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Dementia, Etiologies, and Implications on Communication

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Learning Objectives

By the end of this chapter, you will be able to:

- Better understand and identify different dementias, their symptoms, and causes;
- Appreciate the neurological and social changes that persons with dementia may be experiencing.

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Thinking Points: Vignette 1

- Clara has been exhibiting gradual, progressive word finding difficulties and general forgetfulness for over three years. Her late husband wasn't troubled by this. Instead, during this time, he simply had taken over most of the driving, household chores, and financial management. Still, these accommodations changed their relationship, and those changes occasionally led to Clara feeling more of a dependent, child-like, than a partner.
  Question: What may be causing these changes seen in Clara?
- After their father's death one year ago, Clara's children now had the opportunity to notice these behaviors in their mother. They assumed that these changes along with increased anxiety and seemingly "initial" bouts of confusion were due to grief. They were not aware of how long their mother had been experiencing these symptoms or the many responsibilities that their father had assumed due to Clara's increasing difficulties negotiating the world around her.
  Question: What else may Clara's children have been missing?
- The children watched as Clara's conversations were increasingly challenging—for her and them—as her difficulties expressing herself led to frustration and irritability. They became increasingly concerned when their mother confused their names with her siblings' names, did not acknowledge special days, like their birthdays, and failed to pay her bills. One child stepped in to help manage finances; another visited with her every few days. The children's relationship with their mother was changing. When she got lost while driving, one finally uttered the word and voiced their fear: ***dementia***.
  Question: How should the family best intervene on Clara's behalf?

Introduction

Geriatric psychiatrists routinely treat the cognitive, emotional, functional, and interactional effects of dementia as depicted in Clara's story and the consequence of those changes in the relationships between patients, their families, and the community. From our clinical experience and current research, we focus this chapter on how various neurocognitive disorders (NCD)—their symptoms and progression—affect the interactional abilities of people with dementia and their relationships. We also provide a clinical review of the common forms

of dementia with their neurocognitive effects and symptoms. For a complete listing of stages and symptoms, see *The Diagnostic and Statistical Manual of Mental Disorders*, 5th ed. (American Psychiatric Association, 2013). Specifically, this chapter will compare and contrast common dementias and their effects on communication. We will reflect on how such clinical observations can help researchers, various practitioners, care-giving staff, as well as family members, in order that they may better serve people experiencing dementia.

Some Preliminaries: Clinical Description of Dementia

Our understanding and recognition of defining characteristics of dementia continue to evolve. Previous editions of *The Diagnostic and Statistical Manual of Mental Disorders* (DSM III and IV) identified the condition "Dementia" primarily as a group of disorders in which the cardinal symptom was a deficit in short term memory (American Psychiatric Association, 1980, 1994). Now, however, the conditions relating to "dementia or dementias" refer to the conglomerate of symptoms involving one or more cognitive domains and are grouped under the label Major Neurocognitive Disorders (American Psychiatric Association, 2013). Domains affected go beyond memory to include language, executive function, complex attention, perceptual-motor, and social cognition. Per the *DSM 5* definitions, these deficits, whatever the etiology, must be acquired and demonstrate significant decline from a previous level of performance severe enough to interfere with independence in everyday activities. Different etiologies of dementia (Alzheimer's, Lewy body, frontotemporal, and vascular) have additional criteria to differentiate them. Additionally, each type of neurocognitive disorder is classified by severity: Mild or Major. Major severity is further divided into stages of mild, moderate, or severe. Each is distinguished further by the presence or absence of behavioral symptoms. The import of this change is that "dementia" is now categorized as a broader based collection of disorders, all with particular, albeit sometimes overlapping, deficits and declines in functional impairments.

A Brief Look at Common Dementia Etiologies

The most common forms of NCD/dementia include Alzheimer's disease (AD), Lewy body dementia (DLB), frontotemporal dementia (FTD), and vascular dementia (VaD) (Kaufman, Geyer, & Milstein, 2016). The first three are neurodegenerative processes that result from the progressive loss of nerve cells in the brain. VaD may be a sole cause of dementia, but it often co-exists with another neurodegenerative type. Many less frequent causes, such as Huntington's disease (see also Chapter 10), Parkinson's disease, alcohol-related or other injuries, account for the remainder. Additionally, dementia-like symptoms may arise from reversible causes (e.g., infection, pharmacological interactions). While a definitive identification of a dementia cause for a given patient is sought, for better understanding, treatment, and planning, it is not always achievable. Confounding factors include symptom overlap and limitations in our gradually developing brain scan and genetic testing technology.

