependence. Death usually results from aspiration pneumonia or another infectious process.

INTERDISCIPLINARY CARE

There is no cure for Huntington's disease, and treatment addresses the disease's manifestations. Nurses provide care to clients with Huntington's disease in a variety of community settings. Initially, clients and families can manage care needs at home, but as the disease progresses, the client requires constant supervision, such as that provided in day care facilities. Eventually, skilled long-term care is needed. Clients who develop acute problems may be hospitalized until the crisis is managed. Because of the inevitable total multisystem debilitation of clients with Huntington's disease, nurses and other caregivers face many challenges.

Diagnosis

Genetic testing is the only test available to diagnose clients suspected of having Huntington's disease. Both blood and amniotic fluid may be tested for the presence of a gene mutation on chromosome 4 using DNA analysis. The test can predict with 95% accuracy which offspring have the disease.

Medications

The following medications are given for the manifestations of Huntington's disease:
- Antipsychotics, specifically phenothiazines and butyrophenones, are effective in Huntington's disease because they block dopamine receptors in the brain. The therapeutic goal is to restore the balance among the neurotransmitters.
- Antidepressants are prescribed in the early stage of the disease; however, medications are no substitute for intense follow-up counseling for clients and families.

NURSING CARE

Nurses are faced with a multitude of challenges when caring for families who have Huntington's disease, including physiologic, psychosocial, and ethical problems. Physiologic problems are related to the progressive and eventually debilitating nature of the disease. Psychosocial concerns occur as a result of the client's personality and mental changes, the family's responsibility for providing care, and the guilt implicit in a genetically transmitted disease. Ethical difficulties relate to the genetic nature of the disease: DNA testing for the marker on chromosome 4 can determine whether the person is a carrier of the disease before he or she begins to exhibit manifestations. Children of people with Huntington's disease are thus faced with the choice of finding out whether they will eventually be affected. If they choose not to be tested, they may pass the disease on to yet another generation; and if a fetus is affected, they may face the decision of whether to undergo an abortion.

MEDIALINK Case Study: Huntington's Disease

Nursing Diagnoses and Interventions

Initially, much of the nursing care focuses on teaching about the disease, psychologic support, and genetic counseling. As manifestations become more severe, nursing considerations center on problems related not only to immobility and altered nutrition, but also to the increasing self-care deficits. Families and clients experiencing Huntington's disease face many psychosocial issues. Nurses must be prepared to listen actively as well as to provide comfort and encouragement throughout the lengthy illness. There are many possible nursing diagnoses for the client with Huntington's disease; this section focuses on nursing diagnoses related to aspiration, nutrition, skin integrity, and communication.

Risk for Aspiration

Uncoordinated movements and swallowing and chewing problems put the client at high risk for aspiration.

- Maintain in an upright position while the client eats; support the head. *Proper positioning may prevent aspiration during mealtime.*
- Teach the Heimlich maneuver to caregivers and family members. *Aspiration is a real possibility; caregivers must be prepared to reestablish the client's airway.*
- Provide food that is thick enough to manage, such as thick soups, mashed potatoes, stews, or casseroles. *These foods are more readily tolerated and manipulated by the tongue than liquids.*
- Make sure food is swallowed before giving another spoonful of food. *The automatic phase of swallowing may be disrupted in the client with Huntington's disease; providing adequate time and smaller bites may improve the ability to manipulate foods.*
- Provide a calm, relaxing eating environment. *Stress worsens choreiform movements and inappropriate behaviors.*

Imbalanced Nutrition: Less than Body Requirements

Clients with Huntington's disease have unpredictable choreiform movements of the extremities and decreased ability to control muscles involved with chewing and swallowing. Families and caregivers are challenged to provide sufficient calories to maintain the client in positive nitrogen balance.

- Evaluate current weight and nutritional status, including serum prealbumin and transferrin levels. *Establishing a baseline is crucial for meeting individual caloric, protein, vitamin, and mineral needs.*
- Assess ability to swallow and manipulate eating utensils. *Aspiration is an ever-present danger that must be avoided; utensils may need to be adapted to client's abilities, if client is able to assist at all.*
- Continue feeding even if the client physically turns away from the meal. *Involuntary choreiform movements should not be interpreted as a refusal to eat.*
- Provide high-kilocalorie, nutritious foods and sufficient snacks; request input from a dietitian. *The constant movement of Huntington's disease increases caloric requirements.*
- Avoid milk; provide frequent oral hygiene. *Milk tends to thicken secretions. Decreasing thick secretions may improve ability to swallow and enable the client to ingest more calories.*

Impaired Skin Integrity

Skin integrity is only one component of the client's general need for protection and avoidance of injury. Several factors increase the risk for impaired skin integrity, including poor nutritional status, eventual total immobility, and incontinence.

- Evaluate the skin for actual and potential areas of breakdown. *Establishing a baseline is necessary to modify care and provide prophylactic protection of high-risk pressure areas.*
- Determine nutritional status, especially serum prealbumin level and vitamin, mineral, and kilocalorie intake. *Optimal nutritional status and positive nitrogen balance help prevent skin breakdown and formation of pressure ulcers.*
- Turn and inspect the skin at least every 2 hours, giving special consideration to areas that are most prone to breakdown, such as heels and coccyx. *Pressure points are particularly susceptible to skin breakdown.*
- Provide ROM exercises on a regular schedule in the daytime. *Movement stimulates circulation, which provides oxygenation and allows nutrients to reach muscles and skin.*
- Keep the skin clean and dry; pay particular attention to the perineal area if incontinent. *Skin in close proximity to the perineal area, such as the sacral area, is highly susceptible to breakdown due to exposure to wet, acidic urine and fecal material.*
- Place on an alternating-pressure mattress with foot board. *Decreasing pressure on bony prominences and preventing shearing forces serve to prevent skin breakdown.*
- Pad side rails and headrests of special chairs; have the client wear a football-type helmet. *The client's violent movements can cause trauma to the head and extremities.*

Impaired Verbal Communication

The inability to control muscles related to speech, swallowing, and facial movement contributes to problems of verbal communication. Because Huntington's disease affects fine motor movement, especially the distal portion of the extremities, the hands are not effective in communication. As the disease progresses, mental abilities are also compromised, making both receptive and expressive communication impossible.

- Choose alternative methods of communication while the client is able to participate. *Anticipatory planning may facilitate communication and decrease anxiety.*
- Continue to incorporate therapeutic communication techniques, even though client is not responsive: Maintain eye contact, use touch, and talk directly to the client rather than to others in the room. *These techniques enhance the individual's dignity and worth.*
- Seek input from family about client's usual preferences and how they are communicated; be alert for subtle cues. *Nonverbal communication techniques may be individualized and more readily recognized by the family member or caregiver who usually provides care.*

- Continue talking to the client, even though there is no apparent response. *Hearing may not be impaired, even though the client cannot speak.*

Community-Based Care

Clients with Huntington's disease and their families may know how devastating the illness is because they may have cared for a parent or other close family member who had the illness. Many families are overwhelmed with just the thought of the physical and psychosocial debilitation that the disease brings. Fear, anxiety, and hopelessness leading to depression are common reactions. Teaching ways to cope effectively with the psychosocial and physical changes is an integral part of the nurse's responsibilities. Referrals to appropriate agencies, such as adult day care centers, the Huntington's Disease Foundation, and local support groups or a psychologist should be part of the nursing plan.

Another aspect of client teaching concerns the genetic transmission of Huntington's disease; refer clients and family members to a geneticist. Nurses are frequently involved with clarifying information, especially concerning the transmission, course of illness, and prognosis. A caring, sensitive approach is crucial. Information about transmission of an autosomal-dominant trait is discussed in Chapter 8 ∞ .

THE CLIENT WITH AMYOTROPHIC LATERAL SCLEROSIS

Amyotrophic lateral sclerosis (ALS), or *Lou Gehrig's disease,* is a rapidly progressive and fatal degenerative neurologic disease characterized by weakness and wasting of muscles under voluntary control, without any accompanying sensory or cognitive changes. The name is derived from the pathophysiologic processes of muscle atrophy (*amyotrophy*) resulting from lower motor neuron involvement and sclerosis of the corticospinal tract in the lateral column of the spinal cord resulting from upper motor neuron involvement. Death results in 2 to 5 years after onset of the manifestations (although some people live 10 years or more), usually due to respiratory failure.

ALS is the most common motor neuron disease in the United States. As many as 20,000 people in the United States have ALS, and approximately 5000 new cases are diagnosed each year. In up to 90% to 95% of cases, the disease occurs at random without clearly associated risk factors. About 5% to 10% of all cases are inherited in what is termed familial ALS (NINDS, 2005a).

Most people are between 40 and 60 years of age at diagnosis; the incidence is higher in men in the earlier ages but becomes equal with women after menopause. Most of the health problems a client with ALS encounters are related to swallowing and managing secretions, communication, and dysfunction of the muscles used in respiration.

Pathophysiology

ALS results from the degeneration and demyelination of both upper and lower motor neurons in the anterior horn of the spinal cord, brainstem, and cerebral cortex. Death of the motor neurons results in axonal degeneration, demyelination, glial proliferation, and scarring along the corticospinal tract. In the early stages of the disease, surviving motor neurons sprout new branches to reinnervate affected muscle fibers, preserving muscle strength. However, when more than half of the lower motor neurons are affected, reinnervation fails and weakness is evidenced.

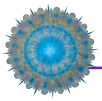
Although the pathogenesis of ALS is not clear, abnormal glutamate metabolism and hydrogen peroxide production are being studied. Echovirus RNA has also been isolated in spinal cord tissue in some clients with nonfamilial ALS. Environmental factors, excess intracellular calcium, and antibodies to calcium channels are also being researched.

Manifestations

The initial manifestations may relate to dysfunction of upper motor neurons, lower motor neurons, or both. Dysfunction of upper motor neurons results in spastic, weak muscles with increased deep tendon reflexes. Dysfunction of lower motor neurons results in muscle flaccidity, paresis (weakness), paralysis, and atrophy.

Weakness and paresis are common early manifestations. The weakness may initially affect only one muscle group. Manifestations vary according to the particular muscle group involved; *fasciculations* (twitching) of involved muscles are common in the early stage of the disorder. With the loss of muscle innervation, the muscles atrophy, and paralysis results. Muscle mass decreases, and clients complain of progressive fatigue. Typically, the disease first affects the hands, then the shoulders, upper arms, and finally the legs.

Increasing brainstem involvement causes progressive atrophy of the tongue and facial muscles with eventual dysphagia and dysarthria. Emotional lability and loss of control occur, but dementia is not part of the pathologic progression of ALS. Vision, hearing, sensation, and cognitive ability usually remain intact. A summary of manifestations and complications is presented in the box on the next page.



INTERDISCIPLINARY CARE

Because many treatable disorders may cause manifestations similar to those that appear in the initial stage of ALS, a thorough evaluation is required. Once ALS is diagnosed, the primary goal is to support the client and family in meeting physical and psychosocial needs, particularly as the disease progresses.

Medical and nursing care for clients with ALS is primarily supportive. Referral for home health management is indicated. Occupational, physical, speech, and respiratory therapy are major supportive and rehabilitative treatments. As the disorder progresses and swallowing becomes ineffective, a gastrostomy tube may be necessary to provide adequate nutritional intake. Ventilatory assistance should be discussed with clients before the need occurs.

Diagnosis

There is no specific test to diagnose ALS. Rather, diagnosis is made based on manifestations and tests to rule out other diseases. A number of disorders may mimic early ALS, including

MANIFESTATIONS AND COMPLICATIONS of ALS

MUSCULOSKELETAL SYSTEM
- Weakness and fatigue
- "Heaviness" of legs
- Fasciculations
- Uncoordinated movements, loss of fine motor control in hands
- Spasticity
- Paresis
- Hyperreflexia
- Atrophy
- Problems with articulation
- Complications: paralysis, loss of ability to perform ADLs, total immobility, aspiration, loss of verbal communication

RESPIRATORY SYSTEM
- Dyspnea
- Difficulty clearing airway
- Complications: pneumonia, eventual respiratory failure

NUTRITIONAL EFFECTS
- Difficulty chewing
- Dysphagia
- Complication: malnutrition

EMOTIONAL EFFECTS
- Loss of control, lability
- Complication: depression

hyperthyroidism, hypoglycemia, compression of the spinal cord, toxic agents, infections, and neoplasms.

Medications

Riluzole (Rilutek), an antiglutamate, is the first medication developed to treat ALS. It inhibits the presynaptic release of glutamic acid in the CNS and protects neurons against the excitotoxicity of glutamic acid. This oral medication is administered without food at the same time each day. Clients are regularly monitored for liver function, blood count, blood chemistries, and alkaline phosphatase. They should be warned to report any febrile illness to their healthcare provider and to avoid alcohol.

NURSING CARE

Nursing care focuses on current health problems and on anticipating future difficulties. As with other disorders causing incapacitation and dependence, individualized nursing goals and interventions relate to decreasing complications, especially those associated with loss of muscular function and immobility; promoting independence to the extent possible; initiating referrals, particularly to a support group for both client and family; and providing physical and psychosocial support as indicated.

Of special consideration is planning for the client's eventual inability to communicate. Because the client's eye muscles and movements remain intact, signals can be prearranged before the loss of speech.

