CHAPTER 18

Delirium and Dementia

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Disorders causing a decline in cognitive function affect approximately 15% of the population older than 65 years. Cognitive deficits are seen in 5% of the elderly older than 65 years and in 20% of the elderly older than 75 years who live in the community. One third to one half of hospitalized elderly people exhibit cognitive changes, and more than 50% of those residing in nursing homes are cognitively impaired (Kane et al., 1994). Depending on the degree of cognitive decline, the consequences for an older person can range from minor problems in performing day-to-day activities to devastation in terms of the person's self-identity, level of independence, relationships with others, and overall quality of life.

The most common conditions associated with cognitive decline in the elderly are dementia and delirium (Berkow et al., 1995). Other conditions affecting cognitive function include depression, paranoid states and other psychoses, amnestic syndromes, and age-associated memory impairment (benign senescent forgetfulness) (Kane et al., 1994; Mayeaux et al., 1993). This chapter addresses the causes, clinical presentation, assessment, and management of delirium and dementia.

DELIRIUM

Delirium, also referred to as *acute confusional states,* is a serious, often unrecognized neuropsychiatric syndrome (Lipowski, 1994; Tune & Ross, 1994). It is especially prominent in older adults. Delirium is to the elderly what fever is to the young, in that the majority of individuals presenting with delirium suffer from a specific physical illness. Delirium may occur during the course of a dementing illness owing to the development of a new medical condition, which may evolve "silently" without a fever or other physical signs (Berkow et al., 1995). Table 18–1 lists the diagnostic criteria for delirium.

Epidemiology

Exact figures on the incidence of delirium are difficult to obtain for several reasons: the disorder is often undiagnosed, there is a failure to use consistent diagnostic criteria, terms are used inconsistently, and various methods of case finding are used by researchers (Francis, 1992; Lipowski, 1984; Miller & Lipowski, 1991).

TABLE 18-1

Diagnostic Criteria for Delirium

A. Disturbances of consciousness (i.e., reduced clarity of awareness of the environment) with reduced ability to focus, sustain, or shift attention.

B. A change in cognition (e.g., memory deficit, disorientation, language disturbance) or the development of a perceptual disturbance that is not accounted for by a preexisting, established, or evolving dementia.

C. The disturbance develops over a short period (usually hours or days) and tends to fluctuate during the course of the day.

D. There is evidence from the history, physical examination, or laboratory findings that the disturbance is caused by the direct physiological consequences of a general medical condition.

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In studies using the *Diagnostic and statistical manual of mental disorders* (DSM-III or DSM-IIR) criteria, prevalence rates of medical inpatients varied from 11 to 33%, and incidence rates varied from 4.2 to 10.4% (Levkoff et al., 1991; Tune & Ross, 1994). Acute confusional states have been reported in 10 to 15% of surgical patients, 30% of open heart surgical patients, and more than 50% of hip fracture patients (Francis, 1992).

Etiology

Causes of delirium can be classified as either predisposing or precipitating factors. Age itself is a predisposing factor. Thus, anything that affects brain function in the elderly can exacerbate the predisposition to delirium (Zisook & Braff, 1986). At high risk are people who have cardiac disorders or who abuse alcohol or drugs as well as elderly patients who have cognitive or memory disorders and have undergone trauma, surgery, or sudden environmental changes. Intoxication with medications, especially cholinergic drugs, is probably one of the most common causes of delirium in elderly people (Lipowski, 1994). Oncology patients are at risk owing to metabolic and nutritional imbalances, metastases, and infections. Patients with known neurological disturbances, such as strokes, head trauma, and brain tumors, and those with fever and dehydration are also at high risk. Table 18–2 presents some of the most frequent causes of delirium.

Clinical Presentation

People suffering from delirium have a decreased ability to attend to environmental stimuli and often show highly disruptive and variable behavior. The onset of delirium is acute, ranging from a few hours to a few weeks. Delirium is usually worse at night, and patients with delirium may have lucid intervals during which it is difficult to detect any disorder. Patients with delirium may present with a range of psychopathological symptoms, including delusions, hallucinations, confabulations, and a variety of emotions such as fear, anger, and apathy (Lipowski, 1994). Observers

TABLE 18-2

Systemic and Central Nervous System Causes of Delirium

Systemic Causes

- Cardiovascular disease
  - Congestive heart failure
  - Arrhythmias
  - Cardiac infarction
  - Hypovolemia
  - Aortic stenosis
- Infections
  - Pneumonia
  - Urinary tract infection
  - Bacteremia
  - Septicemia
- Medications
  - Analgesics
  - Anticholinergics
  - Antidepressants
  - Antihistamines
  - Antiparkinsonian agents
  - Cimetidine
  - Digitalis glycosides
  - Diuretics
  - Neuroleptics
  - Sedatives or hypnotics
- Neoplasm
- Postoperative state
- Substance abuse and poisons
  - Alcohol
  - Amphetamines
  - Sedatives or hypnotics
  - Heavy metals
  - Solvents
  - Pesticides
  - Carbon monoxide
- Trauma
  - Head injury
  - Burns
  - Hip fracture
- Metabolic
  - Electrolyte and fluid imbalance
  - Hepatic, renal, or pulmonary failure
  - Diabetes, hyperthyroidism or hypothyroidism, and other endocrinopathies
  - Nutritional deficiencies
  - Hypothermia and heat stroke

Central Nervous System Causes

- Infection
  - Meningitis
  - Encephalitis
  - Septic emboli
  - Neurosyphilis
  - Brain abscess
- Neoplasm
  - Primary intracranial
  - Metastatic (bronchogenic or breast)
- Trauma
  - Subdural hematoma
  - Extradural hematoma
  - Contusion
- Vascular disorder
  - Transient ischemic episodes
  - Stroke
  - Chronic subdural hematoma
  - Vasculitis
  - Arteriosclerosis
  - Hypertensive encephalopathy
  - Subarachnoid hemorrhage
- Seizure
  - Ictal and postictal states

From Zisook, S., & Braff, D. L. (1986). Delirium: Recognition and management in the older patient. *Geriatrics, 41*(6), p. 73. Copyright 1986 by Edgell Communications, Inc.

have suggested that delirious patients differ in their levels of alertness. Some patients may be confused and unable to focus attention or respond to their environment appropriately but may nevertheless appear relatively alert. Other patients may be similarly confused but appear drowsy or stuporous (Tune & Ross, 1994). Several researchers have classified such patients as *active/somnolent* (Ross et al., 1991) or *hyperalert/hyperactive* and *hypoalert/hypoactive* (Lipowski, 1989). Characteristic features of delirium, such as clouding of consciousness, disorientation, memory impairment, incoherent speech, and perceptual disturbances, all give rise to behavior that appears confused. To ensure the early recognition and treatment of delirium, frequent assessments of mental status should be performed in high-risk patients at the first signs of inappropriate or labile behavior. Evidence of disturbances in attention and arousal as well as disorientation and abnormal behavior is essential in establishing the presence of delirium (Zisook & Braff, 1986). Sullivan and Fogel (1986) cautioned against overemphasizing disorientation as a presenting sign for the diagnosis of delirium. They described four early cases of delirium that masqueraded as a violent personality disorder, a factitious illness, uncooperative behavior, and psychotic suicidal ideas.

Differential Diagnosis

Dementia must be ruled out in the differential diagnosis of delirium. Dementia usually follows a relatively stable course of impairment, whereas delirium is often a variable waxing and waning syndrome (Lipowski, 1994). In a large study of admissions to a general medical service, Erkinjuntti and associates (1986) found that 41.4% of the patients with dementia had delirium on admission and that 24.9% of all delirium patients had a dementing illness (Tune & Ross, 1994). If in doubt, the syndrome should be treated provisionally as delirium. Dementia patients may become delirious, but when the delirium clears the dementia remains. Table 18–3 shows the characteristics used to distinguish delirium and dementia.

Management

Delirium can present a life-threatening situation and must be recognized and treated

TABLE 18-3

Differentiating Delirium from Dementia

	Delirium	Dementia
Onset	Sudden, acute	Insidious
Course	Marked contrasts in level of awareness	Not seen in such contrast Slow, progressive decline
Duration	Hours to weeks	In progress at least 1–2 yr
Level of awareness, alertness, and attention	Hypoalert or hyperalert and aware Level fluctuates	Not affected
Cognitive function, including orientation, thinking, and memory	Preserved during lucid intervals Focal cognitive deficits Orientation impaired for a time Immediate and recent memory impaired	Consistent loss and decline Global cognitive deficits Impaired orientation Recent and remote memory impairment
Affect	Intermittent fear, perplexity, or bewilderment	Flat or indifferent affect
Perceptual disturbances	Hallucinations often disturbing and very clearly defined	Hallucinations vague, fleeting, ill defined; in many cases, it is difficult to make a clear judgment that they exist
Thought disturbances		
Paranoid states	Prominent, while cognitive impairment is mild or variable	More consistent with degree of impairment; less-prominent paranoia
Persecutory delusions	Ordered and cohesive	Vague, random, contradictory
Cause	Usually medical illness or drugs	Alzheimer's disease and multiple infarcts are most common causes
Treatment	Requires immediate evaluation and treatment	Not required immediately Requires ongoing monitoring and evaluation with attention to preventable and treatable conditions that may occur

Data from Berkow et al. (1995); Cummings et al. (1997); and Kane et al. (1994).

promptly. It is essential to determine and treat the underlying medical causes and to provide supportive care. When treated promptly, delirium is usually completely reversible. If the underlying factor is not reversed, however, delirium can lead to chronic brain impairment and death. Studies estimate the in-hospital fatality rate for elderly patients with delirium to be 25 to 33% (Gottlieb et al., 1991; Inouye et al., 1990). Delirium itself is not the cause of death, but the underlying medical condition may be. Delirium should always be treated as a medical emergency.

Treatment efforts should focus on determining and treating the cause or causes of the delirium and maintaining physiological balance with hydration, nutrition, oxygen supply, and electrolyte balance. All unnecessary medications should be discontinued until the problem has been resolved. The use of a low-dose neuroleptic such as haloperidol (0.5–2.0 mg twice a day) may be necessary if the patient is agitated, but it should be discontinued as soon as the patient has recovered. *Neuroleptic malignant syndrome*, a rare, potentially lethal side effect of neuroleptics, can occur in elderly, delirious patients (Francis & Kapoor, 1990). Neuroleptic malignant syndrome is associated with a change in consciousness, fever, and rigidity. Careful monitoring of the patient's neurological state is essential to identify possible side effects. Owing to the fluctuating course of delirium, patients must be deemed stable for 48 hours before recovery can be considered certain (Zisook & Braff, 1986).

Nursing management of delirium may include prevention, detection, and intervention. Using knowledge of the predisposing factors

as a guide, nurses can anticipate delirium and institute preventive measures. The following specific measures may benefit an elderly patient who is hospitalized with a preexisting dementia or whose cerebral function is compromised (Campbell et al., 1986; Tueth & Cheong, 1993):

1. If the patient is being hospitalized for scheduled elective surgery, family members should be encouraged to have a familiar person stay with the patient around the clock for the first few postoperative days. A familiar person can provide reality feedback, comfort, and a sense of continuity with the patient's life before hospitalization.
2. Attention should be paid to nonpharmaceutical comfort measures, as analgesic medications predispose patients to delirium, and medications with anticholinergic effects have cumulative effects and can cause delirium.
3. The patient's reliance on medication may be decreased through proper positioning, massage, maintaining comfortable temperature, and allowing maximum mobility.
4. The patient's orientation and alertness may be maximized by decreasing unnecessary noise, explaining all actions, providing orienting sensory input with brief but frequent contacts, and using staffing patterns that allow for continuity of care.
5. The incidence of dehydration or electrolyte imbalance will decrease with carefully monitored intake, output, skin turgor, and laboratory test results.

