

The Assessment and Treatment of Older Adults

THE WATCH AND WAIT HOLISTIC MODEL

Edited by
LEE HYER

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*The Watch and Wait
Holistic Model*

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**Cambridge
Scholars
Publishing**



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This book first published 2020

Cambridge Scholars Publishing

Lady Stephenson Library, Newcastle upon Tyne, NE6 2PA, UK

British Library Cataloguing in Publication Data

A catalogue record for this book is available from the British Library

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ISBN (10): 1-5275-3859-1

ISBN (13): 978-1-5275-3859-7

TABLE OF CONTENTS

Preface	vii
Contributors	xi
Chapter 1	1
Watch and Wait Model	
Lee Hyer	
Chapter 2	35
Meta-Trends of Aging	
Lee Hyer and Lauren Lee	
Chapter 3	99
Assessment	
Lee Hyer and Kristin Wagner	
Chapter 4	153
Watch and Wait: Perspective and Practical Issues	
Lee Hyer	
Chapter 5	202
Screen Measures of Five Domains	
Lee Hyer	
Chapter 6	265
Application of Watch and Wait to Chronic Medical Conditions and Comorbid Behavioral Health Problems	
Catherine A. Yeager, Ian A. Yeager and Lee Hyer	
Chapter 7	344
Cognition	
Christine Mullen and Lee Hyer	

Chapter 8	417
Depression	
Lee Hyer, Catherine A. Yeager and Ian A. Yeager	
Chapter 9	486
Anxiety	
Lee Hyer and Krystal Jackson	
Chapter 10	527
Life Adjustment	
Lee Hyer	
Chapter 11	574
Personality	
Lee Hyer	
Chapter 12	628
Perspectives on Health Care	
Lee Hyer	
Appendices	653

PREFACE

We expand on our first book in 2014 (Hyer, “Treatment of Older Adults: A Holistic Model”). In that book we laid out plans for a holistic perspective on assessment and care of older adults. Here we update that book, unfold the complexity of the core domains, specify assessment plans, argue for necessary treatment, and place holistic care in perspective. Treatments work at best only 50% of the time and most often not permanently. Older adults persist as both system and self-change failures. We need better models. For older adults with the usual problems of depression, anxiety, cognitive decline, an admixture of somatic ills (health), or the plethora of life adjustment issues of later life, a focus on integrated care seems not only reasonable but now required. Treating “depressive symptoms” in isolation of the whole person, especially one with cognitive and physical limitations, risk a less-effective reduction in overall symptoms. Evidence for such multi-faceted approaches to treatment is growing, but we must borrow from the depths of literature for each problem and look at the efficacy for older adults, as well as what works for younger adults.

What is the best way to address the modal psychosocial problems of late life taking into account what science has to offer, what seems the clinical case has featured, and what can be done, for ONE person. What are the reasonable concepts and learnings required for care of older adults? In the long run to benefit elderly patients in the community, personalization of care must employ comprehensive common sense care algorithms targeting both modifiable predictors of poor outcomes and organizational barriers to care.

This book argues for a case-based model with differences in the approach and in the interventions. We need an all-encompassing model for change. The care algorithms based on this model should of course target clinical/biological predictors of adverse outcomes of depression (or the core problem), but also address unmet needs through linkage to appropriate social services, enhance the competencies of elderly persons so that they make use of their resources, and attend to patient psychotherapy issues of psychoeducation, behaviors, thoughts, and emotions. We provide a model of care then that advocates for something more than a diagnosis.

Over the last 30 years, there has been a dramatic change in the treatment of older adults where mental health is concerned. Aging then is not a meaningful explanation by itself as to why one might experience a problem like cognitive decline or impairment. We need to look at the whole person who is older and has accumulated stuff. What are the variables of most concern? The focus should be on identifying markers that place the person on a continuum, not whether or not that person is in a specific group. This involves the idea that there is a mystery unfolding, where risk factors in genes and the environment interact and accumulate.

Given these concerns, what is a healthcare provider to do? This book addresses an effort to simplify the mental health problems at later life around five core areas with some flexibility regarding assessment and treatment. The five areas include depression, anxiety, cognition, medical concerns, especially pain and sleep, and finally life adjustment. Life adjustment, the last variable, involves all of the day-to-day reality-based concerns that unfold for older adults. Based on this model, it is believed that better psychological and psychiatric care can be attained in the context of primary care.

Several rubrics will drive the thinking where treatment is concerned. We believe in a “Watch and Wait” model; that is, people are assessed and a careful monitoring time is instituted where the patient is given hope, psychoeducation, support, and a belief that change will occur with careful preparation. This is most of all based on validation and connection. From this, there evolves a clear case-based, person-centered care process that leads to the application of care with an admixture of best evidence (empirically supported tenets) as well as practical clinical common sense. This is another way of looking at a stepped care model. In this effort, teams are used and of course monitoring and special efforts are made toward addressing all of the five areas, not just the one that is most of concern.

This is a book for the psychosocial and medical disciplines who wish to address the new care of older adults. Health care professionals, especially in psychology, social work, and nursing, are targeted. It is intended to address a “just sufficient” level of data and extant research, to be integrative, to be practical, and to challenge professionals to assess and treat this population inside and outside the box. Importantly, it stresses the need for an interdisciplinary activity and primary care involvement. It stresses the need for seat-of-the pants interventions. It is also case-loaded and applies an assessment program that sets the stage for care.

Book Perspective

While this is my work, I have had the luxury of colleagues and many students. Many have helped here and are featured in the chapters. They have been magnificent with input, case correction, citations, and ownership. At the end of my career this book like the previous one has been what I called a “labor of love.” In the transition of the first book to this one, the words of Maya Angelou are apt: “Do the best you can until you know better. Then when you know better, do better.”

Chapters

Chapter 1 provides a backdrop to the field of gerontology and geriatrics. It addresses the changes in the last decade, the current clinical field, and the need for a change. It provides needed background information for the care of older adults. Importantly, it argues for the Watch and Wait Model and the five core, “just-sufficient,” domains of aging care.

In Chapter 2 (with Lauren Lee, intern) we discuss the 10 meta-trends of our society and professional organizations that set the backdrop of aging assessment and care. These are the meta-factors that set the stage for the care of older adults for the last decade. We include in this the importance of psychological care and general life adjustment and quality of life.

In Chapter 3 (with Krissy Wagner, intern) we consider assessment. This is a critical and under-represented aspect of aging. We discuss the unique needs and dilemmas/background for older adults. We argue for screens in each of the five domains as well as a recommended expanded assessment package.

Chapter 4 expands on the model. We discuss the importance of Watch and Wait and how this adds to the evaluation/treatment of older adults. The Watch and Wait model is then outlined in an in-depth manner. This is then a core chapter for the working of the Watch and Wait model.

Chapter 5 unpacks the five domains and reasons for their importance. We address the need for each and interrelationship among them. Since the modal problems at late life, anxiety, depression, somatization (health) and cognitive decline, as well as adjustment, are inter-connected, they are best dealt as a group (a profile of the person on the five domains) with modular interventions. There is, therefore, a unified approach to treating problems at late life.

Chapter 6 considers health (Dr Catherine Yeager and Ian Yeager); Chapter 7 addresses cognition (Christine Mullen, intern); Chapter 8 tackles

depression (myself along with Dr Yeager and Ian Yeager); Chapter 9 unfolds anxiety (Dr Jackson, former intern); and Chapter 10 explicates life adjustment. Assessment makers are outlined for each domain and cases provided. Most patients fail treatment because their problems exceed the ability of the effectiveness of our science, or they or we expect too much from our therapies. We address the problems of diagnosis, problems with extent treatment, and problems with aging in each domain. We highlight treatment and seek integration with the other factors. Cases are liberally presented.

In Chapter 11 we present personality as the bedrock of the core domains just discussed. The person responds to life problems based on their temperament/personality and this information is seen as important for the understanding and treatment of the older person. Personality at later life is not only intriguing but informative and highly relevant.

The last chapter (Chapter 12) makes an effort at perspective. The effort is made to see the problem of aging from a societal and policy perspective. We summarize the book and its importance in psychosocial care of older adults.

Any one of the problems in this book can by itself be tumultuous. The desire to find a clear and identifiable cause including factors that could have prevented this is seductive. But simple cause-and-effect decisions imply that it is possible to trace a straight line from some specific event/act in the past leading to the emergent problem(s). Then we can rewind the film to the beginning and see the problems. This is quixotic. Nonetheless, this process is relevant and important as we approach an aging person with a plan and a model that can assist in the care. Then outcomes become the marker. We hope we do this in this book.

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CHAPTER 1

WATCH AND WAIT MODEL

In 2014 we published the book *Treatment of Older Adults: A Holistic Model*. This book articulated a Watch and Wait model of care and posited that there are five core domains necessary for the understanding of older adults. We have had a few years to muse over this effort, have watched the field of geriatrics and geropsychology mature, and have made several additions to that tome. We have noted that over the last 30 years there has been a dramatic change in the understanding and treatment of older adults, especially where mental health is concerned. This continues. We believe that it has been reasonably confirmed that the efficacy of psychopharmacological and psychological approaches is small. If anything, multiple interventions are attempted for the many problems and some cohesion is certainly helpful. Usually there results a “response” to a problem, not a remission. If truth be told, older adults improve because they and their caregivers commit to them and optimize life style and avoid negative behaviors. The utility of a psychiatric classification as determining the course of treatment is poor. In fact, psychiatric/psychological care is less than efficient relative to other medical disciplines. Perhaps it is more complicated or has more causal density. It can be argued further that there are few in the way of unique geriatric syndromes. Fortunately, we can build on the work at earlier ages.

This book attempts to expand on that Watch and Wait model that, again, pays some attention to the nuanced differences in treatment (one antidepressant vs another, one psychotherapy vs another, medication vs psychotherapy, etc.), but that more importantly devotes time to the whole person and their world. In effect, we expand on our model and argue that traditional actuarial foundations are necessary but not sufficient for treating older adults.

Background: The Bad News

We now know more about aging itself. Biological aging is largely determined by the internal biological clock and an accumulation of insults through living. Where the lifespan of the organism is closely related to biological aging, individual longevity is always a function of specific environmental circumstances, the accumulated insults. The two operate at every level of the bio-hierarchy – genes, proteins, cells, organs, and organisms. As we shall see, epigenetics reigns as it accounts for changes in gene expression that are not mediated by mechanisms of the DNA. In fact, we are best considered products of the biopsychosocial model where there are varying levels of physical, cognitive, emotional, behavioral, and environmental factors that contribute in the formulation of the older person (Andrasik, Goodie, & Peterson, 2015).

The complexities of addressing the psychosocial variables responsible for later life issues are readily apparent. The realistic constraints of living into later life make outcomes worse for older adults. Health now rests on our daily behavioral routines. The average health care costs per year are directly related to income: Those living in poverty cost almost twice that compared with those with more means. We believe that the biomedical model is reductionistic and dualistic. Added to this big cost picture, mental health by itself accounts for only 6% of all US health care costs: It influences 50% of all medical illnesses. Over 90% of people over 65 take some form of medication. Psychiatric drugs exceed all other mental health costs. Most older adults have a chronic disease, which is the cause of seven out of 10 deaths. In 1965, the average life expectancy was 70.2 years. By 2012, it had risen to 78.8 years. In 1965, the Medical Board of Trustees projected the Medicare costs for 1990 would be around 9 billion dollars. The actual cost was just short of 70 billion. In 1970, the ratio of tax payers to Medicare beneficiaries was 4:1 and Medicare represented 3.5% of the Federal budget. By 2030, the ratio is expected to be 2.3:1 and Medicare likely to represent over 20% of the budget. Americans today collect Medicare benefits for two and one-half times longer than they did in 1965. In 2012, lifetime Medicare contributions from the average American worker came to about \$114,000. Between retirement and death, the same worker used \$355,000 in Medicare benefits. The first baby boomers began receiving Medicare in 2011. When the last boomer starts receiving Medicare in 2029, there will not be enough tax payers in the work force to cover the shortfall. Compared to other age groups, older adults have the highest numbers of doctor visits and hospital stays, and the highest prescription medication usage. Left unchecked, healthcare

expenditures will likely rise from the current level of ~15% to 29% of gross domestic product (GDP) in 2040.

As intimated, medication use is high among the elderly. Adverse drug reactions account for a substantial amount of emergency room use, hospital admissions, and other healthcare expenditures. Only 50% of medication is taken properly, and there are 1.9 million drug-related injuries per year (Cogbill, Dinson, & Duthie, 2010). Taking just blood pressure medication as an example, only 25% of older patients remain in treatment and consistently take their medications in sufficient amounts for blood pressure control. Poor medication use has been attributed to several barriers. These include physical illness, medication side effects, cognitive dysfunction, psychiatric conditions such as mood disorders, functional loss, social loss, and inability to afford the medication at full dosing (Cogbill et al., 2010). As people age, their activities of daily living (ADLs) decline dramatically between 65 and 74 years – a fourfold change in ADLs generally and a threefold change in independent activities of daily living (IADLs). Looking more closely at functional decline, adults older than 75 years account for 59% of fall-related deaths, but make up only 5% of the population.

Perhaps dealing with aging issues is both a biological problem, where knowledge of geriatrics is concerned, and an ethics problem, as most problems involve values. Medicine becomes a help or a hindrance, but is not curative. Therefore, the need to expand the moral imagination regarding the older adult becomes critical. Healing will involve reweaving the most primal of connections to this sacred web. This is not “psycho”therapy. But it is close. Older adult problems are essentially fragmented and a meaningful intervention can be the glue for personal and group coherence. The therapist often becomes an ethicist, translating values and goals into medical care (life prolongation, maximization of function, or maximization of comfort). The ethical parameters of autonomy, beneficence, justice, and non-maleficence become the new paradigm of care. After all, psychiatric care does best with mild-moderate problems of any sort. Why? Because it is something human contact and intervention can change, if done reasonably well – validation, hope, and a script as well as follow-up. Good care in later life is a creative admixture of common sense, perhaps as deliberated by ethics, and science. In some ways it is as much a philosophical or psychological enterprise as a disease process.

This book attempts to walk the line for healthy aging and validated psychological care rubrics. This crucible is built on a model that hopes to unfold the many real dilemmas of our living longer and making it work with what we know about health and human struggle. It is a holistic, even a person-centered program. The Watch and Wait model is alive and well.

“...the physician’s primary duty is to provide the best possible care. It should be whatever is best and reasonable for the patient. I do not believe that patients want their physicians to shrink from making recommendations. Like me, most patients want their physicians to explain to them the options and recommend the best the best course of action. This is done by discussing risks and benefits within the context of mutual decision-making. This provides the means for putting caring ethics into practice.”