Diagnostic Procedures

When any form of dementia is suspected, a thorough diagnostic evaluation is mandated. Age of onset, family history, and underlying medical history and risk factors are strong diagnostic indicators. Full evaluation includes physical and neurological examinations, laboratory studies, brain scans, and often neuropsychological testing to evaluate potentially reversible causes of cognitive decline (Chen et al., 2016). (The use of biomarkers is still primarily in research stages, not readily available for clinical evaluation.) Evaluation of reversible dementia typically includes testing for vitamin B12 deficiency, thyroid abnormalities, infection, and assessment of medication side effects or interactions. Psychiatric diagnoses, particularly Major Depression or other Mood Disorders must also be ruled out, as they may present as a primary neurocognitive disorder. Normal aging and senescence are often ruled out as neither impairs communication (Bayles, 1982).

Key, then, to diagnosing dementia is the marked and increasing communication impairments. Most important to this chapter's as well as

the text's focus is the semantic system's vulnerability to the effects of a particular progressive dementia. While language abilities of persons with dementia are resilient, particularly the phonological (sounds) and syntactic (grammar) systems, losses in their semantic, pragmatic, and lexical retrieval systems progressively affect their ability to effectively communicate (Murdoch, Chenery, Wilks, & Boyle, 1987).

Thinking Points: Vignette 2

- In addition to Clara's progressive short term memory impairments and loss of independent functioning, she experienced further decline in her communicative abilities. Her sentences have become very simple; she often searches for the next word, often skipping it or using a generic word like "thing" or "it"; and she sometimes misuses pronouns, calling her daughter "he" or her brother "she". Fortunately, her family is still able to understand most of what she is attempting to communicate.
  Questions: How would you as a family member adjust your conversations with Clara? How would you as a clinician advise Clara's family?
- One-on-one interactions are often easier than group conversations. Addressing one topic at a time, being more agreeable and not overly correcting errors and misstatements is suggested (see Chapters 5, 6, 7, and 11).
- Conversations are often helped by being briefer and more focused, particularly as dementia progresses (see Chapters 8, 9, and 10).

A Closer Look at the Individual Etiologies of Dementia

Alzheimer's Disease

AD accounts for 60–80% of all dementias, globally, either as a singular etiology or concomitantly with cerebrovascular disease (Alzheimer's Association, 2018). It is chronic and progressive and ultimately fatal. No preventive, curative, or disease modifying treatments exist. As reported in *2019 Alzheimer's Disease Facts and Figures,* one in ten Americans aged 65 or older has AD, with a total of 5.8 million Americans currently affected (Gaugler, James, Johnson, Marin, & Weuve, 2019). Gaugler and colleagues project that, in the U.S. alone, the prevalence is expected

to reach 14 million by 2050 (2019). Worldwide, over 46 million people have dementia (Wang et al., 2016), significantly increased from 36 million (Batsch & Mittelman, 2012). The number is expected to reach 75 million people worldwide by 2030 (Prince et al., 2015).

Neurocognitive Causes and Changes

AD was first identified over 100 years ago by its pathognomonic findings at autopsy of neurofibrillary tangles and neuritic plaques. The tangles are composed of hyperphosphorylated tau and the plaques are made of beta-amyloid. Recent advances have led to a biomarker categorization system, A/T/N, (amyloid/tau/non-specific) based on measures of brain beta-amyloid deposits, phosphorylated tau found in cerebrospinal fluid, and other nonspecific biomarkers of neurodegeneration per brain imaging (Jack et al., 2016). AD neurodegenerative biomarkers are associated with decreased cognitive function but not beta-amyloid in cognitively normal older individuals (Wirth et al., 2013). These have been incorporated into the National Institutes of Aging (NIA) staging guidelines (Sperling et al., 2011) but have yet to become useful in daily practice. A literature into the genetics of AD is emerging, and while a full review is beyond the scope of this chapter, we will briefly summarize.

The only gene identified with Late Onset Alzheimer's Disease (LOAD), defined as onset after age 60, is apolipoprotein E (APOE). APOE is considered a "risk factor" gene because inheriting a certain allele may increase or decrease a person's risk of developing AD: APOE-2 appears to be protective; APOE-3 is the most common allele and appears to be neutral; APOE-4 appears to increase the risk for developing LOAD. Still, the presence or absence of APOE 2, 3, or 4 will not definitively cause or prevent AD (see Shivani, 2015).