Nursing Diagnoses and Interventions

Two nursing diagnoses that frequently apply to clients with ALS are *Risk for Disuse Syndrome* and *Ineffective Breathing Pattern*.

Risk for Disuse Syndrome

Clients with ALS are at risk for developing problems associated with bed rest not only because they cannot move and reposition themselves, but also because they frequently have altered nutritional and hydration status. Nursing interventions focus on preventing skin breakdown and infections, such as urinary tract infections.

- Assess current condition for baseline parameters, particularly skin over bony prominences, lung sounds, and vital signs. *Understanding client's current condition allows accurate future assessment and realistic planning.*
- Assess skin; provide skin care, and obtain an alternating-pressure mattress. *Pressure points are at risk for breakdown; early detection is crucial to instituting appropriate care.*
- Institute active ROM exercises, as the client is able. Perform passive ROM exercises every 2 hours, when the client is turned. *Contractures can develop within a week because extensor muscles are weaker than flexor muscles.*
- Maintain positive nitrogen balance and hydration status: Monitor prealbumin levels, hemoglobin and hematocrit levels, and urine specific gravity. *Adequate protein is required to maintain osmotic pressure and prevent edema; positive nitrogen balance promotes optimal body functioning.*
- Monitor for manifestations of infection; for example, assess urine, especially if a urinary catheter is present. *Urinary catheters place clients at high risk for sepsis; bed rest places the client at greater risk for urinary stasis.*

PRACTICE ALERT

Urinary tract infection is indicated by cloudy, foul-smelling urine, pain on urination, fever, and general malaise.

Ineffective Breathing Pattern

As the muscle weakness of ALS continues, clients become less able to breathe. The respiratory muscles are affected, and clients eventually may require ventilatory assistance. The nurse must initiate measures to support the existing respiratory effort.

- Obtain a baseline assessment of breathing pattern, air movement, and oxygen saturation. *Assessments indicating the client's current condition provide data to plan individualized interventions.*
- Turn at least every 2 hours. *Movement enhances the ability to move pulmonary secretions and prevents stasis.*
- Elevate the head of the bed at least 30 degrees, suction as indicated, and provide oxygen. *This supports ventilation and enhances lung expansion as the client's condition changes.*
- Monitor temperature and lung sounds routinely; obtain sputum culture as indicated. *Early detection of a possible infectious process leads to prompt treatment.*

PRACTICE ALERT
A pulmonary infection is indicated by respiratory difficulty, crackles and/or wheezes, cough productive of yellow or green sputum, fever, and malaise.

Community-Based Care

Initial teaching centers on explaining the disease process, expected course, and prognosis. Referral to a social worker to determine home care needs and financial assistance is helpful. Counseling and referrals to a home health agency, dietitian, and physical, speech, and occupational therapists can help the family meet the client's changing needs and abilities. The realistic anticipation of needs cannot be overemphasized.

As the client becomes more debilitated, family members or other care providers focus on preventing complications. For example, family members need to know how to suction the client and perform the Heimlich maneuver to prevent aspiration. Teaching the family how to prevent problems related to immobility is a primary consideration for the nurse.

Another focus of teaching is basic care needs, such as care required to meet elimination needs. Teach families methods to establish a bowel routine, considerations related to a urinary catheter, and the need to promptly report manifestations of an infection.

Throughout the early stage and continued care of the client and family with ALS, much consideration is given to psychosocial concerns. Depression, anger, and denial may be initial reactions; refer the client and family to an ALS support group, social worker, psychologist, or psychiatrist as indicated.

PERIPHERAL NERVOUS SYSTEM DISORDERS

Many etiologic agents are responsible for peripheral nervous system disorders. Autoimmune disorders, viruses, environmental toxins such as heavy metals, and nutritional deficiencies can affect the peripheral nervous system.

THE CLIENT WITH MYASTHENIA GRAVIS

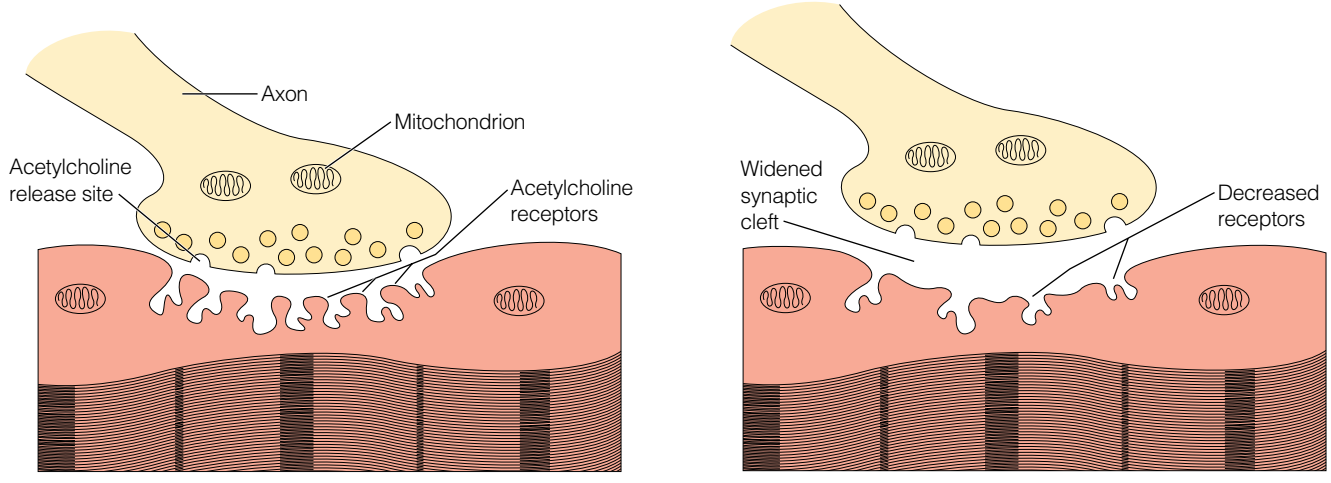
Myasthenia gravis is a chronic autoimmune neuromuscular disorder characterized by fatigue and severe weakness of skeletal muscles. Clients experience periods of remission and exacerbation, and mild forms of the disorder exist. Weakness may remain limited to a few muscle groups, especially the ocular muscles, or may become generalized with all muscles eventually becoming weakened.

Women are affected three times more frequently than men. The age of onset for most clients is between ages 20 and 30 (Porth, 2005). Treatment with anticholinesterase medications has greatly improved the prognosis and symptom management.

Pathophysiology

The axons of motor neurons divide as they enter skeletal muscles, and each axonal ending forms a neuromuscular junction. Although the axonal ending and the muscle fiber are extremely close, they are separated by the synaptic cleft. The transmission of nerve impulses from the nerve to the muscles occurs at the neuromuscular junctions. The neurotransmitter acetylcholine is released from the axonal ending, crosses the synaptic cleft, attaches to acetylcholine receptors on the muscle fiber, and stimulates the muscle.

In myasthenia gravis, antibodies destroy or block neuromuscular junction receptor sites, resulting in a decreased number of acetylcholine receptors. Structural changes also result in diminished acetylcholine uptake. The net result is a decrease in the muscle's ability to contract despite a sufficient amount of acetylcholine. A comparison of a normal neuromuscular junction and one affected by myasthenia gravis is shown in Figure 46–4 ■.



A Normal neuromuscular junction

B Myasthenia gravis

Figure 46–4 ■ *A,* A normal neuromuscular junction and *B,* one showing the changes seen in myasthenia gravis. These changes interfere with the transmission of nerve impulses to the muscle.

In about 75% of clients with myasthenia gravis, the thymus gland, which is usually inactive after puberty, continues to produce antibodies because of hyperplasia of the gland or because of tumors. It is believed that the thymus is a source of autoantigen that triggers an autoimmune response in myasthenia gravis. The exact mechanism and reason for the thymus gland's antibody production are unknown.

Myasthenia gravis is sometimes associated with a tumor of the thymus, thyrotoxicosis (hyperthyroidism), rheumatoid arthritis, and lupus erythematosus. The disorder is often diagnosed when a client seeks treatment for a coincidental infection that exacerbates manifestations. Exacerbations may also occur before the menstrual period and during or soon after pregnancy.

Manifestations

The manifestations of myasthenia gravis correspond to the muscles involved. Initially, the eye muscles are affected and the client experiences either diplopia (unilateral or bilateral double vision) or ptosis (drooping of the eyelid) (Figure 46–5 ■). Next, the facial, speech, and mastication muscles become weak, and clients may have periods of dysarthria and dysphagia. Fatigue is evident even when the client tries to eat a meal; the muscles of chewing tire, and the client is forced to stop eating momentarily. A smile becomes a snarl or grimace, and the voice is weak with a muffled nasal quality. Problems performing fine motor movements of the hands, such as writing, appear early in the disease.

As the disease progresses, the muscles of the neck and extremities are affected. When the muscles of the neck become affected, the head juts forward. Deep tendon reflexes are usually normal, however, even in weak muscles. Fatigue and weakness are exacerbated with stress, fever, overexertion, and exposure to heat and are relieved by rest. Manifestations vary on a daily basis. Manifestations and complications of myasthenia gravis are listed in the box on this page.

Complications

Complications are directly related to the degree of muscle weakness and the specific muscles involved. For example, when the pharyngeal and palatal muscles are affected, the client cannot manage swallowing and may aspirate food or fluids. The client is at increased risk for pneumonia because weakness of the diaphragm and muscles of respiration compromises gas exchange. Clients with myasthenia gravis can develop life-threatening emergencies, including myasthenic crisis and cholinergic crisis.

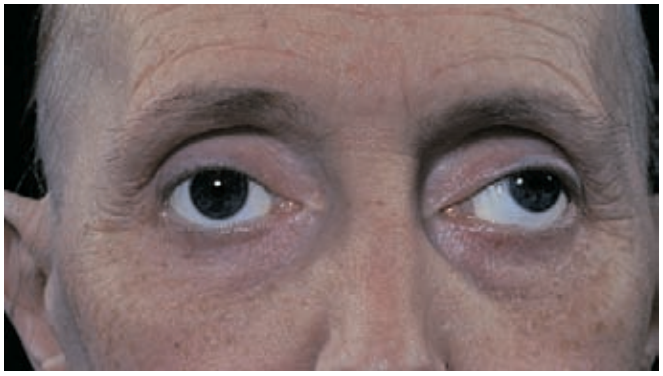


Figure 46–5 ■ In myasthenia gravis, the client experiences unilateral weakness of the facial muscles. Note the drooping of one eyelid.

Source: Custom Medical Stock Photo, Inc.

MANIFESTATIONS AND COMPLICATIONS of Myasthenia Gravis

OCULAR AND FACIAL
- Ptosis
- Diplopia
- Facial weakness
- Dysphagia
- Dysarthria
- Complications: difficulty closing eyes, aspiration, impaired communication and nutrition

MUSCULOSKELETAL
- Weakness and fatigue
- Decreased function of hands, arms, legs, and neck muscles
- Complications: inability to perform ADLs and self-care activities, complications related to immobility, myasthenic and cholinergic crises

RESPIRATORY
- Weakening of intercostal muscles
- Decrease in diaphragm movement
- Breathlessness and dyspnea
- Poor gas exchange
- Complications: decreasing ability to walk, eat, and perform other ADLs, pneumonia

NUTRITIONAL
- Inability to chew and swallow
- Decreasing ability to move tongue
- Impairment of fine motor movements: inability to feed self
- Complications: weight loss, dehydration, malnutrition, aspiration

Myasthenic Crisis

Myasthenic crisis is a sudden exacerbation of motor weakness putting the client at risk of respiratory failure and aspiration. Myasthenic crisis most often is due to undermedication, missed doses of medication, or a developing infection. Manifestations of myasthenic crisis include tachycardia, tachypnea, severe respiratory distress, dysphagia, restlessness, impaired speech, and anxiety.

Cholinergic Crisis

Cholinergic crisis is the result of overdosage with the anticholinesterase (cholinergic) medications used to treat myasthenia gravis. Gastrointestinal manifestations, severe muscle weakness, vertigo, and respiratory distress are signs of cholinergic crisis. Both types of crises are emergency, life-threatening situations; clients frequently require ventilatory assistance. Differentiation is based on the client's response to edrophonium chloride (Tensilon). In myasthenic crisis the test is positive, and in cholinergic crisis the test is negative (see the discussions that follows under Diagnosis).

INTERDISCIPLINARY CARE

Care of the client with myasthenia gravis focuses on providing appropriate treatment, preventing complications, and supporting the client and family in meeting physical and psychosocial needs, especially as the disease progresses.

Diagnosis

Diagnostic tests are conducted following a thorough history and physical examination, with special attention to the facial, oculomotor, laryngeal, and respiratory muscles. Diagnostic tests include the anticholinesterase (Tensilon) test, nerve stimulation studies, and an analysis of antiacetylcholine receptor antibodies.

In the Tensilon test, the client is injected with edrophonium chloride (Tensilon), a short-acting anticholinesterase. Clients with myasthenia gravis show a significant improvement in muscle strength that lasts approximately 5 minutes. This test is also used to differentiate myasthenic crisis (caused by insufficient medication, so the client shows improvement with the drug) from cholinergic crisis (caused by overmedication, so the client does not show improvement).

Single-fiber electromyography can detect delayed or failed neuromuscular transmission in muscle fibers supplied by a single nerve fiber. Serum assay of circulating acetylcholine receptor antibodies, if increased, is diagnostic of myasthenia gravis with a sensitivity of 80% to 90%.