William Branch, MD JAMA Apr 14, 2015

Outline

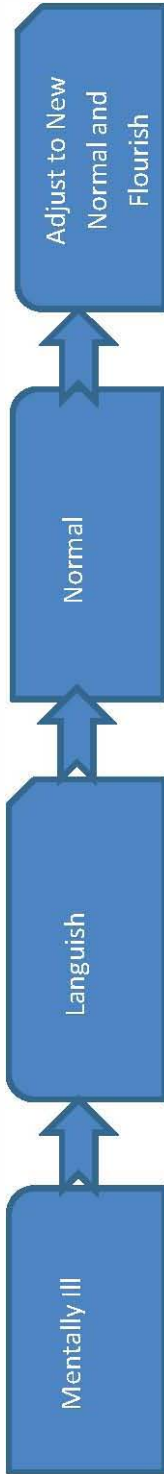
This book is intended to be practical, based on the modal clinical problems of older adults. It is the problem list of the older adult (perhaps) that is most relevant for targeted care and change. We explicate this especially with an guided assessment of older adults and unfold this with therapy suggestions. We have previously discussed our model, Watch and Wait (Hyer, 2014), but address this again in chapter 1 and further in chapter 4. In chapter 2 we discuss the meta-trends in aging and health care over the past decade leading to this book. Then we address the value of assessment and why assessment screens are important in chapter 3. Chapter 4 unfolds the model more completely and expresses psychotherapy perspectives. We then devote chapter 5 to the five key areas posited by the model. For each domain we recommend unique measures. For each area we cover some background, recommend a specific battery, and discuss its role in care. We also set the stage for each domain to be covered separately, providing a distinct chapter for each one (chapters 6-10). Always we interweave medical and health issues. We conclude this consideration of the domains with a discussion regarding personality (chapter 11) as this highlights the expression of the five core domains. It is the scaffold for the profile of problems. We end with some perspective on aging and a summary in chapter 12.

We seek to be clinical and practical. The issue with older adults has now morphed into multiple mid-level problems played out in a complex environment and beset with a unique set of strengths and weaknesses. Our intent, then, is to cover core areas that represent the Watch and Wait model. We over-focus on assessment and treatment for older adults. This is a book about practical clinical problems and their assessment in regard of older adults.

Model: Watch and Wait

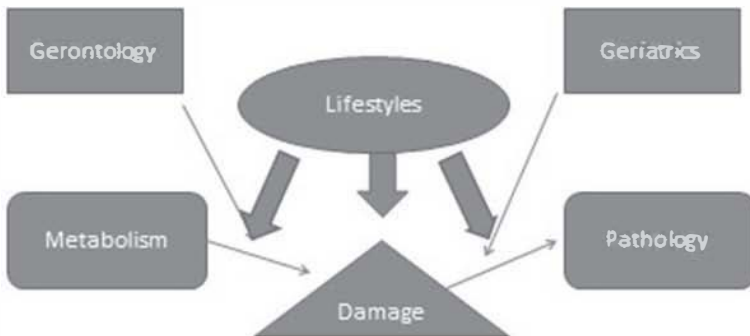
● **Overview:** The Watch and Wait model requires some introduction. The schema below best accounts for change within a more progressive medical model. It is not a disease model. Mental disorders continue to be identified as variegated syndromes and may never be amenable to specific diagnostic tests. Remission is partial and short-lived, and as many as one third do not respond. Cures are a misnomer. In the Flourish Model of mental health (Keyes, 2007), positive psychological functioning (self-acceptance, personal growth, purpose in living, environmental mastery, autonomy) and positive social functioning (self acceptance, social actualization, social contribution, social coherence, social integration) are key. This model does not operate in a world where not being mentally ill is healthy. The absence of depression is not happiness. The ideal is not going from -1 to 0 but -1 to +1. It thrives on baby steps with a new habit taking form over time. Languishing, the non-remitted state concerning clinical problems, is not a status of chronic human imperfection but one of many negative and few positive emotions. The ideal is to extend beyond normal and to flourish, to be optimally responsive to living, in essence to be happy. This can involve a meaningful attachment to accepting life as the new normal and, by acceptance, flourishing.

The model for the care of older adults requires more explanation. ● One way to view this is through the lens of gerontology and geriatrics (below). The slow alteration in the accommodation and assimilation of life is first attached to the elements of gerontology, an element of care that is based on awareness of the components of good living, meaning that the phenomenology and epigenesis of culture and psychosocial factors impact the phenomenology of living. This is most influenced by lifestyle. Health care providers need to give respect to both gerontology and geriatrics: Homage to the latter masks the subtle power of the former. Gerontology leads the way. Aging is the result of an ongoing metabolism that collects insults and degrades because it becomes less efficient at house cleaning. Inflammation, oxidation, impaired immune reactions and the like become enemies of cells. Intra-cell and extra-cell damage occur. Amyloid and tau, the building blocks of plaques and tangles, are often the result. Gerontology impacts the pre-processes for damage and geriatrics provides tertiary cures when possible. But the factor that activates change is lifestyle and a consequential committed attitude to living well. It is best done in mid-age and continuing, but is helpful at any age.



We get more bang for our life buck in gerontology than in geriatrics: knowledge, prevention and discipline before medical treatment. The former is the biopsychosocial dynamic of life; the latter is the secondary prevention or fixing of its maladies. The dynamic for both is to optimize the culture of positive living and growth. This includes having a healthy lifestyle and a positive attitude. But age and disease eventually win out and how this interaction is slowed and softened becomes another key to better living. Damage will occur and have to be attended to. The unfolding of problems leads to aspects of the disability model and negative consequences mandating secondary and tertiary care.

Gerontology and Geriatrics



Lifestyle is the most salient gerontological and geriatric influence. Dhalwani et al. (2017) studied the association between lifestyle factors and the incidence of multi-morbidity in an older English population. There was clear evidence of a temporal association between the combination of different unhealthy lifestyle factors with multi-morbidity. Population level interventions, therefore, were encouraged to include the reinforcing of positive lifestyle changes in a population to reduce the risk of members of the population developing multiple comorbidities. The multiple comorbidities included such things as diabetes, hypertension, stroke, myocardial infarction, congestive heart failure, angina, lung disease, chronic obstructive pulmonary disease, asthma, arthritis, osteoporosis, cancer, hearing problems, Parkinson's and Alzheimer's disease, other dementias, macular degeneration, and glaucoma. This group found a dose-response association between unhealthy lifestyle factors and multi-morbidity. Physical inactivity increased the risk of multi-morbidity by

32% on its own and inadequate fruit and vegetable intake increased the risk by 65% in women. When physical inactivity was combined with obesity or smoking, the risk increased by 2 or 3 times and by more than 4 times when combined with both smoking and obesity. Data sets showing these results are almost commonplace now.

Watch and Wait: This model targets five areas of adjustment for older adults. By addressing core psychosocial problems, including depression, anxiety, cognitive impairment, general health and life adjustment, we believe that we provide for the necessary focus to help the patient adapt and cope with their problems, promoting successful outcomes. So, if the older adult has depression, then we seek to treat depression in the context of the person. This means creating a profile, regarding the five domains, which provides the best understanding of the person, but a depression focus is primary for the moment.

●f course, evidence for such multi-faceted approaches to treatment is nascent, so we borrow from each problem and look at the efficacy for older adults, as well as what works for younger adults. Due to the complexity of patients, the normal application of empirically supported therapies (ESTs), the nuances of the research, and the use of predictor variables in care, while helpful, are not robust enough to warrant allegiance beyond just some respect. We maintain that the differences between one antidepressant and another, one psychotherapy and another, or one medication versus another or a psychotherapy provide us very little help. As we noted in the first book (Hyer, 2014), published reports suggesting that attending to novel “significantly better,” or “evidenced-based,” will result in better patient outcomes is helpful, but doing so with older adults often diverts attention from the real world issues, and has only marginal evidence of benefit. We believe that a comprehensive algorithm for treating more than one problem in older adults is more important (Thielke, Vannoy, & Unützer, 2007). This is also the thinking and position of the transdiagnostic model (reviewed later).

We explicate the Watch and Wait model around case-based care. We emphasize a case-based plan, applying information, validation, assessment, and a plan of treatment modules. A careful and slow process of care is an improvement over fast-paced primary care and psychiatric clinics. The therapist does not pick one best treatment at the outset. Mistakes are often made at the gate (e.g., deciding too quickly to initiate care, under-dosing, inadequate trial duration, poor frequency of follow up, and lack of monitoring). Problems actually better confess themselves over time. According to the model, patients are carefully assessed and monitored. The patient is given hope, a humane context through psychoeducation and

support, and a belief that change will occur with careful preparation. This is a case-based, person-centered model that leads to the application of best evidence in the real world. In 3-5 sessions, the health care provider recognizes how the patient presents with and experiences the five problems, validates and builds alliances, provides necessary psychoeducation, carefully selects treatment options, applies objective measures for a treatment response, and monitors. Changes are made with equal deliberation. One does not get better if the person does not have preparation for and then “experience” in the intervention. We also know that in the complex treatment of older adults, success depends on patient beliefs and organized extra-therapy variables, as much as the actual treatment plan and monitoring.

FAILURE POINTS:

1. Deciding too quickly to initiate care
2. Under-dosing
3. Inadequate trial duration (6 weeks necessary)
4. Poor frequency of follow up
5. Lack of monitoring
6. No team or family involvement
7. Wrong Rx: Only SSRIs, for example
8. Insufficient time: Noncompliance with meds, dropout of psychotherapy
9. Wrong dosage: Too little or not in the “window”
10. Interference from other Rxs: Med side effects
11. Adherence issues: Pt does not do tasks
12. Complex families
13. Use of substances: Opioids, other meds
14. Wrong diagnosis

Case Formulation: A Watch and Wait strategy is a deliberative process of care. It involves assessment, psychoeducation, trust building, concerns about treatment options, team interaction needs, and then availability of modules. As noted, the therapist does not pick one best treatment at the outset. Rather the therapist recognizes how patients present with and experience depression or other problems, carefully selects treatment options, and applies objective measures for a treatment response. Changes are made with suitable deliberation where monitoring and an exposé of the issues dictate change. We also know that in the complex treatment of older adults, the choice of treatment plan matters and needs to be tweaked and

re-tweaked over time. In fact, the case formulation marches through standard tasks. The model provided by Barlow (2003) is most helpful: The patient is assessed, monitored, and followed over time. Nomothetic treatment is identified and applied. Problems are noted and, when prophecy fails, the person-based characteristics are entered. Should change not be seen, then the therapy can be altered, perhaps in the service of a functional analysis.

In the unfolding of the Watch and Wait model, the deliberation by the clinician involves two things: case formulation (step care) and assessment. In mental health case formulation this has been formally recommended for over four decades. In later life any symptom can be generated by multiple permutations of multiple causal factors amid multiple causal paths. The health care provider formulates cases based on confirming and disconfirming data to determine whether selected empirically supported causal variables (e.g., cognitive distortions, medically related problems, poor self control, ineffective problem solving, low rate of positive reinforcement) are relevant, operative, and meaningful to this particular patient. Case formulation is always individualized and multivaried. It is also malleable. Life adjustment interventions can be easily assessed as the outcomes become apparent quickly. Cognitive restructuring too can be tested by decreasing self-defeating thinking, using behavioral experiments to test the validity of a belief, bibliotherapy, modeling, mild refutation, didactic explanations, homework assignments, visualization, and the use of caregivers, to name a few.

Process of Evidence-Based Practice

1. Monitor problems over time
2. Diagnose and formulate a case
3. Match patient problems to nomothetic, empirically supported treatments
4. Based on psychopathology and patient characteristics, use behavioral targets and apply idiographic interventions
5. Apply a functional analysis on non-responsive, deteriorating cases

Evidence-based practice then needs to be personal. It involves using the best available scientific evidence, individualizing the evidence for unique needs and preferences for each person, and a commitment to an ongoing expansion of the evidence in clinical expertise. In this context, clearly informing patients and families about the evidence, engaging them in the process of informed shared decision making, and protecting their rights to self-determination has generally lagged behind this goal. The

2003 President's New Freedom Commission on Mental Health report indicated that nearly every consumer of mental health services expressed the need to fully participate in his or her plan of recovery. Person-centered care and accountability are clearly high on the list for effectiveness in care. In the Institute of Medicine (IOM) landmark report, *Crossing the Quality Chasm: A New Healthcare System for the 21st Century*, person-centeredness was cited as one of the six primary aims of a transformed quality of healthcare delivery system. Not only does the report identify core goals or domains, it also identifies rules and principles that should guide and shape the provider in this behavior (New Freedom Commission on Mental Health. *Achieving the Promise: Transforming Mental Healthcare in America*, 2003).

Understanding Watch and Wait

From a Watch and Wait perspective several features are worth highlighting. First, a slow process of validation is implied. This includes not only joining but motivational interviewing with a clear objective to challenge subtly and often indirectly. It can involve many "therapies." Acceptance/commitment and mindfulness (Acceptance and Commitment Therapy) are in play early. Mindfulness and acceptance do not change reality; they encourage the heart to accept it. Additionally, psychoeducation emphasizes that the older person be encouraged to accept the reality of the issue. This encourages problem solving. Later there are many components for "real psychotherapy interventions" involving CBT, PST, self-instruction, exposure, distraction, and many other curative features in standard psychological therapies. There is a constant interplay also with the environment (with caregivers if necessary) and health care issues. The provider can pick and choose among the empirically supported techniques. As we have implied, reality often asserts a strong influence over psychological methods. Also, consistently there is an ear to on-going monitoring and assessment. Assessment is treatment.

Second, a case formulation (again) is always core to a model for change with Watch and Wait. Tritely but importantly, these always involve being empathic and non-judgmental in reviewing assessment data, working collaboratively with the individual in the family to create and sustain a helping partnership, using respectful first-person language, and avoiding the use of jargon and labels, engaging the individual and the family through motivational interviewing, encouraging the individual to determine who is present during the assessment and planning of meetings, being culturally competent and sensitive to the influence of cultural factors, implementing a team approach, being actively supportive with the

individual and the family's choices, and seeking feedback on his or her performance. The model that has often been used in identifying the components includes creating a narrative summary of the history of the patient, presenting symptoms, the precipitating events, predisposing factors, the perpetuating factors, and previous treatment and response. This goes to the core of the case formulation and implies everything that has to do with care.

Third, person-centered care focuses on targets that are distinct from traditional care. It is then person centered and not practitioner-based (Mast, 2012). It involves a strength-based and not problem-based focus. It clearly endorses a skill acquisition and not a deficit focus. Collaboration, community integration, and quality of care are core. Community-based and not facility-based care is also empowered. The least restrictive path to outcomes and preventative actions is clearly implied.

Fourth, Watch and Wait is a form of step care approach. Older patients pose additional difficulties in disease management due to multiple comorbidities, cognitive issues, and lack of social support. A step care approach to treatment delivery systems represents a reasonable attempt to maximize the efficiency of the resource allocation of ESTs. This is a kind of pyramid. In mental health and with older adults less is more and slow is good. Less intensive treatments are used to treat greater numbers of patients at the bottom of the pyramid, while fewer patients are treated with progressively more intense treatments at the top of the pyramid. The entry level is simple, cost efficient, and the least intrusive. Succeeding levels become more intrusive and expensive. The "stepped up" care is progressive and programmatic. This stepped up care can mean a change to more treatment or adding an additional mode of care. There is no cure for most maladies of later life adults, and the progressive nature of diseases requires effective disease management. Disease management is challenging due to exacerbations, complicated treatment regimens, and hospitalizations.

Step Care Models

There are several (watch and wait) models that assist in the biopsychosocial model. These are stepped care models. These models have been shown to be especially valuable in primary and secondary healthcare systems, such as outpatient mental health systems where demand for service outweighs supply. Originally developed for primary care in the United Kingdom, stepped-care has recently been reimagined (Donohue and Draper, 2011) for rapid access to mental health services in a wide range of settings. The model offers the lowest level of intervention intensity warranted by single or ongoing assessments;

treatment intensity can either be stepped up or down, depending on the level of patient distress or need.

