Cognitive-Communication Disorders of MCI and Dementia

Definition, Assessment, and Clinical Management

Third Edition

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Clinical Management Guide: Assessments and Interventions for Cognitive-Communication Disorders of Dementia

Preface

Since publication of the second edition of *Cognitive-Communication Disorders of Dementia*, a tsunami of dementia research has been published. More than ever, the focus is MCI, or mild cognitive impairment, typically a harbinger of a dementia producing disease. Historically, however, the term MCI was primarily associated with Alzheimer's disease, though scientists and clinicians recognized that all dementing diseases create subtle early cognitive impairments. This problem was remedied with the publication of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (2013). The American Psychiatric Association introduced the term "minor neurocognitive disorder" to refer to mild cognitive impairments that are insufficient to be considered frank dementia, and they characterized dementia as a "major neurocognitive disorder." Nonetheless, clinicians and researchers still use the term MCI but with the understanding that it is associated with myriad diseases, and not just Alzheimer's.

Global interest in early identification of individuals with MCI is intense because drug and cognitive stimulation therapies, as well as lifestyle changes, show promise for delaying or preventing development of dementia. *Highly relevant to speech-language pathologists is the well-documented finding that language performance problems are among the earliest signs of MCI*. Moreover, the new recommendations for diagnosing MCI, also known as a "minor neurocognitive disorder," include the evaluation of language. As experts in language and communication science, speech-language pathologists are uniquely qualified to evaluate and treat the cognitive-based communication disorders of MCI and dementia. Thus, in this new edition, greater emphasis is given to the characteristics of MCI, its assessment, and clinical interventions.

The third edition opens with an overview of cognition and communication and why individuals with dementia have serious cognitive-communication disorders. This is followed by a rationale for building cognitive reserve in those with MCI and therapy for individuals with dementia. Chapter 2 provides an overview of cognition, memory, and communication, and how they are interrelated. Thereafter, clinicians and students in training will find up-to-date information about the characteristics of all major dementia-producing diseases and Down syndrome.

The last segment of the book is composed of the new Clinical Guide and is extensive. It begins with a discussion of the process of assessment and a review of reputable tests. Subsequent topics include:

- Cognitive stimulation programming for MCI
- Clinical techniques supported by the principles of neuroplasticity
- Indirect interventions that facilitate communication, quality of life and the safety of individuals with dementia, and caregiver counseling
- Care planning, goal setting, reimbursement, and required documentation

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Speech-Language Pathology, Mild Cognitive Impairment, and Dementia

Introduction

Individuals with mild cognitive impairment (MCI) and dementia are the fastest-growing clinical population, nationally and globally. In fact, every 65 s someone in the United States develops Alzheimer's disease (AD), the leading cause of dementia (Alzheimer's Association, 2018; Ferri, Prince, Brayne, Brodaty, & Fratiglioni, 2005). Currently, 47 million individuals worldwide are believed to be living with dementia; however, by the year 2050, approximately 131.5 million people will have AD or another form of dementia (Alzheimer's Disease International, 2016). More than five million Americans live with AD, and that number may rise to 16 million by 2050. Characterized another way, one in 10 Americans 65 years or older has AD, two-thirds of whom are women. Individuals with dementia need care and an estimated 15 million Americans provide unpaid care. The cost of care in 2018 may exceed $277 billion and a trillion dollars by 2050 (Alzheimer's Association, 2018).

Dementia-associated diseases, such as AD, can begin decades before they are clinically obvious and, once diagnosed, endure for many more years. Most patients are cared for at home by family, typically with serious financial, social, and emotional consequences for all involved. Those patients and families who have the support of professionals have a higher quality of life (Gaugler, Roth, Haley, & Mittelman, 2008; Mittelman, Roth, Coon, & Haley, 2004) and speech-language pathologists (SLPs) are among the professionals who have an important role in the management of affected individuals. Language performance deficits occur early and worsen as the disease progresses. As experts in language and communication science and the evaluation and treatment of communication disorders, SLPs are uniquely qualified to diagnose and treat the cognitive-communication disorders associated with dementia-producing diseases. Additionally, SLPs provide counseling to professional and personal caregivers about how to best communicate with affected individuals.