Medications

The primary group of medications used to treat myasthenia gravis is the anticholinesterases. These drugs act at the neuromuscular junction and allow acetylcholine to concentrate at the receptor sites, thus promoting muscle contraction. Pyridostigmine (Mestinon) is the most commonly used acetylcholinesterase inhibitor for myasthenia gravis. The client's decrease in manifestations guides dosage.

Immunosuppression with glucocorticoids, typically prednisone, is another pharmacologic therapy aimed at improving muscle strength. Clients must be aware of the need to stay on the drug at the prescribed dose to determine the least amount required for efficacy. If clients do not respond to prednisone alone, it may be combined with other immunosuppressive agents, such as cyclosporine or azathioprine (Imuran). Medications used to treat myasthenia gravis are discussed in the Medication Administration box below.

Surgery

Approximately 75% of clients with myasthenia gravis have dysplasia of the thymus gland. Therefore, thymectomy is often

MEDICATION ADMINISTRATION **The Client with Myasthenia Gravis**

ANTICHOLINESTERASES/CHOLINESTERASE INHIBITORS
Neostigmine (Prostigmin)
Ambenonium (Mytelase Caplets)
Pyridostigmine (Mestinon, Regonol)
For diagnosis: edrophonium chloride (Tensilon)

Cholinesterase inhibitors are used in myasthenia gravis to enhance the effects of acetylcholine at the remaining skeletal muscle receptors. Cholinesterase inhibitors do not cure or change the underlying pathophysiologic processes, but they can provide effective, lifelong improvement of symptoms. Because the cholinesterase inhibitors are nonselective, the neuromuscular, muscarinic, and ganglionic junctions are each affected.

Adjusting the dose to obtain maximum benefit with minimal side effects is a major consideration when administering cholinesterase inhibitors. Initially, small doses are given followed by incremental increases until optimal muscle strength is obtained. The dose may need to be adjusted when activities result in symptoms of undermedication, such as increased ptosis. Severe undermedication results in myasthenic crisis. Although a sustained release form of pyridostigmine is available for bedtime use, it should not be used during the day because of its inconsistent absorption.

Cholinesterase inhibitors should not be administered to clients experiencing obstruction of the intestinal or urinary tract. Caution is advised when administering these drugs to clients with asthma, hyperthyroidism, bradycardia, or peptic ulcer disease. Cholinesterase inhibitors can cross the placenta; reproductive counseling is indicated.

Nursing Responsibilities

- Obtain a baseline assessment of muscle strength and abilities, concentrating on swallowing and ptosis.
- Administer the medication parenterally if the client has dysphagia. Check the dose of the medication carefully when changing from oral to parenteral routes.
- Evaluate the effectiveness of the medication and document the response, for example, time when fatigue occurs in relation to activities.
- Promptly recognize and respond to manifestations of excessive stimulation of muscarinic receptors: excess salivation, urinary urgency, bradycardia, gastrointestinal hypermotility, diaphoresis. Atropine can be administered to combat these manifestations. Respiratory depression and failure can occur and require mechanical ventilation.
- Have a muscarinic antagonist (e.g., physostigmine) readily available to treat poisoning.

Health Education for the Client and Family

- Balancing symptom control with dosage is crucial; record time of dose and response in a journal. Note the time of day when fatigued and any adverse effects, such as excess salivation, sweating, slow heartbeat, and diarrhea.
- Take the medication about 30 minutes prior to meals to enhance swallowing and chewing.
- Report manifestations of myasthenic crisis immediately: severe muscle weakness, fast heartbeat, restlessness, difficulty breathing, increasing difficulty swallowing or speaking.
- Report slow heartbeat, increased salivation or sweating, and/or decreased blood pressure immediately.
- Review possible causes of myasthenic crisis: physical or emotional stress, infection, or reduction in the medication dosage.
- Wear or carry Medic-Alert identification.

NURSING CARE OF THE CLIENT HAVING A Thymectomy

PREOPERATIVE CARE

- Reinforce the physician's explanation of the procedure, and prepare the client for chest tubes and tracheostomy. *Realistic preparation of what to expect postoperatively encourages compliance and allays anxiety.*
- Anticipate the need for alternative communication. *The client may have a tracheostomy; preoperative planning facilitates communication after surgery.*
- Allow sufficient time for questions. *Thymectomy is a major surgery requiring either a thoracotomy and sternal split or transcervical approach. The client is usually anxious, and adequate time must be allocated to preoperative instruction.*

POSTOPERATIVE CARE

- Provide meticulous pulmonary hygiene: turning, deep breathing, and coughing at least every 2 hours; use an incentive spirometer. *Regardless of surgical approach, measures are aimed at preventing pulmonary complications of atelectasis and pneumonia.*
- Clients with a thoracotomy and sternal split procedure will require care of the anterior chest tube. Observe for complications; such as pneumothorax. *Air may enter the thoracic cavity—be alert for sudden chest pain and dyspnea, decreased breath sounds, and early signs of shock, such as restlessness.*
- Manage pain with scheduled analgesic therapy. *Maintaining a therapeutic blood level of analgesic provides better pain control than waiting until the client requests medication, as on a prn basis.*

recommended for clients younger than 60. The two surgical approaches used are the transcervical approach, which is considered less invasive, and the transsternal approach. The latter approach allows a more extensive removal of the gland; however, it also poses more potential complications because it involves splitting the sternum.

Preoperatively, clients may be tapered from steroid therapy. Usually, pyridostigmine is administered to prevent muscular manifestations during the perioperative period. Postoperative nursing care focuses on preventing complications and controlling pain. Nursing implications for the client undergoing thymectomy are presented in the box on this page. Remission is obtained in about 40% of clients but may take several years to achieve. Refer to Chapter 38 for care of the client having a thoracotomy and chest tubes. A tracheostomy may be required when the diaphragm or intercostal muscles are involved.

Plasmapheresis

Plasma exchange in myasthenia gravis may be used in conjunction with other therapies; for example, it may be performed prior to surgical intervention. The goal of therapy is to remove the antiacetylcholine receptor antibodies, thus improving severe muscle weakness, fatigue, and other manifestations. The procedure is frequently performed when respiratory muscle involvement is evident. See Figure 46–6 and the box below for nursing care of the client having a plasmapheresis.

NURSING CARE OF THE CLIENT TREATED WITH Plasmapheresis

PREPROCEDURE CARE

- Teach about the procedure and what to expect, including what the machine looks like, the need for arterial and venous insertion sites, and the length of time of the procedure (2 to 5 hours). *Giving information, answering questions, and addressing concerns decrease anxiety.*
- Check with physician about holding medications until after the procedure. *Medications may be removed from the body as an incidental part of the plasmapheresis process.*
- Assess vital signs and weight. *Baseline parameters are necessary to evaluate for fluid imbalances and response to therapy.*
- Assess CBC, platelet count, and clotting studies. *Clients undergoing plasmapheresis are at high risk for anemia and coagulation problems secondary to hemolysis of cells.*
- Check blood type and crossmatch for replacement blood products. *Hypersensitivity reactions can occur, and close monitoring is important.*

CARE DURING AND AFTER THE PROCEDURE

- Observe for dizziness or hypotension. *Hypovolemia is a complication of plasma exchange, especially during the procedure when up to 15% of the client's blood volume is in the cell separator.*
- Apply pressure dressing to access site(s). *Direct pressure helps decrease or prevent bleeding.*
- Monitor for infection and bruises at the intravenous port site. *The site of vascular access is at risk for complications and must be routinely and carefully assessed for signs of infection and for bleeding or hematoma formation.*
- Monitor electrolytes and signs of electrolyte loss. Report imbalances, and replace electrolytes as ordered. Observe for circumoral tingling, Chvostek's and Trousseau's signs if calcium levels are low, and cardiac dysrhythmias and leg cramps if potassium levels are low. *Hypocalcemia and hypokalemia may occur. Hypocalcemia occurs because the anticoagulant citrate dextrose binds with calcium.*
- Reevaluate preprocedure laboratory data, especially CBC, platelet count, and clotting times. *The cell-separating process can damage cells; anticoagulation is part of the procedure.*

Figure 46–6 ■ Plasmapheresis is a procedure used to separate the blood's cellular components from plasma. About 50 mL per minute is withdrawn to the centrifuge in the plasmapheresis machine. The plasma is replaced with donor plasma or colloids and returned to the client.

Courtesy of Baxter Healthcare Corporation.

NURSING CARE

Because avoiding fatigue is a major part of teaching, it is important to incorporate interventions to enhance rest and conserve energy (Box 46–4). For example, suggest sitting while preparing meals and while performing hygiene and grooming. Anticipating problems, such as impaired communication, and developing alternative solutions can be helpful in promoting independence. A Nursing Care Plan for the client with myasthenia gravis is given on the next page.

Nursing Diagnoses and Interventions

Nursing care of clients with myasthenia gravis focuses not only on present problems but also on anticipated needs. Preventing myasthenic and cholinergic crises and providing psychologic support to clients and families are two important aspects of care. Individualized care depends on the specific therapy instituted. This section discusses the nursing diagnoses related to ineffective airway clearance and impaired swallowing; other nursing diagnoses that commonly apply, such as *Fatigue*, are addressed in other sections of this chapter.

BOX 46–4 Client and Family Teaching: Myasthenia Gravis

- Schedule periods of rest and avoid stress; conserve energy when possible.
- Avoid cigarette smoke, alcohol, and beverages with quinine (e.g., tonic water).
- Take medications as prescribed. If manifestations change, consult the physician; the dose may need to be adjusted.
- Avoid extremes of temperature; an environment that is too hot or too cold may cause an exacerbation of myasthenia gravis.
- Avoid people with upper respiratory infections; infections can result in an exacerbation and extreme weakness.

Ineffective Airway Clearance

The underlying causes of ineffective airway clearance for the person with myasthenia gravis include poor cough mechanism, decreased rib cage expansion, diminished diaphragm movement, and decreased expiratory effort. The following interventions require particular attention if the client undergoes a thymectomy.

- Assist with turning, deep breathing, and coughing at least every 2 hours. Teach proper coughing techniques; use an incentive spirometer every 2 hours while the client is awake. *Position changes promote lung expansion; coughing helps clear secretions from the tracheobronchial tree.*
- Place in a semi-Fowler's position. *This position expands the lungs and alleviates pressure from the diaphragm, especially important considerations if the client is obese.*
- Maintain hydration status and monitor for dehydration; use a humidifier as needed. If needed, teach family how to perform percussion, postural drainage, and suction. *Interventions to liquefy secretions, such as ensuring a daily fluid intake of up to 2500 mL (perhaps via feeding tube or parenteral route), help the client mobilize and expectorate sputum.*
- Monitor lung sounds, the rate and character of respirations, and pulse oximetry readings at least every 4 hours or as indicated by client's condition. *Frequent assessments are critical to early identification of ineffective respirations and oxygenation of tissues.*

Impaired Swallowing

Clients with myasthenia gravis have weakness of the laryngeal and pharyngeal muscles involved with swallowing. Alterations in swallowing place the client at risk for poor nutrition as well as for possible aspiration. Family members need to be included in teaching, particularly the person who prepares and assists with meals.

- Assess the ability to safely manage various consistencies of foods; consult with a speech therapist for evaluation. *Dysphagic clients are at risk for aspiration; matching food consistency to the client's ability to swallow enhances safety.*
- Plan meals to promote medication effectiveness. *Pyridostigmine should be given 30 minutes before the meal to provide optimal muscle strength for swallowing and chewing.*
- Have the client eat slowly, using small bites of food. Schedule meals during periods when the client is adequately rested; develop a daily schedule incorporating rest periods. *Fatigue may add to dysphagia, putting the client at greater risk for aspiration.*
- If necessary, give cues while eating, such as "Chew your food thoroughly; swallow." *Keeping client focused may enhance swallowing.*
- Teach caregivers the Heimlich maneuver and how to suction. *Knowing specific measures to take in case of aspiration decreases both the client's and family's anxiety and promotes confidence in managing potential problems.*

Community-Based Care

Teaching for the client and family with myasthenia gravis focuses on prevention and recognition of crisis situations,

NURSING CARE PLAN A Client with Myasthenia Gravis

Kirsten Avis, a 44-year-old homemaker and mother of two teenage sons, was diagnosed with myasthenia gravis 2 years ago. She takes an anticholinesterase medication, pyridostigmine (Mestinon), four times a day. Over the past month she has been experimenting with decreasing the dose of her pyridostigmine because she has "felt so good." She was prescribed 60 mg of pyridostigmine three times a day before meals and one-half of a long-acting 180-mg pyridostigmine tablet at night.

Three days ago, she began having chills and fever and her myasthenic symptoms became markedly worse. Mrs. Avis is easily fatigued and has been experiencing increasing weakness, bilateral ptosis, and mild dysphagia in the late afternoon and evenings. She is admitted to the hospital.

ASSESSMENT

Lela Silva, RN, is caring for Mrs. Avis. Physical examination of Mrs. Avis reveals severe muscle weakness bilaterally in her hands, arms, and thorax. Her voice is nasal, and she speaks slowly; the longer she speaks, the more difficult it becomes to understand her. She is anxious and dyspneic. Her complaints of weakness, dysphagia, dysarthria, problems with mobility, and ptosis are more pronounced later in the day. Vital signs are as follows: BP 138/88, P 88, R 28, T 102.4°F (39°C).