Many promising online mental health tools have been developed and are available for purchase or licensing and can be developed at various levels within the stepped-care model. Traditionally, evidence-based mental health treatment interventions have been designed to be intensive and to be offered one-on-one by a highly paid specialist. Lower intensity and less expensive care that could address mental health concerns before they become acute or chronic are virtually non-existent in North America. Such lower intensity care may be seen as more palatable to a larger portion of those in need, who are not quite ready to accept all of the challenges of ongoing psychotherapy or of making needed life changes. Less intensive online programs can also allow users to test whether the actions include assessment, monitoring, psychoeducation, interactional health modules, therapist-assisted mental health actions, or intensive therapy for specific mental health problems, as well as several interventions in the family and in the community. Healthcare is also involved at all of these levels since it is central to older adult problems. Case management is also an essential referral issue.

Cornish et al. (2017) noted that the stepped-care model integrates a range of established and emerging online mental health programs systematically along dimensions of treatment intensity and associated needs for patient autonomy. Program intensity then can be stepped up or down, depending on the level of need. In the context of monitoring, which is configured to both the healthcare provider and the patient, feedback unfolds and the stepped-care model empowers patients to participate actively in the traditional process.

The T_{IP} intervention (Treatment Initiation and Participation) is one example of a step approach. It is based on the premise that when a patient can articulate their goals, barriers, or concerns, and has an understanding that good care is dependent on working with their physician, they are more likely to participate in care (Sirey et al., 2017). T_{IP} includes five steps and is delivered in three 30-minute sessions during the six weeks just after an antidepressant is prescribed: (1) review symptoms and antidepressant regimen, and conduct a barriers assessment; (2) define a personal goal that can be achieved with adherence; (3) provide education about depression and antidepressant therapy; (4) collaborate to address barriers to treatment participation; and (5) create an adherence strategy, and empower the older adult to talk directly with the health care provider about treatment. This type of patient participation may mitigate concerns about stigma and reluctance to report side effects, and will hopefully reduce early drop out.

Trans-Power

Person-centered care focuses on targets that are distinct from traditional care. It's of course person centered and not practitioner-based. As noted, it involves a strength-based and not a problem-based focus. It clearly endorses a skill acquisition and not a deficit focus. Collaboration, community integration, and quality of care are core. Community-based and not facility-based care also is empowered. The least restrictive path to outcomes and preventative actions is clearly implied.

First, we address the transtheoretical model. This is a friendly model that provides help for the overall intervention to be applied. This is applied as a conceptual formulation for motivation with patients who are struggling to make changes. Prochaska, Norcross, and Diclemente (1995) noted the importance of having some understanding of the stages of change that people go through. They posited that therapy works through a process: first is pre-contemplation where the individual has no formulation set to attack the problem in any meaningful way; then contemplation when this alters, and the person is somewhat ambivalent, but willing to look at the issues of the pros and cons of the problem; next comes preparation, which is the deciding to change and the building up of the confidence to do so; then comes action, which is actually changing; and last is maintenance, which usually occurs after about 6 months of the action phase. Lapses (temporary failures) can occur and the model repeats. This model serves as a nice understanding of the way in which people struggle to make their life go in a more meaningful way. It is implied too in our Watch and Wait formulation.

Enter the transdiagnostic model. The treatment of psychiatric disorders is entering a new phase characterized by a greater concern with the integration of treatment principles and methods across therapies. This supports our therapy-ideals of empathic, nonspecific validation procedures that build across therapies. The use of eclectic and pragmatic treatment strategies and the emergence of more modular and trans-diagnostic approaches, focusing on specific domains of pathology rather than global diagnoses, is one example. Livesley, DiMaggio, and Clarkin (2016) labeled this approach the Integrated Modular Treatment. They suggested individual patients present with a unique array of problems spanning multiple domains of functioning and that treatment should utilize an integrated array of strategies and techniques to address these diverse impairments. Domains of impairment such as symptoms, problems with emotion and impulse control, interpersonal patterns and self-identity problems, and overall severity of dysfunction, stand out as the focus of the

intervention rather than a more globally conceptualized categorical disorder.

A positive outcome is more a function of a structured approach and change mechanisms common to all effective treatments than to treatment-specific interventions. In this sense, the results of the outcome of treatments for a personality disorder, for example, converged with the results of psychotherapy outcomes generally: For more than 40 years outcomes have been similar across therapies, which suggest that different therapies share common elements associated with successful outcomes. The evidence is clear that outcome studies do not evaluate the mechanisms of action. The guiding principle then is that treatment should start not from a narrowly focused disorder-specific manual, but from a detailed analysis or deconstruction of the patient's psychopathology into domains of dysfunction and that treatment methods should be selected on the basis of what works for a specific problem and domain that are the focus of the therapeutic intervention.

All of this implies that a global diagnosis based on current diagnostic categories is really insufficient. The ICD-10 now has over 70,000 diagnoses and as many procedures. In order to select appropriate interventions, a psychiatric entity needs to be decomposed into different functional domains. This reveals an additional benefit of integration and accommodates the considerable heterogeneity among patients with a given disorder and permits treatment to be tailored to the individual. The importance of tailoring treatment to the individual is illustrated by an outcome study of personality disorders by Gullestad et al. (2012) that assessed pre-treatment mentalizing abilities. Patients with lower pre-treatment "mentalizing" skills fared worse in day hospital treatment than in individual therapy. Mentalizing involves cognitive/affective strategies for change. This can be taught. It is both transdiagnostic and reflective of care based on other processes than diagnoses.

The model incorporates varying degrees of three routes of integration that have traditionally been described in general psychopathology literature. These include common factors, technical eclecticism, and theoretical integration. The common factors approach seeks to identify principles of change common to all therapies and uses these principles to establish the basic structure of treatment. Technical eclecticism uses treatment methods from diverse treatment models without the adoption of their associated theories. Most experienced clinicians show a degree of technical eclecticism as they use methods they have found to work, even though they may not subscribe to the theoretical position on which they are based. Theoretical integration is more complex; it seeks to combine

major components of two or more therapies to create a more effective model.

Watch and Wait Specifics

Truth is confirmed by inspection and delay; falsehood by haste and uncertainty.

Tacitus

Assessment: The belief here is that the basics of care for older adults require special consideration. There is a need to deliberate over the case, proceeding from the real world, targeting practical issues, and entering mental health treatment. This requires assessment and monitoring, as well as flexibility. There is no desire here to usurp the scientist-practitioner model as we need to do formal assessments and to attend to the input of science. There are empirically supported methods. But, as noted before, we need more. Mast (2012) argues for a “Whole Person” approach where the value of “the person” of the diagnostic category becomes as important as the process of the diagnosis and treatment plan. We agree, but add that we need to apply the better known canons of our sciences to the person and formulate real plans that are titrated to tangible outcomes. Empathy and science together rule.

At some point something has to happen too. The patient needs to feel some relief or at the least a firm belief in the process. Context is a friend here. Often this can be done early in the process. In general, the type of problem for most older adults is not at the severe level. Problems are mostly mild or moderate. This includes extended problems with dementia issues. As such, the interventions generally need to be tailored to one who has multiple problems and who is mildly suspicious about the process. We need models of care then that encompass more than one diagnosis.

Clinical and psychosocial predictors of response to single antidepressants or comprehensive interventions have been identified. Less helpful factors include resistant anxiety, hopelessness, executive dysfunction, limitations in physical and emotional functions, chronicity of the current episode, and low income. There are of course more. But, such predictors can help in personalizing the first step of treatment for a given patient. Accordingly, a patient with one or more predictors of poor outcome may receive interventions targeting each modifiable predictor, as well as a more vigilant follow-up. For example, a low-income depressed elderly patient whose symptoms did not respond to an adequate trial of an antidepressant and who is experiencing hopelessness may benefit from a trial of

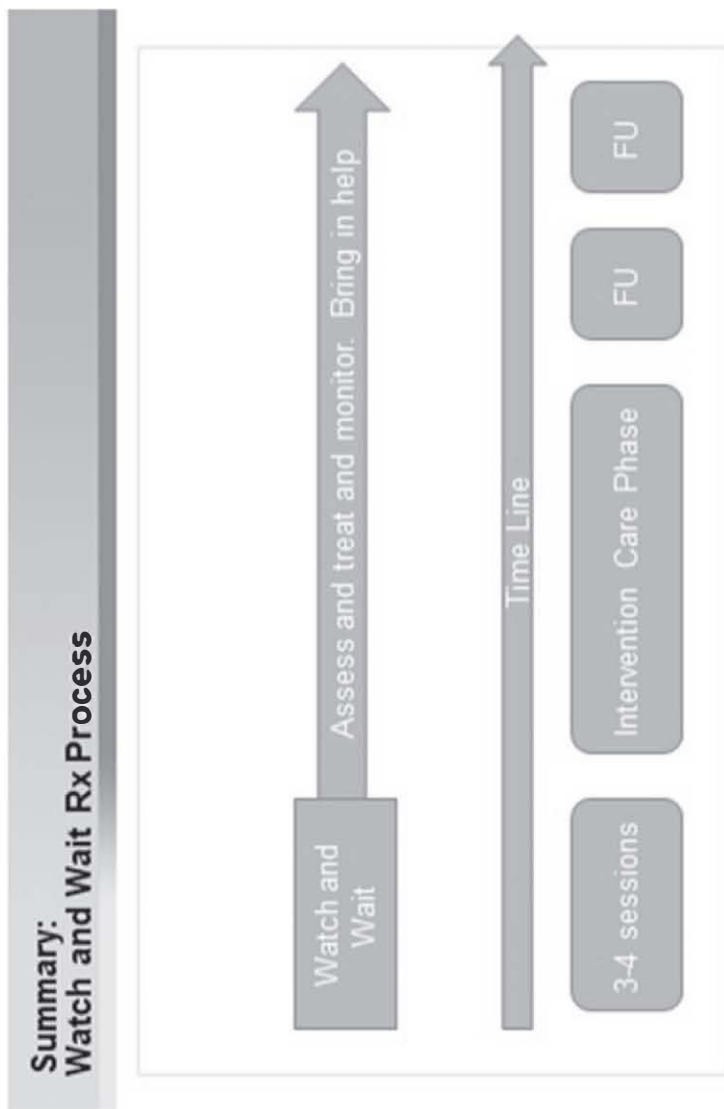
psychotherapy focusing on hopelessness, as well as case management connecting him or her with social services. This intervention will play out in the other 4 domains.

The critical issue in the Watch and Wait model is what to do before the total plan is activated. The application of this model hinges on the challenge of when to shift from the wait point to the activate point: When does the therapist pull the trigger for more active care? In the interim the health care provider can make a difference. The Watch and Wait model below advocates for the necessary and often sufficient conditions of the “psycho”social and “psycho”therapy process. There is always validation, empathy, and problem formulation. This can be stepped up or down through monitoring and mild experiments. We will dialogue about this throughout the book.

The process itself involves three or four sessions of assessment. Assessment of course continues beyond that but the case-based plan is enacted. Diagnosis is involved but it is only one factor. We espouse a problem-based approach. Problems include the five domains (below). The stage is set with care and monitoring, assessing, and even borrowing from other models. We use the traditional predictors but also personalize treatment for a given patient. Accordingly, a patient with one or more predictors of poor outcome may receive interventions targeting each modifiable predictor, as well as a more vigilant follow-up.

We address assessment formally in chapter 3. We start with screenings. Screenings are a necessary evil and have some limits. They do not form a diagnosis. They do not tell us definitively about the quantity of the problem. They also do not reflect the strengths either. Assessment needs reasonable anchoring beyond usual scales. All testing for Mr. X applies to Mr. X and his specific issues: his age, his education, his marriage, his occupation or lack thereof, his place in the country, his comorbidities, his nuanced ethnicity... you get the picture. There are no valid norms then for anyone of us. We get a person-picture, a helpful view of complexity, and a series of targets that, with other data, apply to Mr. X in such a way that we know more about him than otherwise. This involves heuristics. It is part of treatment, part of the unfolding story somewhere between a marker of the specifics of a blood pressure and the general trend of an A1C. The goal then is to take a variety of test-derived pieces of information obtained from multiple sources and place them in the context of historical information, referral information, and behavioral observations to obtain a cohesive and comprehensive understanding of the person being evaluated. Tests are imperfect tools representing the construct variability of the variable of interest (cognition for example), the interaction of age (with its moderating

variables), and real world or practice effects. They are, however, a good enough approximation of the real world of the older adult.



There is an important message here. The concept of “assessment as treatment” lives! This is a core aspect of the Watch and Wait model. It holds that the initial period of assessment is necessary for good treatment. The component of testing is more than test items and reading the results. It is actionable information and data that provides feedback immediately as well as over time. Assessment is always a part of the process “over time.” In addition, with assessment the patient is engaged and has clear targets for change. Therapy feedback has been applied in psychotherapy clinics to good effect for many years. Feedback is helpful to both patient and provider. The Watch and Wait model depends on this mechanism as a change agent by itself.

Domains: We have identified the five areas, health, cognition, depression, anxiety, and life adjustment (unmet needs in the community). We start by showing the common problem at our clinic. We experienced what we believe is a usual occurrence in modern clinics. Patients presented for memory problems and were found to have other problems – encompassing the five core domains. The modal values for each domain was greater than 50%. The table below reflects the values of problems for over 500 patients. Recall that patients presented with memory complaints. The table below gives the percentages of problems *in addition to* cognition.

Mercer Archive Data

Factor	% Problem
• Depression	67% (GDS >4)
• Anxiety	51% (SAST >21)
• Health Problems	57% (SF-12)
• Pain	64% (Pain scale >4)
• Social Problems	48% (MBMD Social)
• Stress – Illness App	62% (MBMD)
• Stress – Functional Def	79% (MBMD)

GDS is Geriatric Depression Scale; SAST is Short Anxiety Screening Test; Pain Scale is 1-10 metric; MBMD is Millon Behavioral Medical Diagnostic. A scores reflect standard metrics for that scale.

This complexity applies within domains. Regarding the cognitive domain, we separated patients complaining about memory into types of mild cognitive impairment (MCI) diagnosis. Here we present data on standard and non-standard MCI diagnoses. Of 198 patients who had memory problems and only very slight or no functional problems, thereby meeting the diagnosis of MCI, the diagnosis itself was variable depending on the criteria applied. For standard criteria, such as use of delayed memory or the overall MoCA, the percentage of MCI designations was variable. When we applied non-standard definitions (using scales from the Repeatable Battery for the Assessment of Neuropsychological Status) like verbal recall, story recall, figure recall, or recognition, these rates also were variable. Of interest, we note too that recent years have placed the markers for problems upstream; subjective memory impairment is now a prodrome for dementia. More on this in other chapters.

MCI Variability in Archive: Different Definitions of MCI	
• Standard	
• Delayed Memory (RBANS <85)	51%
• MoCA(<23)	50%
•	
• Specific Cognitive Domains	
• List Recognition (RBANS <18)	33%
• List Recall (RBANS <4)	50%
• Story Recall (RBANS <6)	40%
• Figure Recall (RBANS <9)	48%
• All 3 RBANS Memory low	20.6%
•	
• Archive =198 with no function problems but	
• cognitive problems. Different people in each group.	