The goal of this book is to provide practicing professionals and graduate students the knowledge needed to evaluate and treat individuals who have mild cognitive impairment (MCI), AD, or another type of dementia, as well as counsel professional and personal caregivers. Toward that end, the first order of business is answering the question, why do individuals with dementia have a communication disorder. To answer that question, we need to first define communication.

Communication Defined

Communication is the sharing of information by means of a symbol system. We call communication linguistic when words are used and nonlinguistic when other symbol systems are used, such as mathematical notation. To communicate, either linguistically or nonlinguistically, an individual must have an idea to share and a symbol system through which to express the idea. For example, symphony conductors communicate their ideas about tempo and loudness to orchestra members by moving a baton in prescribed ways. Football coaches communicate plays to players by hand signals. These are examples of nonlinguistic communication, and although nonlinguistic communication can be impaired as a consequence of a dementing disease, the focus of the SLP is impairment in linguistic communication. *Nonetheless, both nonlinguistic and linguistic communications are impaired in AD because both are cognitive processes for sharing and interpreting information and information processing is progressively disrupted.* Another distinction critical to characterizing the effects of dementia on communicative function is the difference between "speech" and "language." For our purposes, the term "speech" refers to the motor production of sounds, and the term "language" refers to the symbol system by which sound is paired with meaning for a particular purpose. As previously noted, "linguistic communication" is the cognitive process of intentionally sharing ideas through language and in *dementia it is the ability to communicate that is typically most affected.*

"Meaningful" communication requires the production and comprehension of ideas. The act of speaking, in and of itself, does not constitute communication because that which is spoken may be structurally and semantically meaningless. Similarly, knowing the grammar of a language does not ensure the ability to communicate. Communication only occurs when words are structured in such a way that the listener comprehends the speaker's idea. Having made this distinction, the question of why communication is affected in dementia can be answered. Communication is affected because the pathophysiologic processes that disrupt multiple cognitive functions and produce dementia also disrupt information processing.

Clients with dementia are said to have a "cognitive-communication" problem because progressive deterioration of cognition interferes with communication. The fact is, *the production and comprehension of language cannot be separated from cognition.* Consider just the simple act of naming an object; for example, a parsnip. First, you must perceive the features of the parsnip. They must be matched to those in long-term memory for recognition to occur. Thereafter, you must form an intention to say the object's name. The linguistic representations of objects are part of long-term lexical memory and must be retrieved and brought to consciousness. Perhaps you are uncertain about how a parsnip looks and therefore are unsure whether you are perceiving a turnip, parsnip, or rutabaga. If so, you have to decide whether to indicate your uncertainty. To articulate uncertainty about the object's name or identity, a motor plan must be formed. Thus, the simple act of object naming requires perception, access to long-term memory, association, recognition, lexical retrieval, decision making, motor planning, and self-monitoring.

Persons with dementia have difficulty *producing* linguistic information because the information-processing capabilities of declarative and working memory systems are compromised, as is the case in AD (Hornberger, Bell, Graham, & Rogers, 2009; Rogers & Friedman, 2008), and in part because of progressive degradation of knowledge (Landin-Romero, Tan, Hodges & Kumfor, 2016), as is the case in semantic dementia. They have difficulty *comprehending* language because of deficits in the cognitive processes of perception, recognition, attention, and memory, as well as degradation of knowledge (Macdonald, Almor, Henderson, Kempler, & Andersen, 2001).

Rationale for Therapy

In the not too distant past, clinicians thought little could be done to improve the functioning

of individuals diagnosed with dementia. Early identification of those affected was not the priority it is today; however, as the number of dementia patients skyrocketed, interest in early detection and intervention also skyrocketed. Worldwide, researchers in neuroscience, as well as the behavioral and cognitive sciences, have focused on understanding dementia-associated diseases and their management. Research has revealed that individuals with AD experience subtle cognitive deficits years before the disease impairs their ability to live independently. When these individuals are identified, much can be done to prevent evolution to frank dementia or slow the course of the disease, among them pharmacologic interventions, lifestyle changes, and cognitive stimulation. Consider the following facts that comprise a rationale for intervention:

1. The human brain is plastic and many of the factors that advantage neuroplasticity are known.
2. Humans have multiple systems for learning and information representation that are not equally vulnerable to the pathology of the common dementia-producing diseases and spared systems can be strengthened to compensate for impairments.
3. Individuals with greater cognitive reserve exhibit dementia later than those with less.
4. Cognitive stimulation can produce learning and thus greater cognitive reserve in individuals with MCI.