Patients in the community are equally susceptible to delirium. Particularly at risk are those patients who are frail, impaired, and living alone and those who have one or more chronic illnesses. Nurses may take an active role in caregiver education, teaching caregivers to be alert to subtle changes and to seek medical attention promptly for any sudden appearance of agitation, hallucinations, lethargy, somnolence, or sleep disturbance, even in patients with a preexisting dementia. The latter patients are particularly likely to have an undiagnosed delirium because the sudden change is interpreted simply as worsening of the existing dementia. Community health and homecare nurses should monitor frail, community-dwelling elderly people routinely for adequate nutrition and hydration, alcohol use, or misuse of medications. The same interventions are applicable to patients in long-term care settings.

Because nurses have continual contact with the elderly in many settings, they are often the first to detect delirium. The appearance of signs and symptoms that might indicate delirium should lead to immediate further investigation. These symptoms, all of which can fluctuate, can include difficulty with thinking and remembering, disoriented perception, disordered attention, and somnolence or night wakefulness. Vital signs should be taken and compared with baseline values. Assessment includes neurological, cardiovascular, and respiratory evaluations and a review of current medications. Findings from a mental status test should be compared with the patient's baseline function. The patient is screened for fluid volume deficit and hypoglycemia or hyperglycemia. Laboratory studies should include complete blood count with differential; blood urea nitrogen; and levels of creatinine, electrolytes, and glucose. If there are physical findings of respiratory insufficiency, a specimen for arterial blood gas should be drawn (Foreman, 1984). The presence of signs of delirium combined with positive findings in any of the above tests indicates that delirium is a likely diagnosis.

The first step in treating delirium is to find and treat or remove the causative factor. This process involves time. In the interval, providing nursing care for the patient with delirium presents a challenge. Primary nursing goals are to ensure patient safety and comfort and to intervene to mitigate psychiatric symptoms. Patients with delirium require hospitalization; if possible, they should be in a private room with continual supervision. The immediate environment should be quiet and as uncluttered and simple as possible. Patients with delirium are highly excitable and irritable and are prone to misinterpret stimuli. The presence of familiar objects, such as a large calendar and clock, is important. Ideally the room should have a view to provide cues to time and place. Light-

ing should be soft and diffuse to avoid sharp contrasts and shadows that can be misperceived. Patients who normally use hearing aids or glasses should be allowed to keep them (Levkoff et al., 1986; Tune & Ross 1994). The use of restraints should be avoided whenever possible (see Chapter 25).

Interactions with the patient should be kept to a minimum during periods of agitation. Medication may be used to treat symptoms of sleep-wake disturbance, hallucinations, and illusions present during agitated periods. Short–half-life benzodiazepines such as temazepam can be used to correct disturbed sleep-wake cycles. Because a patient with delirium is already impaired, care should be taken to use the lowest effective dose. As a rule of thumb, the starting dose should be one third the usual adult dose. The necessary nursing care, attending to hygiene, hydration, and nutrition should be given during periods of relative calm and lucidity. At these times the nurse can provide corrective sensory input while administering care.

The patient may need frequent reorientation to the surroundings and situation. Patients with delirium may not remember where or why they are hospitalized and may need to be retold the location and duration of the hospital stay and the events leading to it. Consider telling the patient that he or she is confused and disoriented. This can be done in a gently reassuring manner, emphasizing the positive aspects of the situation. Small improvements, such as a change from parenteral to oral nutrition, ambulating for greater distances, or taking a shower independently can be praised in a way that encourages the patient. A conversational tone and style by the nurse conveys respect and concern without being condescending. Successful nursing management of delirium includes monitoring and reporting the patient's condition and providing protection, support, and basic physical needs.

DEMENTIA

Clinicians have long recognized that dementia is a common clinical syndrome seen in the elderly; however, it is only in the past decade that the condition has been the subject of intensive systematic study (Katzman, 1992; Schoenberg, 1986). The knowledge gained has resulted in definitions and diagnostic criteria for this condition as well as a greater accuracy in its diagnosis.

Dementia is a symptom complex characterized by intellectual deterioration occurring in the presence of a clear state of consciousness. The intellectual deterioration is severe enough to interfere with social or occupational functioning and represents a decline from a previously higher level of functioning. Memory impairment, a prominent early symptom, is required to make the diagnosis of dementia. Dementia involves progressive deficits not only in memory but also in other cognitive areas, such as language, perception, praxis, learning, problem solving, abstract thinking, and judgment. Personality characteristics may be maintained or exaggerated in some patients and may be altered in others. Social withdrawal, fearfulness, and anxiety are common features. Paranoid symptoms and delusions can sometimes occur (American Psychiatric Association, 1994). Irritability, agitation, and verbal and physical aggression toward family members may develop as the dementia progresses and the individual experiences an increasing loss of control of his or her environment. Table 18–4 lists the diagnostic criteria for dementia.

Age-associated memory impairment (AAMI), also known as *benign senescent forgetfulness*, is distinctive from the changes caused by dementia. AAMI refers to the mild memory loss seen in elderly people. Memory loss in AAMI is not progressive, and other areas of cognitive function are not affected. Healthy older people with AAMI should be differentiated from those with early signs of a dementing illness. This important distinction can be made by observing over time for increased memory problems and cognitive decline as well as through psychometric testing (Kane et al., 1994; Mayeux et al., 1993). People with depression may appear demented; therefore, it is important to rule out depression in establishing the diagnosis of dementia (see Chapter 17). It is also critical to differentiate dementia from delirium, which is a more acute condition in which the level of

TABLE 18-4

Diagnostic Criteria for Dementia

A. Development of multiple cognitive deficits manifested by both
   1. Memory impairment (impaired ability to learn new information or to recall previously learned information)
   2. One (or more) of the following cognitive disturbances:
      a. Aphasia (language disturbance)
      b. Apraxia (impaired ability to carry out motor activities despite intact motor function)
      c. Agnosia (failure to recognize or identify objects despite intact sensory function)
      d. Disturbance in executive functioning (i.e., planning, organizing, sequencing, abstracting)
B. The cognitive deficits in criteria A1 and A2 each cause significant impairment in social or occupational functioning and represent a significant decline from a previous level of functioning.

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alertness and awareness fluctuates (Berkow et al., 1995; Larson et al., 1992).

Recognizing the dementia is only the first step; to care for the patient, one must also understand the impact on the family and the environment. This chapter examines dementia not only as a diagnostic entity but also from a psychosocial perspective involving both patient and family.

Epidemiology

By the year 2040, an estimated 11.8 million people in the United States will be afflicted with some form of dementia (Evans, 1990). Similar trends are expected for many developed countries (Cooper, 1991; Schoenberg, 1986). The prevalence of dementia increases with age. Community surveys demonstrate that 4.6% of individuals older than 65 years have severe dementia and 10% have mild to moderate dementia. Prevalence rates vary significantly between the ages of 65 and 85 years. Severe dementia is thought to be present in fewer than 1% of those who are 65 years of age but in more than 15% of those who are older than 85 years (Bachman et al., 1992). In the age range of 75 to 85 years, severe dementia is as frequent as myocardial infarction (Katzman, 1986).

Documenting the onset of dementia, as is required for studies of incidence or survival, is difficult because of the slow progressive nature of the condition. Mortality tabulations do not provide a reliable estimate, as deaths among demented individuals are often attributed to other underlying causes. Despite the prevalence of dementia in the elderly, the diagnosis is frequently missed (McCartney & Palmateer, 1985; U'Ren, 1987). Because many patients in the early stages of dementia may not be seen by a physician or are not diagnosed, the true magnitude of the problem may be underreported (Katzman, 1992).

Types of Dementias

Dementia can occur from a multitude of causes that may occur singly or in various combinations. These causes are broadly classified as *reversible* or *irreversible*. Table 18–5 lists the most common causes of dementia under these categories.

Some causes of dementia can be treated and ameliorated, whereas others are irreversible given the current state of knowledge. In a critical review of 32 studies investigating the prevalence of the causes of dementia, Clarfield (1988) found that 13.2% of all cases were potentially reversible. However, these studies did not always follow up the more important question of whether patients with potentially reversible causes actually achieved a reversal in their mental status. In 11 studies that did provide follow-up, 11% of dementias were resolved, either partially (8%) or fully (3%). The common reversible causes were drugs (28.2%), depression (26.2%), and metabolic disorders (15.5%). The true incidence of reversible dementias in the community is believed to be even lower (Clarfield, 1988, 1995).

Major Dementias in the Elderly

At present, it is estimated that more than 70 disorders cause dementia, with acquired im-

TABLE 18-5

Reversible and Irreversible Causes of Dementia

Potentially Reversible Causes
Cardiopulmonary disorders
Hypertension
Severe cardiac failure
Cardiac arrest
Depression
Drug toxicity, including alcohol
Normal pressure hydrocephalus
Space-occupying lesions
Subdural hematomas
Primary brain tumor
Metabolic-endocrine derangements
Hyperthyroidism or hypothyroidism
Hyperparathyroidism
Hypernatremia or hyponatremia
Hypoglycemia
Nutritional deficiency states
Thiamine deficiency
Folate deficiency
Cobalamin deficiency (vitamin B_{12})
Infections
Bacterial meningitis or encephalitis
Brain abscess
Neurosyphilis
Probable Irreversible Causes
Alzheimer's disease
Dementia with Lewy bodies
Pick's disease
Huntington's disease
Multi-infarct dementia
Amyotrophic lateral sclerosis
Parkinson's disease
Cerebellar degenerations
Alcoholism—Korsakoff's syndrome
Infections
Creutzfeldt-Jakob disease
Acquired immunodeficiency syndrome

Data from Berkow et al. (1995); Feldman & Plum (1993); Kane et al. (1994); Katzman (1992); and Miller (1997).

mune deficiency syndrome being the latest cause identified (Katzman, 1992). The most common dementias seen in the elderly are Alzheimer's disease (AD), multi-infarct dementia (MID), and dementias secondary to metabolic endocrine derangements, with alcohol and medications being major contributors (Feldman & Plum, 1993; Katzman, 1992). AD accounts for 50 to 60% of all cases (Cummings, 1995), and vascular disease, specifically MID, accounts for 10 to 15%. Some patients may have both disorders. Other causes include alcoholic dementia (5–10%), normal pressure hydrocephalus (6%), intracranial masses (5%), and Huntington's chorea (3%). In 5% of the cases, the cause remains unknown (Chui et al., 1992; Kase, 1991; Katzman, 1992).

ALZHEIMER'S DISEASE

Alzheimer's disease, named after Alois Alzheimer, who first described its neuropathology in 1907, is a neurological disorder of the brain that occurs primarily in middle or late life, although it may occur earlier. Its primary characteristic is a progressive dementia. The pathology includes the degeneration and loss of nerve cells (neurons), particularly in those regions essential for memory and cognition, and the presence of neuritic plaques and neurofibrillary tangles. Aggregates of amyloid protein can be seen adjacent to and within the walls of many cerebral and leptomeningeal blood vessels (Brousseau et al., 1994; Selkoe, 1991). Alterations occur in the neurotransmitter and neuromodulator levels, especially in the cholinergic system and frequently in the somatostatinergic, glutamatergic, and noradrenergic systems (Ingram et al., 1994; Katzman, 1984).

Diagnostic criteria for AD were established by the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA) (Table 18–6). At present, a definitive diagnosis of AD can be made only through an examination of brain tissue obtained during either postmortem examination or a brain biopsy. In most cases, the risks of brain biopsy far outweigh the benefits. Therefore, the diagnosis is made by a systematic exclusion of all other possible causes of dementia. If the symptoms are progressive, follow a pattern over time, and are attributable to no other cause, a clinical diagnosis of probable AD should be made. In patients with an atypical presentation, a diagnosis of possible AD may be made and the patient followed at regular intervals to assist in clarifying the diagnosis (McKhann et al., 1984).