Some improvement in muscle weakness is noted following a restful night's sleep; however, the respiratory distress is more evident, and Mrs. Avis is increasingly restless. She is moved to the intensive care unit for advanced monitoring and possible ventilatory assistance. The medical diagnosis is myasthenic crisis secondary to pulmonary infection.

DIAGNOSES

- *Impaired Gas Exchange* related to ineffective breathing pattern and muscle weakness
- *Risk for Aspiration* related to difficulty swallowing
- *Fatigue* related to increased energy needs from muscular involvement

EXPECTED OUTCOMES

- Pulse oximetry readings will be maintained at 92% or above.
- No aspiration will occur.
- Will verbalize decreasing fatigue when performing ADLs.
- Will state the correct method of medication dosing and demonstrate how she will maintain schedule.

PLANNING AND IMPLEMENTATION

Mrs. Avis's manifestations improve following administration of edrophonium chloride (Tensilon) to verify myasthenic crisis. She is placed on oxygen by mask and suctioned as needed; equipment for possible intubation and ventilation is made readily available. She is placed in a semi-Fowler's position, and vital signs are assessed every 5 minutes during the acute exacerbation. The nurses in the intensive care unit remain in constant attendance throughout the crisis period and provide explanations to Mrs. Avis in an effort to decrease her stress and to avoid further severity of manifestations.

Three days after the crisis period, Mrs. Avis is moved to a progressive nursing care unit. Nurses follow up on teaching her the manifestations of both myasthenic and cholinergic crises. They discuss the need to wear Medic-Alert identification and review medication administration techniques with Mrs. Avis. The nurses emphasize in particular that Mrs. Avis must not split time-released medications.

Within 5 days, Mrs. Avis's condition stabilizes, and her weakness decreases sufficiently to allow discharge home. Although her temperature has returned to normal and her respiratory status has improved, she still has a productive cough. Oral antibiotics are prescribed for 2 weeks, after which she will have a follow-up visit with her primary care provider. She is instructed to seek treatment promptly if respiratory symptoms or temperature indicate recurrence of infection.

EVALUATION

Mrs. Avis is discharged without developing aspiration pneumonia or any symptoms of aspiration. Her airway was maintained throughout the myasthenic crisis, and her pulse oximetry readings remained above 92% once oxygen therapy was initiated. On discharge, pulse oximetry is above 95% without oxygen therapy. Mrs. Avis states that her fatigue and weakness have significantly improved.

Both Mrs. Avis and her husband are able to explain the difference between myasthenic and cholinergic crises and to identify methods to avoid both problems. Mrs. Avis correctly relates her proper medication regimen and makes an appointment for a follow-up visit with her physician.

CRITICAL THINKING IN THE NURSING PROCESS

1. What is the rationale for administering Tensilon to evaluate a myasthenic crisis?
2. Develop a plan to teach Mrs. Avis how to avoid fatigue when preparing and eating meals.
3. Develop a nursing care plan for Mrs. Avis for the nursing diagnosis *Ineffective Role Performance.*

See Evaluating Your Response in Appendix C.

understanding the disorder, and methods for coping with both physical and psychosocial problems. Setting realistic goals with the client and family provides opportunities for self-assessment and promotes active participation in rehabilitation.

Address the following topics:

- The importance of maintaining consistency in medication dosage and management
- Realistic expectations
- Methods to avoid fatigue and undue stress; specific measures for avoiding upper respiratory infections and exposure to extreme heat or cold
- Birth control measures or referral for counseling (Pregnancy can exacerbate manifestations; also, medications used to control myasthenia gravis, such as neostigmine bromide [Prostigmin], cross the placenta.)
- Referral to support groups
- Helpful resources such as the Myasthenia Foundation.

THE CLIENT WITH GUILLAIN-BARRÉ SYNDROME

Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating disorder of the peripheral nervous system characterized by an acute onset of motor paralysis (usually ascending). The classification of Guillain-Barré subtypes includes acute inflammatory demyelinating polyradiculoneuropathy, acute axonal motor neuropathy, and acute motor and sensory axonal neuropathy.

Guillain-Barré syndrome is one of the most common peripheral nervous system disorders, affecting about 3500 people in the United States and Canada each year (Porth, 2005). The cause is unknown, but precipitating events include a respiratory or gastrointestinal viral or bacterial infection 1 to 3 weeks prior to the onset of manifestations, surgery, viral immunizations, and other viral illnesses. In 60% of cases, *Campylobacter jejuni* is identified as the cause of the preceding infection. Approximately 80% to 90% of clients with GBS have a spontaneous recovery with little or no residual disabilities.

The disease is characterized by progressive ascending flaccid paralysis, accompanied by paresthesias and numbness. About 20% of clients have respiratory involvement to the point that ventilatory assistance is required. GBS is often a medical emergency.

Pathophysiology

The primary pathophysiologic process in GBS is the destruction of myelin sheaths covering the axons of peripheral nerves. The demyelination is thought to be the result of both a humoral- and cell-mediated immunologic response. The loss of myelin results in poor conduction of nerve impulses, causing sudden muscle weakness and loss of reflex response. Other manifestations occur when nerve conduction to various muscles is interrupted. The stages of Guillain-Barré syndrome and their usual manifestations are presented in Box 46–5.

Manifestations

Muscles, sensory nerves, and cranial nerves are commonly affected in clients with GBS. Most people experience symmetric muscle weakness, initially in the lower extremities. The weakness and sensory loss then ascends to the upper extremities, torso, and cranial nerves. Sensory involvement includes severe pain, paresthesia, and numbness. Cognition and level of consciousness are not affected. Facial nerve involvement results in the inability to change facial expressions and close the eyes. Muscles involved with chewing, swallowing, and speaking may be affected.

Paralysis of intercostal and diaphragmatic muscles may alter respiratory function. These clients require ventilatory assistance and supportive care. Involvement of the autonomic nervous system is characterized by fluctuating blood pressure, cardiac dysrhythmias and tachycardia, paralytic ileus, syndrome of inappropriate antidiuretic hormone secretion, and urinary retention.

The weakness usually plateaus or improves by the fourth week. Strength then improves slowly over weeks or months. Women who have had Guillain-Barré syndrome are at increased risk for relapse in the first trimester of pregnancy.

BOX 46–5 Stages of Guillain-Barré Syndrome

I. Acute Stage

- Characterized by severe and rapid weakness, especially in the lower extremities; loss of muscle strength progressing to quadriplegia and respiratory failure; decreasing deep tendon reflexes; decreasing vital capacity; paresthesias, numbness; pain, especially nocturnal; facial muscle involvement (inability to wrinkle forehead or change expressions).
- Involvement of the autonomic nervous system manifested by bradycardia, sweating, fluctuating blood pressure (notably hypotension) which may last for 2 weeks.

II. Stabilizing/Plateau Stage

- Occurs 2 to 3 weeks after initial onset.
- Marks the end of changes in condition; characterized by a "leveling off" of symptoms.
- Generally, the labile autonomic functions stabilize.

III. Recovery Stage

- May take from several months to 2 years.
- Marked by improvement in symptoms.
- Generally, muscle strength and function return in descending order.

INTERDISCIPLINARY CARE

Interventions during the acute phase (1 to 3 weeks) focus primarily on ensuring oxygenation via ventilatory assistance and preventing complications from immobility. Rehabilitation time to regain muscle strength and function varies; most people return to full presyndrome muscle function within 6 months to 2 years.

Care of the client with GBS requires a team approach. From the initial acute phase through rehabilitation, many members of the healthcare team are involved. An accurate and rapid diagnosis is needed to ensure prompt supportive treatment, particularly if there is respiratory involvement combined with widespread paralysis.

Diagnosis

Diagnosis of GBS is made after a thorough history and clinical examination. It must be differentiated from several disorders, among them influenza, heavy metal poisoning, Lyme disease, and cranial hemorrhage. Diagnosis is made based on manifestations, history of a recent viral infection, elevated CSF protein levels, and EMG studies reflecting decreased nerve conduction. Although there is no specific test to diagnose this syndrome, several findings support and confirm the diagnosis.

Medications

No medications are available for the specific treatment of Guillain-Barré syndrome. Other medications may be prescribed to provide support or prophylaxis, or to combat concurrent problems; for example, antibiotics may be prescribed for urinary tract or respiratory infections. Morphine is commonly administered to control muscle pain. Anticoagulation therapy is usually instituted to prevent thromboembolic complications, such as deep venous thrombosis and pulmonary

embolism, which are associated with prolonged bed rest. If hypotension is a problem, vasopressors are prescribed.

Surgery
Tracheostomy is performed if respiratory failure occurs. Clients who need ventilatory support are usually able to be weaned after 2 to 3 weeks, but the time frame varies greatly. When the client's vital capacity reaches 8 to 10 mL/kg, he or she may be weaned from the ventilator (Hickey, 2003). Insertion of a temporary pacemaker may be indicated for bradycardia.

Plasmapheresis
Plasma exchange has been beneficial, particularly when performed within the first 2 weeks of the syndrome's development. Antibodies are removed, and immunosuppressive agents are administered concurrently. Clients typically have five exchanges during an 8- to 10-day period.

Nutrition and Fluids
Nutritional support for the client who is immobilized for prolonged periods of time is crucial. Maintaining positive nitrogen balance, ensuring sufficient fluid intake and electrolyte balance, and ensuring recommended caloric intake are goals of therapy. When swallowing problems occur, total parenteral nutrition may be indicated if feeding via a nasogastric or gastrostomy tube is ineffective.

Physical and Occupational Therapy
Long-term physical and occupational therapy are crucial to recovery. Clients with Guillain-Barré syndrome usually require prolonged rehabilitation care, which begins during the acute phase and focuses on preventing complications and limiting the effects of immobility. The severe muscle atrophy and loss of muscle tone require that clients relearn many functions and skills, such as walking. Compromise in respiratory function may delay physical rehabilitation; clients need positive reinforcement when they make even small gains in their progress. Continued attention to pain control is essential because paresthesia and pain can interfere with physical therapy.

NURSING CARE
Many of the nursing interventions for clients with this syndrome involve monitoring neurologic function, preventing problems of immobility, ensuring adequate hydration and nutrition, and promoting respiratory function. Anticipating needs of both the client and family is an important aspect of care. For example, developing an alternative method of communication before it is necessary may decrease anxiety. It is important that nursing care focus on preventing complications that may be fatal by following a rigorous predetermined schedule for turning and respiratory care (e.g., coughing, deep breathing, suctioning), using strict aseptic technique, and providing continuous psychosocial support.

Nursing Diagnoses and Interventions
Anxiety and powerlessness are major nursing considerations. The client is almost always admitted to the ICU for care, and is mentally alert but suddenly mute, ventilator dependent, and immobile. Refer to previous nursing care sections in this chapter for interventions related to anxiety, imbalanced nutrition, impaired swallowing, impaired verbal communication, and ineffective airway clearance. This section focuses on the nursing diagnoses related to pain and risk for impaired skin integrity.

Acute Pain
Pain experienced with Guillain-Barré syndrome varies. Frequently, there is a "stocking–glove" pattern, with pain in the hands, feet, and legs. Pain and tenderness in muscles can be severe; interventions must be individualized to client needs. The intense pain combined with altered sensations leads to anxiety; nursing interventions can make a difference in breaking the cycle of increasing pain that leads to increased anxiety and in turn causes more pain.

- Listen to the description of pain; determine presence of triggers or a pattern. *Acknowledging the client's perception of pain is a basis for treatment; listening establishes trust.*
- Use a pain scale for determining extent of pain. *Consistent measurement is essential to evaluate degree of pain and effectiveness of intervention.*
- Use complementary therapies to help manage pain:
  - Application of heat/cold
  - Guided imagery
  - Relaxation techniques
  - Massage.

  Presenting options for managing pain gives the client control over the situation and helps reduce anxiety. Noninvasive interventions may augment the therapeutic benefit of medications.
- Provide analgesics as indicated; administer on a regular schedule rather than waiting until pain becomes severe. *Anticipating and managing pain before it becomes severe decreases anxiety and averts the cycle of increased anxiety leading to increased pain.*
- Monitor for side effects of analgesics, particularly respiratory depression; assess respirations and lung sounds. Perform routine pulmonary care measures and monitor for aspiration. *Clients with Guillain-Barré syndrome have weakened thoracic muscles; frequent respiratory monitoring is indicated.*

Risk for Impaired Skin Integrity
During the acute and plateau stages of Guillain-Barré syndrome, clients are at risk for problems related to immobility and malnutrition. Impaired skin integrity is one such problem. Preventing areas of skin breakdown is important. Prophylactic interventions will help ensure that ingested protein and calories are used to maintain ideal body weight and other body functions rather than to heal an avoidable problem. Implicit in interventions is maintenance of adequate nutrition.