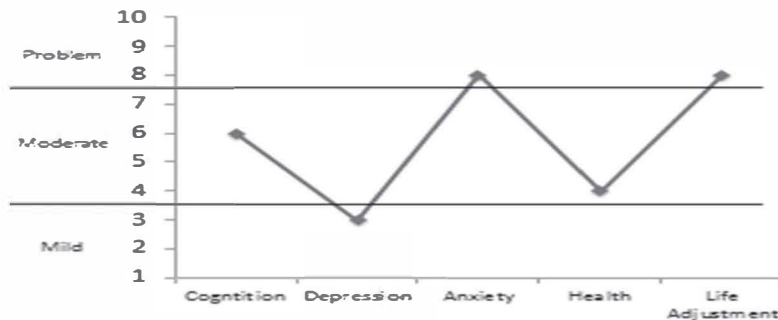
When we separated patients according to scores or ratings on their domains, the results suggested considerable variability in the domains, as well as problems in one domain leading to problems in another domain. We separated domains into two groups – High and Low: For Health, a health rating (cut off at 7/10); for Cognition, MoCA (20 as cut off); for Depression, BDI-II (12 as cut off); for Anxiety, GAD-7 (8 as cut off); and for Life Adjustment, FAQ (10 as cut off). As can be seen (below), there

were differences between High versus Low ratings in the other domains. This suggests that there is considerable variability in the groups and also that problems in one domain lead to problems in others: People high or low in depression, for example, have scores that reflect problems on the other domains. Clearly there is a need to individualize by patient. The point again is that there is considerable variability for any one diagnosis; there are multiple comorbidities and problems associated.

High versus Low Domains Assessed Against Other Domains					
Domain	High vs Low Health	High vs Low Cognition	High vs Low Depression	High vs Low Anxiety	High vs Low Life Adjustment
Health Rating		High > Low	High > Low	NS	High > Low
Cognition (MoCA)	High > Low		High > Low	High > Low	NS
Depression (BDI-II)	Low > High	Low > High		Low > High	Low > High
Anxiety (GAD-7)	Low > High	Low > High	Low > High		Low > High
Life Adjustment (FAQ)	Low > High	Low > High	Low > High	NS	

High and Low Health determined by Health Rating (cut off at 7); High and Low Cognition determined by MoCA (cut off at 20); High and Low Depression determined by BDI-II (Beck Depression Inventory-II, cut off at 12); High and Low Anxiety determined by GAD-7 (General Anxiety Disorder-7, cut off at 8); High and Low Life Adjustment determined by FAQ (Functional Adjustment Questionnaire, cut off at 10).

As noted, we developed an assessment battery for each domain using standard screenings and a short psychological battery, and secondly, a metric for designating whether the patient met criteria for each of the five domains: Mild/Normal, Moderate, or Problem. The figure below identifies one who has problems in anxiety and life adjustment, but is rated as Moderate in cognition and health and Mild in depression. After the identification of the profile, we apply an empirically supported plan of selected modules for the more problematic domain(s) and monitor these.



These results suggest that a sole traditional diagnosis and treatment of older adults leaves something to be desired. Treating symptoms in isolation, especially cognitive and physical limitations, risks slower or less-effective reduction in symptoms for overall care. Targeting an organized patchwork of psychological problems, notably depression, anxiety, cognitive impairment, adjustment, and health, provides for a concerted focus to help the patient adapt and cope with their problems. Again for the health care clinician for older adults, most often the presenting problem is memory. In the main we believe that mental health providers have over-compartmentalized expertise. The Watch and Wait model provides the infrastructure for care plans to be activated meaningfully and deliberately. It is served economically by the five problem-based components that require an understanding for their assessment and eventual treatment plans. This model effectively represents a best fit for the content of the older patient's problems and needed interventions to be applied. It melds further stage-treatment matching and patient-treatment matching. Careful work is done up front.

We also note two things. First, it is possible, even common, for a domain to be "problematic," as for example the cognitive domain being in the dementia range, but not be a "Problem" because this is handled well. This is actually common. Second, profiles change. It is very usual for the profile to change over time. Typically, an older person presents for memory problems and, after a few sessions, the target alters. Many older adults will show problems with a lower cognitive score but be able to function and have a more pressing problem of health or depression. The most common alteration involves the change from cognition to life adjustment or depression. Again, the person "becomes" the profile and the interventions flow from this model. Profiles are living and changing documents.

There are precedents for this. As one example, the Lust for Life program is a promising program designed to relieve the depressive symptoms of older adults in primary care in the short term. Providing one single clinical intervention in accordance with the participant's goals shows a stepped care process that could be life changing. Van Beljouw et al. (2015) examined the effects of Lust for Life and compared this with the usual care for depressive symptoms in older adults in a community. Eighteen general practices in a home-care organization in the Netherlands were assessed. Two hundred and sixty-three community-dwelling 65-year-olds with depressive symptoms according to patient PHQ-9 were looked at. After three months of watchful waiting, participants could sequentially choose between following evidence-based interventions of self-guided, self-help program problem solving or self-referral to a general practitioner. Applying the stepped care program Lust for Life, results showed a significant short-term positive effect on depressive symptoms in the first three months after implementation, in which the average PHQ scores dropped. Watching and waiting as well as step care, therefore, appears to be an effective program.

In sum, this model holds to the ideal that the patient is not broken, is not a medical anomaly, and does not need to be rescued. The patient is trapped in rigid patterns of living but is not a mechanistic creature who requires someone to figure out their feelings and eliminate causes of problems. Rather, each lives in a context with learning that can be used for the better, given the right atmosphere and support. The medical model has failed where the older adult is concerned. As implied, in recent years it has given way to the biopsychosocial model. This latter model is now over 40 years old. It nicely espouses the ideal that the person of the patient is complex but understandable. For care to be effective, a comprehensive approach is necessary. The variables for clarity involve the biological, the psychological, and the social. This means that care is encompassing and alterable. In a sense the model is an personal model, an epigenetic unfolding for each person. Each case is unique and there are many idiosyncratic turning points and complexities.

Watch and Wait: Lifestyles

We introduce lifestyles as a major marker for change. We will address this over and over. A number of modifiable risk and preventive factors (for AD) have been described in observational studies. Risk factors that have been found in several studies include diabetes mellitus, hypertension, renal dysfunction, alcohol and smoking patterns, high cholesterol, coronary

heart disease, depression, sedentary life style, low cognitive activity, and diet. These factors combined account for more than half of the attributable risk for AD (O'Donnell et al., 2015). The most difficult of these factors to address is diet because it is highly dependent on income and access to fresh foods. In a 16-year observational study of 949 individuals using the Lifestyle for Brain Health (LIBRA) measure of modifiable risk factors, a 1-point increase in LIBRA score was associated with a 19% greater risk of dementia (Schiepers et al., 2017). Bad diets results in poor cognition and behavior. In a meta-analysis of 19 studies, cognitive leisure activities, including crossword puzzles, card games, computer use, arts and crafts, life-long learning, group discussions, and music playing, had a protective effect (Yates et al., 2016). Physical activities may lead to a 20% to 65% risk reduction depending on the type and intensity of activity through mechanisms involving lower vascular disease risk, better respiratory function, stimulation of trophic factors, and lower oxidative stress and inflammation. Also, objective measurement of midlife vascular risk factors demonstrated a greater risk of dementia in late life (Sindi et al., 2015). Stress in addition plays a role. In a study of 2,000 individuals aged 71 to 78, home- and work-related stress increased the risk of MCI, dementia, and AD (Sindi et al., 2017). Finally, we will also emphasize cognitive training (CT). Despite many confounds in methods, samples, outcomes and transfer effects, CT motivates, focuses, and activates, all good events.

Although age is the single greatest risk factor for AD, AD itself is not inevitable. The best estimates suggest that at age 85 there is 42% risk of developing AD (Alzheimer's Association, 2017). The reasons are unknown, but may be explained in part by a host of modifiable and non-modifiable risk factors. Up to 30% of AD cases may be preventable through the modification of risk factors. Simple behavioral changes can mitigate the effect of those risk factors that are modifiable (Ashby-Mitchell et al., 2016). There is an ongoing debate as to whether the current evidence base is sufficient to initiate prevention programs because it is difficult to prove causation from observational studies, and it is difficult to pool multiple RCTs because of differences in study design, measurements used, and anticipated outcomes. That said, the value of lifestyle enhancement is now beyond reproach and its judicious application is needed for best health.

Interventions: “Psycho”therapy

●ne can rest assured that this statement is never uttered by a patient.

“I want to be less depressed than the average no-treatment control patient at the .05 level.”

A brief review of the efficacy and value of psychotherapy will make the point that older adults require more than the usual therapies. A holistic view of the therapies is required for change at later life. The five domains require more than psychotherapy. The issues are varied and overlapping. In the ensuing chapters we speculate on the many varieties of interventions necessary for change. For now we can see the many forms of problems, as well as change differences, coming from problems of older adults. Clearly a broader perspective is a necessity.

General Treatment Ideas for ●lder Adults

- ●lder patients do not get adequate treatment
- Treatment works BUT...mediators reign
- In general, treatment is less effective than in younger groups
- ●lder patient may be different requiring the doctor to be the authority.
- Cognition: MMSE (Mini Mental State Exam) of 18 is weak but best for a cognitive focus
- ●lder age involves chronic conditions.
- Mental and Physical Illness are conjoined.
- Current psychological treatment is inadequate.
- Current assessments address dichotomous problems but a focus on continuous and individual symptoms is required.
- Primary care is the new psychiatric care.
- Professional silos are a problem: teams are complex and necessary but messy and imprecise.
- Current models of care (e.g., IMPACT, PEARLS) espouse case based models.
- Step care works: Specialty clinics are applied last.
- Patient-centered care is under-performed and critical.
- Public health models work well, especially for older adults.
- The clinician’s attitude and skills are critical.
- Psychoeducation and perspective are always among the curative agents.

- Alternate forms of psychotherapy provide as much or more help as the one form of psychotherapy alone.
- Pre-treatment preparations make a difference; pre-training, experiential pre-training; e.g., TIPS (Treatment Initiation Program) for meds; Diabetes--> needs of the patient and patient role.
- Motivational Interviewing works.

That said, psychotherapy and its core elements are necessary parts of this curative endeavor. The older adult is after all (many times) in a forced compliant setting where diagnosis and help are often met with skepticism or begrudging acceptance. The health care provider must connect. It is becoming increasingly clear that randomized control trials (RCTs) and meta-analyses, the bread and butter of data collection, are not sufficient for good therapy practice, especially as these apply to older adults. Treatment outcome studies are of course wanting. Differences are often modest, the sample size is usually inadequate, and questions have been raised about whether or not two treatments have been delivered in comparable ways. Overall outcome studies have suggested there are few empirical grounds for selecting one evidence-based supported approach over another.

However, the specialized therapies are generally more efficacious than treatment as usual. Although this finding appears to suggest that advantages accrue from using a specialized therapy, there are reasons to question this conclusion. Treatment as usual is a rather modest standard because it is limited to whatever routine outcome is available in the setting in which the study occurs, and in some settings this care can be quite limited. Differences between specialized treatments and treatment as usual are also decreasing with time, presumably because treatment as usual is itself improving. Moreover, the specialized treatments are compared with well-specified, manualized, general psychiatric care related to specific identities or problems. This is intense but hardly the real world in practice.

Perhaps there is a need for practice-based evidence. This is a chiasmic counterpart to evidence-based practice, which over the past 20 years has come up short. Evidence-based practice leaves open issues regarding the transportability of everyday practice. Rather than simply carrying out effectiveness studies as a logical extension of RCTs, there is a need for a research paradigm that not only fulfills this function, but that is also sufficiently robust to initiate research from practice settings that might then lead to more specific investigation, even by RCTs. In this way, evidence-based practice and practice-based evidence are complimentary to each other. Therefore, there is a need to readjust our view of science and shift to what might be called a good enough level of practice-based science

that can be owned by practitioners, driven by clinical and service utility, and then accepted by academics and policy makers alike as a legitimate compliment to the trial's methodology. In fact, to find a therapy better than an existing one with an adequate effect size, sample sizes of over 400 are often needed.

Related, in a meta-analysis of 70 N-of-1 trials, 50 of 57 completed trials provided definitive clinical or statistical answers, with 39% prompting physicians to change the plan of care (see Galvin, 2017). In another meta-analysis examining 108 trials involving 2,154 participants it was found that 54% of participants had subsequent treatment decisions changed based on the results (Galvin, 2017). These results suggest that the use of traditional predictors starts the process. The clinician then personalizes treatment for a given patient. A patient with one or more predictors of poor outcome may receive interventions targeting each modifiable predictor, as well as more a vigilant follow-up. This is of course the ABC model of behavioral therapies – a function-based approach to change. Things are also likely to change.

At a societal level, we still want the quick fix. The medical model rules. According to the American Psychiatric Association, the rate of antidepressant use in the United States has vastly increased since the introduction of selective serotonin reuptake inhibitors (SSRIs), such as Prozac in the late 1980s (Yan, 2017). Based on data from the Centers for Disease Control, antidepressant use has increased in the United States from 6.4% between 1999 and 2002 to 10.7% between 2011 and 2014 (CDC, 2016). Fewer patients receive psychotherapy during this period. In general, we are changing in a medical direction as we seem to seek ready-made solutions that are not labor intensive.

We note too that the border wars of the psychotherapies are waning. Cuijpers (2015) assessed 400 randomized trials on psychotherapies for adult depression that have been conducted. Results are less than what has been promised and effect sizes lower than expected. Many new psychotherapies have been promised to be more effective than existing ones, usually without success (Meichenbaum & Lilienfeld, 2018). Recent research on, for example, Acceptance and Commitment Therapy (ACT) and cognitive bias modification concluded that both have not been shown to be more effective than existing therapies (Cuijpers, 2015). A growing number of studies have also focused on therapies that may be successful in further reducing disease burdens, such as treatments for chronic depression and relapse prevention. Studies that involve postpartum depression, comorbid somatic disorder, heart disease, cancer, comorbid alcohol problems, and subclinical depression tend to show that there is no

evidence that one psychotherapy is more or less effective than another, even in different ethnic groups. This might apply also to group psychotherapy, self-help groups, supportive Internet groups, and the like. The evidence suggests that extra-therapy factors and activation of the patient hold the brunt of the variance for better outcomes.

But here is the point: Even if the entrails of psychotherapy are not proven, psychotherapy itself has several new gifts. Its value exceeds its science. One gift is an understanding of the brain. Increasing data show that the brain changes with empirical psychotherapy; a reregulation of neuronal networks parallels symptomatic changes in psychotherapy. Changes as a result of CBT cause commensurate changes in the brain, as is the case with medications. This seems to apply to mindfulness also (Davidson et al., 2003; Davidson & Kaszniak, 2015).

Perhaps psychological problems at late life then are best dealt with by modular interventions. This is so because the modal problems at late life, anxiety, depression, somatization (pain), and cognitive decline, interact such that emotional disorders have a similar underlying structure. In this sense one size can fit all. The modal empirical therapies for older adults, CBT, problem solving therapy (PST), and interpersonal psychotherapy (IPT), have several overlapping modules that result in behavioral change at least. The search is for the right mix of modules for change. This mixture will change with each person. The process of psychotherapy may indeed be “made for” older adults: Validation and a wide scope for care make a difference.