Epidemiology. The prevalence of AD in-

TABLE 18-6

NINCDS-ADRDA Criteria for Clinical Diagnosis of Alzheimer's Disease

I. The criteria for the clinical diagnosis of PROBABLE Alzheimer's disease:

- Dementia established by clinical examination, documented by the Mini-Mental Test, Blessed Dementia Scale, or some similar examination, and confirmed by neuropsychological tests:
- Deficits in two or more areas of cognition
- Progressive worsening of memory and other cognitive functions
- No disturbance of consciousness
- Onset between the ages of 40 and 90 years, most often after age 65 years
- Absence of systemic disorders or other brain diseases that in and of themselves could account for the progressive deficits in memory and cognition

II. The diagnosis of PROBABLE Alzheimer's disease is supported by the following:

- Progressive deterioration of specific cognitive functions such as language (aphasia), motor skills (apraxia), and perception (agnosia)
- Impaired activities of daily living and altered patterns of behavior
- Family history of similar disorders, particularly if confirmed neuropathologically
- Laboratory results of the following:
  - Normal lumbar puncture as evaluated by standard techniques
  - Normal pattern or nonspecific changes in electroencephalogram, such as increased slow-wave activity
  - Evidence of cerebral atrophy on computed tomography, with progression documented by serial observation

III. Other clinical features consistent with the diagnosis of PROBABLE Alzheimer's disease, after exclusion of causes of dementia other than Alzheimer's disease:

- Plateaus in the course of progression of the illness
- Associated symptoms of depression; insomnia; incontinence; delusions; illusions; hallucinations; catastrophic verbal, emotional, or physical outbursts; sexual disorders; and weight loss
- Other neurological abnormalities in some patients, especially with more advanced disease, including motor signs such as increased muscle tone, myoclonus, or gait disorder
- Seizures in advanced disease
- Computed tomography normal for age

IV. Features that make the diagnosis of PROBABLE Alzheimer's disease uncertain or unlikely:

- Sudden, apoplectic onset
- Focal neurological findings such as hemiparesis, sensory loss, visual field deficits, and incoordination early in the course of the illness
- Seizures or gait disturbances at the onset or very early in the course of the illness

V. Clinical diagnosis of POSSIBLE Alzheimer's disease:

- May be made on the basis of the dementia syndrome, in the absence of other neurological, psychiatric, or systemic disorders sufficient to cause dementia, and in the presence of variations in the onset, in the presentation, or in the clinical course
- May be made in the presence of a second systemic or brain disorder sufficient to produce dementia, which is not considered to be *the* cause of the dementia
- Should be used in research studies when a single, gradually progressive severe cognitive deficit is identified in the absence of another identifiable cause

VI. Criteria for diagnosis of DEFINITE Alzheimer's disease:

- Clinical criteria for probable Alzheimer's disease
- Histopathologic evidence obtained from a biopsy or autopsy

VII. Classification of Alzheimer's disease for research purposes should specify features that may differentiate subtypes of the disorder, such as:

- Familial occurrence
- Onset before age of 65 years
- Presence of trisomy-21
- Coexistence of other relevant conditions such as Parkinson's disease

NINCDS-ADRDA, National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association.

Adapted from McKhann, G., Drachman, D., Folstein, M., et al. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA work group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology, 34,* 940.

creases with age, and the rate more than doubles between the ages of 60 and 80 years (Mayeaux & Schofield, 1994). There is a higher prevalence of AD in women than in men. The underlying reason may be the longer life expectancy of women, although other factors may be involved (Berkow et al., 1995; Civil et al., 1993). A few studies have found that cultural or ethnic differences may exist in the prevalence and incidence of AD (Mayeaux & Schofield, 1994).

Theories of Causation. The cause of AD remains unknown. Several theories have been proposed and form the basis for current research:

1. Genetic theory (Levy-Lahad et al., 1995; Pericak-Vance et al., 1991; Reichman, 1994; St. George-Hislop et al., 1987; Schellenberg et al., 1992; Tanzi et al., 1992; Zoler, 1994)
2. Slow virus theory (Pruisner, 1984; Wurtman, 1985)
3. Aluminum theory (Bolla et al., 1992; Deary & Whalley, 1988)
4. Cholinergic theory (Bowen et al., 1976; Davies & Maloney, 1976; Ingram et al., 1994; Larson et al., 1992)
5. Amyloid theory (Beyreuther et al., 1991; Rosenberg, 1993; Rumble et al., 1989)
6. Autoimmune theory (Aisen & Davis, 1994; Wurtman, 1985)
7. Trauma theory (Clinton et al., 1991; Drachman & Lippa, 1992; Heyman et al., 1984; Mortimer et al., 1991; van Duijn et al., 1992)

Clinical Presentation. AD is a progressive neurological disorder that has an insidious onset and progresses over time, leaving the individual in a vegetative state. A wide variety of symptoms can progress. The most typical initial changes include lapses of recent memory, a decreased ability to learn new information, an altered attention span, agnosia, and a lack of spontaneity. Clinically, these patients show relatively good preservation of their ability to recall remote events, and they tend to more readily recall incidents that are associated with important life events, such as births, marriage, and wars (Fromholt & Larsen, 1991; Sagar & Sullivan, 1988). Sentence structure is not usually affected during the early stages, and there may be little or no evidence of a deficit in casual conversation.

Progression of the disease involves personality changes, mental status changes, social role dysfunction, difficulty with activities of daily living, language deterioration, visuoperceptual deficits, incontinence, and gait disorders. Drawing ability and the construction of three-dimensional figures become affected. Drawings are small and cramped, key features tend to be omitted, and patients may "close in" on figures (i.e., draw figures on top of the existing ones). Spatial disorientation, reading difficulties, and anomia are also noted when objects are presented visually (Huff et al., 1986). Figure 18–1 shows examples of constructional apraxias.

Rigidity of the extrapyramidal type occurs in about 30% of people with AD and may be associated with a greater severity of illness (Mayeux & Schofield, 1994; Sagar & Sullivan, 1988). Balance and gait disturbances are fairly frequent. The most frequent abnormalities seen are decreased arm swing, reduced stride length, and postural instability. Dyskinesia and myoclonus are common. Myoclonic jerks and generalized seizures may occur in the later stages of the disease (Katzman, 1992). Oculomotor function is typically impaired. There may be deficits in upward gaze, causing a perseveration of downward gaze, and severely impaired and demented patients show gaze perseveration (Sagar & Sullivan, 1988). During the advanced stages of the disease, the patient becomes mute, is unable to walk, is incontinent of bowel and bladder, and becomes dependent on others for all activities of daily living.

Patients with early dementia are often aware of and frightened by their deficits. Changes in affect and impulse control as well as motivational and general interest occur as the disease progresses. Behavioral symptoms frequently seen in patients with AD include wandering, aggressiveness, sleep disturbances, nocturnal confusion, tearfulness, and anxiety (Civil et al., 1993). Delusions have been reported in 13 to 53% of AD patients and tend to be primarily paranoid in nature, involving misbeliefs about infidelity or theft (Civil et al., 1993; Cummings & Benson, 1983). About 40% of AD pa-

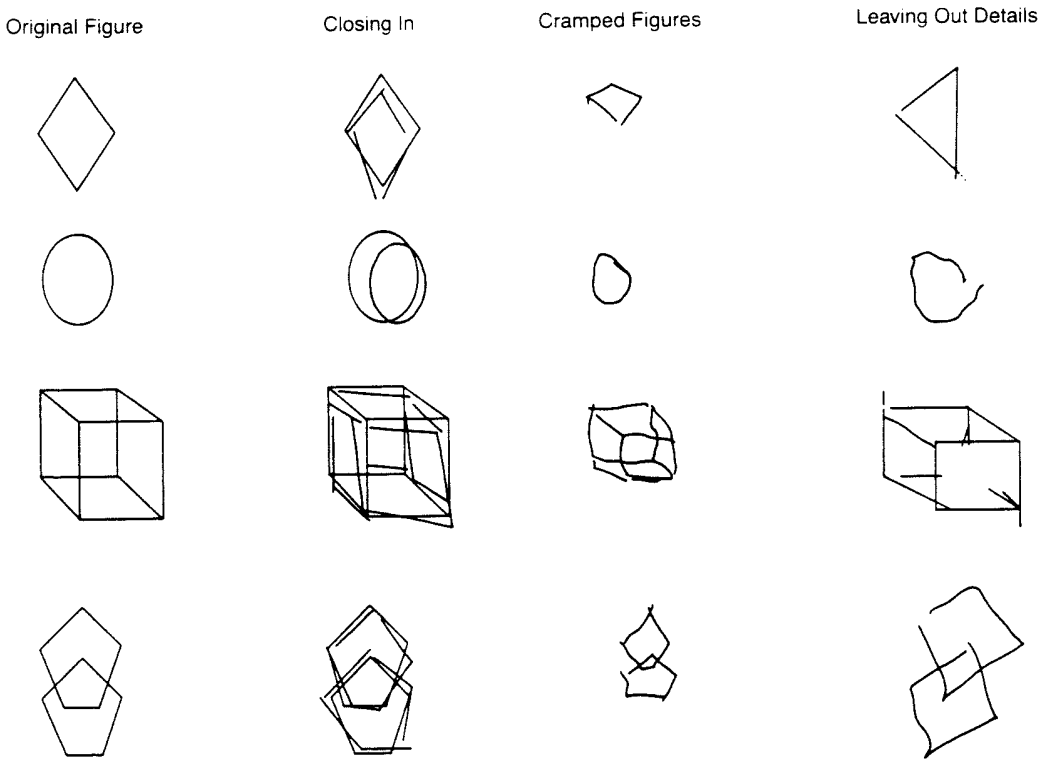

FIGURE 18–1. Examples of constructional deficits seen in patients with Alzheimer's disease.

tients have depressive features, usually early in the disease, which contribute to cognitive dysfunction (Rosser, 1993).

Approximately 10% of patients may have an atypical presentation, with a deficit predominately in one area of cognition. For example, the first presenting symptoms may be a difficulty with finding words, visuospatial deficits, isolated personality changes, or pure memory loss (Katzman, 1992). These focal symptoms may occur many years prior to the typical generalized progression seen in most AD patients. The rate of deterioration differs among patients and within patients over time. The Global Deterioration Scale developed by Reisberg and colleagues (1982) is one of several scales available to measure the changes and to identify the stage of the disease (Table 18–7).

When tested, patients in the early stages of AD make continuous attempts to answer questions. In contrast, patients with depression complain of memory deficits and an inability to recall but, if encouraged, may do well on testing. Aware of their deficits, patients with AD frequently try hard to minimize or rationalize their errors in testing. Denial, confabulation, perseveration, and avoidance of questions are typical interview behaviors of patients in the later stages of AD.