- Inspect bony prominences and provide skin care at least every 2 hours. Reposition the client and clean, dry, and lubricate the skin as needed. *These activities stimulate circulation and ensure even distribution of body weight; baseline observations allow discovery of early signs of altered integrity.*

MEDIALINK Care Plan Activity: Guillain-Barré Syndrome

- Pad bony prominences, such as sacral area, heels, and elbows. *This decreases shearing tears on these pressure points.*
- Use an alternating-pressure mattress or water bed. *Relieving pressure stimulates circulation and promotes oxygenation of tissues.*
- Monitor for incontinence and provide thorough skin care following each episode of incontinence. *Urine is caustic to the skin, and the moisture promotes skin breakdown.*

Community-Based Care

Clients and family members are frequently stunned by the rapid deterioration of function and fear that the paralysis will be permanent. Regularly reinforce teaching because the client's high anxiety level may interfere with listening and understanding. When possible, include the client and family in decision making; for example, seek their input when planning a daily schedule of care that incorporates various therapies.

Teaching the rationales for preventive measures reinforces the client's and family's understanding and may promote compliance during the lengthy rehabilitation. For example, because of autonomic nerve involvement, clients need to be monitored for cardiac dysrhythmias and taught to avoid changing position suddenly to prevent orthostatic hypotension.

Referrals to appropriate therapists are a component of anticipating needs; speech, nutritional, occupational, and physical therapists are an integral part of rehabilitation. Another focus of care is teaching both the client and family; incorporate explanations for interventions aimed at promoting self-care. For further information, refer the client and family to the Guillain-Barré Syndrome Foundation, International.

CRANIAL NERVE DISORDERS

Disorders of the cranial nerves may be caused by intracranial trauma or by pathologic processes. The pairs of cranial nerves, described in Chapter 43 ∞, are numbered in the order in which they arise in the brain and are named according to their anatomic characteristic or primary function. The most common cranial nerve disorders are those affecting the trigeminal (cranial nerve V) and the facial (cranial nerve VII) nerves. These disorders, discussed in the following sections, result primarily in pain or loss of sensory or motor function.

THE CLIENT WITH TRIGEMINAL NEURALGIA

Trigeminal neuralgia, also called *tic douloureux,* is a chronic disease of the trigeminal cranial nerve (V) that causes severe facial pain. The trigeminal nerve has three divisions: the ophthalmic, the maxillary, and mandibular (Figure 46–7 ■). The ophthalmic division supplies the forehead, eyes, nose, temples, meninges, paranasal sinus, and part of the nasal mucosa. The maxillary division supplies the upper jaw, teeth, lip, cheeks, hard palate, maxillary sinus, and part of the nasal mucosa. The mandibular division supplies the lower jaw, teeth, lip, buccal mucosa, tongue, part of the external ear, and the meninges. Sensory fibers of the nerve conduct impulses for touch, pain, and temperature; motor fibers innervate the temporal and masseter muscles used for chewing and lateral movement of the jaw. The maxillary and mandibular divisions are the divisions of the trigeminal nerve affected in almost all cases of this disorder.

Trigeminal neuralgia occurs more commonly in middle-age and older adults and affects women more often than men.

Pathophysiology

The actual cause of trigeminal neuralgia is unknown; however, contributing factors include irritation from flulike illnesses, trauma or infection of the teeth or jaw, and pressure on the nerve by an aneurysm, a tumor, or arteriosclerotic changes of an artery close to the nerve (Hickey, 2003).

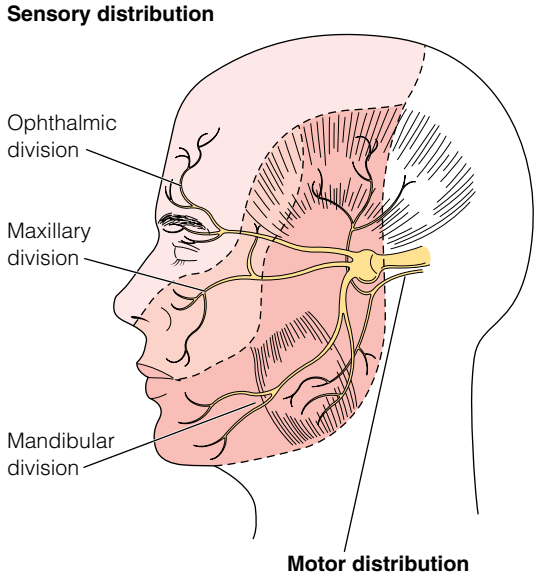


Figure 46–7 ■ Sensory and motor distribution of the trigeminal nerve. The three sensory divisions are ophthalmic, maxillary, and mandibular.

Stimulating specific areas of the face, called *trigger zones,* may initiate the onset of pain. These trigger zones usually parallel the distribution of the nerve and typically follow a track leading from just over the eyebrow to the ridge of the cheekbone, along the nasolabial fold, around the corner of the mouth, and down the side of the chin. The episodes of pain are initiated by many factors, including light touch, eating, swallowing, talking, sneezing, shaving, chewing gum, brushing the teeth, or washing the face. Other factors that may trigger a pain episode include changes in temperature and exposure to wind. In an attempt to control the pain, clients may refuse to wash, shave, eat, or talk.

The episodes of pain may recur for several weeks or months. The disease then spontaneously goes into remission, and the client is free of pain for periods lasting from days to years. As

the client grows older, the remissions tend to become shorter, and a dull ache may be present between episodes of acute pain.

Manifestations

Trigeminal neuralgia is characterized by brief (lasting a few seconds to a few minutes), repetitive episodes of sudden severe (usually unilateral) facial pain. The pain may occur as often as hundreds of times a day to as infrequently as a few times a year. The pain is experienced over the surface of the skin. It most often begins near one side of the mouth and rises toward the ear, eye, or nostril on the same side of the face. Clients describe the pain as stabbing or lightning-like and often respond to the pain by wincing or grimacing.

INTERDISCIPLINARY CARE

There are no specific diagnostic tests for trigeminal neuralgia. The disorder is diagnosed by the characteristic location and type of pain. The disorder is treated by pharmacologic or surgical interventions.

Medications

The drug most useful in controlling the pain is the tricyclic anticonvulsant carbamazepine (Tegretol). If carbamazepine is ineffective, other medications such as the anticonvulsants phenytoin (Dilantin) or gabapentin (Neurontin) or the skeletal muscle relaxant baclofen (Lioresal) may be used. These drugs are administered to decrease paroxysmal afferent impulses and stop the pain. Drugs in this category may cause side effects of dizziness, nausea, and drowsiness. Liver function, bone marrow function, and blood levels of the medications should be monitored on a regular basis.

Surgery

If medications do not control the pain, surgical procedures may be performed, including various types of *rhizotomy,* the surgical severing of a nerve root. Closed surgical interventions by percutaneous rhizotomy involve inserting a needle through the cheek into the foramen ovale at the base of the brain and partially destroying the trigeminal nerve with glycerol (an alcohol), by radiofrequency-induced heat, or by balloon compression of the trigeminal ganglion. These procedures carry less risk and result in shorter hospital stays than do open procedures, but there is a possibility of recurrence of pain. Following surgery, the client may have some facial numbness, but there usually is no residual paralysis. The involved side of the face is insensitive to pain. The client will have some loss of facial sensation (e.g., to temperature and/or touch) and is at risk for loss of the corneal reflex. Closed procedures provide long-term pain relief and are well tolerated by the older adult. Nursing care of the client undergoing a percutaneous rhizotomy is presented in the box below.

It has been found that some structural abnormalities (such as an artery or vein compressing the nerve) may cause the neuralgia, and if so, decompression and separation of the blood vessel from the nerve root produce lasting relief of the pain (Tierney et al., 2005). The Jannetta procedure involves locating and lifting the involved vessel and placing a small piece of silicone sponge between the vessel and the nerve. Possible complications of the procedure include headache and facial pain.

NURSING CARE

Nursing care for the client with trigeminal neuralgia involves teaching self-management at home after medical or surgical intervention. Primary client concerns are managing pain, maintaining nutrition, and preventing injury.

Nursing Diagnoses and Interventions

Interventions for managing pain and improving nutritional intake are addressed here; teaching to prevent injury following surgery is discussed under client and family teaching.

Acute Pain

The client with trigeminal neuralgia has excruciating pain and often avoids ADLs and socializing with others in an attempt to

NURSING CARE OF THE CLIENT HAVING A Percutaneous Rhizotomy

POSTOPERATIVE CARE

- Follow routine postoperative interventions for clients having surgery (see Chapter 4 ∞).
- Monitor cranial nerve function every 2 to 4 hours:
  a. Assess the corneal reflex by lightly touching the cornea with a wisp of cotton. If the reflex is intact, the client will blink. *Severing the ophthalmic division of the trigeminal nerve destroys the corneal reflex and leaves the cornea at risk for dryness and injury.*
  b. Assess the facial nerve by asking the client to blow out the cheeks, wrinkle the forehead, frown, wink, and close both eyes tightly. Test taste by placing bitter, salty, and sweet substances on the anterior portion of the tongue. *Facial weakness is evidenced by changes in movement in the involved side of the face. The facial nerve also innervates the anterior two-thirds of the tongue.*
  c. Assess the function of the oculomotor muscles by asking the client to follow your finger through the cardinal positions of vision (see Chapter 47 ∞). *The eyes should move together; alterations in movement indicate an abnormal response.*
  d. Assess the motor portion of the trigeminal nerve by asking the client to clench the teeth while you palpate the tightness of the contracted masseter and temporal muscles. *Loss of motor function is indicated by loss of bulk and tightness of these muscles.*
  e. Apply as prescribed; an ice pack to the jaw on the operative site. *Cold decreases bleeding and swelling.*
  f. Teach the client to avoid rubbing the eye on the involved side. *Loss of the corneal reflex removes protection because the client no longer has the sensation of pain in the involved eye. Rubbing the eye could cause corneal abrasions.*

prevent the onset of pain. Pain management is fully discussed in Chapter 9 ∞. Nursing interventions for pain in clients with this disorder focus on strategies for self-management.

- Identify factors that trigger an attack, and discuss strategies to avoid these precipitating factors. *Most clients can clearly identify trigger zones and triggering factors. Identification is the first step in pain control.*
- Determine usual response to pain. *Sensitivity and reaction to pain are influenced by previous experiences with pain and by age, gender, emotional factors, and cultural background.*
- Assess factors that affect the ability to influence pain tolerance, including the knowledge and cause of the pain, the meaning of the pain, the ability to control the pain, cultural background, and support systems. *Pain tolerance, which is the duration and intensity of pain a person is willing to endure, differs greatly among individuals and may also vary within particular clients in different situations.*
- Monitor the effects of the medication prescribed for the neuralgia. *If the prescribed medication does not provide relief, other medications or methods of treatment may be used to control the pain.*

Risk for Altered Nutrition: Less than Body Requirements

Clients often refuse to eat during periods of pain attacks, fearing that the movements of chewing may precipitate the pain. In addition, the chronic nature of the illness often causes depression, which may depress the appetite.

- Monitor dietary intake and weight at each visit, and ask the client to keep a weekly weight record. *Ongoing assessments are necessary for early detection of nutritional deficiencies.*
- Discuss the temperature and consistency of foods eaten, and suggest referral to a dietitian if necessary. *Hot or cold foods may trigger an attack; soft, warm, or cool foods are less likely to act as triggers.*
- Suggest chewing on the unaffected side of the mouth. *Chewing on the unaffected side is less likely to trigger an attack of pain and so facilitate food intake.*
- If unable to tolerate oral food, tube feedings may be necessary. *Adequate kilocalories and nutrients for metabolic processes are essential.*

Community-Based Care

The client with trigeminal neuralgia who is receiving medical treatment and providing self-care at home requires teaching about the disease process, the medication(s) being taken, and ways to reduce the incidence of attacks or pain. Diet teaching and assistance with self-management of pain are also important. For example, if the home setting is drafty and attacks of pain are triggered by wind blowing across the face, it may be necessary to encourage the client to put weather stripping around windows and doors. To prevent injury to affected areas, the topics in the Meeting Individualized Needs box below should be addressed.

THE CLIENT WITH BELL'S PALSY

Bell's palsy, also called *facial paralysis,* is a disorder of the seventh cranial (facial) nerve, characterized by unilateral paralysis of the facial muscles. The facial nerve is primarily a motor nerve that supplies all the muscles associated with expression on one side of the face. The sensory component innervates the anterior two-thirds of one side of the tongue.

This disorder can occur at any age but is seen most often in adults between 20 and 60. The incidence is equal in men and women. Eighty percent of clients recover completely within a few weeks to a few months (and three-fourths recover without any treatment). Of those remaining, 15% recover some function but have some permanent facial paralysis; these clients are usually older, have diabetes mellitus, or have more severe manifestations, such as vertigo, a sensitivity to noise, and deep head pain.

Pathophysiology

The exact cause of the disorder is unknown, although inflammation of the nerve and a relationship to the herpes simplex virus have been suggested (Tierney et al., 2005).

Manifestations

The onset of Bell's palsy is usually sudden and almost always involves one side of the face. Pain behind the ear or along the jaw may precede the paralysis. The client initially notices

MEETING INDIVIDUALIZED NEEDS **Teaching for Home Care of Trigeminal Neuralgia**

EYE CARE

- Do not rub the eyes; use artificial tears four times a day if the eyes are dry or irritated.
- Wear an eye patch at night.
- Wear protective sunglasses or goggles when outside, when working in dusty areas, when mowing the lawn, and when using any type of spray material (e.g., hair spray, cleaning materials, paint, insecticides).
- Remember to blink frequently.
- Check your eyes for redness or swelling each day.
- Schedule regular eye examinations.

