Benjamin T. Mast
Brian P. Yochim

Advances in Psychotherapy –
Evidence-Based Practice

Alzheimer's Disease and Dementia

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From B. T. Mast & B. P. Yochim: *Alzheimer's Disease and Dementia* (ISBN 9781616765033) © 2018 Hogrefe Publishing

Alzheimer's Disease and Dementia

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Library of Congress Cataloging in Publication information for the print version of this book is available via the Library of Congress Marc Database under the Library of Congress Control Number 2017955719

Library and Archives Canada Cataloguing in Publication

Mast, Benjamin T., author

Alzheimer's disease and dementia / Benjamin T. Mast, University of Louisville, KY, Brian P. Yochim, VA St. Louis Health Care System, St. Louis, MO.

(Advances in psychotherapy--evidence-based practice ; v. 38)

Includes bibliographical references.
Issued in print and electronic formats.
ISBN 978-0-88937-503-1 (softcover).--ISBN 978-1-61676-503-3 (PDF).--ISBN 978-1-61334-503-0 (EPUB)

1. Dementia. 2. Alzheimer's disease. 3. Dementia--Treatment. 4. Alzheimer's disease--Treatment. I. Yochim, Brian P., 1976-, author II. Title. III. Series: Advances in psychotherapy--evidence-based practice ; v. 38

RC521.M377 2017 616.8'31 C2017-906573-4
C2017-906574-2

Cover image: © monkeybusinessimages – iStock.com

http://www.hogrefe.com

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USA: Hogrefe Publishing Corporation, 7 Bulfinch Place, Suite 202, Boston, MA 02114
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EUROPE: Hogrefe Publishing GmbH, Merkelstr. 3, 37085 Göttingen, Germany
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USA: Hogrefe Publishing, Customer Services Department,
30 Amberwood Parkway, Ashland, OH 44805
Phone (800) 228-3749, Fax (419) 281-6883; E-mail customerservice@hogrefe.com

UK: Hogrefe Publishing, c/o Marston Book Services Ltd., 160 Eastern Ave.,
Milton Park, Abingdon, OX14 4SB, UK
Phone +44 1235 465577, Fax +44 1235 465556; E-mail direct.orders@marston.co.uk

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SWITZERLAND: Hogrefe Publishing, Länggass-Strasse 76, CH-3000 Bern 9

Format: PDF

ISBN 978-0-88937-503-1 (print) • ISBN 978-1-61676-503-3 (PDF) • ISBN 978-1-61334-503-0 (EPUB)
http://doi.org/10.1027/00503-000

Contents

Dedication

This book is gratefully dedicated to our mentor, friend, and colleague, Dr. Peter Lichtenberg.

1

Description

1.1 Terminology

Dementia refers to a syndrome of cognitive and behavioral declines that are severe enough to interfere with daily functioning. Dementia is a broad category of cognitive changes with a variety of causes (or types) including Alzheimer's disease, cerebrovascular disease (vascular dementia), and/or Lewy bodies (Lewy body dementia). Dementia is distinct from normal age-related declines in cognitive functioning.

Dementia is a cognitive syndrome with many causes or types; Alzheimer's disease is the most common cause of dementia

Although there are reversible forms of dementia, this volume focuses on dementias that are irreversible. **Alzheimer's disease** is the most common cause (or type) of dementia, with 60–80% of dementia cases caused by the neuropathology of Alzheimer's disease. The *Diagnostic and Statistical Manual of Mental Disorders,* 5th edition (DSM-5) adopted the term *major neurocognitive disorder* for this condition (American Psychiatric Association [APA], 2013).

DSM-5 uses the term *major neurocognitive disorder* instead of dementia

Mild cognitive impairment (MCI) (also known as *mild neurocognitive disorder*) represent less severe forms of cognitive change that may or may not develop into dementia over time. MCI is not considered normal aging or dementia, but has often been conceptualized as a transitional condition between the two. There are multiple forms of MCI, including amnestic MCI (isolated memory impairment), nonamnestic MCI (impairment in isolated cognitive ability that is not memory), and multiple domain MCI, in which multiple areas of cognition are mildly impaired, but the person retains relative independence and does not meet the criteria for dementia. **Mild neurocognitive disorder** is the term used for MCI in the DSM-5. It does not differentiate between amnestic or nonamnestic subtypes, but involves specification of the possible or probable etiology.