General Treatment Ideas for Older Adults

Success is related to the extent treatment matches shared social constructions about what it means to be remoralized or cured.

Success depends on the conviction by the patient that the therapist cares and is competent. Mackover (1992) long ago noted that continued alteration of therapy leads to a better “approximation of truth.”

Quality of the patient’s participation is most determinate of outcome. What the patient does in this setting and outside of the therapy determines how well the therapy will go.

If the patient buys into the model – competent therapist, some change, empathic reactions, and monitoring – change stands a good chance.

Therapy is people helping people with requisite belief systems, encouraged behaviors, and some rituals.

Techniques may mostly be placebo delivery devices.

Activating the patient is critical to success.

Interpersonal empathy is at the heart of the change in most conditions.

At the risk of repeating, a biopsychosocial model applies best at late life. Older adults require more: Medication and psychotherapy alone have limits. The nature of change for psychology may be grounded in the empirically supported treatments (ESTs) but also equally in the practical concerns of the person, medical and psychosocial. For medication, we know that there is little relationship between dosage and plasma levels of an antidepressant and outcome; for psychotherapy, there is even less. Successful doctors who implement a Watch and Wait attitude get better results. This occurs with SSRIs/SNRIs/TCAs/anxiolytics/placebo/psychotherapy or whatever. The key is a careful therapist connection and accepting therapist belief system. The whole here (Watch and Wait, open therapist, selected modules) is greater than the sum of its parts.

Both context and outcomes do of course matter with older adults. "Truth" is elusive where best practice psychotherapy is at issue. For outcomes, the issue is never just symptom abatement. Rather, therapy should aim at symptom relief *and* improving overall quality of life (QoL). Moreover, although evaluation questions necessarily focus on the reasons why an elder is seeking treatment, such a narrow focus is only helpful for limited targets; for understanding process changes over time or other longer term and broader concerns, more is demanded. In translating ESTs to older adults, and in targeting this to the most researched psychotherapy, CBT, we must concentrate on more general outcomes (not just diagnosis) as well as specific markers associated with the identified problem. With older adults, the therapy also demands a scientific attitude, a skillful and flexible delivery of services, quantitative monitoring of the client's progress, and an awareness of the personal, interpersonal, and cultural characteristics of the client as well as quality of life themes.

Applying the Contextual Adult Lifespan Theory for Adapting Psychotherapy (CALTAP) model, Knight and Pachana (2015) noted that the techniques of therapy with older adults differ little from the work with younger clients when the problems and their context are similar. The accurate understanding of older adults presenting problems, however, is complex for many reasons, including the need to have an accurate view of normal and developmental aging and other sources of age-related differences as well as the ability both to identify the effects of specific challenges, including late-life illnesses and the side effects of treatments, and to recognize the neurocognitive impairments common to older ages. Age-related differences are typically more related to social context, cohort differences, and interacting influences of the culture and the cohort rather than developmental aging per se.

In this context Judd (1999) offered the closest to a melding of lifespan, clinical psychology, and neuropsychology approaches to assessment and intervention with patients. His theoretical approach, labeled neuropsychotherapy (Judd, 1999) pointed out the need for clinical psychology knowledge about a broader formulation of cases and empirical intervention strategies to be paired with neuropsychological assessment and rehabilitation techniques for best outcomes. With respect to older adults, he pointed to the high rates of comorbidity between psychiatric conditions, such as depression and cognitive decline, and progressive neurological conditions and suggested that both needed to be assessed and treated in tandem, using best practice.

Geropsychology principles (for example multidisciplinary approaches) endorse this position. Therapeutic geropsychology highlights the idea that assessment is a collaborative exercise between the patient and the psychologist and that part of the goal of the assessment is always to answer the specific queries of the patient. This approach requires that the health care provider consult more extensively about the patient's own goals, fears, and hopes with respect to what could be achieved by such an assessment. This approach uncovers potential misgivings about both testing and its outcomes as well as assisting the professional in structuring both the assessment and the feedback from the assessment to best match patient goals which are sometimes lost in the service of answering the referral. This is then part of the whole process.

“Psycho”social therapies are at base generally more flexible for older adults. Hans Strupp, a premier psychotherapy researcher, noted that “patients progress at their own pace.” In fact, all forms of therapy address two features at least: a new understanding and a new experience. Change percolates from within and from without. The therapist validates the reality of the symptom and develops a functional explanation, suggests a script and places emphases on a lack of harm and a vision for change.

From this larger perspective there is then a soft consensus on a unified approach to treating problems at late life. Psychotherapeutic issues like experiencing emotion, changing cognition, and behaviorally acting are important, but may be limited at later life. We are asking for something more. As we have noted, for older adults medication and psychotherapy have their limits. The nature of change is less grounded in the ESTs as it is in the practical concerns of the person, both medical and psychosocial. As we have just noted, for medications we know that there is little relationship between dosage and plasma levels of an antidepressant and outcome; the same is true for psychotherapy. The key is a careful therapist connection and accepting therapist belief system. We will discuss this more in later chapters.

Recall the construct “aptitude/treatment interaction” that was born on the heels of poor education techniques and that then became relevant considering personality. The clinical idea is for the professional to strive to give the right treatment to the right patient at the right time. The challenge is to determine which characteristics of the older individual (for example, biological, cognitive, symptomatic, and ethnic) or treatment features (for example, intervention intensity or dosage, mix of client and therapist expectation, therapeutic engagement and alliance) strongly influence the outcomes of different interventions. This construct is the ideal point for the assessment and care of older adults.

As an aside, we present the particulars of a personality scale, MBMD in the Appendix A. This can be used as a reference for the remaining chapters. We liberally apply it. In the Appendix A we identify the MBMD markers and variables. It is further discussed in other chapters.

Conclusion

The older adult is now different. Probably 85% of people over the age of 60 have some form of coronary atherosclerosis. Cohorts now even 30 years later than a decade or so ago have better biomarkers. These reflect metabolism, inflammation, and organ function, as well as blood pressure and breath capacity. But, living longer, we are lonelier and more stressed, and our knowledge has not made us happier. We need to somehow get a grip on the good, the bad, and the ugly of the aging experience. We need better ways to assist our brains in the processing of information as unconscious but intelligent.

We have the technology and have more recently acquired the data/information. Given what we know about migraines and heart problems, each of us knows basically what we need to do for change. This might not be sufficient for change but it leads in the right direction and sets up success in other areas. It is probable too that older adults have a premonitory phase in an illness when we can sense problems will unfold. We now have clear evidence that we can make a difference in life with better lifestyles and information. Robert Sapolsky noted that we will all have the luxury of dying of a stress related disease. If only we can change ourselves, have a positive attitude toward aging or even be more upbeat.

(As we begin we note that we have provided many citations and allowed for some repetition in the chapters. We left other citations out. We do this intentionally. We want to create a book environment that allows the

reader sufficient information for references but not be overwhelmed by this. At times this may be insufficient for the reader.)

References

- Andrasik, F., Goodie, J.L., & Peterson, A.L. (Eds.). (2015). *Biopsychosocial assessment in clinical health psychology*. New York, NY: Guilford Press.
- Ashby-Mitchell, K., Burns, R., Anstey, K. J., & Shaw, J. (2017). Proportion of dementia in Australia explained by common modifiable risk factors. *Alzheimer's Research & Therapy*, 91-8. doi:10.1186/s13195-017-0238-x
- Barlow, D (2008) *Clinical handbook of psychological disorders* (4th ed). New York, NY: Gilford Press.
- Centers for Disease Control (2016). *Selected prescription drug classes used in the past 30 days, by sex and age: United States, selected years 1988-1994 through 2011-2014*. [Data file]. Retrieved from <https://www.cdc.gov/nchs/hus/contents2016.htm#080>.
- Cogbill, E., Dinson, K., & Duthie, E. (2010). Considerations in prescribing medication to the elderly. In V. Hirth, D. Weiland, & M. Dever-Bumba (Eds.), *Case-based geriatrics: A global approach*. New York, NY: McGraw-Hill.
- Cuijpers, P. (2015). The future of psychotherapy research: Stop the waste and focus on issues that matter. *Epidemiology and psychiatric sciences*, 25, 291-294.
- Cuijpers, P. (2015). Psychotherapies for adult depression: Recent developments. *Current Opinion In Psychiatry*, 28(1), 24-29. doi:10.1097/YC0.0000000000000121
- Davidson, R. J., & Kaszniak, A. W. (2015). Conceptual and methodological issues in research on mindfulness and meditation. *Am Psychol*, 70(7), 581-592. <http://doi.org/10.1037/a0039512>
- Dhalwani, N. N., Fahami, R., Sathanapally, H., Seidu, S., Davies, M. J., & Khunti, K. (2017). Association between polypharmacy and falls in older adults: a longitudinal study from England. *BMJ open*, 7(10), e016358.
- Galvin, J. E. (2017). Prevention of Alzheimer's Disease: Lessons Learned and Applied. *Journal of the American Geriatrics Society*, 65(10), 2128-2133. <http://doi.org/10.1111/jgs.14997>
- Gullestad, F. S., Johansen, M. S., Høglend, P., Karterud, S., & Wilberg, T. (2013). Mentalization as a moderator of treatment effects: Findings

- from a randomized clinical trial for personality disorders. *Psychotherapy Research*, 23(6), 674-689.
- Judd, T. (1999). Neurology and Neuropsychology. In *Neuropsychotherapy and Community Integration* (pp. 17-29). Springer, Boston, MA.
- Hyer, L. (2014). *Psychological treatment of older adults: A holistic model*. Springer Publishing Company.
- Keyes, C. (2007). Promoting and protecting mental health as flourishing: A complementary strategy for improving national mental health. *American Psychologist*, 62 (2), 95-108.
- Knight, B.G. & Pachana, N.A. (2015). *Psychological assessment and therapy with older adults*. USA: Oxford University Press.
- Livesley, W. J., Dimaggio, G., & Clarkin, J. F. (Eds.). (2015). *Integrated treatment for personality disorder: A modular approach*. Guilford Publications.
- Mast, B.T. (2012) Methods for assessing the person with Alzheimer's disease: Integrating person-centered and diagnostic approaches to clinical assessment. *Clinical Gerontologist*, 35(5), 360-375.
- Meichenbaum, D., & Lilienfeld, S. ●. (2018). How to spot hype in the field of psychotherapy: A 19-item checklist. *Professional Psychology: Research and Practice*, 49(1), 22-30.
<http://doi.org/10.1037/pro000172>
- Donohue, W. T., & Draper, C. (2011). *Stepped care and e-health: Practical applications to behavioral disorders*. New York, NY: Springer Science + Business Media. doi:10.1007/978-1-4419-6510-3
- Prochaska, J. ●., Norcross, J. C., & DiClemente, C. C. (1995). *Changing for good*. New York: Avon Books.
- Sindi, S., Calov, E., Fokkens, J., Ngandu, T., Soininen, H., Tuomilehto, J., & Kivipelto, M. (2015). Diagnostic Assessment & Prognosis: The CAIDE Dementia Risk Score App: The development of an evidence-based mobile application to predict the risk of dementia. *Alzheimer's & Dementia: Diagnosis, Assessment & Disease Monitoring*, 1328-333. doi:10.1016/j.dadm.2015.06.005
- Sindi, S., Hagman, G., Håkansson, K., Kulmala, J., Nilsen, C., Kåreholt, I., & ... Kivipelto, M. (2017). Midlife work-related stress increases dementia risk in later life: The CAIDE 30-year study. *The Journals Of Gerontology: Series B: Psychological Sciences And Social Sciences*, 72(6), 1044-1053.
- Sirey, J. A., Banerjee, S., Marino, P., Bruce, M. L., Halkett, A., Turnwald, M., ... & Kales, H. C. (2017). Adherence to depression treatment in primary care: a randomized clinical trial. *JAMA psychiatry*, 74(11), 1129-1135.

- Schiepers, G. G., Köhler, S., Deckers, K., Irving, K., O'Donnell, C. A., van den Akker, M., & ... O'Donnell, C. A. (2018). Lifestyle for Brain Health (LBR): a new model for dementia prevention. *International Journal of Geriatric Psychiatry*, 33(1), 167-175. doi:10.1002/gps.4700
- Thielke, S., Vannoy, S., & Unützer, J. (2007). Integrating mental health and primary care. *Primary Care: Clinics in Office Practice*, 34(3), 571-592.
- Van Beljouw, I.M.J. et al. (2015). Does an outreach stepped care program reduce depressive symptoms in community-dwelling older adults? A randomized implementation trial. *The American Journal of Geriatric Psychiatry*, 23(8), 807-817.
- Yan, J. (2017). Percentage of Americans taking antidepressants climbs. Retrieved from <https://psychnews.psychiatryonline.org/doi/full/10.1176/appi.pn.2017.pp9b2>.
- Yates, L. A., Ziser, S., Spector, A., & Orrell, M. (2016). Cognitive leisure activities and future risk of cognitive impairment and dementia: Systematic review and meta-analysis. *International Psychogeriatrics*, 28(11), 1791-1806. doi:10.1017/S1041610216001137

CHAPTER 2

META-TRENDS OF AGING

LEE HYER AND LAUREN LEE

Perspective on Clinical Aging

In 2002, McGinnis, Williams-Russo, and Knickman published a paper in *Health Affairs* that broke down the determinants of health by percentages. On average, our genes account for 30% of our health. Behavioral patterns account for about 40% of our health. Environmental exposure to toxins accounts for about 5% of health and social circumstances (education, income, etc.) account for 15%. Health care accounts for only 10% of our health. This model has actually been updated to reflect less for genes and more for behavioral influences (Lyubomirski, 2008). In other words, the behavioral and social determinants of health account for greater than 50% of what is causing a person to get sick or stay well. In 2005, about 133 million Americans suffered from at least one chronic disease and it is estimated that this will increase to 157 million in 2020. About 50% of these people will have multiple chronic conditions and each year seven out of 10 deaths are the result of a chronic condition. In this context we need to know what aging is and how it unfolds.

“What is aging?” is the question asked over the last 70 plus years with some scientific panache at bio-gerontology meetings. To most of us the question is easy; as Justice Potter said of pornography, “We know it when we see it.” To the scientist the question is too complex for simple musings and needs hours of perspective taking. The idea that small sets of gene mutations could precipitate or delay multiple distinct aspects of aging now seems unlikely and even off-putting. In fact, where do we even start to provide answers to this issue. Aging is most probably an emergent construct. Like the weather it defies facile measurement and definition. “Emergence” is defined as a state that derives its essence from multiple synergies of its components. Aging is now “new and more” and, as Philip Anderson says, “More is different.” We reiterate that biological aging is

largely determined by the interaction of the internal biological clock and an accumulation of the insults of life. These operate at every level of the bio-hierarchy – genes, proteins, cells, organs, and organisms. The gene expression of age is clearly only one marker of life change. As we shall see, epigenetics rules! We live in both a genetic and genetic-modifying world. They shape the environmental influences on brain and behavior. In time, we need to know how the aging person mixes genes with their environment, no easy task.