Prognosis. Patients with AD have a reduced life expectancy (Terry & Katzman, 1992). The disease may have a course as short as 18 months or as long as 27 years, but the average duration of illness is 10 to 12 years (McLachlan et al., 1984; Miller et al., 1994). The leading causes of death in patients with AD have been found to be related to diseases of the heart (cardiopulmonary arrest, arrhythmia, myocar-

TABLE 18-7

Global Deterioration Scale for Age-Associated Cognitive Decline and Alzheimer's Disease

GDS Stage	Clinical Phase	Clinical Characteristics	Diagnosis
1 = No cognitive decline	Normal	No subjective complaints of memory deficit. No memory deficit evident on clinical interview.	Normal
2 = Very mild cognitive decline	Forgetfulness	Subjective complaints of memory deficits. No objective deficits in employment or social situations. Appropriate concern with respect to symptomatology.	Normal aged
3 = Mild cognitive decline	Early confusional	Earliest clear-cut deficits. Decreased performance in demanding employment and social settings. Objective evidence of memory deficit obtained only with an intensive interview. Mild to moderate anxiety accompanies symptoms.	Compatible with incipient Alzheimer's disease
4 = Moderate cognitive decline	Late confusional	Clear-cut deficit on careful interview. Inability to perform complex tasks. Denial is dominant defense mechanism. Flattening of affect and withdrawal from challenging situations occur.	Mild Alzheimer's disease
5 = Moderately severe cognitive decline	Early dementia	Patients can no longer survive without some assistance. Patients are unable during interview to recall a major relevant aspect of their current lives. People at this stage retain knowledge of many major facts regarding themselves and others. They invariably know their own names and generally know their spouse's and children's names. They require no assistance with toileting or eating but may have some difficulty choosing the proper clothing to wear.	Moderate Alzheimer's disease
6 = Severe cognitive decline	Middle dementia	May occasionally forget the name of the spouse on whom they are entirely dependent for survival. Patients are largely unaware of all recent events and experiences in their lives and require some assistance with activities of daily living. Personality and emotional changes occur.	Moderately severe Alzheimer's disease
7 = Very severe cognitive decline	Late dementia	All verbal abilities are lost. Frequently there is no speech at all—only grunting. Incontinent of urine; requires assistance toileting and feeding. Loses basic psychomotor skills (e.g., ability to walk).	Severe Alzheimer's disease

From Reisberg, B., Ferris, S. H., de Leon, M. J., et al. (1982). The global deterioration scale for assessment of primary dementia. *American Journal of Psychiatry, 139,* 1136–1139. Copyright 1982 by the American Psychiatric Association.

dial infarction) and to respiratory conditions or bronchopneumonia (Olichney et al., 1995). Chandra and associates (1986) reported that AD patients are at a greater risk for infections, trauma, nutritional deficiency, Parkinson's disease, and epilepsy. Although there is no cure for AD at present, there are many preventable and treatable conditions that, with appropriate management, may extend this population's life span and improve their quality of life (Kane et al., 1994; Katzman, 1992).

Research in Alzheimer's Disease. Clinical and research interest in dementia and its causes has greatly increased in recent years, and researchers have begun to study a broad spectrum of areas associated with the problem. It is still uncertain whether AD is an infectious process, a toxic disorder, a biochemical defi-

ciency, or an exaggeration and acceleration of the normal aging process, with dementia appearing when the neural reserves are exhausted and compensatory mechanisms fail (Drachman & Lippa, 1992).

One of the most important research issues in AD is accurate diagnosis. The National Institute on Aging Task Force (1980) estimated incorrect diagnosis to be 10 to 30% in the general medical population. With the advent of newly developed diagnostic criteria, the reported diagnostic accuracy for dementia is now 95%, and the diagnostic accuracy of probable AD is approximately 90% (Katzman, 1990). Standarized criteria for the diagnosis of ischemic vascular dementia (VD) have been developed (Chui et al., 1992). An early and accurate diagnosis is essential for research on AD and other dementing disorders to progress.

The development of diagnostic criteria has in turn led to broader epidemiological studies. Longitudinal studies, which are necessary to advance research on the diagnosis of AD, are currently being conducted. Researchers in this area are collecting detailed information on people with the disease as well as on normal aging populations in an effort to identify premorbid events and conditions that may predispose some individuals to developing the disease and to gain a better understanding of the normal aging process. One of the major difficulties in diagnosing AD involves the variations in presentation and the need to understand the relationships among neuropsychological, neuropathological, and neuroradiological findings. Neuroimaging techniques and the development of more precise neuropsychological tests are high priorities.

The role of environmental factors is another central research theme. Memory loss was for many years considered a normal consequence of aging. However, the process of normal aging is not clearly understood, and until a better knowledge is gained in this area, diagnosing AD will continue to be a difficult and somewhat imprecise process. For some time, it has been clearly established that there is a serious defect in the cholinergic system of patients with AD. This finding has continued to generate a tremendous amount of interest. Research in this area has focused on understanding the cause and effect of the defect and looking for pharmacological approaches to treat, prevent, or stop the disease process. Along with pharmacological studies, there is an interest in developing new ways of dispensing medications (e.g., by pumps and skin patches) that will be useful not only in AD but in many other disorders as well.

It is not yet known why brain cells die in AD. Many researchers are studying the chemistry of the neurofibrillary plaques and tangles found in the brains of AD patients. Data supporting a causal role of amyloid in AD have led to a wide range of studies aimed at delineating the biochemical pathways involved in the deposition of amyloid deposits in patients with AD or other dementias. Others are studying the membrane structures and how changes in the membrane influence transport and homeostasis of essential ions. Pedigree studies investigating the familial genetic aspects of AD are essential in understanding the disease and in helping predict which individuals are at risk for the disease. A high priority of current research is identifying genetic markers and biological correlates of AD. Recent animal studies have shown that nerve growth factor can promote the survival of central nervous neurons in adult life. Researchers are looking for therapeutic approaches that will use this information. Studies are currently investigating the use of estrogen, antioxidants, and nonsteroidal antiinflammatory or immunosuppressive drugs as a means of reducing the risk of and treating AD (Barrett-Connor & Kritz-Silverstein, 1993; Brietner, 1996; McGreer et al., 1992; Sano et al., 1997; Yaffe et al., 1998).

VASCULAR DEMENTIA

VD is a clinical syndrome of acquired intellectual and functional impairment resulting from the effects of cerebrovascular disease. After AD, cerebrovascular disease is the most common contributor to dementia. VD is characterized by a wide range of neurological and neuropsychological signs and symptoms that reflect the wide variety of responsible lesions. Depending on the cause of VD, the onset may be abrupt, insidious, static, remitting, or progressive (Reichman, 1994; Roman et al., 1993).

Ischemia, hemorrhage, and anoxia can all lead to dementia. Accumulations of ischemic cerebral infarctions can produce a condition in which dementia is the dominant symptom. Dementia resulting from the accumulation of ischemic cerebral infarctions is known as MID (multi-infarct dementia). Chronic ischemia, without frank infarction, may also contribute to cognitive deterioration, and ischemic changes may coexist with other pathology (Chui et al., 1992).

The extent and type of cognitive dysfunction found in patients with MID depend on the localization and size of the lesions. Small bilateral infarcts in the hippocampus or thalamus may produce considerable dysfunction in recent memory, whereas infarcts of the same size in the optic radiation may produce only minor visual field limitations. Some studies have found that multiple infarcts at times do not produce any cognitive dysfunction (Heyman, 1978; Kane et al., 1994; Kase, 1986). Patients with MID tend to have a more severe drop in motor functions than do patients with AD (Erkinjuntti et al., 1986, 1987).

There have been few reports of changes in specific cognitive function in patients with MID, probably because the nature of the deficit is so closely dependent on the location and size of the infarcts. To compound the difficulty in measuring dysfunction, AD and MID coexist in about 15% of patients with dementia (Berkow et al., 1995). The cognitive deficits of the two conditions may be additive, so that in patients who already have AD changes in the brain, higher cerebral dysfunction may be seen following a stroke (Sagar & Sullivan, 1988).

Epidemiology. As with most causes of dementia, the incidence of cerebrovascular disease increases with age. Neuropsychological studies have shown that approximately 12.5% of dementias in the elderly are caused by MID, and 13.6% by a combination of both MID and AD or a mixed dementia (Meyer et al., 1986). There is a higher incidence of MID in men than in women. Hypertension is a major risk factor (Marshall, 1993). In countries with a high incidence of hypertension, such as Japan and Finland, a higher proportion of dementia may be attributed to MID (Katzman, 1992).

Etiology. Several causes for vascular dementia have been proposed. These are summarized in Table 18–8.

Clinical Presentation. The clinical features of VD depend on the underlying cause and/or the size and location of the responsible lesions. General features can include all or some of the following:

Abrupt onset
Fluctuating course
Focal neurological signs and symptoms
Depression
Emotional lability
Somatic complaints
Nocturnal confusion

TABLE 18-8

Multi-Infarct Dementia: Theories of Causation

Cause	Theory
Lacunae	Small focal infarcts result from occlusion of branch arteries that penetrate the brain. Accumulation of these lesions can cause progressive mental deterioration.
Binswanger's disease	Infarcts are limited to the subcortical white matter. Hypertension is thought to be major cause.
Multi-emboli	Any vessel can cause emboli, but extracranial arteries and the heart are most likely to produce the widely distributed lesions that produce multi-infarct dementia.
Vasculitis	May be idiopathic, infectious, granulomatous, or the result of toxins or immune complex. Lupus erythematosus is one of the most frequent causes.
Blood dyscrasias	Uncommon, but known to cause vascular lesions.
Hypoperfusion	Systemic hypotension, or occlusion of a major vessel, can cause damage to areas of the brain perfused by the most distal vessels.
Anoxic episodes	Caused by cardiac arrest, anesthesia, or other similar causes resulting in neuronal loss, leading to dementia without the presence of infarctions.

Data from Read & Jarvik (1984).

Patients with MID experience a stepwise decline with a fluctuating clinical course. The onset tends to be more acute with focal neurological signs and symptoms, and patchy cognitive losses are often seen (Marshall, 1993). Dysarthria, hemineglect of visual space, movement disorder, or a subtle paresis may be evident. Patients may exhibit an inability to distinguish left from right, to reproduce two-dimensional drawings, and to identify letters traced on the hand. Delusions may occur. Studies have found delusions in as many as 40% of patients with MID (Cummings et al., 1987). A history of transient ischemic attacks, hypertension, strokes, diabetes mellitus, vasculitis, and cardiac arrhythmias is frequently associated with MID. A family history of stroke or cardiovascular disease may be present (Read & Jarvik, 1984; Reichman, 1994).

A mental status examination is important in delineating patchy deficits. Focal slowing may be seen on electroencephalogram in patients with infarcts too small to be visible with computed tomography (Benson et al., 1982; Read & Jarvik, 1984). The Hachinski Ischemia Rating Scale is a useful tool in differentiating AD from MID (Table 18–9).

Disease Progression. The course of MID can be intermittent and fluctuating. Patients may have episodes of clouded sensorium or may plateau for long periods. Hypertension is the most important risk factor for stroke in patients with MID. Specific cardiovascular diagnosis and management are key to arresting progression and reducing mortality (Hachinski et al., 1974; Marshall, 1993). When deterioration proceeds rapidly following a single cerebrovascular accident, there has likely been slowly advancing subclinical change, now potentiated by infarction, which has served to push the neuronal damage beyond the threshold point.

Prognosis. The chances of social survival are thought to be better in patients with VD than in those with AD. Treatment of the underlying illness and the secondary psychiatric and medical conditions can substantially improve the prognosis. Selected patients with MID may benefit from anticoagulation therapy or surgery. Some cases of VD are remediable, and

TABLE 18-9

Hachinski Ischemic Rating Scale

Instructions: Record the presence or absence of the clinical features of dementia listed below and add the point values assigned each feature (value in parenthesis) whenever "Present" is checked. Summation of points produces an Ischemic Score. Scores of $< +4$ indicate patients with pure primary degenerative dementia (Alzheimer's type dementia). Scores of >4 indicate patients with multi-infarct dementia.