FACE AND MOUTH CARE

- Chew on the unaffected side of the mouth.
- Avoid eating hot foods or drinking hot liquids.
- After every meal, brush your teeth and inspect the inside of your mouth for food that may collect between the gums and cheek.
- Have regular dental examinations; you will not be able to feel pain associated with gum infection or tooth decay.
- Use an electric razor to shave the face.
- Protect your face from very cold or windy conditions.

numbness or stiffness of one side of the face that distorts the appearance. As the disease progresses, the distortion becomes more obvious, and the face appears asymmetric. The facial paralysis causes the entire side of the face to droop, and the client cannot wrinkle the forehead, close the eye, or pucker the lips on the affected side. When the client attempts to smile, the lower facial muscles are pulled to the opposite side of the face. Some clients have only mild manifestations, whereas others have complete facial paralysis (Figure 46–8 ■). Clients often believe they have had a stroke. Manifestations of Bell's palsy are listed in the box on this page.

INTERDISCIPLINARY CARE

There are no definitive laboratory or diagnostic tests for Bell's palsy, nor are there any specific treatments. Treatment includes medications and physical therapy. Recent studies have shown that antiviral drugs such as acyclovir combined with an anti-inflammatory drug such as prednisone may be effective by limiting damage to the nerve. Physical therapy to stimulate the facial nerve and help maintain muscle tone may help prevent permanent contractures before recovery takes place. Moist heat applied to the affected side of the face may decrease pain.

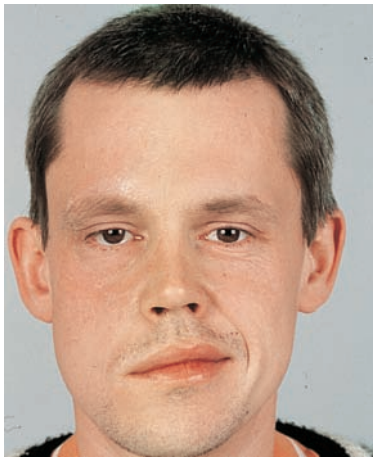


Figure 46–8 ■ The client with Bell's palsy shows the typical drooping of one side of the face.

Source: NIH/Phototake NYC.

MANIFESTATIONS of Bell's Palsy

- Paralysis of the facial muscles on one side of the face
- Paralysis of the upper eyelid with loss of the corneal reflex on the affected side
- Loss or impairment of taste over the anterior portion of the tongue on the affected side
- Increased tearing from the lacrimal gland on the affected side

NURSING CARE

Although clients provide self-care at home, the nurse plays a key role in teaching the client and family about the disease and how to prevent injury and maintain nutrition. The client is often anxious about his or her appearance and may require counseling if any deficits in facial expression become permanent. The following topics should be addressed:

- Use artificial tears four times a day to lubricate the eye; wear an eye patch or tape the eye shut at night. Wear sunglasses or goggles when outside, when working in dusty conditions, and when using any type of spray.
- Massage combined with warm, moist heat often is effective in relieving the pain.
- A soft diet that does not require chewing and six small meals a day are helpful. Chew slowly on the unaffected side and avoid hot foods. Clean the mouth and carefully inspect the area between the gums and cheek for food after each meal.
- As function returns, practice wrinkling the forehead, closing the eyes, blowing air out of the puckered mouth, and whistling for 5 minutes three or four times a day.

DISORDERS RESULTING FROM INFECTIONS AND NEUROTOXINS

A variety of disorders of the nervous system may have infectious or toxic causes. Although these disorders are not common, those included here require significant nursing care when they do occur.

THE CLIENT WITH CREUTZFELDT-JAKOB DISEASE

Creutzfeldt-Jakob disease *(CJD)* (also called *spongiform encephalopathy*) is a rapidly progressive, degenerative, neurologic disease that causes brain degeneration without inflammation. The disease is transmissible and progressively fatal. The causative agent is believed to be an abnormal form of a cellular glycoprotein known as the prion protein. Transmission of the agent is by direct contamination with infected neural tissue, such as during eye and brain surgery. The injection of contaminated human growth hormone from cadaveric pituitaries has also been implicated.

A different form of the disease, called *new variant CJD (vCJD)* is also a rare, degenerative, fatal brain disorder, but is not the same as the classic form of CJD. New variant CJD, referred to as "mad-cow disease," is believed to result from consumption of cattle products contaminated with bovine spongiform encephalopathy (BSE). This form primarily affects young adults. Because the illness is fatal and is associated with infected cattle, severe restrictions have been placed on the importation of cattle, sheep, and goats and on products from these animals from countries in which BSE is known to exist.

The disease occurs worldwide, but clusters occur in several areas, more often in England, Chile, and Italy. The incidence is approximately 1 or 2 cases per 1 million population (Centers for Disease Control and Prevention [CDC], 2005). Classic CJD affects adults over the age of 50; vCJD affects younger adults. The median age of death for people with classic CJD is 68 years. In contrast, the median age of death with vCJD is 28 years.

Pathophysiology

Creutzfeldt-Jakob disease is characterized by degeneration of the gray matter of the brain. The spongiform degeneration (involving the formation of tiny holes and resembling a sponge) produces severe dementia, myoclonus (muscle contractions), and characteristic changes in brain waves. On autopsy or biopsy of brain tissue, the brain shows loss of neurons and a proliferation of astrocytes (indicating destruction of nearby neurons).

Manifestations

The disease has characteristic stages and manifestations. The onset is characterized by memory changes, an exaggerated startle reflex, sleep disturbances, and nervousness. The person then experiences rapid deterioration in motor, sensory, and language function. Tremors, hyperreflexia, rigidity, and a positive Babinski reflex are often present, and confusion progresses to dementia in almost all cases. Clients in the terminal state are comatose and exhibit decorticate and decerebrate posturing. The median duration of illness for CJD is 4 to 5 months; the median duration of illness for vCJD is 13 to 14 months (CDC, 2005).

INTERDISCIPLINARY CARE

No specific treatment is available to stop or slow the progression of CJD. Collaborative interventions focus on the disease's manifestations. The disease is diagnosed by a thorough neurologic examination, specific EEG changes, and a CT scan. However, the final diagnosis of CJD can be made only by postmortem examination. It is often difficult to differentiate this disease from Alzheimer's disease, especially in the early stages.

NURSING CARE

The nurse may identify the manifestations of Creutzfeldt-Jakob disease when conducting a health history and total physical assessment. Include questions about familial history, cultural and geographic risk, and high-risk occupations or procedures in the history. Assessment of mental function, reflexes, and cranial nerve function may provide information to assist in diagnosis.

Nursing care focuses on maximizing comfort, preventing injury, preventing transmission, and providing support. The following guidelines are useful in designing the plan of care:

- Although comfort is difficult to assess in clients with impaired cognitive function, interventions that provide a quiet environment and analgesia are important.
- Communication is essential, even if the client is unable to respond.
- Institute seizure precautions, and pad side rails.
- Provide skin care, changes in position, and pressure-relief mattresses to decrease the risk of pressure ulcers, venous stasis, and pneumonia.
- Use standard precautions for blood and body fluids when providing care. Disinfect surfaces with a solution of 5% bleach. Sterilize contaminated equipment by autoclave, or soak in 5% bleach solution for 1 hour. Label all specimens as biohazardous. Teach staff members and family members guidelines for care, including careful hand washing. It is not necessary to place the client in isolation, however.
- Provide time for family members to verbalize grief and loss, which may be manifested as anger and frustration with the healthcare system.
- Provide information to family members about all procedures and the plan of care.
- Refer family members to sources of support, such as social services and the appropriate clergy.

THE CLIENT WITH POSTPOLIOMYELITIS SYNDROME

Postpoliomyelitis syndrome is a complication of a previous infection by the poliomyelitis virus. This disease was epidemic in the 1940s and 1950s, but has largely been eradicated through immunization with oral live trivalent virus vaccine. However, it is thought that nearly 50% of the estimated 1.63 million people in the United States who had the disease are reexperiencing manifestations of the acute illness (NINDS, 2005g). These people have struggled for years to rehabilitate themselves and lead productive lives. Now, as they reach retirement age, they are again experiencing manifestations which may be physically and psychologically incapacitating.

The poliomyelitis virus destroys some of the motor cells of the anterior horn cells of the spinal cord, causing neuromuscular effects that range from mild to severe flaccid paralysis and atrophy. The primary cause of death is respiratory arrest (Tierney et al., 2005).

Manifestations of motor neuron degeneration and weakness may emerge 10 to 40 years after the initial infection. Most clients with postpoliomyelitis syndrome initially had a more severe case of polio and required hospitalization, contracted the disease after the age of 10, required ventilator assistance for respiration, and had paralysis in all four extremities. The incidence is slightly higher in women. As the population ages, it is projected that the number of older adults with postpoliomyelitis syndrome will increase.

Pathophysiology

The pathophysiologic process in postpoliomyelitis syndrome is not known.

Manifestations

The manifestations of postpoliomyelitis syndrome include fatigue, muscle and joint weakness, loss of muscle mass, respiratory difficulties, and pain. The manifestations are most often seen in muscles affected by the initial infection, but new muscle groups may also be affected. In addition to neuromuscular manifestations, the client may experience cold intolerance, dizziness, headaches, urinary incontinence, and sleep disorders.

INTERDISCIPLINARY CARE

Postpoliomyelitis syndrome is diagnosed by a previous history of polio and the current manifestations. Diagnostic studies of nerve conduction, muscle strength, and pulmonary function determine current physical status. Treatment addresses the manifestations, and often involves physical therapy and pulmonary rehabilitation programs.

NURSING CARE

The client with postpoliomyelitis syndrome faces the challenge of unexpected physical changes. Clients are often anxious about how others will react or what the future holds. Respiratory dysfunction may result in the need for oxygen. Muscular weakness and decreased pulmonary function may make walking difficult, if not impossible. Activities of daily living, independent self-care, and careers are threatened.

Many clients have not fully recovered psychologically from having polio and may respond to a recurrence of manifestations with denial and disbelief. Older clients may not know they had polio as children. Nurses are responsible for assessing and identifying the manifestations of postpoliomyelitis syndrome. It is essential to question middle to older adults about a past history of polio when conducting the health history and to ask specific questions about manifestations that the client may be experiencing.

The nurse individualizes teaching to meet the physical and psychosocial needs of the client and family. Provide candid explanations, and teach the client how to prevent fatigue, promote optimal respiratory function, meet self-care needs, modify ADLs, and maintain safety. Follow-up care with nurses, physicians, physical therapists, respiratory therapists, and counselors is indicated. Referral to a support group can make a positive difference in the client's and family's ability to cope with the disorder.

THE CLIENT WITH RABIES

Rabies is a rhabdovirus infection of the central nervous system transmitted by infected saliva that enters the human body through a bite or an open wound. If untreated, rabies is a fatal viral encephalitis that causes 30,000 to 70,000 deaths worldwide each year. In the United States, 25,000 to 40,000 people are treated annually for exposure to rabid or potentially rabid animals (Hankins & Rosekrans, 2004). This is a critical illness that almost always causes death if untreated. The rabies virus is carried by both wild and domestic animals, including bats, skunks, foxes, raccoons, cats, and dogs. After an incubation period that may last from 10 days to many years (norm is 3 to 7 weeks), the virus travels to the brain of the infected animal via the nerves. It multiplies and migrates to the salivary glands.

Pathophysiology

The client with rabies usually has a history of an animal bite but may also become infected through an abrasion or open wound that is exposed to the infected saliva. The virus spreads from the wound to local muscle cells and then invades the peripheral nerves. It eventually travels to the central nervous system. The incubation period in humans varies according to the severity and location of the bite. For example, bites on the face may result in manifestations in 10 days to a few weeks, whereas bites on the lower extremities may incubate for as long as 1 year.

Manifestations

The manifestations occur in stages. During the initial, or prodromal, stage, the site of the wound is painful and then exhibits various paresthesias. The infected person is anxious, irritable, and depressed. General manifestations of infection (such as headache, loss of appetite, and sore throat) may appear. The person may also have increased sensitivity to light and sounds, and the skin is especially sensitive to changes in temperature.

The prodromal stage is followed by an excitement stage. The infected person has periods of excitement that alternate with periods of quiet. Attempts to drink cause such painful laryngospasms that the person refuses to drink (a phenomenon called *hydrophobia*). Large amounts of thick, tenacious mucus are present. The client experiences convulsions, muscle spasms, and periods of apnea. If untreated, death occurs approximately 7 days from the onset of manifestations and is usually due to respiratory failure.

INTERDISCIPLINARY CARE

Animals that bite are kept under observation, if possible, for 7 to 10 days to detect rabies manifestations. Sick animals should be euthanized and their brains examined for presence of the rabies virus, which is detected by fluorescent antibody testing. The blood of an infected person can also be tested with the same diagnostic study to demonstrate the presence of rabies antibodies.

NURSING CARE

Nursing care for clients with rabies is provided in an intensive care unit, with the client in a quiet, darkened room to decrease stimulation as much as possible. The client requires interventions to maintain the airway, maintain oxygenation, and control seizures. Standard precautions are essential, because the rabies virus is present in the saliva of the client. If an open wound of a healthcare provider is contaminated with infected saliva, the provider must receive postexposure immunizations.

Health Promotion

Client and family teaching focuses on the importance of immunizing pets, providing proper care of wounds, seeking immediate medical attention for animal bites, and obtaining treatment after any suspicious bite.