One even hears that there is no such thing as aging. Aging is not a causal variable. Rather it is a marker on a temporal axis along which various exposures and disease processes operate. Aging then is not a meaningful explanation for why one might experience decline, especially cognitive decline or impairment. Dementia itself may be considered a non-event. It is an end stage of a process of an accumulation of poor health, behaviors, and genetics. We again argue that both genetic factors and health-related behaviors influence survival to old age and good health. Longitudinal cohort studies demonstrate that lower levels of cardiovascular risk factors measured by midlife or early older years predict survival and healthy survival to 85 years of age and beyond. But it is also true that longevity has also been observed to cluster within families such that parents and siblings of centenarians have a greater likelihood of attaining advanced age. An offspring of centenarians may even have a delay in age-related diseases.

This issue sets the background for this chapter. There is a paradigm shift under way in the Kuhnian sense regarding the care and understanding of older adults (given the direction of newer work regarding the physiological/psychological unfolding of aging issues). This applies to psychopathology as well as the aging process itself. In effect, we are beset with several newer meta-trends that interact and complicate simple answers. There is little in uniqueness when it comes to geriatric syndromes: No new geriatric syndromes exist psychiatrically. Aging people lose gray and white cells but we are good compensators. We have the ability now to alter to some extent the onset of and course of dementia. Mild cognitive impairment (MCI or mild Neurocognitive Disorder) is now a passage in time, a “teachable moment” for a commitment to renewed living, not a death sentence.

Medicine likes one answer solutions: We have a clogged artery, for example. Complex diseases are different and medicine offers answers that are less crisp. In fact, most patients fail treatment because their problems exceed the effectiveness of our science, or they or we expect too much from our therapies. The majority of patients with behavioral

problems seen in primary care settings do not receive evidence-based care for their conditions. A number of factors that lead to quality of care gaps between the usual care and evidence-based management include the patient's reluctance to seek treatment, failure to detect and diagnose core issues like depression, insufficient dosage or duration of the intervention, failure to recommend evidence-based psychotherapy, limited access to behavioral health specialty clinicians, lack of regular monitoring, failure to integrate medical and behavioral care, failure to provide education to patients and families, and failure to use community influence and support (●Donnell, Cummings, & Cummings, 2014). Yes, aging is an ineluctable decline process, but we are living longer and longer. We are arguing now that living well can make a difference in this process.

Commonly Studied Aging Factors

- Delay initiation of cell senescence
- Change Lifestyle Habits (smoking, ET●H use, exercise, cognitive training, diet)
- Stabilize telomere length
- Control of apoptosis
- Explore calorific restriction
- Intervene in insulin-like growth factor 1 pathway (IIS)
- Enhance adenosine monophosphate-activated protein kinase and forkhead (F●X●)
- Promote systemic antioxidant activities
- Inhibit target of rapamycin signaling
- Diminish activation of inflammatory cytokines
- Express longevity enhancing genes
- Identify new candidate genes and pathways

Stessman et al., 2009

Professional societies are taking notice at the policy level. There has been a spate of guideline papers from the American Psychological Association regarding older adults (APA, 2014). Several of these speak to aging and assessment. As suggested in chapter 1, clinical work with older adults involves developmental issues specific to late life, a cohort perspective, comorbid physical illnesses, problems with polypharmacy, cognitive and sensory impairments, and histories of mental or medical disorders. ●lder adults' developmental pathways are shaped by the adaptation of normative-earlier and -later life transitions such as retirement and bereavement, as well as loneliness. Increasingly too it is becoming apparent that stability of personality gives way to plasticity across the

second half of life. Aging is of course a reflection of the interaction of the person with the environment. Often there are no mental health services for older adults. Clearly this is an issue as the menu of medical problems always generates a pressure for adjustment for older adults. This battle, unfortunately, is often played out with medication according to the medical model.

Competence in and Attitudes toward Working with Older Adults
(American Psychological Association)

General Knowledge About Adult Development, Aging, and Older Adults. This includes diversity and knowledge about biological and health related aspects of aging

Clinical Issues

Cognitive Changes – PET is now approved, as is the yearly physical and cognitive evaluation

Functional Capacity – everyday competence with IADLs especially

Psychopathology at Late Life – 20% plus of older adults will meet criteria for an Axis I disorder.

Assessment

Relevant methods involve clinical interviewing, self-report scales, cognitive performance measures, direct behavioral observation, role playing, psychophysiological techniques, neuro-imaging, and use of informant data.

Interdisciplinary assessment is optimal

Knowledge of normal aging is important.

Integration of objective measures of cognitive performance includes historical, neurological, psychiatric, medical, and other diagnostic information.

Older adult's premorbid functioning is a needed and important area of evaluation for determination of current problems, especially cognitive ones. Knowledge of education, occupation, current interests, and cognitive practices are all relevant.

Psychometric theory, test standardization, and the importance of using reliable and valid measures.

Norming is messy and critical. No assessments are validated for older adults on personality or psychophysiological assessments are noted.

Knowledge of the brain is an important area of focus. Special targets for research are biomarkers that will be more important each year as algorithms improve.

Knowledge of function is critical. This includes ADLs and IADLs and their interaction with cognitive skills and the environment.

Cross cultural issues are very relevant and often prepotent in determining outcomes. Considerable within and between group differences can be found. Multicultural competence includes explicit consideration of the older adults ethnic, racial, and cultural issues. Multicultural issues and aging are linked.

Use of other informants is also important and requires a context for best understanding.

Repeated assessments may be required.

Use of performance-based evaluations are important.

Assessments are also needed for specific issues like competence in various areas. Targeted evaluations are necessary.

Accommodations are also important and need both study and consideration

Knowledge of the core psychotherapies are most relevant for assessment. This involves an evaluation of outcomes but more specifically these involve psychotherapy-specific outcomes (e.g., CBT).

Telehealth may be increasing but has been applied sparingly to older adults.

Meta-trends

Our attention is now directed to 10 trends applicable to the clinical status of older adults. We start this section with a reminder that behind the scenes are more basic and social sciences addressing aging. Many researchers are seeking new ways to combat aging-related issues one at a time. Some experts in the field of geroscience are uniting to develop preventive and therapeutic approaches to fight multiple diseases. Seven have been noteworthy.

1. Individual and environmental stressors affect our physical and psychological wellbeing over time.
2. Genetics: How our environment can affect gene activity.
3. Inflammation: The body's natural response to a range of conditions, although chronic inflammation plays a contributing role in accelerated aging and chronic disease.
4. Macromolecular damage: The causes and effects of damage over time to the large molecules that comprise our bodies; DNA for example, which drives the development of chronic disease and age-related conditions.

5. **Metabolism:** Metabolic changes due to aging may play a role in cardiovascular problems, neurodegenerative diseases, diabetes, cancer, and other chronic conditions.
6. **Proteostasis:** This is the housekeeping process that regulates and maintains the proper protein function within ourselves.
7. **Stem cells and regeneration** have been noted. The adult stem cells in our bodies are well-known for their ability to divide, renew, and replace damaged tissue in the body itself.

We set the stage by noting that aging is the largest risk factor for most chronic diseases, including stroke, heart disease, diabetes, arthritis, metabolic syndrome, blindness, and frailty, to name the most common. Aging may also be a modifiable risk factor. Now care for the elderly accounts for 43% of the total health care spending in the US and the percentage will rise. A modest increase in lifespan and healthspan can measurably reduce these expenses. We address 10 of the more salient of these factors here.

1. Brain Assistance and Complexity

We start with brain complexity. The BRAIN Initiative, the national White House Grand Challenge involving more than 100 laboratories in the United States, has already made progress in establishing large scale neuroscience goals and developing shared tools. The technological challenges that must be surmounted are sufficiently complex that they are beyond the reach of a single investigator effort. This group believes that they can only be surmounted through highly coordinated, multi-investigator, cross-disciplinary efforts. The BRAIN Initiative outlined four areas that are critically dependent on new technology that would be unlikely to be quickly realized outside of the center-based approach. These include connectomics, the systematic reconstruction of neural circuits, neural mental probe systems, new resonance imaging techniques, and computational data mining. ICNs (intrinsic coherence networks) are now of interest: These are groups of brain regions whose activity levels regularly fluctuate in synchrony at common frequency to core problems at later life, like depression. Things are changing.

The two most notable differences of the new criteria relating to brain degeneration are factors related to Alzheimer's disease (AD). These are criteria published in 1984 and represent the incorporation of underlying disease biomarkers and the formulization of different stages

of disease; preclinical AD, MCI, and dementia. Regardless, we know that pathology is not destiny. During the Rush Memory and Aging Project and the Religious Order Study, 3000 older adults were tracked over 2 decades. Autopsies showed that some mentally spry individuals had extensive signs of cellular damage and others with substantial problems of cognitive decline in their later years had few signs of these cellular abnormalities. There was, then, a disconnect between cellular pathologies and cognitive decline. Other work has implicated the value of exercise, social interaction, conscientiousness, and sense of purpose in cognitive resilience (Wilson et al., 2013). The authors also note that, even if successful, standard medical care for AD is helpful for only about one third of age-related cognitive decline problems. Large scale genomic, proteomic, and epigenomic studies are required for more direction and help.

In recent years there have been several drug trials in pre-symptomatic patients: the Alzheimer's Prevention Initiative, Autosomal-Dominant Alzheimer's Disease Treatment Trial (ADNI), the Dominantly Inherited Alzheimer Network Trial (DIAN), the ApoE4 Treatment Trial, and the Anti-Amyloid Treatment in Asymptomatic Alzheimer's Disease (A4) Trial. The first three test anti-amyloid drugs in subjects with strong genetic risk factors for AD and the A4 study examines anti-amyloid treatment in subjects who are cognitively normal, but have imaging evidence of beta amyloid brain plaques. In addition, new radioligands are being developed that allow the PET imaging of tau tangles. Like amyloid imaging, tau imaging has even more potential to redefine the way clinical trials are created and conducted and could in the future become a useful tool for diagnosis and for tracking both disease progression and modification. Tau imaging may also untangle the complicated relationship of tau, amyloid, and cognition. Tau normally builds up very slowly in the medial temporal lobe with age and in general it does not have a major deleterious effect, but when beta-amyloid comes along, it somehow exacerbates this increase and tau moves out into the temporal cortex (see Smith & Farias, 2017). Additionally, Hammes, van Eimeren, and Drzezga (2016) studied 10 subjects with AD who had PET (F-18 FDG and Pittsburgh B-11) for both tau and amyloid. Results revealed that tau is directly associated with hypometabolism (reflecting neuronal dysfunction): Nothing for amyloid. Tau is more closely related to neuronal injury; amyloid provides data many years ahead of the onset of symptoms.

To place this in perspective, in addition to tau and a-beta, other "usual suspects" of biomarkers for AD involve white matter hyperintensities (WMH), proinflammatory cytokines, and number of neuritic plaques. In 2014, there were 244 drug compounds investigated in 413 clinical trials:

99.6% failures. Aducanumab, levetiracetam, a nasal spray of insulin with anamides (antioxidant), and ultrasound (clear toxic clumps) have been reviewed more positively. PAN2401 is another such agent now under consideration. All are wanting. Small and Greenfield (2015) iterated the markers of dementia; presenile dementia, rare mutations, cholinergic deficits, ApoE, beta and tau. No disease modifying drug or symptomatic treatment has been discovered. The cholinergic hypothesis has been tested because other populations of noncholinergic neurons have been prone to neuro-degeneration. Amyloid and tau theories are suspect also, as in both cases APP (from which amyloid is abnormally cleaved) and tau protein are virtually ubiquitous features of all neurons; hence we need to identify a constraining additional feature that would explain why certain neurons are vulnerable. The drugs that target tau or beta amyloid may help but the underlying mechanism is not in evidence. Regarding Parkinson's disease, neither amyloid or tau could account for: (1) a frequent co-morbidity with Parkinson's disease; (2) a selectivity of cells that are prone to degeneration located in the basal forebrain, midbrain, and brainstem nuclei. These cells have different embryological origins. Neurodegeneration could then be an aberrant form of development. There is also still no exemplary animal model.

The clinical features of AD are non-specific and cannot be easily assayed until death. Biomarkers in the CSF or saliva do not directly reflect the brain disease process. Neuroimaging helps and the atrophy of the entorinal and hippocampal volumes (especially with an Apoe-4 allele) suggest a heightened risk but regional alterations of the brain can be many things. Scans (fMRI and PET) are critical for the detection of neurodegenerative problems. Combining other factors like genetic risk factors (APOe allele) with scans (FDG-PET) can increase detection. Pittsburgh Compound B and florbetapir are also both helpful. Unfortunately, despite the large variety of compounds available as candidates for therapy, their mechanisms of action are few: general neuronal health and conventional receptor agents, tau hyperphosphorylation and amyloid. The time frame for someone experiencing cognitive problems to MCI and then dementia can be as long as 15-20 years. Understandably few studies have this type of follow-up data due to time and economic constraints. What appear, therefore, as negative results in the short term could translate into positive benefits much later in time, particularly if participants continue to actively engage in material provided in interventions.

In one of the more important studies on the transition from MCI to dementia, BIOGARD was conducted (Alpert et al., 2014). This involves a combo of evaluations: cognition, brain structure, and brain proteins. Of 1995, 349 Ss with a mean age at admission of 57 were followed for 17 years. Biannual CSF punctures, MRI, clinical evaluations, and cognitive testing were conducted. Results showed that at baseline lower A-beta and higher tau, lower right entorhinal cortex, and lower hippocampal volume were associated with the onset of AD. Cognitive measures of paired associates and symbol digits were also in the mix. The sensitivity was .85; specificity = .75. Psychological measures made a difference.

In the previous book (Hyer, 2014) we emphasized the use of the brain as a metaphor with an anchoring in psychopathology. The brain is complexly present and increasingly understood in the context of the aging process as well as neurodegeneration. We can use it as an explanation for decline and problems. It is unfortunately for assessment purposes very individual and experienced as unique, hence less amenable to assessment and norming. We do not need a new Tower of Babel with misplaced brain information or metaphors. Fortunately, current data suggest that the brain can be reasonably understood and used for change. Science increasingly emphasizes a view of the brain as a set of information processing circuits of systems, not isolated neurons or regions. Brain disorders then are not deficits in one brain region but problems of information flow across circuits. Deep brain stimulation (DBS) is a good example. Circuit-guided interventions have the potential to become more widespread. CNS abnormalities, including generalized brain atrophy, small vessel disease, cerebral infarcts, Lewy bodies, neuritic plaques, neurofibrillary tangles, and white matter hyperintensities (WMH) are very common in older adults who do not have clinical neurological disease, and each of these individually or in combination may adversely affect motor function and gait.

There have been surprising trends in the prevalence of AD in recent years. First, the prevalence of dementia has decreased in the 65 and older generation. Second, people with more education have dramatically lower prevalence of dementia relative to those with less education. This especially applies to those with 16 or more years of education. Third, cardiovascular diseases are more successfully treated but this may not extend healthy life at older ages. In fact, there may be an increased risk of acquiring dementia in later life. Therefore, prevention of chronic disease may generate health and longevity but does not reduce the burden of dementia (Zissimopoulos, Tysinger, St Clair, & Crimmins, 2018).

Cross Cutting Dementia Themes

Dementia is becoming less prevalent overall in the US but not for all groups.

There is a worsening of cognitive skills, however, in blacks and whites older than 67.

Some groups of older adults are living fewer years with dementia.

People with good cognition are living longer.

Racial and SES disparities in dementia are large and not diminishing. High school dropouts suffer most.