Mild cognitive impairment is not normal aging or dementia

1.2 Definition

1.2.1 Dementia

The American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders,* fourth edition (DSM-IV; APA, 1994) defined *dementia* as impairment in memory and one other area of cognitive functioning, including aphasia, apraxia, agnosia, or executive functioning. These declines must be more severe than normal age-related declines to warrant a diagnosis of demen-

Cognitive changes in dementia interfere with daily functioning

tia. More specifically, they must be severe enough to interfere with daily functioning, including occupational functioning or leisure activity. Clinicians often use **instrumental activities of daily living** (IADLs) as an index for this portion of the diagnostic criteria among older people who are no longer working (e.g., medication management, driving, shopping, housework, management of finances, and using the telephone or technology).

Dementia involves changes in a variety of cognitive abilities, not just memory

The DSM-5 indicates that performance on neuropsychological testing is typically 2 or more standard deviations below the mean compared with people of similar background (i.e., of similar age, education, and ethnic/cultural background) in individuals with dementia (major neurocognitive disorder) and 1 to 2 standard deviations below the mean among people with MCI (mild neurocognitive disorder). DSM-5 also broadened the potential areas of cognitive impairment by including impairment in social cognition, complex attention, and/or perceptual-motor functioning. DSM-5 criteria no longer require impairment in memory, which was a key criterion in DSM-IV. DSM-5 diagnoses of major neurocognitive disorder or dementia rest upon quantifiable impairment (2 standard deviations below normative levels) in one or more abilities, whether in memory or other cognitive domains. **Neurocognitive disorder (NCD) due to Alzheimer's disease** is the only NCD diagnosis that requires impairment in two or more domains (unless there is evidence of a relevant genetic mutation). This is an important advance because many clinicians believed that the requirement of memory impairment, previously described as the "Alzheimerization" of dementia (Royall, 2003), led to underdetection of types of dementia that are not characterized by prominent memory impairment in the earliest stages, such as frontotemporal dementia, vascular dementia, and Lewy body dementia.

The cognitive changes must be a decline from prior levels of functioning to warrant a diagnosis of dementia

According to the DSM-5 criteria, the cognitive changes in the individual still need to be severe enough to interfere with daily living to be diagnosed as a major NCD (dementia), must be a decline from prior levels of functioning, must not be accounted for by another DSM condition (e.g., major depressive disorder), and must not be caused by another medical condition (such as delirium).

Emerging diagnostic research suggests growing interest and value in biomarkers for Alzheimer's disease

The NIA/AA diagnostic guidelines for dementia due to Alzheimer's disease (McKhann et al., 2011) are a revision of the McKhann et al. 1984 guidelines. Several scientific advances are reflected in the new criteria: (1) the pathology of Alzheimer's disease occurs across a broad clinical spectrum, ranging from normal to MCI to dementia; (2) other neuropathologies (e.g., Lewy body disease) can also cause dementia; (3) the development and use of biomarkers in research on Alzheimer's disease; (4) the observation that memory impairment is not always the predominant deficit in Alzheimer's disease; (5) advances in the understanding of genetic risk factors and mutations in Alzheimer's disease; and (6) the removal of age cutoffs for diagnosis of dementia – with the growing awareness that Alzheimer's disease is the same disease whether it strikes someone in their 40s or 90s, there is no need for "pre-senile" and "senile" categories.

These new guidelines for dementia (McKhann et al., 2011) require cognitive *or* behavioral symptoms that interfere with work or usual activities and represent a decline from a previous level of functioning. Occasionally patients retain the ability to perform IADLs such as paying bills or cooking, but they have become impaired in their work. Evidence of two areas of cognitive

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Theories and Models of Alzheimer's Disease and Dementia

Before exploring the most common causes of dementia, it is important to understand that many, if not most, patients with dementia have more than one contributing etiology, and this is typically documented only after autopsy. For example, in one autopsy study, 20% of patients with Alzheimer's disease were also found to have Parkinson's disease, and 25% of patients with Alzheimer's disease were also found to have cerebrovascular disease (Gearing et al., 1995). In another study (Schneider, Arvanitakis, Leurgans, & Bennett, 2009), 54.5% of people with MCI had autopsy-confirmed Alzheimer's disease, and 19.4% had mixed pathologies. In reality, it is common for a person with a MCI or dementia to have more than one underlying disease. Therefore, a diagnostic evaluation can be considered a way of ruling out various possible causes before settling on two or more etiologies that cannot be ruled out. A person is unlikely to show cognitive or behavioral symptoms of a neurological disease process until enough tissue is damaged; it may take the compilation of two or more pathological processes to reach this point.

2.1 Alzheimer's Disease

2.1.1 Neuropathology

Amyloid plaques and neurofibrillary tangles are the primary brain changes in AD

Alzheimer's disease is the most common cause of dementia among older adults. The hallmark **neuropathological characteristics** of Alzheimer's disease include the development of plaques, composed mainly of amyloid beta (Aβ), and neurofibrillary tangles, consisting mainly of tau. These changes are accompanied by synapse loss and cerebral atrophy, or wasting away of tissue.