In sum, SLPs now have evidence-based techniques that stimulate neuroplasticity and the building of cognitive reserve in individuals with MCI to delay conversion to dementia. Furthermore, they have evidence-based techniques for maximizing the functioning of those with clinically apparent dementia.

Neuroplasticity

Neuroplasticity is the lifelong ability of the brain to reorganize as a result of experience (Kleim & Jones, 2008; Nudo & Bury, 2011). Learning is the byproduct of neuroplasticity. Intuitively, we know this to be true because we add to and refine our knowledge throughout life. Said another way, *neuroplasticity is experience-dependent and behavioral training is key to promoting brain reorganization after brain damage* (Raskin, 2011). Table 1–1 contains a list of empirically demonstrated factors that can be clinically manipulated to support neuroplasticity (Kleim & Jones, 2008; Kolb & Gibb, 2008; Shaffer, 2016). Not listed are hormones and drugs that also affect the capacity for recovery, but are not the province of SLPs.

Of particular significance to clinicians is the fact that the *type* of experience matters (the specificity principle). Learning can be negative or positive. An example of negative learning is the learned nonuse of a paretic limb. An example of positive learning is improvement in a language skill through language therapy.

To trigger neuroplasticity, sufficient stimulation is needed and the *type* of stimulation influences the way in which the brain reorganizes. For example, the presentation of an intensive program to incrementally challenge the auditory processing system can create structural changes in the network of cells that support auditory processing. Visual stimuli influence cell networks that support visual processing. A clinician who knows a client's profile of processing deficits and strengths can design a personalized stimulation program to influence brain response in a positive way. In the case of individuals with a neurodegenerative disease, such as Parkinson's disease or AD, the goal is to strengthen residual knowledge and skills, and if possible, build additional cognitive reserve.

Memory Systems and Their Selective Vulnerability to Disease

In Chapter 2, the various memory systems with their putative neuroanatomical substrates are described. Of significance to clinicians is how the neuropathology of the different dementia-associated diseases affects each disease. For example, as previously noted, the various memory

Table 1–1. Principles of Neuroplasticity

PRINCIPLE	DEFINITION
Attention	Learning requires attention. Attention is a function of stimulus relevance to the individual.
Specificity	Nature of stimulation dictates the nature of brain reorganization. For example, language stimulation produces changes in neuronal networks supporting language.
Use or Lose	Lack of use of knowledge or skills causes both to degrade.
Use and Improve	Use of knowledge and skills strengthens both.
Stimulation	Sensory and/or motor stimulation of sufficient intensity produce changes in brain.
Simultaneity	Concepts, words, actions that occur together become linked in nervous system.
Novelty/Challenge	New, enriching experience stimulates neurogenesis and increases gray matter volume and health of white matter.
Positive Emotion	Enhances mental performance.
Reward	Increases attention and frequency of desired behavior and creates positive emotion.
Constrain	Forced use stimulates the brain to reorganize.
Repetition	Repeated stimulation is essential for creation of long-term memory and skill building.
Intensity	Intense experience is needed for significant brain change.
Duration	The stimulation/experience must be of sufficient duration to create lasting change.
Interference	Brain reorganization in response to one experience can interfere with learning of a similar behavior.
Transference	Brain reorganization in response to one experience can enhance learning of a similar behavior.
Sleep	Regular sleep of 7–8 hours is necessary for consolidation of new information and skills.
Diet	High antioxidant, low fat, low sugar diet nourishes brain cells and reduces damaging free radicals.
Exercise	Exercise increases blood flow to the brain, increases gray matter volume, and energizes motor responses to improve speed of reaction.
Age	Plasticity is greater in childhood.
Time	Different forms of plasticity occur at different times.