Feature	Absent	Present	Point Value
1. Abrupt feature	_____	_____	2
2. Stepwise deterioration	_____	_____	1
3. Fluctuating course	_____	_____	2
4. Nocturnal confusion	_____	_____	1
5. Relative preservation of personality	_____	_____	1
6. Depression	_____	_____	1
7. Somatic concern	_____	_____	1
8. Emotional incontinence	_____	_____	1
9. History of hypertension	_____	_____	1
10. History of strokes	_____	_____	2
11. History of associated atherosclerosis	_____	_____	1
12. Focal neurological symptoms	_____	_____	2
13. Focal neurological signs	_____	_____	2

From Hackinski, V. C., Iliff, L. D., Zilhka, E., et al. (1975). Cerebral blood flow in dementia. *Archives of Neurology, 32,* 634. Copyright 1975 by the American Medical Association.

occasionally some are reversible (Meyer et al., 1986; Read & Jarvik, 1984; Wade, 1991). Other individuals with VD may experience further cognitive decline.

ALCOHOLIC DEMENTIA

Alcohol abuse gives rise to a large and diverse group of mental disorders. Unfortunately, alcoholic dementia is not well studied, and confusion still exists concerning its diagnosis. Studies have shown that (1) mild forms of cerebral dysfunction are common in heavy drinkers, (2) a smaller but still significant proportion of heavy drinkers will have more severe dysfunction, and (3) a similar proportion of patients who present with dementia will have a history of heavy drinking or alcoholism (Willenbring, 1988). The effects of alcohol on the brain range from acute or reversible conditions (e.g., acute intoxication, alcoholic hallucinosis, and pathological intoxication) to withdrawal states (e.g., delirium tremens and withdrawal seizures) to chronic, largely irreversible conditions reflecting a long-term derangement of metabolism, as seen in Wernicke-Korsakoff's syndrome, alcoholic pellagra, and hepatic encephalopathy (Feldman & Plum, 1993; Willenbring, 1988).

Wernicke-Korsakoff's syndrome is one of the major alcoholic dementias. Wernicke's encephalopathy and Korsakoff's syndrome are considered to be stages of one syndrome and not separate diseases (Victor, 1993). However, there may be cases of Korsakoff's syndrome with no apparent history of Wernicke's (Carlen & Neiman, 1990). Thiamine deficiency is the specific nutritional factor thought to be responsible for this condition. Neuropathological findings include lesions in the medial thalamic region, pons, medulla, and cerebellar cortex. The lesions responsible for memory loss are structural rather than biochemical. Korsakoff's syndrome is most often associated with alcohol but may be a symptom of various other disorders (Victor, 1993). Other alcoholic dementias are described in Table 18–10.

Epidemiology. Alcohol abuse is a major health problem. One out of eight adults is considered to be a heavy drinker. Alcohol contributes to nearly 7% of all dementias. Approximately 50% of excessive drinkers show neuropsychological impairment, and 3% of alcoholics have clinically defined dementia

TABLE 18-10
Alcoholic Dementias

Disease	Cause	Treatment	Progression	Prognosis
Wernicke-Korsakoff's	Thiamine deficiency	Thiamine, adequate diet	With treatment improvement begins in a few weeks to 3 months. Recovery is slow, 1 year or longer.	Varies. Complete or almost complete recovery in about 20% of patients. Small percentage show no recovery, and the majority fall in between.
Alcohol-induced pellagra	Poor nutrition over time; deficiency of niacin or tryptophan	Niacin, adequate diet	Degree of improvement depends on severity of brain lesions.	Aggressive treatment can produce considerable improvement. Fatal if untreated.
Hepatic encephalitis	Chronic alcoholism resulting in liver disease, leading to increased ammonemia	Treat liver disease	Symptoms evolve over days or weeks. Coma may occur. Symptoms may regress completely, become chronic, or worsen with repeated comas.	Symptoms are reversible if treatment is timely. Fatal if untreated.

(Feldman & Plum, 1993). The alcoholic dementias have a high mortality rate and can produce crippling effects.

Etiology. The role of alcohol is considered to be secondary in the pathogenesis of these dementias. The adverse effects on the nervous system are the result of nutritional and liver diseases engendered by the chronic abuse of alcohol. Alcoholic dementias are thought to differ in this respect from other pathological intoxication states that directly result from the effect of alcohol on the central nervous system or the withdrawal of alcohol following a period of chronic intoxication (Willenbring, 1988).

Clinical Presentation. In the chronic alcoholic patient, Korsakoff's syndrome (or Korsakoff's disease) usually begins with an acute attack of Wernicke's disease (presenting with global confusion, apathy, ataxia, and ocular abnormalities). With adequate diet and thiamine, patients become increasingly alert, responsive, and gradually less confused, and the major remaining impairment is a decreased retentive memory. If not treated, Wernicke's is thought to become chronic and persist as a Korsakoff's syndrome (Allen, 1994; Carlen & Neiman, 1990). Korsakoff's syndrome is unique and is characterized by two abnormalities that always occur together: *retrograde amnesia* (losing information acquired in the past) and *anterograde amnesia* (having a decreased ability to acquire new information). Confabulation is not consistently present and is not required for diagnosis. In an otherwise alert and responsive patient, retentive memory is impaired out of proportion to other cognitive functions. Other cognitive functions that are not memory dependent may be impaired but to a lesser degree. Limited insight, apathetic behavior, inertia, and indifference to people and events may also be present (Carlen & Neiman, 1990; Victor, 1993).

Prognosis. Improvement begins anywhere from a few weeks to 3 months after treatment, and the maximum degree of recovery may not be obtained for a year or longer. Recovery proceeds very slowly and is governed by both the inherent slowness of recovery of damaged brain tissue and the extent of damage present (Allen, 1994). Once Korsakoff's disease symptoms are established, complete or almost complete recovery occurs in approximately 20% of patients. A few patients show no recovery. The majority fall somewhere between slight and almost complete recovery (Carlen & Neiman, 1990; Victor, 1993). The syndrome is preventable.

The Dementia Workup

The workup for establishing a diagnosis of dementia and identifying its cause consists of a detailed medical and family history, a physical examination, a full mental status examination, and biochemical studies. Computed tomography or magnetic resonance imaging, neuropsychological evaluation, and additional diagnostic procedures may also be indicated in selected cases (Katzman, 1992; National Institutes of Health Consensus Conference, 1987). Even with this extensive workup, a definitive diagnosis may not be reached, and follow-up examinations for dementia may be required. A period of several months should be allowed between these evaluations to detect any progression or emergence of symptoms. Table 18–11 summarizes the dementia workup.

HISTORY

General Considerations. In the early stages of a dementing illness, many patients maintain their social skills and conversational abilities. Patients may be unaware of symptoms or deny that there is a problem. It is essential when taking a history to have someone present who knows the patient well to verify the patient's responses and supply additional information. If possible, clinicians should interview the patient with a family member present and also see the patient and the family member separately. This process gives the advantage of revealing the interactions between the patient and the family member and also provides each family member an opportunity to discuss information and concerns that he or she may be reluctant to divulge in the other's presence. The content of the history is listed in Table 18–11.

When working with the older population, it is of the utmost importance to be empathic, patient, and observant. Interviewers must wait for the patient's response to questions. Interviewers must *look as well as listen,* noting

TABLE 18-11
The Dementia Workup

History	Diagnostic Tests
Chronological account of current problem Onset Duration Specific cognitive, memory, and behavior changes Changes in functional status	Standard Complete blood count Electrolyte panel Screening metabolic panel Thyroid gland function tests Vitamin B_{12} and folate levels Syphilis and additional tests based on history: Human immunodeficiency virus titer Lyme disease antibody titer Urinalysis Electrocardiogram Chest roentgenogram
Medical history Relevant systemic diseases Trauma Surgery Psychiatric disorders Nutrition Alcohol and substance abuse Exposure to environmental toxins Medications—prescribed and nonprescribed	Other Neuropsychological evaluation Imaging Computed tomography (without contrast) Magnetic resonance imaging Positron-emission tomography Single photon emission computed tomography Electroencephalogram Lumbar puncture
Family history Dementia Down's syndrome Psychiatric disorders	
Physical Examination	
Neurological examination	
Mental status examination	

Data from Katzman (1992); Mayeaux & Schofield (1994); National Institutes of Health Consensus Conference (1987).

whether the patient is easily distracted and whether he or she can comprehend the question but is having difficulty with expressive speech. Does the person look depressed or restless and unable to sit still? Are there myoclonic movements, tremors, or gait disturbances?

Questions. Table 18–12 outlines some of the key questions asked in the dementia assessment. The dementia workup should include a detailed medical, social, and family history as well as a review of systems to identify infectious, metabolic, cardiovascular, and nutritional problems. Taking a detailed inventory of medications is also important, as they have long been known to cause fatigue, apathy, mental slowing, and confusion in the elderly (U'Ren, 1987).

Establishing the date of onset of symptoms may be difficult. Patients are often not aware of the changes, and family members may not note the early deficits or changes until the symptoms have become more pronounced. Nevertheless, it is important to use specific questions to establish the date of onset as closely as possible, especially when there may be more than one condition present. A good way to assist families in establishing a date of onset is to ask of them, "Given what you now know about the individual's problems, think back and try to remember when you first may have seen changes but didn't think they were serious."

FUNCTIONAL STATUS

Three areas are crucial in assessing the patient: (1) cognitive state, (2) the ability to perform basic and instrumental activities of daily living, and (3) the frequency and quality of emotional symptoms and behaviors. Patients' strengths as well as weaknesses must be sought and used to assist them in compensating for their deficits. Patients need to maintain some power over their lives. Cognitive dysfunction does

TABLE 18-12

Questions for Assessing Dementia

Is there a history of hypertension, transient ischemic attacks, syncopal episodes, strokes, or cardiovascular disease?
Is there a family history of dementia, depression, psychiatric illness, or other neurological disorders?
Psychiatric signs and symptoms: Is there evidence of depressive symptoms, a sad mood or affect, lack of energy, loss of interest and enjoyment in former activities, changes in sleep patterns, loss of weight, loss of appetite?
Is there a past history of depression or psychiatric disorders?
Has the individual experienced any recent major losses, environmental changes, or traumas?
Have there been any changes in memory, intellect, or personality?
Did the memory loss come before the depression?
Did the personality changes occur before or after the stroke?
What medications is the patient taking?
What over-the-counter preparations and vitamins are used?

not mean a lack of feeling, and patients should be allowed to do as much as possible for as long as possible. Table 18–13 illustrates specific areas of functional ability that should be included in the assessment (see also Chapters 7 and 9).

FAMILY AND SOCIAL SUPPORT

Because the patient with a dementing illness increasingly relies on family support and assistance, assessment must of necessity include the family (see also Chapter 8). The five most critical areas to be covered when performing a family assessment are as follows:

1. Who is the family?
2. Who is the primary caregiver?
3. What is the family history?
4. What are the family coping styles?
5. What is the meaning of the illness and its symptoms?

PHYSICAL EXAMINATION

The physical examination should include a detailed neurological assessment to evaluate the presence or absence of focal neurological signs and symptoms and to assist in defining the cause of the dementia (Table 18–14). Patients in the early stages of AD are comparatively free of neurological changes, apart from the occasional presence of snout reflex, rigidity, and myoclonus. These symptoms may also be seen in nondemented elderly people (McKhann et al., 1984). As the disease progresses, myoclonic movements and rigidity increase, and patients in the later stages may develop seizures. MID patients show a higher frequency of focal neurological signs and symptoms and higher scores on the Hachinski Ischemic Rating Scale than patients with AD (Hachinski et al., 1974).