Because the untreated disease is almost always fatal, the best intervention is prevention. Preventive activities follow:

- Immunize household dogs and cats; immunize people who are exposed to animals.
- Local treatment of animal bites and scratches:
  - Carefully and thoroughly clean and flush wounds with soap and water to remove the saliva and dilute the viral exposure.
  - Immediately take the person with the bite for emergency treatment.
- Postexposure care:
  - Rabies immune globulin (RIG) is administered for passive immunization. Up to 50% of the globulin is infiltrated around the wound, and the rest is administered intramuscularly. At the same time, an inactivated human diploid cell vaccine (HDCV) is administered intramuscularly, with 1 mL given on the day of exposure and on days 3, 7, 14, and 28 after exposure (Tierney et al., 2005). RIG and HDCV should never be given in the same syringe or at the same site. Local and mild systemic reactions include itching, tenderness, headaches, muscle aches, and nausea.
  - If RIG is not available, equine rabies antiserum may be administered after testing the client for horse serum sensitivity.

THE CLIENT WITH TETANUS

Tetanus, more commonly called *lockjaw,* is a disorder of the nervous system caused by a neurotoxin elaborated by *Clostridium tetani.* This anaerobic bacillus lives in the soil. Spores of the bacillus enter the body through open wounds contaminated with dirt, street dust, or feces (animal or human). The wounds may result from punctures, scratches or abrasions, bee stings, abortions, surgery, trauma, burns, or intravenous drug use. Incidence is highest among people who have never been immunized, older adults whose immunity has been lost, and women. The majority of cases occur in people over age 50. Tetanus has a high mortality rate, with death occurring in over 40% of all cases. Contaminated lesions of the head and face are more dangerous than those in other parts of the body.

Pathophysiology

When the spores of *C. tetani* enter the open wound, they germinate and produce a toxin called tetanospasmin. The incubation period averages 8 to 12 days but can range from 5 days to 15 weeks (Tierney et al., 2005). The toxins are absorbed by the peripheral nerves and carried to the spinal cord, where they block the action of inhibitory enzymes at spinal synapses and interfere with transmission of neuromuscular impulses. As a result, even minor stimuli cause uncontrolled muscle spasms.

Manifestations

The manifestations begin with pain at the site of the infection. The infected person has stiffness of the jaw and neck and dysphagia. There is often profuse perspiration and drooling from increased salivation. As the infection progresses, the person experiences hyperreflexia, spasms of the jaw muscles (*trismus*) or facial muscles, and rigidity and spasms of the abdominal, neck, and back muscles. Generalized tonic seizures are caused by even minor stimuli, and the person assumes a typical opisthotonic position during the seizures: The head is retracted, the back is arched, and the feet are extended. The muscle spasms are painful. The person may be unable to breathe from spasms of the glottis and respiratory muscles. Despite these physical effects, the client has no change in mental status.

The complications of tetanus include urinary retention and airway obstruction from the spasms. Cardiac and respiratory failure are late, life-threatening complications.

INTERDISCIPLINARY CARE

There are no specific diagnostic tests for tetanus; diagnosis is based on manifestations. Tetanus is completely preventable by active immunization. Immunization for children includes tetanus toxoid, administered as part of the diphtheria-pertussis-tetanus (DPT) immunization series. In adults, immunization is obtained by administering tetanus toxoid as two doses 4 to 6 weeks apart, with a third dose in 6 to 12 months. All individuals should have a booster dose every 10 years throughout life or at the time of a major injury if the last booster dose was given more than 5 years prior to the injury.

If a wound is contaminated or if the person's immunization status is uncertain, passive immunization with tetanus immune globulin is administered. Active immunization with tetanus toxoid is begun at the same time. The wound is carefully and thoroughly debrided and antibiotics administered.

The client with tetanus requires intensive care in an area of minimal stimulation. Penicillin is administered to help destroy the toxin-producing organism. Muscle spasms and seizures are controlled by chlorpromazine (Thorazine) or diazepam (Valium), often combined with a sedative. Anticoagulants may be prescribed to prevent venous thrombosis. In severe cases, seizures and spasms are controlled with paralysis by a curare-like medication, and airway obstruction is managed by mechanical ventilation.

NURSING CARE

Nursing care for the client with tetanus is intensive and focuses on assessments and interventions to promote safety, prevent injury, maintain nutrition, and maintain pulmonary and cardiovascular function. The client usually requires in-hospital care for 2 to 5 weeks. The nursing care plan commonly includes the following:

- Place in a quiet, darkened room to decrease stimuli that cause muscle spasms and seizures.
- Provide only necessary physical care, and do so during periods of maximal sedation to decrease tactile stimulation that causes muscle spasms.
- Maintain oxygenation through mechanical ventilator and frequent suctioning of secretions.
- Maintain intravenous access for the administration of fluids and medications.
- Administer prescribed antibiotics, anticonvulsants, and sedatives. In the case of cardiovascular complications, administer prescribed beta-adrenergic blocking agents such as propranolol (Inderal).
- Provide adequate nutrition through prescribed nutritional support, such as total parenteral nutrition.
- Monitor respiratory and cardiovascular status and provide immediate interventions for respiratory or cardiovascular failure.
- Monitor fluid and electrolyte status. Ensure adequate fluid intake to maintain hydration and urinary output.
- Monitor urinary output, which should be maintained at 1.5 to 2 L per day.
- Monitor for the hazards of immobility, including constipation, pneumonia, deep venous thrombosis, and pressure ulcers.

Health Promotion

Tetanus is a preventable disorder, and nurses have a major role in promoting immunizations for all children and for educating adults about the need for booster doses. The older population is especially at risk for never having been immunized or for letting immunizations lapse. Information for this age group can be provided through activities such as community health fairs and programs at senior citizen groups.

It is also necessary to teach the proper care of wounds. All wounds, no matter how small, should be thoroughly washed with soap and water. All foreign material should be carefully flushed out or removed from a wound, and medical care should be sought for wounds that are more extensive or contaminated.

THE CLIENT WITH BOTULISM

Botulism is food poisoning caused by ingestion of food contaminated with a toxin produced by the bacillus *Clostridium*

botulinum. This anaerobic spore-forming bacillus is found in the soil. Most cases of botulism occur from eating improperly canned or cooked foods, especially home-canned vegetables and fruits, smoked meats, and vacuum-packed fish. The mortality rate is high if the disease is untreated.

Pathophysiology

The toxins liberated by *C. botulinum* are absorbed by the gastrointestinal tract and bound to nerve tissues. They block the release of acetylcholine from nerve endings and thus cause respiratory paralysis due to paralysis of skeletal muscles.

Manifestations

Manifestations appear 12 to 36 hours after ingestion of the contaminated food. They usually begin with visual disturbances such as diplopia, loss of accommodation, and fixed, dilated pupils. Ptosis is often present. Gastrointestinal manifestations include nausea and vomiting, diarrhea, dysphagia, and dry mouth. Involvement of the larynx is manifested by dystonia (impaired muscle tone). Paralysis of all muscle groups progresses throughout the body, with respiratory paralysis causing death if the client is not placed on a mechanical ventilator. There is no effect on mental status.

INTERDISCIPLINARY CARE

Infection with the *Clostridium* toxin is verified by laboratory analysis of the serum and stool and of suspected food, if possible. If botulism is suspected, the state health department and the CDC should be notified for assistance with laboratory assays and procuring botulism antitoxin. All people who may have eaten the contaminated food must be located and observed.

Any toxins in the gastrointestinal system are removed by cathartics, enemas, and gastric lavage. The client with respiratory paralysis is placed on a mechanical ventilator and may require a tracheostomy. Botulism antitoxin is administered to eradicate toxins in the circulation. Nutritional support is often provided with total parenteral nutrition. Intravenous fluids are administered to prevent dehydration and renal failure. If ventilation can be maintained, the client often recovers without further neurologic deficits.

NURSING CARE

The client with botulism is hospitalized, and interventions focus on monitoring for respiratory failure and providing ventilatory assistance if necessary. Ongoing assessments are also made for manifestations of paralytic ileus and urinary retention. The client will be NPO until able to swallow and breathe; therefore, hydration and nutritional status are monitored. Teach the client and family that fatigue and weakness may persist for up to a year. During this time, the client may need to modify ADLs and take rest periods throughout the day.

Health Promotion

Education of the public to prevent botulism is important. Address the following topics at health fairs and community programs:

- Home-canned foods must be processed in a pressure cooker rather than in boiling water because the organism is difficult to kill.
- Do not eat home-processed foods that have a change in color, are soft, contain gas bubbles, or have a bad odor.
- Always heat both home-processed and commercial foods at temperatures over 248°F (120°C) or boil for 10 minutes before tasting or eating them.
- Discard home-processed or commercially canned or bottled foods with defective seals.
- Discard commercially prepared canned foods that are damaged or have bulging sides or leaking contents.

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CHAPTER HIGHLIGHTS

- Alzheimer's disease (AD) is a form of dementia (a disease of the brain) of older adults with progressive irreversible deterioration of general intellectual functioning. The disease is characterized by atrophy of brain tissue, loss of neurons, neurofibrillary tangles, and amyloid plaques. AD finally leaves the person unable to communicate, maintain continence, and recognize self or others. Caregivers need much teaching and support to not only provide care, but to also avoid caregiver burden.
- Multiple sclerosis (MS) is a chronic demyelinating neurologic disease of the CNS (brain, optic nerves, and spinal cord). Occurring in young to middle-age adults, it is believed to be due to an autoimmune response to a prior viral infection. The loss of myelin leads to axon dysfunction, which slows and distorts nerve impulses. Medications (ACTH, immunosuppressive agents, interferon, and glatiramer acetate) are used to slow the progression of the disease, decrease the number of exacerbations, and treat manifestations.
- Parkinson's disease (PD) is a progressive degenerative neurologic disease characterized by tremor, muscle rigidity, and bradykinesia. The loss of voluntary motor control is the result of pathologic processes resulting in a decrease of dopamine (an inhibitory neurotransmitter) so that it can no longer inhibit acetylcholine (an excitatory neurotransmitter). Medications are used to treat manifestations and include MAO inhibitors, dopaminergics, dopamine agonists, and anticholinergics. Other treatments include deep brain stimulation and surgery.
- Huntington's disease (chorea) is a progressive, degenerative inherited neurologic disease characterized by increasing dementia and chorea. There is an excess of dopamine, causing excessive, uncontrolled movement.
- Amyotrophic lateral sclerosis (ALS) is a rapidly progressive and fatal degenerative motor neuron disease characterized by weakness and wasting of voluntary control muscles, but without sensory or cognitive changes. The client eventually loses the ability to communicate and breathe. Riluzole, antiglutamate medication, is used to treat (but not cure) manifestations.
- Myasthenia gravis (MG) is a chronic autoimmune peripheral nervous system disorder characterized by fatigue and severe skeletal muscle weakness. It results from a decreased number of acetylcholine receptors at the neuromuscular junction, so muscles are unable to contract. Life-threatening emergencies include myasthenic crisis (sudden increase in motor weakness) and cholinergic crisis (from an overdose of the anticholinestease medications used to treat MG).
- Guillain-Barré syndrome (GBS) is an acute inflammatory demyelinating disease of the peripheral nervous system characterized by an acute onset of flaccid motor paralysis that begins in the lower extremities and ascends to involve the upper extremities, torso, and cranial nerves. Paralysis of intercostal and diaphragmatic muscles often necessitates ventilatory assistance. The acute phase lasts from 1 to 3 weeks, followed by recovery, which takes from 6 months to 2 years.
- Cranial nerve disorders include trigeminal neuralgia (tic douloureux) and Bell's palsy. Trigeminal neuralgia is a chronic disorder of cranial nerve V and causes severe facial pain. Bell's palsy is an acute disorder of cranial nerve VII, characterized by unilateral paralysis of the facial muscles. Clients provide self-care at home and require teaching to help prevent complications.
- Neurologic disorders resulting from neurotoxins or viruses include Creutzfeldt-Jakob disease, postpoliomyelitis syndrome, rabies, tetanus, and botulism. Community-based health education can not only provide information but can also prevent illness in the case of rabies, tetanus, and botulism.

TEST YOURSELF NCLEX-RN® REVIEW

1 Which of the following statements is true of dementia? (Choose all that apply.)
   1. Dementia is a general term used to describe manifestations of damage or death of neurons.
   2. Dementia is the term used to describe the cognitive and behavioral manifestations of AD.
   3. Dementia is an acute disorder, resulting from an injury to the brain.
   4. Dementia is the primary manifestation of Guillian-Barré syndrome.

2 What manifestation is usually the first indication of the onset of AD?
   1. inability to perform ADLs
   2. sundowning at night
   3. subtle memory deficits
   4. inability to communicate

3 Which of the following nursing diagnoses is appropriate for clients with MS, regardless of type or severity?
   1. *Fatigue*
   2. *Risk for Aspiration*
   3. *Acute Pain*
   4. *Impaired Gas Exchange*

4 You are preparing information to teach a client about medications for MS. What drugs would you expect to be used?
   1. antibiotics
   2. antihistamines
   3. interferon
   4. levodopa

5 Which of the following topics is important when teaching a young adult with MS?
   1. how to prevent sexually transmitted infections
   2. how pregnancy can improve manifestations
   3. what can be done to cure the disease
   4. why it is important to avoid extremes of heat and cold

6 The manifestations of Parkinson's disease are the result of:
   1. autoimmune responses to a viral infection.
   2. the failure of dopamine to inhibit acetylcholine.
   3. effects of a neurotoxin.
   4. a genetic defect.