Sources: Kleim & Jones (2008); Kolb & Gibb (2008); Shaffer (2016).

systems are not equally vulnerable to the effects of AD, especially early in the disease course. The neural structures that support working and declarative memory, particularly episodic memory, are affected early, whereas those supporting conditioning, motor procedural, and habit memory are relatively spared until advanced dementia (de Vreese, Neri, Fioravanti, Belloi, & Zanetti, 2001; Salmon, Heindel, & Butters, 1992). In Parkinson's disease, the neural structures supporting nondeclarative and working memory are more vulnerable early, whereas those supporting declarative memory are relatively spared. *Clinicians can use early spared systems to help individuals compensate for disease effects and inform caregivers about how to reduce demands on impaired systems.*

The discovery of the differential vulnerability of the brain's representation systems to AD motivated investigations of the potential of procedural learning treatments and conditioning for improving function and quality of

life for AD patients. A considerable literature now exists documenting improved skill learning in AD patients through programs that capitalized on spared procedural memory systems and conditioning (de Werd, Boelen, Rikkert, & Kessels, 2013; Deweer et al., 1994; Deweer, Pillon, Michon, & Dubois, 1993; Dick, Hsieh, Bricker, & Dick-Muehlke, 2003; Dick et al., 1996; Grober, Ausubel, Sliwinski, & Gordon, 1992; Keane, Gabrieli, Fennema, Growdon, & Corkin, 1991; van Halteren-van Tilborg, Scherder, Hulstijn, 2007; Verfaellie, Keane, & Johnson, 2000). For individuals with MCI, who have not evolved to dementia, strengthening their knowledge and skills (cognitive reserve) is the primary goal. Their ability to learn new factual information will be greatly influenced by the degree of their episodic memory impairment. Early on, when episodic memory is minimally affected, new fact learning is easier. As the disease progresses, more emphasis can be placed on using the spared nondeclarative memory/learning systems than on the more impaired declarative systems. Regardless of stage, however, consistent use of retained skills and knowledge helps maintain them.

Cognitive Reserve

The term "*cognitive reserve*" *refers to the mind's ability to cope with brain damage.* One cannot assume that people with similar amounts of brain damage, by virtue of disease or injury, have similar cognitive abilities. This fact is apparent in individuals with AD. Research has shown that some individuals with extensive brain pathology display few, if any, cognitive deficits in life (Katzman et al., 1988). In fact, approximately 25% of individuals with AD pathology whose brains undergo postmortem examination were symptom free in life (Ince, 2001). Why the discrepancy?

Scientists theorize that some individuals may have had more neurons to begin with; others suggest that some internal or external mechanism prevents the extensive neuronal loss typical of the disease. Yet, others suggest that a richer network of interneuronal connections, as a result of education and life experiences, have had a neuroprotective effect. All of these theories are true.

Katzman and colleagues (1988) found an association between brain size and degree of AD symptomatology. Patients who had few symptoms and extensive pathology had higher brain weights and more neurons. More recently, Perneczky et al. (2012) reported that clinical and epidemiologic studies suggest that AD patients who have larger head sizes have better cognitive performance than those with smaller head circumferences, even though the degree of neuropathology is the same.

One "external mechanism" known to influence susceptibility to the effects of AD is amount of education. Individuals with greater education have a reduced risk of developing AD (Anttila et al., 2002; Evans et al., 1993; Evans et al., 1997; Letenneur, Commenges, Dartigues, & Barberger-Gateau, 1994; Stern et al., 1994; White et al., 1994; Zhang et al., 1990). Furthermore, slower decline in cognitive function has been reported in people with more education (Albert et al., 1995; Butler, Ashford, & Snowdon, 1996; Chodosh, Reuben, Albert, & Seeman, 2002; Christensen et al., 1997; Colsher & Wallace, 1991; Farmer, Kittner, Rae, Bartko, & Regier, 1995; Lyketsos, Chen, & Anthony, 1999; Sando et al., 2008; Snowdon, Ostwald, & Kane, 1989). Similarly, people with more education and cognitively challenging careers have better cognitive reserves that reduce risk of dementia (Cheng, 2016; Katzman, 1993; Stern, 2002; Valenzuela & Sachdev, 2005).

Education provides cognitive stimulation and cognitive stimulation results in synaptogenesis and a richer network of interconnected neurons, or brain reserve. Cognitive reserve is related to brain reserve (physical characteristics of brain, e.g., more neurons). Brain reserve can be characterized in any number of ways including brain size, number of neurons, synapse count, and degree of dendritic branching.