Amyloid plaques develop between neurons as byproducts of neuronal degeneration, and interfere with communication between neurons. The amyloid cascade hypothesis suggests that amyloid plaque build-up subsequently leads to neurofibrillary tangles, but this hypothesis has increasingly been called into question. Cognitive decline and impairment correlate most with the number of neurofibrillary tangles (Nelson et al., 2012).

Neurofibrillary tangles develop within neurons, and neurons lose their structural integrity as a result. These tangles develop when the microtubules that carry substances from the neuronal cell body to the end of the axon become twisted. The twisted microtubules condense into tangles. The tangles

develop in the entorhinal cortex, hippocampus, and other parts of the temporal lobe, before spreading to other parts of the brain, including the nucleus basalis of Meynert in the forebrain. The nucleus basalis of Meynert is involved with the production of acetylcholine, and as this area degenerates, the depletion of acetylcholine also impairs memory. Current pharmacological treatments therefore attempt to prevent the breakdown of acetylcholine; this will be discussed later in the treatment section of this volume (see Section 4.1.2 "Medication Treatment for Cognitive Symptoms"). More recent evidence suggests that the disease process, particularly tau build-up, begins in neurons in the locus coeruleus in the brainstem that project to the cerebral cortex, and that this process may begin in childhood or puberty (Braak & del Tredici, 2012). Mechanisms as yet unknown enable neurons to function even when tau pathology is present.

Because of the risk inherent in obtaining a biopsy of brain tissue in a living person, Alzheimer's disease cannot be diagnosed definitively until autopsy of the affected individual's brain; hence, 100% certainty can only be achieved after death. Therefore, clinicians make diagnoses such as "major NCD probably due to Alzheimer's disease" or "dementia of the Alzheimer's type" when Alzheimer's disease seems the most likely cause. At least three sets of criteria exist to gauge the severity of Alzheimer's disease in an autopsied brain. First, the Consortium to Establish a Registry for Alzheimer's disease (CERAD) provided criteria for staging the disease (Gearing et al., 1995), and several neuropsychological tests have emerged from this consortium's work that are still used today (e.g., the CERAD drawings test, which includes having the patient copy a cube). The Braak and Braak (1991) criteria for staging the disease propose a progression of the disease from the transentorhinal cortex (Stages I and II) to the hippocampus (Stages III and IV), ending with involvement throughout the neocortex (Stages V and VI). Finally, after the death of Ronald Reagan, who died of complications related to Alzheimer's disease, the National Institute on Aging–Reagan Institute (NIA-Reagan) criteria were developed (Newell, Hyman, Growdon, & Hedley-Whyte, 1999). The NIA-Reagan criteria provide a high, intermediate, or low likelihood that a person's dementia was due to Alzheimer's disease.

Brain autopsy is used to confirm Alzheimer's disease

Amyloid plaques also develop in the arteries and capillaries of the brain, a condition called **amyloid angiopathy**. Amyloid angiopathy is also known as *congophilic angiopathy*, and when carefully sought may be found in as many as 85–95% of the brains of people with Alzheimer's disease (Vinters, 2015). It is a common cause of spontaneous intracerebral hemorrhages in older adults as well as of the progressive build-up of multiple cerebral microinfarctions (Vinters, 2015). Conceptually, one can wonder whether a patient with this condition should be diagnosed with dementia due to Alzheimer's disease, vascular dementia, or both.

Clinical Pearl
Challenges in How to Frame Evaluation and Diagnosis for Patients

Many people who are cognitively normal until death are nonetheless found to have the neuropathological features of Alzheimer's disease in their brains at autopsy (Vinters, 2015). Indeed, abnormal tau levels may be found as

3

Diagnosis and Treatment Indications

Clinicians should assess more that just cognition

Dementia is more than a neurological entity. Though the initial changes are neuropathological, the subsequent effects that clinicians and family members deal with are neuropsychological and psychosocial. There are four assessment and treatment targets in dementia and MCI (regardless of underlying neuropathology or dementia type) that can be adopted as multiple levels to understanding dementia: neurobiological, cognitive, psychological, and behavioral symptoms, and caregiving and family issues. Each of these levels is critical to understanding dementia and MCI, and should be considered in the evaluation process and subsequent intervention (Figure 2).