Demographic Factors

Age is the key predictive factor: the older persons are, the higher their risk for developing AD becomes. As noted in the 2019 Alzheimer's Disease Facts and Figures report, 3% of people age 65–74, 17% of

people age 75–84, and 32% of people age 85 and older have AD; with women experiencing the highest incidence; among people age 71 and older, 16% of women have AD or other dementias compared with 11% of men (Gaugler et al., 2019). African Americans and Hispanics have a higher incidence than Caucasians or Asians with incidence rates for African Americans about twice as likely and Hispanics about 1.5 times as likely as non-Hispanic Whites (Gaugler et al., 2019).

Prognostic Information

AD is viewed as a three-stage continuum (Gaugler et al., 2019). The preclinical stage is defined as having the presence of brain changes, per imaging, without clinical symptoms. Mild Cognitive Impairment (MCI) due to AD is characterized by both brain changes and mild cognitive symptoms that do not significantly affect everyday living. Dementia due to AD presents as brain changes and significant problems with memory, thinking and/or behavior which interfere with daily activities.

AD Symptoms

AD involves progressive short-term memory decline, particularly impacting executive functioning, language, and visuospatial abilities. Anomia, or word finding difficulties, is a frequent early symptom. A decrease in spontaneous verbal output manifests early and worsens as the dementia progresses. Likewise, paraphasic errors (the use of incorrect words or syllables) and certain symptoms of aphasia, such as circumventing forgotten words, are exhibited. Mild AD causes mild word finding problems, word substitution, repetition, and reluctance to speak, generated from fear of mistakes, Moderate AD shows midrange symptoms, with increased repetition, disorganization, gaps in speech, and confabulation. Severe AD causes significant difficulties to be understood, reduced speech often resulting in mutism (for a review, see Kindell, Keady, Sage, & Wilkinson, 2017).

Synchronic memory and neurocognitive decline occur with increasing impairments in functional abilities and behavioral/neuropsychiatric symptoms. Correlating with memory and communication declines, behavioral changes are common, often social withdrawal, apathy, depression and irritability early in the course, with aggression and psychosis in later stages of the disease (Jost & Grossberg, 1996).

Thinking Points: Vignette 3

- Clara's family moved her to a memory care facility. The transition was made easier by incorporating a number of family photos and Clara's personal items. Her children allowed her to talk and repeat herself without interruption. She rarely shows awareness of her deficits.
  Question: How could you as a family member maximize Clara's communication?
  Question: How could you as a clinician guide Clara's family on further expected communicative declines?
- Use of smiles, gestures, and other nonverbals can greatly aid communication. Gently assisting a person who is struggling to find a word is often helpful. However, taking over the conversation can be met with increased frustration and anger. Accept repetition. Allow the person with dementia to lead the conversation with your appropriate guidance. See Chapters 6 and 11.

Lewy Body Dementia

DLB, sometimes referred to as Dementia with Lewy Bodies (DLB), is more frequently recognized, and, due to post-mortem exams, speculated as the most common progressive dementia (Outeiro et al., 2019). While DLB may present as the single cause, it often co-occurs with other dementias. Disambiguating single versus mixed dementia is near impossible as DLB's symptoms overlap with AD and Parkinson's disease. These include memory impairment plus the classic triad of early presentation of prominent visual hallucinations, Parkinsonian symptoms, and fluctuating levels of consciousness or confusion.

DLB Causes

Rather than the plaques and tangles seen in AD, the microscopic findings in DLB are "Lewy bodies" or accumulations of alpha-synuclein, a presynaptic neuronal protein (Stefanis, 2012). These structures are also found in the brains of persons with Parkinson's disease (PD), where their density and locations result in identifiable types and severities of symptoms. For instance, in the basal ganglia, they cause classic PD motor symptoms; in the cortex, they are responsible for memory impairment. Distinguishing DLB from AD is a clinical diagnosis, without any specific findings on laboratory testing or scans. Neuropsychological testing may be helpful for diagnostic clarity.

Language Effects of DLB

As with AD, language related symptoms are not seen in early DLB (Gatchel et al., 2015, p. 191; Muangpaisin, 2007), but other symptoms are key to DLB.