MENTAL STATUS EXAMINATION

The mental status examination plays a key role in the diagnosis of dementia. It is especially important during the early stages of impairment. Patients in the very early stages of cognitive decline may be able to carry on a normal conversation and answer questions about their personal history during an interview but may nevertheless show cognitive deficits on a mental status examination (Katzman, 1992; Sagar & Sullivan, 1988). The main components of the

TABLE 18-13

Functional Activities Assessment

Writing checks, paying bills, keeping records
Assembling tax records, making out business and insurance papers
Shopping alone for clothes, groceries, and household necessities
Playing a game of skill, such as bridge, other card games, or chess
Heating water for coffee or tea and turning off the stove
Preparing a balanced meal
Keeping track of current events
Paying attention to and understanding a television program, book, or magazine
Remembering appointments, family occasions, and medications
Traveling away from the neighborhood

TABLE 18-14
Neurological Examination

Cranial nerves II–XII
Motor (tone, bulk)
Sensory (pinprick, fine touch, position, vibration)
Cerebellar (finger to nose, heel to shin)
Frontal lobe (*Suck*—tap upper or lower lip. *Snout*—tap center of closed lips. *Grasp*—place fingers in patient's hand between thumb and forefinger. *Palmo-Mental*—scratch or stroke palm from base of thumb to wrist. *Glabellar*—tap root of nose.)
Gait (standard, tandem, Romberg)
Deep tendon reflexes and plantar response
Speech
Facial expression
Tremor at rest
Action or postural tremor of hands
Rigidity
Finger taps (tap thumb with index finger in rapid succession)
Hand movements (open and close hands in rapid succession)
Leg ability (tap heel on ground rapidly, picking up entire leg)
Arising from chair (rising from straight-backed chair with arms folded across chest)
Posture
Posture stability
Body bradykinesia and hypokinesia (slowness, hesitancy, decreased armswing, small amplitude, general poverty of movement)

mental status examination include orientation, memory, calculation, information and speech comprehension, concentration, and constructional ability. The goal is to not only identify the presence of cognitive deficits but also quantify the changes and establish a baseline to measure further changes over time. The basic strategy of the mental status examination is to start with very simple questions and advance to more complex questions that test the patient's basic cognitive abilities (Katzman, 1992).

Short-term memory and concentration are usually the first areas to be affected in individuals with dementia; other symptoms increase in severity and number as the condition progresses. In the very early stages of dementia, some highly trained individuals with premorbid intelligence quotients in the above-average range can continue to score well on a mental status examination. Impairment in one area by itself does not indicate the presence of dementia. Decline from a premorbid level of function over time is more indicative of a progressive dementing illness than an isolated deficit. Many reliable and validated mental status examinations are available, ranging from the simple to the complex (see Chapter 7); of these, the Folstein Mini-Mental State Examination is the most frequently used by both clinicians and researchers (Folstein et al., 1975; Katzman, 1992). The clock test is also a useful screening instrument for identifying people with AD (Tuokko et al., 1992).

LABORATORY TESTS

The standard laboratory workup for dementia includes a complete blood cell count, electrolyte panel, metabolic screen, and syphilis serology (see Table 18–11). The use of additional laboratory tests depends on the history and clinical findings (Kane et al., 1994; Katzman, 1992). Blood chemistries should be obtained to identify abnormalities that could be contributing to or are the cause of the dementia. Elevated calcium levels and abnormal liver and kidney function can cause memory deficits. Abnormal thyroid function can contribute to memory loss and confusion. Correcting the abnormality may clear or improve the patient's difficulties. In the past, syphilitic dementia was a more common problem; however, the possibility of syphilitic infection should still be ruled out. Obtaining cerebral spinal fluid by means of a spinal tap should be considered where there is some question of infection, malignancy, or toxic substances contributing to the dementia (Berkow et al., 1995; McKhann et al., 1984).

OTHER DIAGNOSTIC TESTS

Neuropsychological Testing. Mental status examinations and neuropsychological testing are essential components of the dementia workup. Brief mental status tests such as the Folstein Mini-Mental State Examination are screening instruments used to detect cognitive impairment. Neuropsychological testing clarifies and refines the presence of cognitive impairment and provides additional information for the differential diagnosis of dementia. In addition, neuropsychological tests, because of

their increasing complexity, are used to identify less severely impaired individuals. Patients may score normally on a Mini-Mental Status Examination, for example, but may show widespread cognitive impairment when tested by detailed neuropsychological examinations (Sagar & Sullivan, 1988). When used in longitudinal assessment, these tests can measure the progression of a disease through comparison with the individual's previous performance on the same test measures. Any correlations between clinical changes, test performance, and the findings of scanning or imaging procedures can be helpful in further identifying areas of deficit and in establishing a diagnosis in cases with atypical symptom presentation (Cronin-Golomb et al., 1993; McKhann et al., 1984).

Neuropsychological testing consists of a comprehensive battery of tests measuring various specific aspects of cognition, memory, language, sensory perception, motor function, visuospatial performance, and psychiatric evaluation (Cronin-Golomb et al., 1993; McKhann et al., 1984). More detailed information and standardized method summaries are provided in LaRue (1992), Lezak (1995), and Kolb and Whishaw (1985).

In progressive dementias such as AD, the range of deficits extends from a mild memory impairment to global brain failure virtually incompatible with life. This wide range creates an additional problem in testing patients over time and at different stages of the disease, as it is difficult to use a single clinical tool over the entire course of the illness. The Alzheimer's Disease Assessment Scale was developed specifically to evaluate the severity of cognitive and noncognitive behavioral dysfunctions characteristic of individuals with AD (Rosen et al., 1984). This standarized instrument is widely used in reporting AD research.

Imaging. Computed tomography and magnetic resonance imaging remain the most commonly used screening tools in establishing a diagnosis of dementia. Positron-emission tomography (PET) and single photon emission computed tomography (SPECT) add important data on the pathophysiology of dementia and assist in the differential diagnosis of more atypical presentations; their cost and availability, however, prevent their use in wide clinical applications at this time. Their ability to measure physiological changes in the brain over time may prove invaluable in the development of new treatment approaches. The electroencephalogram is useful in establishing a diagnosis of dementia in cases with atypical presentations and in clarifying the cause of a dementia.

Treatment Approaches

ALZHEIMER'S DISEASE

At present, there are no known cures for AD. Drug treatment for AD is divided into two types: *symptomatic treatment,* which concerns the management of emerging symptoms, and *specific drug therapies,* which focus on the presumed cause of the basic disorder (Hollister, 1985; Katzman, 1992).

Symptomatic Treatment. Medications can be used to achieve either simple control or the symptomatic relief of behaviors such as aggression, agitation, restlessness, hyperboisterousness, hyperactivity, verbal hostility, and insomnia. Antipsychotic medications can be used to provide symptomatic relief of anxiety, depression, regressed behavior, hallucinations, and delusions (Table 18–15).

Patients with AD are acutely sensitive to central nervous system neurotransmitter alterations; small changes can produce significant effects. Many AD patients have idiosyncratic reactions to medications (Omar et al., 1995; Thornton et al., 1986), and thus medications must be initiated at very low doses and carefully titrated. Less emphasis should be placed on dosage than on the individual patient's clinical response (Maletta, 1990). Some behaviors can be managed or decreased by means of behavioral and environmental changes; these approaches should always be tried first and before medications. A concomitant medical illness or the use of contraindicated drugs must be determined before beginning medications. Clinicians should determine whether the patient had these behavioral problems in the past, whether any medications were effective, and if any side effects developed.

Neuroleptics remain the standard treatment for agitated behavior in patients with AD. Hal-

TABLE 18-15

Medications Used to Provide Symptom Relief

Drug (Generic)	Drug (Brand Name)	Daily Dosage
Antidepressants		
Sertraline	Zoloft	25–150 mg
Paroxetine	Paxil	10–50 mg
Desipramine	Norpramin	25–150 mg
Bupropion	Wellbutrin	75–300 mg
Fluoxetine	Prozac	5–20 mg
Nortriptyline	Aventyl or Pamelor	10–50 mg
Trazodone	Desyrel	25–200 mg
Sedative-Hypnotics		
Temazepam	Restoril	7.5–15 mg
Lorazepam	Ativan	0.5–1 mg
Zolpidem	Ambien	5–10 mg
Antipsychotics		
Haloperidol	Haldol	0.25–2 mg twice a day
Thiothixene	Navane	1–15 mg
Clozapine	Clozaril	75–100 mg
Risperidone	Risperdal	0.5–6 mg
Olanzapine	Zyprexa	2.5–20 mg
Quetiapine	Seroquel	100–250 mg
Antianxiety Agents		
Diazepam	Valium	2 mg every day or twice a day
Lorazepam	Ativan	0.5–1 mg twice a day
Alprazolam	Xanax	0.25 mg two or three times a day
Agents for Agitation or Aggressivity		
Buspirone	Buspar	5 mg twice a day
Trazodone	Desyrel	50 mg
Clonazepam	Klonopin	0.25–0.5 mg twice a day
Carbamazepine	Tegretol	50–100 mg twice a day
Valproate	Depakote	125–250 mg

Data from American Psychiatric Association (1997); Dubovsky (1994); Grossberg & Kumar (1998); Ham (1997); Jeste et al. (1998); Salzman (1998), and Small et al. (1997).

operidol (Haldol) is frequently used to control behavioral problems and has also been used with some success when augmented by buspirone (Buspar) (Caudiex, 1993). In very small doses, Haldol can be effective; however, some patients respond by becoming increasingly agitated, with increasing dosages only escalating the problem. Parkinsonian symptoms can become marked with the use of haloperidol, and the patient can rapidly become severely dysfunctional (Maletta, 1985; Mayeux & Schofield, 1994). Because of these side effects, the newer atypical antipsychotics should be the first drug of choice in this population. Risperidone (Risperdal) and olanzapine (Zyprexa) are the two most widely used. Clozapine (Clozaril) needs to be used with caution owing to the side effect of agranulocytosis, requiring weekly blood counts (Ham, 1997). A rule of thumb in drug therapy with these patients is to use the smallest possible doses and to temporarily discontinue the drug on a regular basis to evaluate response (Mayeux & Schofield, 1994).

Specific Drug Therapies. Specific drugs for AD are intended to treat the disease itself. Table 18–16 lists the specific strategies and medications used in treating AD. Most of these treatment approaches were conducted as clinical drug studies that strictly followed regulated research protocols. Although some studies found small changes in attention, mood, and concentration, there unfortunately have not been any major breakthroughs. Most drug trials are based on animal studies, and although some drugs appear promising in animal trials, the results have not been duplicated in humans. The recent development of transgenic mice with amyloid plaques similar to those seen in humans with AD is an important step in developing new treatment approaches.

OTHER DEMENTIAS

The management of dementia resulting from MID or alcohol abuse is focused primarily on treating the underlying condition and eliminating those factors that predispose or directly lead to the problem. The prognosis and quality of life for patients with MID can be improved by controlling hypertension, stabilizing the cardiovascular system, and treating secondary medical conditions or psychiatric disorders (Katzman, 1992; Read & Jarvik, 1984). Anticoagulation therapy and vascular surgery may benefit some patients with MID.

Shunts are sometimes the treatment of choice for patients with normal pressure hydroceph-

TABLE 18-16

Specific Drug Therapies for Alzheimer's Disease

Generic Name	Brand Name	Action
Tetrahydro-aminoacridine, tacrine	Cognex	Cholinesterase inhibitor
Donepezil	Aricept	Cholinesterase inhibitor
Velnacrine maleate	Mentane	Cholinesterase inhibitor
Physostigmine	Synapton	Cholinesterase inhibitor
E-2020		Cholinesterase inhibitor
Linopiridine	Aviva	Enhances release of acetylcholine, dopamine, and serotonin
Nimodipine	Nimotop	Calcium channel blocker
Sabeluzole		Calcium channel blocker; also effects serotonin
Acetyl-L-carnetine	Alcar	Enhances cell energy production—may help protect nerve cells from damage
Phosphatidylserine	Bovice Cortex-Phosphotydal-Serine	Helps maintain cell membrane integrity
L-deprenyl		Selective MAO B inhibitor
Lazabemide		Selective MAO B inhibitor
Desferrioxamine	Desferal	Reduces aluminum levels (chelation therapy)

MAO B, monoamine oxidase type B.