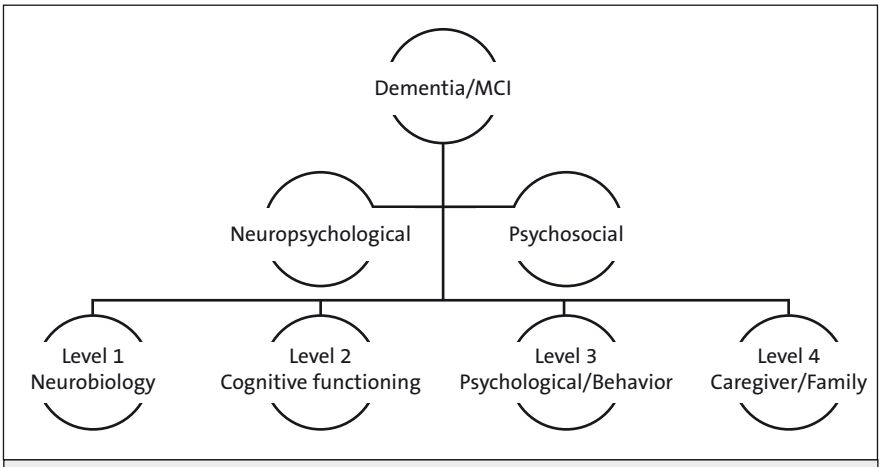

Figure 2
Assessment and treatment targets in dementia and mild cognitive impairment (MCI).

3.1 Level 1 – Neurobiological Changes

As described in Chapter 2, dementia and MCI begin with underlying changes in the brain, including amyloid plaques and neurofibrillary tangles in Alzheimer's disease; cerebrovascular disease, including subcortical ischemia and microbleeds in vascular dementia; Lewy bodies in Lewy body dementia and Parkinson's dementia; and abnormal levels of atrophy across dementias,

An essential guide to assessing and treating people with dementia syndromes

As the number of older adults with dementia continues to skyrocket, every health care professional needs accurate, up-to-date knowledge of these conditions, their prevention, and possible treatments. This compact, evidence-based book discusses essential aspects of the diagnosis, assessment, and interventions of Alzheimer's disease and the syndromes of dementia and mild cognitive impairment. It reviews the diagnostic criteria from the National Institute on Aging, Alzheimer's Association, and the DSM-5 and provides a broad range of treatment options, including psychosocial, educational, and lifestyle interventions. Practitioners will especially appreciate the current overview of caregiver interventions. Practitioners and students alike will find the clear information, the tools for assessment, and other resources provided in this volume extremely useful for helping patients and their families cope with dementia.

"This book provides best practices in assessment and intervention in a clear, concise, and insightful fashion. The experience and insights of the expert authors come through in terms of both the breadth and depth of information they provide. A must-read for every clinician working with people with dementia and those caring for them."
Peter A. Lichtenberg, PhD, Director Institute of Gerontology and Merrill Palmer Skillman Institute, Wayne State University, Detroit, MI

"This is the book on dementia that I've been waiting for – a concise, evidence-based volume that covers the essential aspects of diagnosis, assessment, and treatment of persons with Alzheimer's disease and related conditions. This is the one book that healthcare providers across disciplines should have ready access to in their work with individuals and family members who are struggling with possible or clearly established dementias."
Shane S. Bush, PhD, ABPP, Board Certified in Clinical Neuropsychology and Geropsychology; Coauthor of *Ethical Practice in Geropsychology* and coeditor of *Geriatric Neuropsychology: Practice Essentials*; VA New York Harbor Healthcare System, Brooklyn, NY & University of Alabama, Tuscaloosa, AL

"This book, written by well-regarded researchers, offers a contemporary overview of Alzheimer's disease and dementia that both the seasoned practitioner and the new graduate will find useful. The breadth of interventions covered is impressive, encompassing lifestyle interventions, person-centered treatments, and caregiver and dyadic approaches. A section on multicultural issues is most welcome, and points to the care that must be taken in clinical practice for diverse client groups."
Prof. Nancy A. Pachana, PhD, FASSA, Co-Director, Ageing Mind Initiative at The University of Queensland, Brisbane, QLD, Australia

"The authors have digested a massive amount of the latest research on Alzheimer's and dementia, yielding a state-of-the art understanding of the assessment and treatment of neurocognitive disorders germane to readers from diverse health services disciplines. Particularly impressive is the integration of science and practice from neuropathology, psychopharmacology, gerontology, neuropsychology, and clinical psychology that will benefit both academics and clinicians."
Victor Molinari, PhD, ABPP, University of South Florida, College of Behavioral and Community Sciences, School of Aging Studies, Tampa, FL

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Volume 38
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Florence · Copenhagen · Stockholm
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ISBN 978-0-88937-503-1