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Clinical Management Guide

Assessments and Interventions for Cognitive-Communication Disorders of Dementia

I Assessment

Introduction

Assessment of cognitive function is critical to the early identification of individuals with mild cognitive impairment (MCI) or dementia. For individuals with MCI, early identification has profound benefits because lifestyle changes, cognitive stimulation, and drug therapy can prevent or slow evolution to dementia. Comprehensive evaluation of cognitive and linguistic functions is needed to correctly diagnose and treat those affected and for family counseling. In this section three major topics are addressed:

Assessment procedures that produce valid and reliable data

Tests for diagnosing MCI and dementia

Differentiating the types of dementia

Assessment Procedures that Produce Valid and Reliable Data

The diagnosis of MCI or dementia is made on the basis of history, performance on neuropsychological tests, perceptions of patients and families, and biomarker data from laboratory and imaging results. Currently, however, biomarker data are generally unavailable and results of cognitive-linguistic assessment are the primary basis primarily used for making a diagnosis. Therefore, clinicians should use assessments for which there are normative data on the performance of healthy individuals of a similar age and, if possible, individuals with MCI or dementia. Additionally, clinicians must control for sensory loss and other age-related conditions that can affect cognitive functioning and lead to inaccurate conclusions about mental status and functional abilities.

Prior to Testing

Take a Case History

- What were the presenting symptoms?
- When did they develop?
- Are the symptoms variable? Variability in mental status is characteristic of individuals with Lewy body dementia.
- Is there evidence of more than one neurologic problem? Individuals with Alzheimer's disease (AD) frequently have vascular disease and those with vascular disease often have AD.
- Does the client have other medical problems?
- Does the client have a history of depression?
- What, if any, drugs does the client take?
- What, if any, laboratory tests or neuroimaging has the client had? And, what are the results?
- If the client has a diagnosis of dementia, what is the presumptive cause?
- What is the severity of dementia? Clinicians should be familiar with the commonly used severity rating scales and the scores that correspond to mild, moderate, and severe dementia (Table I–1).

Table I–1. Three Commonly Used Measures of Dementia Severity and Scores/Ratings that Correspond to Mild, Moderate, and Severe Dementia

Measure	Mild Dementia	Moderate-Moderately Severe Dementia	Severe Dementia
Global Deterioration Scale (GDS; Reisberg, Ferris, de Leon, & Crook, 1982): The course of dementia is defined in seven stages that have specific observational criteria. GDS stage 1 represents "normal" aging without evidence of cognitive decline. GDS stage 7 represents "late dementia" with very severe cognitive decline.	3 or 4	5 or 6	7
Clinical Dementia Rating Scale (CDR; Hughes et al., 1982): The CDR provides a rating of global cognitive function based on clinical information about memory, orientation, judgment, problem solving, community affairs, home and hobbies, and personal care. Level of impairment is rated as none (0), questionable (0.5), mild (1), moderate (2), or severe (3).	CDR 1	CDR 2	CDR 3
Dementia Rating Scale (DRS; Mattis, 1976): The DRS contains items that evaluate five cognitive functions: attention, initiation and perseveration, construction, conceptualization, and memory. A maximum of 144 points can be awarded and normal elders obtain scores of 140 or more.	120–130	90–119	<90

- Review the patient's medical history for information about sensory impairment (i.e., macular degeneration, moderate to severe hearing loss) and other conditions that can alter cognitive function (i.e., depression, drug effects).

Arrange for a Good Test Environment

Testing should be conducted in a quiet room with adequate lighting. Illuminate test materials in a way that prevents shadows and remember that elders need two to three times more illumination than young adults. Glare can impair an elder's vision. Avoid sitting with your back to a bright light or window that makes your facial features hard to perceive. Test stimuli should be printed in a font size that is readily perceptible. Black print on a white background is the easiest for elders to see. Avoid putting words in all caps as they are harder to comprehend.

Check Vision

Visual acuity diminishes with age and many elders need glasses to read. If the patient's vision status is unknown, a standard eye chart can be used to screen for a visual deficit or the patient can be asked to read simple words in a print size *smaller* than the test stimuli. If the patient can read text in the smaller print, you can be assured that the patient has adequate visual acuity to see the printed test materials.

Check Hearing

Hearing loss is more common with age (Cruickshanks, 2009; NIDCD, 2017). Indeed, age is the strongest predictor of hearing loss and those with the greatest amount of loss are typically

older than 60 years. Men are twice as likely as women to have hearing loss (Hoffman, Dobie, Losonczy, Themann, & Flamme, 2017). Nearly one-quarter of adults aged 65 to 74 and 50% of those who are 75 and older have disabling hearing loss.