From The Alzheimer's Disease and Related Disorders Association (1992). *Theory to therapy: The development of drugs for Alzheimer's disease.* Chicago, IL: The Alzheimer's Disease and Related Disorders Association.

aly, although statistics show that the success rate for improvement is low and treatment complications are common (Beck et al., 1982; Graff-Radford et al., 1989). Studies indicate that in general, patients best suited for shunting are those who present with the classical triad—gait disturbance, incontinence, and dementia—who have a readily identifiable cause for NPH, and who have a relatively short duration of symptoms (Clarfield, 1989; Reichman, 1994). In alcohol-related dementias, the standard treatments of choice include eliminating alcohol use, improving nutrition, stabilizing the patient's medical status, and using high doses of thiamin, niacin, and vitamin B_{12} (Willenbring, 1988). The pharmacological management of secondary symptoms and behaviors was discussed in the preceding section covering the treatment of AD, and nonpharmacological approaches are discussed later (see *Management of Dementia*).

ALTERNATIVE TREATMENT APPROACHES

Chelation therapy and megavitamin therapy have been promoted as alternative approaches in the treatment of dementias. Statistics have not shown any basis for their therapeutic claims, however, and in fact, the use of megavitamins may cause more difficulties and adverse reactions than improvement (Funkenstein et al., 1981; Thal, 1988). Some preliminary studies (Gold & Stone, 1988; Hall et al., 1989) have shown that circulating glucose may control acetylcholine synthesis and enhance memory in older individuals. Many AD patients develop cravings for sweets, and PET and SPECT data show deficits in glucose metabolism in the brains of these patients. Age-related memory deficits in some elderly individuals might be alleviated by better control of blood glucose levels achieved through dietary control or medications; however, the mechanisms for doing this have yet to be developed. The high intake of glucose on a regular basis has not been effective, and there is no evidence that this would be beneficial in patients with AD. It is important to maintain a balanced nutritional status for the patient, and families should be taught to do so.

Management of Dementia

Dementia is a multifaceted problem. Integrated management focuses not only on the individual but also on the family, the environment, and the community. Assessment and planning must begin early, and planning, implementation, and evaluation should be concurrent and

longitudinal. Because of the complexity of the problem, a multidisciplinary approach should be used to provide diagnosis, pharmacological management, nursing management, and resource management. In working with this population, a commitment to long-term relationships among the patient, family, staff members, and health professionals is essential. Learning to live with a dementing illness is also learning to live with uncertainty. The course of the disease varies among individual patients and may be unpredictable. Families frequently ask the following questions: How long will the patient live? How soon will changes take place? Will the patient become assaultive? Will he or she wander? Will he or she hallucinate? Will our children get the disease? Unfortunately there are no firm answers. Although the outcome of dementia is known, many factors and variations can influence its progression. Some patients have a more gradual decline with periods of plateauing, whereas others have a more rapid rate of decline with more severe behavioral changes.

The responsibilities of the advanced practice nurse are threefold: assessment, planning, and the coordination of care. Plans must be realistic, tailored to the individual, consistent with the psychosocial environment, and financially feasible. Because of the progressive nature of the disease, planning should anticipate and accommodate the patient's decline, with the support system increasingly activated to the escalating need for assistance (American Medical Association Council on Scientific Affairs, 1993; Eisdorfer & Cohen, 1981; Teri & Logsdon, 1990).

TABLE 18-17
Communication Techniques

Avoid arguments—dementia patients reason by a different set of rules and arguments, usually resulting in aggravation for both parties.
Use distractors—the patient's memory deficits can frequently be used to advantage to distract and redirect attention.
Communicate nonverbally—by touching or holding hands.
Praise desired behavior—and make it emotionally meaningful.
Ignore undesired behaviors—only if it is safe to do so.

Data from Thornton et al. (1986).

TABLE 18-18
Environmental Safety Checks

Keep environment simple; remove knickknacks or clutter that may distract or confuse
Remove items that can prove dangerous (e.g., car keys, knives, iron, power tools, matches, cleaning solvents)
Keep all medications out of reach and under lock and key
Install gates on stairs; check handrails
Install grab-bars
Reduce temperature on water heater
Remove rugs that slip
Remove glass tables and unsteady or delicate furniture
If possible, arrange an outside area that is safe and accessible where the patient can walk or sit
Put bright reflector tape on steps

Data from Mace & Rabins (1991).

BEHAVIORAL MANAGEMENT

There are no magic tricks for dealing with problem behaviors. Dementia patients are extremely sensitive to nonverbal cues and tend to mirror the affective behavior of those around them. Nurses can serve as role models to other caregivers in helping to soothe the patient; patience, gentleness, and calmness can be very effective. Models such as the Progressively Lowered Stress Threshold (Hall et al., 1995) can be used in planning and evaluating the management of behavioral symptoms in these patients. Table 18–17 lists effective communication techniques, and Chapter 27 provides additional information on managing problem behaviors.

Although all dementia patients exhibit some form of behavioral disturbance, the exact type and extent of behavioral problems are quite variable. A specific and thorough behavioral assessment is essential for identifying and treating behavioral problems in AD patients (Teri & Logsdon, 1990). Supportive psychotherapy may be helpful in disrupting a cycle of frustration, anger, anxiety, and rage in patients with mild to moderate dementia (Haggerty, 1990; Verwoerdt, 1981).

SAFETY

Individuals with a dementing illness experience changes not only in memory and learning but also in visuoperceptual areas, smell, taste, and balance and gait. Dysphagia is frequently a problem. Table 18–18 notes areas of environmental safety that must be assessed and taken into consideration when caring for these patients.

CAREGIVER EDUCATION AND SUPPORT

Families must be taught to respond to their loved ones' catastrophic and massive emotional overresponses to minor stress; they should remain calm and remove the patient from the threatening situation. Caregivers must learn to work on specific solvable problems within a general framework. By developing strategies for dealing with problems in advance, caregivers can gain a measure of control and thus not approach each problem behavior as a crisis. This also helps stabilize the environment for the patient. Teaching families to remain calm and improve nonverbal communication often helps reduce behavioral disturbances in the patient (Thornton et al., 1986). Responding to feelings rather than to the content of the patient's words is an important practice for caregivers and families and an effective way of dealing with the frequent repetition of questions.

Fear and uncertainty cause caregivers to dread the future. Families can accept uncertainty and be better prepared for emergencies by learning about the disease; learning how to handle specific contingencies as they arise; breaking down dilemmas into smaller, solvable problems; and, most important, taking care of themselves by allowing others to provide support for the patient. Learning that a family member has AD or a related dementing illness is shocking, but once families have made the appropriate legal and financial adjustments (see Chapter 3), they should be encouraged to focus not on the disease and its disabilities but on positive experiences. With early diagnosis and supportive therapies, patients are capable of enjoying many family activities and intimacies for a considerable period. It is tragic when fear and anxiety in both the patient and the family are allowed to deprive them of these years.

Counselors and support groups should be made available to all patients with dementia and their families. For these individuals, there is nothing worse than to be told that "there is nothing to be done" and to be left without any additional support. Families are often tremendously relieved and helped by meeting others who are sharing the same experiences and problems. Several nationally and regionally organized groups provide information, assistance, and regularly held support meetings throughout the country. These groups are often tailored to the type of caregiver or the setting of the care recipient. One of the most helpful things clinicians can do for patients and families is to put them in contact with these groups. More than one group is frequently required as patients and their families move along the various stages of the disease process. The Alzheimer's Disease Association, a national organization with chapters in most major areas of the country, is an excellent resource for information regarding support groups and services (see Appendix 18–1).

Impact of a Dementia Diagnosis

People who have a dementing illness must cope simultaneously with associated psychopathological states, such as depression, anxiety, and paranoia, as well as a number of losses. The loss of memory, loss of a job, loss of a role as head of a household or breadwinner, and loss of a future are only a few of the losses experienced by individuals with a dementing illness. In addition, these people must cope with the reactions of others toward them, perhaps overprotective spouses, or distant children and friends. They may feel embarrassment and shame. The nuclear family may experience fear, anger, helplessness, and hopelessness. Feelings of abandonment and isolation are frequent. Criticism by relatives is a common occurrence, wherein they have difficulty accepting the diagnosis and try to rationalize the patient's symptoms in the earlier

stages of the disease by attributing blame to family members. The patient may deal with fears and anxiety by becoming verbally and physically abusive toward family members.

Spouses are in social limbo; they cannot mourn decently because the patient is still alive and frequently shows few outward signs of change. They cannot divorce with dignity, even if they wish to do so, and are left with no acceptable outlets for their sexual frustrations and emotional needs.

Young children living in the home are frequently ignored by the patient and neglected by the caregiver, who is mentally and physically drained in attempting to cope with the meaning of the diagnosis and the changes in the patient. The patient may bully the children or vie for attention with them. This is seen more often in fathers whose teenage sons try to assume the parent's role and are viewed as a threat by the patient. Younger children frequently incorporate and misinterpret the family's frustrations, fears, and self-recriminations (Lezak, 1978).

Older children living away from home must cope with feelings of guilt, fear, and loss. Torn between conflicting needs, they may become actively involved or distance themselves from the parent and the disease. Some may become oversolicitous to obtain previously unreceived love from a parent. As the disease progresses in the parent, the adolescent and young adult children engage in a sociopsychological process with distinct stages and phases. The stages of this process of sequential resolving are as follows (Davies et al., 1988):

1. Awareness, in which the child comes to realize that something is truly wrong
2. Exploration, in which the child seeks a reason for the parent's behavior and deals with the physical and psychosocial consequences of the diagnosis
3. Definition, in which the child separates the *patient that is* from the *parent that was*

Factors that influence the adjustment process are family dynamics and each child's age, developmental stage, and personal coping style. Compounding these difficulties, the disease can affect insight, judgment, and decision-making abilities. Attempts by caregivers and professionals to deal with evolving deficits may be viewed by the patient as obstructive, interfering, and manipulative (Lezak, 1978).

FAMILY STAGES AFTER DIAGNOSIS

A diagnosis of dementia in a family member results in many changes in the family system. Roles, family expectations, and alliances are altered. In order to cope, families must learn to deal with many new demands. This author's clinical practice has identified several stages that families must move through following a diagnosis; Table 18–19 delineates these stages.

The assessment of a family's ability to care is essential. This assessment should include (1) the organization or disorganization of the family system; (2) individual family members' levels of sophistication about medical and psychological problems; (3) the degree of cooperation; (4) the existence in family members of major chronic or recurring physical illness; (5) the presence of psychiatric problems in any family members; and (6) the dynamics of patient interaction with individual family members (Cohen, 1994).

INSTITUTIONALIZATION AS A FAMILY CRISIS

The decision to seek nursing home placement frequently presents a crisis in the family. Family members often disagree as to the need for and timing of placement. Feelings of anger, guilt, self-recrimination, and failure are common. Most people, particularly older people, have a fear of and deep aversion to nursing homes; in addition, patients often extract promises from their spouses and families that

TABLE 18-19

Family Stages After Diagnosis of Dementia

Coping with diagnosis
Beginning to look at management
Deciding what to do
Coping maximally
Making the transition to institutionalization
Making the decision
Acting on the decision

they will never be placed in a nursing home. Clinicians, therefore, are often faced with a situation in which a spouse insists on caring for the patient at home even though the patient's needs for care far exceed the resources of the caregiver or family—and even if in reality this situation is against both the patient's and family's best interests (Meier & Cassel, 1986). This situation is highly complex for the family and may be compounded by financial issues, a scarcity of resources, and the clinician's own biases and feelings. The sense of guilt and personal responsibility felt when contemplating nursing home placement can be devastating, especially if there is disagreement within the family as to what should be done.