7 When teaching care at home for the client with PD, what would be a major area to discuss with caregivers?
   1. preventing an overdose of medications
   2. avoiding daily baths and showers
   3. preventing falling
   4. increasing appetite

8 What drug classification is the medication used to treat ALS?
1. dopamine agonist
2. anticholinergic
3. anti-inflammatory
4. antiglutamate

9 You are preparing a teaching plan for a client with Bell's palsy. What information would you include?
1. "You will experience severe facial pain during attacks."
2. "The disease affects your muscles so you can't walk."
3. "One side of your face will not move normally."
4. "Be sure to boil all home-canned foods before eating them."

10 How can the nurse prevent tetanus?
1. Teach safe food preparation techniques.
2. Promote immunizations for adults and children.
3. Demonstrate proper disposal of soiled dressings.
4. Promote immunization of household pets.

See Test Yourself answers in Appendix C.

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UNIT 13 BUILDING CLINICAL COMPETENCE
Responses to Altered Neurologic Function

Functional Health Pattern: Cognitive-Perceptual

Think about clients with cognitive-perceptual problems for whom you have cared in your clinical experiences.

- What were the clients' major medical diagnoses (e.g., traumatic brain injury, spinal cord injury, stroke, aneurysm)?
- What manifestations did each of these clients have? (Were these manifestations similar or different?
- How did each of these clients respond to interaction with you as you provided care for them? Was it difficult for them to remember the identity of visitors or family members, that they were in a hospital, the correct year? Was their speech affected (aphasia)? Did the family indicate that the client took longer than normal to "find the words" in conversation? Were their words "jumbled" or confused when they spoke?

The neurologic system regulates and integrates all body functions, mental abilities, and emotions. It is made up of the central nervous system and peripheral nervous system. These two components are made up of two types of cells: neurons (which receive and transmit information) and neuroglia (which protect and support the neurons). The Cognitive-Perceptual Pattern includes functional abilities such as language, memory, judgment, decision making, and sensation. The pathophysiologic factors affecting cognition and perception are:

- Decreased blood flow to and ischemia of neurons (e.g., stroke, ruptured intracranial aneurysm or arteriovenous malformation, spinal cord injury, increased intracranial pressure, cerebral edema)
- Direct injury to neurologic tissues from trauma or compression (e.g., traumatic brain injury, skull fractures, increased intracranial pressure, spinal cord injury, herniated intervertebral disk, brain tumors, spinal cord tumors)
- Alterations in the electrical activity of cerebral neurons (epilepsy)
- Infections of the neurologic system (e.g., meningitis, encephalitis, rabies, tetanus, botulism)
- Degeneration or alteration of neurons, supporting neurologic structures, or neurotransmitters (e.g., Alzheimer's disease, multiple sclerosis, Parkinson's disease, Huntington's disease, amyotrophic lateral sclerosis, myesthenia gravis, Guillain-Barré syndrome, cranial nerve disorders).

One example of the pathophysiologic effects of neurologic disorders, increased intracranial pressure (a response to many disorders of and injuries to the brain) causes cell damage and death and can lead to transient or permanent manifestations such as:

- *Altered level of consciousness* (with decreasing circulation to and oxygenation of neurons → decreased cellular metabolism → Na-K pump failure → edema damage to the RAS)
- *Aphasia or dysphagia* (resulting from changes in the complex neurologic pathways in the speech center through ischemia, decreased oxygen and blood circulation, cell death, and the toxins released by dying cells)
- *Seizures* (seizure threshold is exceeded → abnormal neuronal activity remains localized or spreads to involve the entire brain → causing local or generalized effects).

Priority nursing diagnoses within the Cognitive-Perceptual Pattern that may be appropriate for clients with neurologic disease or injury include:

- *Ineffective Tissue Perfusion: Cerebral* as evidenced by level of consciousness changes, cognitive defects, and inaccurate interpretation of stimuli including confusion, comprehension, problem solving, abstraction, and memory deficits
- *Impaired Verbal Communication* as evidenced by inability/difficulty in speaking or understanding spoken or written words
- *Powerlessness* as evidenced by expressions of frustration regarding inability to control their illness, recovery rate, or care
- *Acute Confusion* as evidenced by restlessness, hallucinations, disorientation, anxiety.

Two nursing diagnoses from other functional health patterns often are a high priority for the client with neurologic disease or injury:

- *Impaired Swallowing* (Nutritional-Metabolic)
- *Impaired Physical Mobility* (Activity-Exercise)

Directions: Read the clinical scenario below and answer the questions that follow. To complete this exercise successfully, you will use not only knowledge of the content in this unit, but also principles related to setting priorities and maintaining client safety.

CLINICAL SCENARIO

You have been assigned to work with the following clients on a neurologic hospital unit. Significant data obtained during report are as follows:

- Louie Hernandez is a 74-year-old male who was admitted from the emergency department to the neurologic unit last night. Mr. Hernandez was found by family in his home. He was unable to speak or move the right side of his body and continues to experience paralysis on the right side in both his arm and leg. He is aphasic with facial drooping, but is able to nod his head to communicate. Mr. Hernandez has a known history of hypertension, CHF, and type 2 diabetes controlled by oral hypoglycemic medications. He has smoked a package of cigarettes a day for 40 years. BP is now 140/105, P 98, R 24. Lungs reported to have crackles in the bases. He is receiving 2 liters of oxygen per nasal cannula. A swallowing evaluation has been ordered to be done today.
- Jane Thomas is a 56-year-old female. She was admitted 4 days ago and underwent a lumbar laminectomy. She was able to ambulate with assistance on day 2 after surgery and now reports no dizziness on ambulation. Her leg strength has progressively increased since surgery. She has equal sensation and is able to wiggle her toes on both feet. Pain is controlled at the client's desired level of 2 to 3 on a scale of 1 to 10 with oral medications.
- Cesar Phillips is a 39-year-old male, admitted 4 days ago after experiencing paralysis and severe headache at work. He has been diagnosed with stroke following an MRI. He has a history of smoking one package of cigarettes per day and hypertension. His wife reported that he had stopped taking his blood pressure medication due to the side effects of impotence. Mr. Phillips is awake and alert, but has exhibited impulsive behavior and agnosia He also has a difficult time remembering words and neglects the left side of his body in ADLs. BP had been 130/85 at the beginning of last shift but has increased to 170/110. He received an additional prn blood pressure medication 1.5 hours ago.
- Tonya Walton is a 29-year-old female. She was involved in a car crash 12 hours before her admission 5 days earlier. She developed a headache, drowsiness, confusion, and pupil enlargement several hours after the crash. Following an MRI, Ms. Walton underwent intracranial surgery to evacuate a subdural hematoma. She has been complaining of a headache that has increased in intensity from a 2 to a 6 on a scale of 1 to 10 on the previous shift. She received two Tylenol ES tabs 1 hour ago. BP was 140–150 systolic and 90–100 diastolic for most of the shift. Her BP at the time of the Tylenol administration was 175/115. She has not experienced any changes in her neuro assessment except for the increasing headache and blood pressure elevation.

Questions

1 In what order would you visit these clients after report?

1. _____
2. _____
3. _____
4. _____

2 What two top priority nursing diagnoses would you choose for each of the clients presented above? Can you explain, if asked, the rationale for your choices?

	Priority Nursing Diagnosis #1	Priority Nursing Diagnosis #2
Louis Hernandez		
Jane Thomas		
Cesar Phillips		
Tonya Walton		

3 After completing Mr. Hernandez's beginning-of-shift baseline assessment, what assessment would be your next priority?

1. rhythm strip interpretation
2. intake and output
3. heart sounds
4. level of pain

4 You are completing the beginning-of-shift assessment for Mr. Phillips. What changes would best indicate that the plan initiated by the previous shift was effective?

1. Mr. Phillips no longer has a craving for cigarettes.
2. Mr. Phillips' respiratory rate is 18–20.
3. Mr. Phillips' LOP is now 1–2.
4. Mr. Phillips' blood pressure has decreased to 145/90.

5 What are the top two education priorities for Jane Thomas as you plan for discharge? (Select all that apply.)

1. appropriate exercise
2. pain management
3. weight control
4. incision care
5. importance of rest

6 You review the lab reports for Jane Thomas. You are most interested in the results for which of the following studies? (Select all that apply.)

1. white blood cell count
2. red blood cell indices
3. serum sodium
4. hemoglobin
5. hematocrit
6. osteocalcin
7. prealbumin

7 You are completing the beginning-of-shift assessment for Ms. Walton. What changes would best indicate that the plan initiated by the previous shift was effective?

1. Ms. Walton has no neurologic changes or deterioration.
2. Ms. Walton's BP is 142/94.
3. Ms. Walton's pain level is now 1–2.
4. Ms. Walton's dressing is dry and intact.

8 If you reevaluated Ms. Walton and her headache pain had not decreased, what should be your next priority?

1. Reposition Ms. Walton on her left side with the head of the bed flat.
2. Call the physician about client report of increasing headache.
3. Decrease stimuli in the room by darkening the room and limiting noise.
4. Check the prn orders to evaluate if a stronger pain medication has been ordered.

9 If you were planning an effective team meeting with all disciplines that should be collaborating to provide care and a discharge plan for Mr. Phillips, which six disciplines should be represented and why?

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____

10, 11, 12 Review the five steps of the abbreviated neurologic assessment as outlined in your text. What are three findings you would expect when conducting a neuro check with Mr. Hernandez?

1. _____
2. _____
3. _____

CASE STUDY

Leticia Hays is a 69-year-old African American female. She was admitted 2 days earlier after being diagnosed with a stroke. She lives with her husband of 52 years and her 14-year-old grandson. Her grandson found her lying on the kitchen floor when he got home from school. When he asked her what had happened, she was unable to say words that were clear or understandable. He tried to help her stand up, but was unable to because she was extremely weak in her left side. He called 911 and an ambulance transported her to the emergency department. Her blood pressure when the ambulance arrived was 220/125, P 105, R 24, and she was very anxious and spoke garbled words loudly at the paramedics. She was diagnosed with acute stroke after evaluation at the hospital and was admitted for treatment. Ms. Hays has a history of hypertension and type 2 diabetes. She recently stopped taking her blood pressure medication because it made her feel "tired."

The pathophysiology of a stroke is dependent on location of the causative factor, size of tissue affected, and time of loss of cellular perfusion and oxygenation. Time is critical when it involves compromised perfusion to any organ, and the brain is no exception. Cerebral neuron cellular metabolism is severely affected within 4 to 5 minutes of compromised perfusion. Compromised cellular metabolism leads to glucose, glycogen, and adenosine triphosphate depletion and sodium-potassium pump failure. Wherever sodium goes, water follows. When the sodium-potassium pump fails, the sodium in the cell draws water into the cell, resulting in edema. Additionally, cerebral vessel walls also swell, further compromising blood and oxygen supply. Severe or prolonged ischemia leads to cellular death. The manifestations of a stroke include, but are not limited to, motor deficits, elimination disorders, sensory-perceptual deficits, communication disorders, and behavioral changes. The manifestations are always sudden in onset, focal, and usually unilateral. Typically, a stroke is manifested by weakness of the face, arm, and leg; loss of vision in one eye; speech and swallowing problems; and difficulties with balance. There are many different complications of a stroke, including increased intracranial pressure, coma, death, chronic long-term confusion, and intellectual changes.

The nursing diagnosis of *Ineffective Tissue Perfusion: Cerebral* is appropriate for implementing care for Mrs. Hays.

affects

- Cellular perfusion and oxygenation of area of brain decreased or stopped
- Ischemia of cerebral neurons leads to cellular death

Ms. H
69 y.o. female
Stroke

leads to

assess

- History of hypertension and type 2 diabetes
- Not taking antihypertensives
- BP 220/125, P 105
- Left side weakness
- Garbled speech
- Confused

generates

- Impaired Verbal Communication related to effects of stroke
- Self-Care Deficits related to left-sided weakness and confusion
- Impaired Physical Mobility related to left-sided weakness
- Risk for Impaired Swallowing related to neurologic deficits from stroke

generate nursing diagnosis

prioritized to

Altered Tissue Perfusion: Cerebral

expected outcomes

- Actively participates in rehabilitation, including physical therapy, speech therapy, and occupational therapy
- Demonstrates improved ability to think, speak, carry out self-care, and ambulate
- Verbalizes necessity of taking prescribed medications
- Transfers to rehabilitation facility when health status improves

evaluate

- PT, OT and ST initiated twice daily.
- Speech is much improved and understandable
- Alert and oriented to person, place and time
- Able to feed self and walking with the assistance of a walker and another person
- Verbalized need for medications
- Transferred to rehabilitation facility for further care

Interdisciplinary Interventions

- Request referrals to physical therapy, speech therapy, and occupational therapy
- Apply nasal oxygen as prescribed
- Administer prescribed hypoglycemic and antihypertensive medications

Independent Interventions

- Schedule nursing activities for care around rehabilitation times
- Monitor neurologic status, including orientation, LOC, and reflexes
- Monitor pulse oximetry levels
- When appropriate, discuss effects and side effects of medications as well as risks of not taking them
- Discuss future transfer to rehabilitation facility with family members