If the patient has hearing aids, have the patient wear them during testing. If a full audiometric evaluation cannot be obtained, a simple speech discrimination test can be given to ensure that the examinee can hear the clinician's voice in the test environment. Have the client repeat words that sound similar (like "cake" and "take") to determine if they can discriminate subtle differences. Use the same intonation and loudness level when presenting the stimulus word pairs. An example of this type of speech discrimination task can be found in the Arizona Battery for Cognitive-Communication Disorders (ABCD-2) (Bayles & Tomoeda, 2019). An error rate of 30% or greater indicates that the patient's hearing will likely impact test performance.

Check Literacy

Check for literacy by having the examinee read two or three simple sentences. Generally, even individuals with moderately severe dementia are able to read aloud if they were literate prior to the onset of dementia.

Take Steps to Reduce Test-Taking Anxiety

Before testing, visit with the examinee and describe what will occur during the session. Position score sheets so the examinee cannot see you record responses. When examinees ask about their performance, tell them the testing is going well. During testing, do not tell examinees if their responses are correct.

Some individuals, particularly those with dementia, become anxious about where their spouse or family member is. Give assurance that the caregiver is nearby. If an examinee will not participate in the evaluation without the caregiver being present, allow the caregiver to be present but refrain from giving cues.

Be Alert to Depression

Depression is common in older adults and can negatively affect test performance. In fact, its effects can mimic dementia. Historically, the term "pseudodementia" was used to designate a dementia-like performance in a cognitively intact but depressed individual (Kiloh, 1961). However, in recent years, the term has fallen out of favor (Alexopoulos, 2003; Dobie, 2002; Emery & Oxman, 2003) because research has shown that depression is frequently an early sign of a dementing disease (Alexopoulos, Young, & Meyers, 1993; Kral & Emery, 1989; Saczynski et al., 2010).

Individuals who are depressed generally convey a sense of distress. Often, they make self-deprecatory comments. Some are uninterested in the testing and its outcome. A subsequent part of this section is devoted to differentiating mild AD from delirium and depression. Table I–2 contains brief descriptions of tests that can be used to screen for depression in adults.

Be Alert to Drug Effects on Performance

Most elders and many middle-aged individuals at risk for dementia take several medications to manage age-associated chronic disease (Lamy, 1986). The potential for adverse drug interactions rises with advancing age because of age-related physiologic change, greater occurrence of comorbid diseases, and an increase in the number of medications prescribed (Hines & Murphy, 2011; Seymour & Routledge, 1998). Sloan (1983) reported that the potential for drug interaction is 5.6% when patients take two drugs and increases to 100% when patients take eight or more drugs. Similarly, Larson, Kukull, Buchner, and Reifler (1987) found that drug reactions that impair cognitive function increase as the number of prescription drugs increases. In a review of 16 studies that included 1,551 patients, Weytingh, Bossuyt, and van Crevel (1995) found that drugs were a frequent cause of reversible dementia.

Table I–2. Screening Tests for Depression

Name of Measure	Reference	Description
Hamilton Rating Scale for Depression (HRS-D)	Hamilton (1967, 1970)	The HRS-D is one of the first-developed and best-known interview-based rating scales for depression. It is a 17-item inventory of symptoms that are rated for severity by an experienced clinician, based on an interview and other available data.
Beck Depression Inventory (BDI)	Beck, Ward, Mendelson, Mock, & Erbaugh (1961)	The BDI is a 21-item inventory of depressive symptoms and attitudes that are rated from 0 to 3 in terms of intensity. The BDI is commonly used as a self-administered measure, although it was designed for administration by trained interviewers.
Zung Self-rating Depression Scales (SDS)	Zung (1965)	The SDS comprises 20 items that evaluate four areas of disturbance: pervasive psychic, physiologic, psychomotor, and psychologic. The patient rates the applicability, within the past week, of each item according to the following terms: "none or a little of the time," "some of the time," "good part of the time," and "most or all of the time."
Zung Depression Status Inventory (DSI)	Zung (1972)	The 20 items of the DSI correspond to the SDS; however, the interviewer rates the severity of symptoms or signs on a four-point scale from none to severe based on the results of a clinical interview.
Dementia Mood Assessment Scale	Sunderland et al. (1988)	This two-part instrument is designed to measure the severity of mood disturbance of demented patients based on direct observation and a semistructured interview by health professionals. The first 17 items evaluate mood and are scaled from 0 (within normal limits) to 6 (most severe). The remaining seven items measure the patient's functional capacities.
Cornell Scale for Depression in Dementia	Alexopoulus, Abrams, Young, & Shamoian (1988)	The Cornell Scale is a 19-item clinician-administered instrument that uses information obtained from interviews with the patient and a member of the nursing staff. This instrument was specifically designed for measuring depression in demented patients.