Thinking Points: Vignette 4

- Ruth is a healthy 76-year-old widow, living independently in a senior apartment complex, with no obvious impairments. Her son became concerned when Ruth began talking about the friendly children who visit her in the evening and at night. He knew no children lived in the building, and immediately was skeptical about Ruth's reports. She was not bothered by "the children" and, in fact, looked forward to their "visits."
  Question: What course of action should Ruth's son pursue in thinking his mother's reports are delusions?
  Question: What causes could delusions be a symptom of?

Egosyntonic (pleasant) visual hallucinations, commonly of children or animals, are often early symptoms of DLB. (In most other forms of dementia, hallucinations do not occur until late stages.) In DLB, people present to their doctor with visual hallucinations or tremors, often before the presence of memory impairment is noted. Prescribing an

acetylcholinesterase inhibitor may assist, a person with DLB to live a fairly independent lifestyle long before moving to an assisted living facility.

Frontotemporal Dementias

FTDs are a large group of somewhat similar dementias that affect various parts of the frontal (primarily) and temporal lobes of the brain. While majority of people with AD are over 65 years of age, most people who develop FTDs do so before age 65 with the average onset at 53 years old (Gatchel et al., 2015). Not only do FTDs typically occur earlier in life than AD, but also progression to death is shorter.

FTDs Cause and Predispositions

FTDs often cluster in families (Autosomal Dominant pattern) with up to one half of people having an affected family member. Recent studies have subdivided types of FTDs based on types of proteins found at particular neuro-locations, with symptoms and extent of neurodegeneration mapping to locations (Gatchel et al., 2015).

FTDs Behavioral Effects

The frontal lobes of the brain function as the control centers for personality, emotions, and executive decisions (Kaufman et al., 2016, p. 123). They also work to inhibit socially inappropriate and impulsive behaviors. Thus, advancing FTDs result in changes often characterized as the coarsening of personality, loss of inhibitions, increasing emotionality and problems with organizing, planning, sequencing, and other executive functions. Clinically, people with FTD may have cognitive symptoms similar to those seen in AD, but with much more prominent and early behavioral symptoms, including disinhibition and impulsivity, along with language deficits (see Chapter 9).

Language Effects of FTDs

In contrast to AD, people with FTDs often exhibit early and progressive paraphasias, anomias, and decreased fluency. When the left hemisphere is heavily affected, language symptoms are primary, and manifest significantly earlier than cognitive symptoms (Gatchel et al., 2015, p. 193). Semantic Dementias (also known as Semantic Variant Primary Progressive Aphasia) are types of FTDs highly affecting the left temporal lobe. People with FTDs lose the ability to match certain words with their images or meanings—verbal and nonverbal—but often retain intact fluency and episodic memory (Bayles, 1982).

Thinking Points: Vignette 5

- Tom is a 62-year-old retired engineer. His wife reports that he was always an active, energetic, "type A" person. About 3 years ago, he began exhibiting increasing irritability with his family. He began having trouble "finding the right word" and with concise expression. For the first time in his career, he was reprimanded for making significant mistakes in his work and for treating colleagues in an angry manner. He was not forgetful, and his family first assumed that he must be experiencing depression. He was prescribed an antidepressant medication.
- Tom did not respond to the antidepressant. His irritability worsened. His word finding problems increased and he began making out-of-character, insensitive, and inappropriate comments in public situations. He had more problems with work performance and lost his job. His primary care doctor's evaluation, including blood work and a brain scan, showed no obvious problems.
- Due to Tom's family's insistence, he underwent neuropsychological testing, which yielded a diagnosis of FTD.
  Question: What conditions other than depression might account for Tom's changing behaviors?
- Self and family advocacy in exploring alternative diagnoses and treatment options can be key to accurate identification.

Vascular Dementia

VaD differ from the previously described dementias. VaD was formerly referred to as Multi-Infarct Dementia (MID). VaD's classic progression includes a step-wise decline in cognitive functioning, along with weakness

and physical impairments due to a series of strokes. The causes of VaD can range from one large, usually unilateral Cerebrovascular Accident (CVA), to many small strokes occurring over time. Physical deficits such as single-sided paralysis often result. Numerous small strokes, however, may falsely present as a progressive neurodegenerative dementia, as no significant neurological deficits may be obvious without a thorough neurological evaluation. The location, number, and severity of each stroke determine the overall symptom profile including cognitive loss, functional and behavioral symptoms, and language effects. VaD may occur as the single cause of dementia or may co-exist with another neurodegenerative dementia.