Rising education levels of current cohort (relative to earlier ones) account for less prevalence of dementia.

Postponing the onset of dementia directly is the most effective way to reduce the number of people with dementia.

Schoeni, Freedman, & Langa (2018)

Finally, we note this: 47 million people have amyloidosis but without clinical symptoms (Brookmeyer, 2017). While most will not progress to a form of dementia, one out of seven will. Based on a math model from Mayo on 1541 Ss, 3% of normals segue to amyloidosis every year. The typical dance is: Amyloidosis at 60 and AD at 75. Amyloid positive status is a risk for AD. Related, in the ADNI study (N=445), 243 were Amyloid- and 202 were Amyloid+. All were normal at the initial assessment (Mean age = 74; Education = 16 years). In 3.1 years, a summative score (Sum of MMSE, CDR, Logical Memory Recall [PACC]) revealed that Amyloid+ had a higher likelihood of cognitive decline. An AP-E was greater in Amyloid+. Amyloid is not a benign phenomenon or reflector of normal aging (20 years before onset of dementia; Donahue et al., 2018).

The brain, then, is the organ of optimal interest. How could it not be so? That said, it is under review and we are a way from final understanding. It seems that weekly we are finding new molecules for possible targets for an arrest of AD or PD. In fact, it is probable that the brain is a massively parallel system of multiple processes with no central theater, no central "I" or self that dictates actions. Human consciousness is a complex set of selves and self-constructed illusions.

2. Epigenesis

“There is now a vast amount of data demonstrating the relationship between gene expression and anatomical features in early brain development... we suggest that similar genetic and epigenetic mechanisms continue to impact the structure and function of the brain throughout life... Late in life, similar genetic mechanisms may be involved in the breakdown of brain microstructure, AND changes in experience, as in early development, can advance and ameliorate the deleterious effects of aging. Data from multiple laboratories around the world suggest that calorific restriction and environmental enrichment can impact on gene expression in the aging brain...”

Huffman, 2012, p 9

Neuroepigenetics, a more precise term, highlights that biological aging (as we have noted) is largely determined by the internal biological clock and the accumulation of insults. Again, where the lifespan of the organism is closely related to biological aging, individual longevity is always a function of specific environmental circumstances, accumulated insults. The two totally interact at every level of the bio-hierarchy. Epigenetics, then, is the study of changes in gene expression that are mediated by mechanisms other than DNA; importantly, the environment and body interact at the behest of the mind (brain). They shape the environmental influences on the brain and behavior.

More complicated scenarios connecting genes, environment, and epigenetics are possible and likely. For example, because epigenetic regulation controls when and where genes are expressed, a mutated gene that could cause disease is irrelevant in the context of a normally low expression. However, that mutated gene may have adverse biological consequences if that regulatory context changes due to epigenetic modifications resulting from age or exposure. A “bad” environment matters. This would appear to be the age by gene or exposure by gene interaction that would be mechanistically explained by epigenetics. There are lots of mediating factors here. It is also plausible that genetic variation contributes to inherent differences in an epigenetic state between individuals, making some people more susceptible to environmental insults that would alter epigenetic regulation resulting in pathogenic biology.

In one study, growing up poor can lead to poor brain development and lower test scores according to a 6-year longitudinal study of 389 economically diverse children and young adults between the ages of 4 and

22. Scientists from Duke and the University of Wisconsin Madison analyzed 839 MRI scans collected from participants every two years. They found that children who came from families below the poverty line showed differences in brain structure from middle class and higher income children. Specifically, children from poor families had differences in the brain areas most crucial for academic development, including the frontal and temporal lobes and the hippocampus. The poorer children also had less brain matter overall than the other children (see Hyer, 2014).

And, we can see changes based on expressions of concern in life. A study by Kirton et al. (2014) showed changes in white matter lesions (WML) over a five year period for different age cohorts. Normal subjects were requested to rate depression or somatic problems on the CES-D at one point in time for their age cohort and then measured in WML volume. Five years later they were assayed on WML volume. Results indicated that ratings of either somatic problems or depression symptoms led to lower WML volume five years later. This was true for all the ages studied. Note that ratings were made blind to brain volume: How adults saw themselves resulted in brain problems.

These findings have applications in many directions. Results indicate that cognitive decline is not due to chronological age per se, but rather reflects the multiple causal factors from a broad range of biological and physical health domains that operate along an age continuum. We look, therefore, for functional biomarkers that can include a number of things, such as index functional capacity, decline with age, fitness, health conditions, and cognitive markers. In addition, objective biomarkers, such as histopathological accounts of plaques and tangles, oxidative stress markers derived from biochemical blood assays, genetic markers such as the ApoE4, WMHs, whole brain atrophy, dopamine receptor binding, and amyloid burden, are also noted. The bioage of a person, therefore, might be very different from others of the same age and have little to do with one's actual chronological age. Two individuals who are 75 years of age who have different biological and functional biomarkers, therefore, are distinct people and have a different bioage. This also seems to be happening as a function of cohort – adults measured on biomarkers 30 years ago have poorer levels of biomarkers (e.g. inflammation) than those adults assessed in very recent years. Finally, “superagers,” youth-like older adults, respond like younger adults and have above normal neurobiology. Importantly. They do not compensate or apply less efficient brain strategies (less dedifferentiation). This may account for

10% of the older population. Non-youth have different mechanisms. They have a less efficient memory, have a default network dysfunction, possess regional specific gray matter loss, and use frontal lobes more. According to Reuter-Lorenz and Park (2010), two core areas of brain efficiency at later life are in play. The juxtaposition of neurophysiological decline (e.g., gray matter loss) and efficiency of scaffold networks (more frontal lobe activation) result in a need to cope better. This takes cortical effort. In effect, as the normal aging brain declines, the older adult compensates and functions reasonably well, but this takes its toll.

Two popular markers are concerning. One is depression. Learned helplessness holds that the inability to have some control of one's life results in depression. Recent work suggests that adult experiences of depression can rearrange epigenetic markers in the brain and thereby change our behavior. Several groups of scientists have mimicked human depression in mice by pitting the animals against each other. If a mouse loses a series of fights against dominant rivals, its personality shifts. It shies away from contact with other mice and moves around less. When the mice were given access to a machine that lets them administer cocaine to themselves, the defeated mice take more. Again, depression may be an epigenetic disease process.

Second, the vascular system is especially vulnerable. Older adults with greater WMH and gait problems are at greater risk for depression and this in turn results in less efficient motor skills and behaviors, and more dementia (Rodakowski, 2018). Studies suggest that a good deal of the problems can be associated with what is known for preventative cardiology, which has a long history of identifying risk patients, initiating treatment, and reducing the impact of the disease. We can learn from cardiology. We should move, therefore, from a focus on dementia as an event that begins with a diagnosis and consider it instead as the possible end-stage of a long pathophysiological process that begins in mid-life with a "brain at risk." We need to identify patients at risk of cognitive decline and then initiate treatment to reduce risk factors and hopefully delay the onset of progression of the disease. Once a dementia diagnosis is given, it is far too late for prevention and remediation to occur. Preventative cardiology has shown the way in this.

We are of course highlighting an age x environment interaction. We know that 5-10 year rates of decline do go down but are different for cohorts and intra-individually. The biggest offenders include gait, IADL, ADL, followed by depression, grip strength, cognition, and others. These also show a downward trajectory as a function of the person and their interactions (Brigs, Carey, Kenny, & Kennelly, 2018). We have known for

some time that CNS disabilities abound, including atrophy, small vessel disease, and infarcts. These too are present as a direct result of the person-environment interface.

It appears that we do need a change in the definition of healthy aging. With age we degrade and have adjustment problems: Life spent with cognitive impairment is fairly constant with increasing age at around 1.4 years in men and 2.5 in women. Education, one of those epigenetic change agents, may reduce this by 13% for men and 22% for women. Several other interventions have an effect on longevity; interventions clearly help, such as yoga, internet use, being able to hear better, as well as all of the markers related to exercise and healthy living. Even climate seems to have a major impact on length and quality of life.

The epigenetic players of concern involve several common ones. They include cognition (subjective memory impairment, mild cognitive impairment, and dementia), affect-based problems (depression and anxiety but also somatic issues), genetics (chiefly ApoE4 allele), lifestyle (virtually everything), and biomarkers (WMH, tau, A-beta, brain weight), as well as health indicators (morbidity and vascular system). The ApoE genotype alone accounts for the clear majority of AD genetic risk. The finding of a relationship between AD and ApoE genotype has been widely replicated. Possession of one ApoE4 allele increases the risk of developing AD 3- to 5-fold, and possession of two ApoE4 alleles increases risk 15- to 20-fold.

Interestingly, using DNA from a saliva sample, a home test kit can determine whether the test subject possesses one or two copies of the Apolipoprotein-E $\epsilon 4$ (ApoE4) allele (Raber et al., 2004). This can create considerable ethical issues – knowledge of ApoE4 status and no counseling. Regardless, epigenetic changes are a key hallmark in aging, and DNA methylation-based biomarkers – often referred to as the epigenetic clock – have been shown to be robust measures of biological age. Incorporating blood cell metrics into the epigenetic measures demonstrates significant association with mortality (Murabito et al., 2018). Brains are energetically costly organs, but fortunately can adapt via social learning.

We add that epigenesis always takes its toll in subtle ways. The allostatic load has revolutionized the biology of disease as it implies that our bodies are constantly challenged by our environment. We remain healthy when we meet these challenges and return to baseline. Allostasis has a perspective in which physiological challenges provoke far flung adaptations throughout the body; the biological grind of an

infected toe, for example, reverberates throughout the body and continues to cause problems. Easily known internal biomarkers, like blood pressure and cholesterol, powerfully predict physical health and mortality. We now know also that low SES is linked to a heavy allostatic load as it appears that the body is in a constant battle to return to a baseline, non-stressed state. In this way childhood allostatic loads cause more long-term problems than one for an adult. In a sense the inside inequality of the outside psychological stress is relentless, impacting the PFC, hippocampus, amygdala, dopamine system, chronic inflammation, metabolism, and the circulatory system, at least. Stress, then, translates to an epigenetic marker that impacts all body systems. It even reduces the length of telomeres; wear and tear impact biology (Sapolsky, 2018).

3. Biopsychosocial Model

The biopsychosocial model has been mentioned. It is a broad view that attributes disease causation or disease outcome to the intricate, variable interaction of biological factors (genetic, biochemical, etc.), psychological factors (mood, personality, behavior, etc.), and social factors (cultural, familial, socioeconomic, medical). The biopsychosocial model counters the medical model, which attributes disease to roughly only biological factors, such as viruses, genes, or somatic abnormalities. The biopsychosocial model applies to disciplines ranging from medicine to psychology to sociology; its novelty, acceptance, and prevalence vary across disciplines and across cultures. Engel, its progenitor, described the commonsense observation that nature is a hierarchically arranged continuum with its more complex, larger units superordinate on the less complex smaller units.

The biopsychosocial model of health is based in part on social cognitive theory. It implies that in the treatment of disease processes the health care team address biological, psychological, and social influences upon a patient's functioning. In a philosophical sense the biopsychosocial model states that the workings of the body can affect the mind, and the workings of the mind can affect the body. The model presumes that it is important to handle the three together as a growing body of empirical literature suggests that patient perceptions of health and threat of disease, as well as barriers in a patient's social or cultural environment, appear to influence the likelihood that a patient will engage in health-promoting or treatment behaviors, such as medication taking, proper diet, and engaging in physical activity. Psychosocial factors can cause a biological effect by predisposing the patient to risk factors. As suggested by the epigenetic

influence, depression by itself may not cause liver problems, but a depressed person may be more likely to have a poor environment or poor vision, and therefore poor adjustment. Perhaps it is this increased risk-taking that leads to an increased likelihood of disease. Most diseases are really biopsychosocial illnesses/disorders.

“Maladjusted people are not truly mentally ill but at risk for becoming so. One sees such people everywhere: they were the socially awkward ones, the introverts, the evasive ones, the “excessively” good ones, or conversely, the rebels. What all these people shared was a deficiency in the ability to effectively “react” or “adapt” to the real demands of life... Mental disorders are disorders of human adjustment, maladaptations, un-hygientic compromises, immature or distressed methods of meeting the real situations of life.”

Adolph Meyer

If there is one thing true about the life process, aging, it is that we live in a biopsychosocial world. We are living longer and everything is connected. According to the death certificates created between 1969 and 2013, an overall decreasing trend in age standardized death rate was observed for all causes combined, heart disease, cancer, stroke, unintentional injuries, and diabetes, where the rate of decrease appeared to have slowed for heart disease, stroke, and diabetes. The WHO Ministerial Conference on Global Action Against Dementia hosted in March of 2015 showed that the focus on finding causes and cures for dementia, including AD, is intensifying since development of a cure for dementia by 2025 or beyond was cautiously noted. This now appears to be highly unlikely. Therefore, risk reduction is the most effective approach to delay onset and potentially reduce new cases.

As noted above, a balance of positive and negative genetic factors affect the brain in early/middle life to determine the degree of cognitive agility or impairment at late life. These factors increase or decrease oxidative stress, inflammation, insulin signaling components, size and frequency of infarcts, and concentration of growth factors, cortisol, and other hormones. It is also reasonably true that the mean cognitive decline over 10 years was initiated at several (putatively 7) years before dementia was diagnosed. During these years global cognitive decline measure will lower slowly at first and then pick up speed before diagnosis. Cognitive decline in AD is nonlinear and precedes dementia onset being established (Wilson et al., 2012). In fact, disability in old age takes form in a biopsychosocial environment.

While cognitive decline is a central part (Rajan et al., 2013), the broad spectrum of care mandates that the biological meet the psychological and social factors in the clinical arena.

The biopsychosocial paradigm forces us to look in many places for a bigger and better understanding of interactions. As people age, activities of daily living (ADLs) decline dramatically between 65 and 74 years. There appears a fourfold change in ADLs and threefold change in independent activities of daily living (IADLs). In a review in 2001, Hanlon et al. called for attention to the association between number of prescription medications and diminished physical and IADLs in community women. Older adults with no chronic conditions filled on average 10.9 prescriptions per year. With one or two chronic conditions, the number increases to 24.6. With three to four chronic conditions, it goes up to 44 prescriptions per year. If an older adult has five or more chronic conditions, the average is 60.6. The number of medications an individual takes concomitantly can have a dramatic effect on health. Clearly, the biological focus that has accompanied the medicalization of mental health is premature, given the primitive state of our psychiatric sciences. There are deficiencies in the logic and evidence base that supports efficacy claims in both psychiatric drug treatment and the specific empirically supported psychosocial treatments targeted as putatively discrete disorders described in the diagnostic manuals (Woolfolk, 2015).

Psychiatry Medication Limits:

Not one psychiatric drug cures anything.

Neurotransmitter deficiency is outdated model of mental illness.

100 known neurotransmitters: Psychiatry addresses 20.