Orange (2001) listed classes of drugs that commonly interfere with speech, language, and cognition in persons with dementia. They include:

- Sedatives
- Antidepressants
- Anxiolytics (antianxiety drugs)
- Antipsychotics
- Anticoagulants
- Antihypertensives
- Narcotic-based analgesics

Massey (2005) cautioned that long-term use of benzodiazepines can interfere with the ability to learn, and long-term use of anticholinergic medications (Artane, Cogentin, Atropine) and antihistamines (Benadryl, Dimetapp, Chlortrimeton) can contribute to confusion. Mental status changes also occur with medications for incontinence (Detrol, Ditropan), motility (Levsin, Bentyl), and pain.

Know the Criteria for Diagnosing MCI and Dementia

The criteria for diagnosing MCI and dementia provide a guideline for the assessment.

Criteria for diagnosing MCI (Albert et al., 2011) are as follows:

1. **Concern regarding a change in cognition.** Concern can be expressed by the person

or an informant who knows the individual well or a skilled clinician.
2. **Impairment in one or more cognitive domains.** Evidence is needed of lower performance in one or more cognitive domains that is greater than would be expected for the patient's age and education. Remember that change can occur in a variety of cognitive domains, including language, attention, visuospatial skills, memory, and executive function. Scores on tests of those with MCI are typically 1 to 1.5 standard deviations (SD) below the mean for age and education.
3. **Preservation of independence in basic functional abilities.**
4. **No dementia.**

Take Into Account Degree of Intelligence and Education in Evaluating Test Performance

The challenge of identifying MCI or mild dementia is that intelligence and degree of education vary considerably in the healthy population. Without taking into account intelligence and education, you can mistakenly conclude that a low-average or average performance is normal in a highly intelligent and educated individual when, in fact, the individual has MCI. Similarly, you can mistakenly conclude that a below average performance of a person, who has below average intelligence, is evidence of impairment. To cope with these challenges, the authors of the diagnostic clinical criteria for MCI established the following performance cutoffs.

> **A performance of 1 to 2 SD below the mean of healthy elders with similar education is considered indicative of cognitive impairment** (Albert et al., 2011).

Clearly, to make this comparison, you need to administer tests for which there are normative data for healthy adults.

Criteria for diagnosing dementia (American Psychiatric Association, 2013) are as follows:

1. **Individual exhibits significant cognitive decline from a previous level of performance.**
2. **The cognitive deficits are sufficient to interfere with independence in everyday activities.** At a minimum, assistance should be required with complex instrumental activities of daily living, such as paying bills or managing medications.
3. **The cognitive deficits do not occur exclusively in the context of delirium.**
4. **The cognitive deficits are not better explained by another mental disorder.**

The National Institutes of Health–Alzheimer's Disease and Related Disorders Association (NIH-ADRDA) working group acknowledges that specifying the definitive points at which individuals transition from MCI to dementia and mild to moderate dementia is difficult. Efforts to refine the criteria for characterizing the various stages of dementing diseases (asymptomatic preclinical, symptomatic predementia, and clinically apparent dementia) are ongoing (Albert et al., 2011; McKhann et al., 2011; Sperling et al., 2011). Thus, some degree of clinical judgment is involved. However, to improve decision making, use tests for which there are normative data on the performance of healthy adults, and, if possible, measures that also have data on the performance of individuals with mild and moderate dementia (for whom etiology is specified).

Severity of Cognitive Impairment Affects Test Selection

We have found, and others have confirmed (Locascio, Growdon, & Corkin, 1995), that tests suitable for early detection of cognitive impairment are unsuitable for documenting abilities in severely demented individuals. If a test is sufficiently cognitively challenging for identifying individuals with MCI, it is usually too difficult for individuals with moderate dementia and *floor effects* emerge. Similarly, tests designed to characterize cognitive-communicative functioning in the later stages of dementia are generally too easy for individuals in the mild stage, and the result is a *ceiling effect.* It is the