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Preface

The second edition of *Cognitive-Communication Disorders of Dementia* contains updated information on the cognitive-communication disorders associated with the most common dementia-producing diseases as well as new chapters on minimal cognitive impairment (MCI) and cognitive stimulation as a means of building cognitive reserve in individuals at risk for dementia. Students in training and practicing clinicians will find up-to-date information about how to diagnose, differentiate, and provide interventions to individuals with MCI and dementia-associated cognitive-communication disorders.

Four general topics are covered:

1. Cognition and communication
2. Dementing diseases, their characteristics, and effects on cognition and communication
3. Assessment of cognitive-communicative communication function
4. Treatment of cognitive-communication disorders

The book begins with an overview of the role of the speech-language pathologist with individuals at risk for and diagnosed with dementia. Chapter 2 provides a foundation for understanding the memory systems that can be selectively impaired by neurodegenerative diseases and their relation to communicative function. Chapter 3 is devoted to minimal cognitive impairment (MCI), a fast-growing population that speech-language pathologists are increasingly asked to serve. Chapters 4 through 10 focus on the common dementia-producing diseases. The longest chapter, Chapter 11, is on assessment and differentiation of the cognitive-communication disorders associated with the dementing diseases. Chapters 12 through 15 focus on therapy, the direct and indirect interventions that clinicians can use to maximize the function of individuals with dementia.

At the end of each chapter is a summary of important points. This summary will provide instructors, students, and practicing professionals a good overview of the chapter’s content.
Our greatest debt is owed to the many people with dementia and their family members who shared their experiences and generously gave their time to participate in research. They are our heroes. We are also indebted to the teachers, colleagues, and students who shaped our views and supported our endeavors. Since publication of the first edition of *Cognitive-Communication Disorders of Dementia*, voluminous research has been published on dementia-associated diseases, their diagnosis, and treatment. For her cheerful help in locating and organizing the publications relevant to the mission of this book and her care in performing myriad assignments, we thank Lesley Skinner.

A new chapter in this edition covers cognitive interventions for individuals with minimal cognitive impairment (MCI). Increasingly clinicians are treating and counseling individuals with MCI and they need information about providing effective cognitive stimulation programs. We thank Kimberly C. McCullough for sharing her expertise on this timely topic.
Introduction

Individuals with mild cognitive impairment (MCI) and dementia are the profession’s fastest growing clinical population, nationally and globally. In fact, every 4 s someone is diagnosed with Alzheimer disease (AD), the leading cause of dementia (Alzheimer’s Association, 2012; Ferri, Prince, Brayne, Brodaty, & Fratiglioni, 2005). Currently, 24 million individuals worldwide are affected; however, by the year 2020, 42 million people will have AD or a form of dementia and 100 million will be affected by 2050 (Alzheimer’s Association, 2012).

Dementia-associated diseases, such as AD, can begin decades before they are clinically obvious and, once diagnosed, endure for many more years. Because the dementia syndrome eventually makes it impossible for affected individuals to care for themselves, they increasingly depend on others for survival. Most patients are cared for at home by family, typically with serious financial, social, and emotional consequences to 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). Speech-language pathologists (SLPs) are among the professionals who can provide support to affected individuals and their families. 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 the disease.

The goal of this book is to provide practicing professionals and graduate students the knowledge needed to evaluate and treat individuals who have MCI or dementia and 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?” However, to answer that question, we need to first define communication.

Communication Defined

Communication is the sharing of information by means of a symbol system. When words are used, we call it linguistic communication 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. Baseball coaches communicate plays by hand signals to players. 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 on impairment in linguistic communication. Nonetheless, both nonlinguistic and linguistic communications are impaired in AD because both are cognitive processes for sharing information.

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 the ability to communicate is affected more than speech and language.

“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 have been structured in such a way that the listener comprehends the speaker’s idea.