VaD Causes and Predisposition

Population based studies document vascular pathologies in 50% of older persons that, consequentially, correlate with other dementia (Kapasi & Schneider, 2016). Genetics can be direct causative factors in VaD, specifically Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL). However, the most common cause of VaD is stroke, either single or multiple, due to primarily preventable causes: high blood pressure, obesity, elevated cholesterol, and poorly controlled diabetes mellitus.

VaD Prevention

Aggressive management of cardiovascular risk factors such as blood pressure, cholesterol, and blood sugar along with diet and exercise, avoidance of smoking, and minimal alcohol consumption are the most effective preventions. Anticoagulation or antiplatelet treatment may either be recommended or contraindicated per stroke type.

Language Effects of VaD

When vascular damage is predominantly in the left hemisphere, language impairment is typically exhibited, whereas predominantly

right sided lesions result in visuospatial problems (Kaufman et al., 2016, p. 155).[1] Location, size, and neurodegenerative comorbidity determine symptom presentation (Gatchel et al., 2015, p. 195).

Thinking Points: Vignette 6

- William is an obese 73-year-old who had smoked two packs of cigarettes daily, and drank alcohol excessively for 40 years. Following a small heart attack at 59 years old, he stopped all cigarette and alcohol use. However, he has slowly gained weight and does not pay attention to his blood sugar or blood pressure. He is increasingly sedentary and just doesn't like to "take pills". His memory and abilities to manage his own finances appear normal. Unfortunately, William has also had a series of strokes. They caused paralysis on one side of his body and significant expressive speech impairments. He appears able to understand other's speech, but communicates best, though not consistently, with gestures, yes/no utterances and pointing to a communication board.
  Question: How do Williams's receptive and expressive communication problems compare with Clara's?
  Question: At this point, would you recommend any specific treatments or interventions?

For degenerative dementias (e.g., Clara's AD, Ruth's DLB), medications (e.g., acetylcholinesterase inhibitors, memantine) can slow progression of communicative, memory, and functional decline. For abrupt and rapid changes caused primarily by VaD, such as William's, these treatments are of no benefit. As highlighted by the many vignettes, cognitive impairment and declines in language ability are the cardinal symptoms of dementia, but physical, behavioral, emotional, and functional impairments likely accompany. Verbal communication with a person with dementia, particularly seen in William's case, can often be enhanced with non-verbal approaches, including gestures, and visual cues and prompts.

[1]The dominant hemisphere controls most language, though the nondominant hemisphere controls prosody, including inflection, rhythm and manner of speaking. The dominant hemisphere also contains the main areas for cognitive activity and emotions (Kaufman et al., 2016, p. 155).

Discussion

No cures for any form of dementia exist. Treatments may result in modest neurocognitive (including language), functional, and/or behavioral improvements, but a more realistic expectation may be a temporary period of stability, followed by a temporary slowing of anticipated decline. (Exceptions are an isolated vascular event, followed by excellent control of future risk factors, or identification of another reversible dementia cause.) Behavioral strategies, especially the setting of regular routines, in simplified familiar environments are highly important. Cognitively stimulating activities are often well accepted by patients and their families, have no adverse effects, and may provide modest help.

What can be done, by those of us who interact with persons experiencing any of these dementias, is to work toward better communication practices. The vignettes provided in this chapter aim to help with that understanding for all stakeholders—fellow practitioners, researchers, caregivers, family members, and the persons struggling with the diseases themselves. These are summarized in the box below.

Practical Highlights

1. Make no assumptions about an individual's cognitive and communicative abilities. But, be acutely aware of progressive declines in a person's memory AND communicative abilities.
2. Reports of "unreal" visualizations can be a warning sign of early DLB or a late sign of other dementias.
3. Drastic changes in personality, particularly lack of inhibition, can be signs of an FTD.
4. Some progressive dementias respond to medications (e.g., AD, DLB) or behavioral changes (e.g., VaD).
5. Advocacy—self or family—is the best assurance for diagnosis and treatment.

Continuing that goal, the research findings that follow in this book aim to provide a window into current interactional and communication strategies beneficial to all who are experiencing the epidemic of dementias: individuals, families, communities—local and global.

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