Clinicians should be aware of all these factors when assisting families in making the decision to institutionalize. Important questions to be considered are the patient's needs and preferences (if known), the caregivers' needs, and the needs of the overburdened family. Have all other alternatives been thoroughly explored? Emotional support, reassurance, and assistance in evaluating existing resources are essential at this time. The use of respite and hospice care is gaining acceptance and may provide an alternative to institutionalization. It is helpful to let the patient and family know that they will not be cut adrift from their previous support systems. Ideally, the issue of possible future placement should be discussed with the patient and family early in the disease process when the patient may be able to discuss concerns and preferences and designate a surrogate decision maker.

SUMMARY

Delirium and dementia are major concerns in geriatric care. They represent two of the most common mental disorders encountered in older adults. Basic and clinical research have resulted in improved diagnostic methods and the clinical management of delirium and dementia.

Delirium is a neuropsychiatric syndrome characterized by reversible changes in attention (clouding of consciousness) and cognition. Onset is relatively rapid and the course typically fluctuates. Some patients may have increased levels of arousal accompanied by hallucinations and delusions, whereas others may appear confused, drowsy, or stuporous. The most consistent high-risk factors for developing delirium are age, concurrent cognitive impairment (dementia), and drug toxicity. Clinical examination and consistent observation are the most important factors in detecting and diagnosing delirium. Delirium can present as a life-threatening situation and should always be treated as a medical emergency.

Dementia is a devastating problem for everyone concerned. A conservative estimate of the cost of AD to society is about $90 to 113 billion per year in medical bills, nursing home costs, homecare costs, and lost productivity (Cramer, 1993; Cummings & Mega, 1996). Prolonging patients' stay in the community and providing social support systems for the family add to the quality of life for both the individual and family. But prolonging the inevitable is not enough. With the rapid changes occurring in the healthcare system, widely recognized standards of care for dementia patients and their families are essential and are currently being developed (California Workgroup for Alzheimer's Disease Management Guidelines, in progress). Additional research is needed for effective treatments, including the prevention of underlying causes of the disorder.

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APPENDIX **18 - 1**

Resources

ORGANIZATIONS

Alzheimer's Disease and Related Disorders Association (ADRDA)
919 N. Michigan Avenue, Suite 1000
Chicago, IL 60611-1676
Phone: (800) 272-3900

The ADRDA is an excellent source of help for patients and their families. The national office maintains an 800 number and provides informational material on specific questions of concern to a caregiver or family and refers them to the local chapter of the association. Local chapters provide direct information from experienced caregivers or staff members and maintain a variety of support groups and services.

Alzheimer's Disease Education and Referral Center
P.O. Box 8250
Silver Springs, MD 20907
Phone: (800) 438-4380
http://www.alzheimers.org/adear

Excellent source for current information on all aspects of Alzheimer's disease.

AREA AGENCIES ON AGING

In addition to voluntary organizations, state governmental agencies such as the local area agencies on aging are often involved in providing information and assistance to dementia patients and their families. To identify resources in your community, contact the National Association of Area Agencies on Aging, phone: (202) 296–8130

DIAGNOSTIC AND TREATMENT CENTERS

In a number of states diagnostic and treatment centers have been established. Most are affiliated with University Medical Centers. In addition to diagnostic evaluations these centers provide social services and other related resources.

ALZHEIMER'S DISEASE RESEARCH CENTERS

A number of centers are supported by grants from the National Institute on Aging and the National Institute of Mental Health. These centers have a responsibility to provide education to professionals and caregivers. Most centers also provide a variety of formal courses open to family members as well as professionals.

BOOKS

Mace, N. L., & Rabins, P. V. (1991). *The 36 hour day* (2nd ed.). Baltimore: Johns Hopkins University Press.
Written for caregivers. Covers all aspects of Alzheimer's disease and is an excellent resource for managing the day-to-day care of dementia patients.

Aronson, M. K. (Ed.). (1988). *Understanding Alzheimer's disease.* New York: Charles Scribner's.
Covers all aspects of the disease, the diagnostic process, and management of the dementia patient. Appropriate for both lay members and professionals. Recommended as a first line of information for newly diagnosed patients and their families.

Cohen, D., & Eisdorfer, C. (1987). *The loss of self.* New York: New American Library.
Lends additional insights into the psychological issues of Alzheimer's patients and their families.

Davies, H. D., & Jensen, M. P. (1998). *Alzheimer's: The answers you need.* Forest Knolls, CA: Elder Books.

Written for individuals in the early stages of Alzheimer's disease and their caregivers. The book provides helpful answers, in an easy-to-read format, to the questions frequently raised by newly diagnosed individuals.

Gwyther, L. P. (1985). *Care of Alzheimer's patients: A manual for nursing home staff.* Chicago: American Health Care Association and ADRDA.

Care guide for nursing home staff; a useful resource for caregivers and professionals.

A wide variety of other educational materials is also available, including video and audio for professionals and caregivers. The ADRDA maintains a current list.

0.01), disposition (change in $R^2 = 0$) and discharge BI (change in $R^2 = 0.02$). These results sugest that factors other than age determine LOS and functional outcomes in stroke rehabilitation. This finding has broad implications regarding functional expectations and costs/benefit issues in rehabilitating older stroke patients.

● 47 Correlation of Geriatric Brain Weight with Age, Sex, Race, and Body Parameters

Khang-cheng Ho, MD, PhD, Uros Roessmann, MD, Ray Hoffman, PhD, Theodore MacKinney, BS, Lawrence Hause, PhD, Alfred J. Anderson, MS, Departments of Pathology, Biostatistics, and Medicine, The Medical College of Wisconsin, Milwaukee, Wisconsin, and the Department of Pathology, Case Western Reserve University, Cleveland, Ohio

Data on brain weight, age, gender, race, body weight, height, and body surface area were obtained for individuals more than 65 years old at the time of death from autopsy reports over a ten-year period. Brains with lesions that substantially affect brain weight were excluded. A total of 1,491 cases were analyzed. Mean brain weights were 1,314 g, 1,250 g, 1,184 g and 1,119 g for white males, black males, white females, and black females, respectively. The differences in brain weights between all four subgroups were statistically significant. When brain weight was adjusted by body parameters, the differences remained unchanged, with few exceptions. There was significant brain weight loss with age for all subgroups beyond 65 years old. The rate of decline was greatest for white males, followed by white females, black males, and black females, although the differences between subgroups were not statistically significant. The average loss of brain weight for those between the ages of 65 and 99 was 3.45 g/yr, 2.5 times greater than that between 25 and 65 years of age found in a previous study. The decline of brain weight was greatest after age 80. The rate of brain weight loss was less than that of body weight loss in the elderly group, contrary to the findings in the younger group from the previous study. This study has defined the brain weight and its rate of decline in the elderly in relation to age, gender, race, and body parameters. In analyzing geriatric brains, especially for brain atrophy, these factors should be considered.

● 48 Neurosyphilis and Dementia

Stephen L. Read, MD, Steven S. Matsuyama, PhD, Bruce L. Miller, MD, Department of Psychiatry, UCLA and West Los Angeles Veterans Administration Medical Center, Brentwood Division, Los Angeles, California

Although Alzheimer disease is a specific clinicopathologic entity, diagnosis of dementia of the Alzheimer type (DAT) has been approached largely by the presence of a progressive dementia with the exclusion of underlying causative medical illness. One such illness that must be "ruled out" is neurosyphilis, the fabled "great imitator." Guidelines developed by the American Psychiatric Association in conjunction with DSM III criteria recommend the use of the serum VDRL for this purpose, although there are reports of active neurosyphilis with a negative VDRL reaction. Case material from an ongoing longitudinal study of Alzheimer-type dementia suggests that this recommendation is inadequate. Failure to identify neurosyphilis is a grievous error. Three measures would produce fewer misdiagnoses of DAT in lieu of specific laboratory tests for that disease: 1. the development of more restrictive phenomenologic criteria for DAT, 2. use of a more sensitive and specific test such as the fluorescent treponemal antibody (FTA) or the microhemaglutination assay for *Treponema pallidum* (MHA-TP) instead of the VDRL, 3. evaluation of the CSF in the work-up of dementia.

● 49 Longitudinal Mental Status Examinations in Patients with Early Dementia

Joe E. Thornton, MD, Helen D. Davies, RN, Cecelia A. Peabody, MD, Terry Miller, MD, Jared R. Tinklenberg, MD, Palo Alto Veterans Hospital, and Stanford University Medical Center, Stanford, California

The authors are compiling, for exploratory data analysis, a retrospective longitudinal clinical data base on a microcomputer system. They discuss findings and changes on individual items of the "Folstein Mini Mental Status Exam" (MMS) over time. The individual items are orientation for time, orientation for place, registration, attention, recall, name, repeat, follow three-part command, read, write, and copy.

The data base at this writing consists of 25 patients who have participated in studies for early dementia. There are 20 males, average age 65, and five females, average age 62. All patients were living in the community and able to take care of most of their activities of daily living. Testing was done every six months. On initial examination the average MMS score was 20 with a range of 10–29. All patients have been followed for at least six months to a year. Twelve patients have been followed for over a year, and six for over two years.

Examination of the exploratory data suggests that the progression of dementia in the ambulatory phase is slower than emphasized in the literature and the morbidity of early dementia is underestimated.

Functional examinations over time with instruments such as the MMS may be useful in correlating clinical findings with the distribution, if not the severity, of pathologic changes in the brain.

● 50 Systematic Screening and Treatment for Depressive Symptoms in Elderly Outpatients: Effects on Depressive Symptoms and Functioning

John P. Fulton, PhD, Dennis H. Novack, MD, Joan Rothney, ACSW, Michael Brown, BA, Jill Summerfield, BA, Marsha D. Fretwell, MD, Tom J. Wachtel, MD, Brown University Program in Medicine, Providence, Rhode Island

Although depressive symptoms are common among elders, they are frequently over-looked or misinterpreted, and may lead to dysfunction and loss of independence. The authors designed a system to screen, refer, and treat elderly outpatients for depression. Of 75 randomly selected elders screened with the Center for Epidemiologic Studies Depression (CES-D) scale, 28 (37 per cent) were found to have significant depressive symptoms (scores of 16+), and were referred to a psychiatric social worker for assessment. In collaboration with an attending physician, the following diagnoses were made: major depression (n = 2), dysthymic disorder (n = 9), and cognitive impairment (n = 12). After discussing diagnoses with primary care physicians, the social worker designed and implemented interventions for the 28 patients. A year later, 60 of 75 of the elders thus screened were re-interviewed (80 per cent follow-up). No one refused follow-up. The 15 patients not interviewed had died or could not be contacted. For the 40 follow-ups who had had CES-D scores of less than 16 initially (time 1), the mean CES-D score changed from 6.4 (t_1) to 8.0 (t_2), $P = 0.14$, paired t-test. For the 20 treated follow-ups who had had CES-D scores of 16+ at time 1, the mean CES-D score decreased from 24.0 (t_1) to 17.4 (t_2), $P = 0.05$, paired t-test. Nine of the 20 (45 per cent) had CES-D scores of less than 16. Using indexes developed at Brown University, functioning was assessed at times 1 and 2 for the 60 follow-ups in the following dimensions: self-care, fine motor skills, lower limbs, stamina, vision, hearing, and thinking (alpha-reliability between 0.8 and 0.9 for all indexes but hearing, = 0.69). Function remained unchanged in the untreated