Now the question of why communication is affected in dementia can be answered. Communication is affected because the pathophysio-logic processes that disrupt multiple cognitive functions and produce dementia disrupt information generation and processing. Patients 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 turnip. First you must perceive the features of the turnip. 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 turnip 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 they have trouble thinking as well as generating and ordering ideas, in part because 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), in part because of progressive degradation of knowledge (Laisney, Giffard, Belliard, de la Sayette, Desgranges, & Eustache, 2011; Laisney, Giffard, & Eustache, 2004) as is the case in semantic dementia. They have difficulty comprehending language because of deficits in the
cognitive processes of perception, recognition, attention, memory, and 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 behavioral and cognitive sciences, have focused on dementia-associated diseases and their management. Collectively, their findings make a compelling case for early detection that can be summarized as follows:

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.
3. Individuals with greater cognitive reserve exhibit dementia later than those with less.
4. Cognitive stimulation can improve function and produce learning (greater cognitive reserve) in individuals with MCI and dementia.

Said another way, SLPs now have evidence-based techniques that advantage neuroplasticity for strengthening cognitive reserve in individuals with MCI to delay conversion to dementia and 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).

Of significance to clinicians is the fact that the type of experience matters. 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.

Table 1–1 contains a list of empirically demonstrated factors known to be influential in recovery of function (Kleim & Jones, 2008; Kolb & Gibb, 2008). Not listed are diet, hormones, and drugs that also affect the capacity for recovery but are not factors that SLPs manipulate.
Memory Systems and Their Selective Vulnerability to Disease

In Chapter 2 the various memory systems with their putative neuroanatomic substrates are described. Of significance to clinicians is how the neuropathology of the different dementia-associated diseases affects them. For example, the various memory 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

<table>
<thead>
<tr>
<th>Principle</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attention</td>
<td>Learning requires attention and attention is a function of stimulus relevance to the individual.</td>
</tr>
<tr>
<td>Reward</td>
<td>Increases attention and increased attention produces better learning.</td>
</tr>
<tr>
<td>Stimulation</td>
<td>Sensory and/or motor experience of sufficient intensity and duration are necessary for brain reorganization.</td>
</tr>
<tr>
<td>Use or lose</td>
<td>Lack of use of knowledge or skills can cause their degradation.</td>
</tr>
<tr>
<td>Use and improve</td>
<td>Use of knowledge or skills produces improvement.</td>
</tr>
<tr>
<td>Specificity</td>
<td>The nature of the stimulation/experience dictates the nature of brain reorganization. Example: Language stimulation produces changes in the neuronal networks that support language.</td>
</tr>
<tr>
<td>Simultaneity</td>
<td>Concepts, words, and actions that occur together become linked in the nervous system.</td>
</tr>
<tr>
<td>Repetition</td>
<td>Repetition of stimulation/experience is necessary for the creation and maintenance of long-term potentiation and learning.</td>
</tr>
<tr>
<td>Intensity</td>
<td>Intense experience is needed for significant brain change.</td>
</tr>
<tr>
<td>Duration</td>
<td>The stimulation/experience must be of sufficient duration to create lasting change.</td>
</tr>
<tr>
<td>Constrain/forced use</td>
<td>Stimulates the brain to reorganize, relearn, or compensate.</td>
</tr>
<tr>
<td>Interference</td>
<td>Brain reorganization in response to one experience can interfere with learning of another behavior.</td>
</tr>
<tr>
<td>Transference</td>
<td>Brain reorganization in response to one experience can enhance the learning of a similar behavior.</td>
</tr>
<tr>
<td>Sleep</td>
<td>Necessary for representation of new information and skills in the nervous system through synaptic and memory system alterations.</td>
</tr>
<tr>
<td>Age</td>
<td>Plasticity is greater in childhood.</td>
</tr>
</tbody>
</table>
those supporting conditioning, motor procedural, and habit memory are relatively spared (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 (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, Groudon, & Corkin, 1991; 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;