Importantly, the biopsychosocial model has strong applicability at later life. If one wants to understand the causes and contributing factors of biological diseases, we need to assess the psychological and social factors. In fact, the skills that are required to practice medicine have changed measurably over the last 100 years. The primary causes of death are not infections but chronic diseases that start slowly and build over time. All of these diseases have a behavioral component. The biopsychosocial model further assumes there are varying levels of physical, cognitive, emotional, behavioral, and environmental factors that contribute to the clinical assessment and conceptualization of the case (Andrasik, Goodie, & Peterson, 2015). Templates provided by Belar and Deardorff (2015) indicate that developing the goals in clinical health psychology includes: Questions about the biological bases of health and disease that are related

to the problem; questions of the cognitive affective basis of health and disease as related to the problem; questions of the social basis of health and disease as related to the problem; questions of the development and individual bases of health and disease as related to the problem; questions of the interaction of the biological effect of cognitive, social, and developmental components as related to the problem; and concerns about choosing empirically supported clinical assessment methods for problems and how the assessment might be affected by information. These are strange questions to those of traditional medicine.

4. MCI and SMI (SCI), The New Cognitive Aging

Yes, this trend extends the focus of the brain noted as Number 1 above. Individual differences and cognitive aging may be associated with three broad factors. These include, firstly, normative age-graded factors both biologically and culturally determined. These are generally thought of as occurring at similar ages and periods for most individuals. The second is that individual differences in cognitive aging may also be associated with history-graded events, such as are illustrated by cohort effects, individuals varying in cognitive aging as a result of history-graded factors, such as medical advances and the treatment of chronic diseases affecting cognition, economic events, and outcomes of war. Finally, there are non-normative or idiosyncratic life events that affect cognitive aging. These are events not determined by age or history. A person may experience a brain aneurysm or be selected into a clinical trial for a new medication.

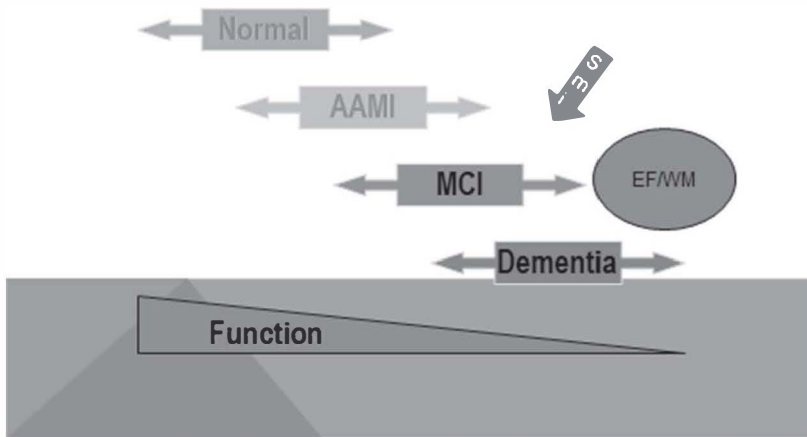
Cognitive aging itself presents as a gradual late life decline in performance typically assessed using cognitive task measures, experienced to a degree by most humans who reach old age. It is both ubiquitous and yet difficult to define, and even more challenging to accurately and reliably measure. Age is linked to smaller regional brain volumes and poor cognitive performance, including for episodic memory. Moreover, neural and cognitive factors completely mediate age differences in episodic memory. Thus, there not only exist multiple paths of influence mediating age effects on memory, but structural brain measures are central in cognitive aging. There are, then, multiple dissociations among specific age-sensitive cognitive skills and their reported neural anatomical substrates that support the view that age-related cognitive declines are unlikely to stem from a single aspect of brain aging (Lockhart & DeCarli, 2014; Marchant et al., 2013).

In normal aging, cognitive decline occurs mostly in speed of processing, working memory, long term memory, controlled processing (elaborative processing and performance at effortful tasks are poor), source memory, senses (visual and verbal), dedifferentiation (less neural specificity), as well as most fluid intelligence areas. The verbal domains remain largely intact. Several hypotheses have been proffered for this process; right brain, accelerated aging, reverse causation, lower IQ declines fastest, brain reserve, frontal lobe, crystallized and fluid intelligence, as well as age markers that pinpoint the age marker where decline modally occurs (age 55).

There are many variants of subclinical health problems related to cognition. We just need to identify the best markers, watch these, and then at some point treat. As with most constructs in search of definition, researchers scan up and down stream for anchors. Downstream of MCI is dementia, and the variability for conversion depends on the number of memory or cognitive problems as well as other factors, like poor education, Apo-E4, and others. Upstream is subjective memory impairment (SMI) which is just “below” normal aging and largely congruent with age associated memory impairment (AAMI). Of interest is that it is quite probable that SMI is a prodrome for MCI and dementia, and, along with depression or sleep problems, seems to be especially noxious and a predictor of a looming dementia.

The most notable issue in the past 20 years related to cognitive decline has been the conceptual space between normal aging and dementia, MCI (now Mild Neurocognitive Decline, mNCD). The figure below represents the taxonomy of the cognitive decline process. This decline process is not ineluctable and can be annoyingly patchy and unclear. Often people iterate between the various types (normal, AAMI, MCI, and dementia). MCI especially is fluid in definition but clear in concept formation.

MCI: DANCE OF COGNITIVE DECLINE



AAMI IS Age Associated Memory Impairment; SMI is subjective memory impairment; EF is executive function; WM is working memory

Cognitive Reserve

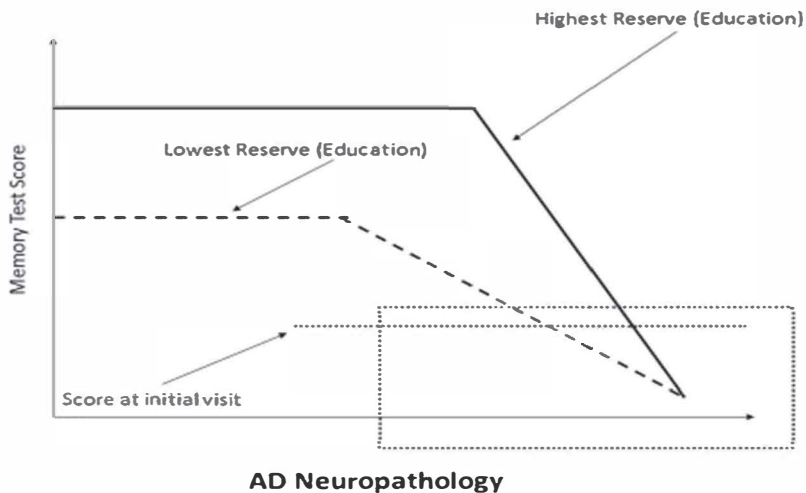
MCI and its variability imply the concept of reserve, cognitive or brain. The construct MCI has bolstered the need for cognitive reserve as it implies a continuum of skills or decline. In the neurodegenerative process the person with reserve has an assist during the early stages of neurodegeneration; symptoms arise later in the process, if at all. After a period of time, the level of symptoms catches up and the person shows a more precipitous trajectory of decline; in fact, cognitive reserve patients have more impaired brains (metabolic and perfusion deficits). The idea of reserve against brain damage stems from the repeated observation that there does not appear to be a direct relationship between the degree of brain pathology or brain damage and the clinical manifestation of that damage. Some time ago Katzman et al. (1989) described 10 cases of cognitively normal elders who were discovered to have advanced AD pathology in their brains at death. They speculated that these subjects (all women) did not express the clinical features of AD because their brains were larger than average.

Similarly, most clinicians are aware of the fact that a stroke of a given magnitude can produce profound impairment in one patient, while having minimal effect on another. Something must account for the disjunction between the degree of brain damage and its outcome. The concept of reserve has been proposed to serve this purpose.

One convenient, although not entirely accurate, subdivision of reserve models revolves around whether reserve is a passive process, or the brain is actively attempting to cope with or compensate for pathology. In passive models, reserve is defined in terms of the amount of damage that can be sustained before reaching a threshold for clinical expression. In the active models, reserve revolves around differences in how the task is processed. These two approaches are not mutually exclusive. Ultimately, some combination of these two approaches might best describe the empirical observations that have prompted the development of the concept of reserve.

One of the key outcomes in the study of reserve is the presence or absence of some clinical entity. For example, in AD many studies have attempted to determine whether there is a relation between some measure of reserve, such as education, and the prevalence or incidence of AD. Many studies have observed a higher prevalence of AD in individuals with lower education. Note that the assumption here is that since education is associated with reserve against the expression of AD pathology, AD should be less prevalent in individuals with higher education. These studies most often contaminate this conclusion by not directly measuring AD pathology, and thereby assuming that its prevalence is relatively equal across education groups.

Cognitive reserve is, however, a rich concept that has heuristic value for clinical work. While reserve is basically a simple idea, upon consideration there can be many layers of theoretical complexity. Reserve cannot be considered as a unidimensional entity. Brain reserve and cognitive reserve produce different predictions about the impact of brain pathology on function. Further, the differentiation of reserve and compensation may have particular practical utility when applying functional imaging. Below we can see the trajectories of a low and a high reserve person. While this figure posits that both low and high reserve show a similar score at one point in time (score at initial visit in figure (e.g., MMSE =17)), clearly they are different people and have different clinical profiles. High and low reserves have different brains as well as different trajectories of decline.



One model that accounts for the taxonomy of cognitive aging and reserve is the scaffold theory (Reuter-Lorenz & Park, 2010). Reuter-Lorenz and Park have established a conceptual model of a scaffolding theory of aging and cognition. We have reported on this model briefly above and in the previous book. It is an important model. Older adults may achieve a preserved cognition status by means of preserved neurobiology or compensatory processes or some combination of these factors. It is a hallmark of successful cognitive aging if maintenance of abilities and underlying neurobiology (rather than reliability of the assessment markers) have adequate longitudinal measures to evaluate the degree of change over time. Neural resource enrichment in this model includes such things as cardiovascular fitness and education, as well as brain reserve, while neural depletion includes such things as ApoE4 or vascular risk factors, heart disease, and stress, among other problem areas.

This model provides us with a good understanding of the problems of compensation and cognitive reserve. A balance of positive and negative genetic and environmental factors affects the brain in early/middle life to determine the degree of cognitive agility or impairment at late life. These factors increase or decrease brain factors, oxidative stress, inflammation, insulin signaling components, size and frequency of infarcts, and the concentration of growth factors, cortisol, and other hormones. Scaffolding or secondary networks are an important part of normal aging. This allows for the maintenance of

cognitive functioning as age associated structural deterioration occurs. This process is very efficient in youth and stands at the ready at late life. With age the scaffolding process may be invoked to perform familiar tasks, whereas in youth these processes are performed with little challenge. Extrinsically, the brain is confronted with novel or increased demands; intrinsically biological aging asserts itself. At some point scaffolding will meet its limit due to pathology as in AD. The pathology increases and the reparative processes wither.

The idea then is that, despite pathology, older adults compensate by scaffolding. This is the recruitment of additional circuitry that shores up declining structures whose functioning is noisy. Over-activation of the prefrontal sites accompanies the under-activation of the posterior sites of the occipital and parietal lobes. Generally this involves bi-lateral activation. Both hemispheres are involved. Scaffolding is not, then, a sign of pathology; rather it is compensatory in design and nature. It has strengths: physical fitness, cognitive stimulation, occupational attainment, education.

Importantly, lifestyle adds to cognitive reserve power. The hypothetical model presented by Cheng (2014) makes the case for cognitive reserve as a functional lifestyle in the context of cognitive decline. MCI, then, is a decline that is expected to trigger the brain to utilize reserve capacities to cope with the decline and stabilize functioning with minimal impairment. As pathology continues to accumulate and crosses the threshold, dementia is manifested along with the more accelerated downhill course. Lifestyle habits make a difference. Cheng espouses cognitive reserve as a marker to alter the clinical trajectory in several ways. Those with higher cognitive reserve are expected to show a delay of the MCI traits as they are more able to tolerate insult to the brain. It is probable that intellectual activity as well as physical activity is congruent with the model of cognitive reserve: That people who are physically active also tend to be cognitively active may explain the lack of independent effects of physical activity in multivariate models.

Additionally, Lojo-Seoane, Facal, Guàrdia-Blas, & Juncos-Rabadán (2014) presented a model of cognitive reserve based on education level and lifestyle. Cognitive reserve is often operationally defined as a complex structure of latent variables. The authors present a structural model that analyzes the effect of cognitive reserve on three cognitive domains: episodic memory, working memory, and general cognitive performance. They developed and analyzed a structural equation model to study cognitive reserve and cognitive performance in 326 participants over 50 years of age with subjective memory complaints. The cognitive reserve

construct was found to consist of two factors: An educational factor and a lifestyle factor. The model revealed that cognitive reserve had significant direct effects on episodic memory, working memory, and general cognitive performance, and indirect effects on episodic memory via working memory. This is important because interest in studying the validity of a cognitive reserve construct and its relation to cognitive function has increased in recent years and has been increasingly shown to be convergently and discriminately valid. It appears, therefore, that education and lifestyle both have a marked effect on cognitive reserve.

Perhaps the biggest reserve factor is education. In the MIDUS study, it was shown that a composite index of a number of adaptive psychosocial and behavioral factors was positively related to cognitive performance and change over and above the role of physical factors, health factors, and cognitive activities. Whereas previous studies had examined these factors individually, Agrigoroaei and Lachman (2011) showed the accumulative association and protective value for cognitive functioning of these variables. Interestingly, the effects of the protective factors were equally beneficial across adults within old age. Another key promising finding was that episodic memory and declined reasoning abilities were significantly attenuated as a function of some selective protective factors: People who engage in frequent cognitive activity can compensate for education differences. Cognitive factors performed over time make a difference. This suggests that the cognitive risks traditionally associated with low education can be attenuated by modifying a large spectrum of lifestyle factors. More on this later.

SCI

As noted, SCI refers to Subjective Cognitive Impairment. The domains of cognition have become increasingly muddy, or at least more diverse. In 2012, The Subjective Cognitive Decline Initiative, SCD-I, working group was formed in response to the challenge to understand the complex and evolving construct of SCD itself. SCD again applies to a syndrome whereby older adults complain or are concerned about perceived declines in cognitive abilities, yet perform within normal limits on standardized neuropsychometric and other clinical measures and retain normal function in instrumental activities of daily living. Rates of cognitive decline in SCD are variable and there are many difficulties in ascertaining the rate and frequency of

decline in persons with SCD. Among these are the sampling window within which estimates are taken and the demography of the sample in question. The terminology of SCI is also varied: subjective cognitive impairment (Reisberg & Gauthier, 2008), subjective memory impairment (Jessen et al., 2010), and cognitive complaints (Saykin et al., 2006).

Jessen and Colleagues (2013) examined the data from the Age – CoDe Study, a general practice registry-based longitudinal study in older adults, designed to identify predictors of cognitive decline and dementia. Among the individuals with SCI and early MCI, risk was assumingly elevated in subgroups of participants who reported concern. This suggests that early in the trajectory of decline, perceived concern has additional prognostic value over and above self-reported cognitive complaints, the presence of which may elevate the risk for subsequent decline to AD and other dementias. Mewton et al. (2014) also looked at over 1900 community dwelling participants between 65 and 85 and measured subjective memory complaints. Those who reported subjective memory problems (35%) reported more psychological distress, poor functioning, service use, and negative self-assessed mental and physical health. Declining memory perception was also related to increased psychiatric problems in the previous five years. This occurred in people with MMSE >27. This also has an impact on the negative perception of aging. Epigenetics anyone?

At the individual client level, there are still no reliable objective measures to corroborate a diagnosis of SCD and differentiate it from normal aging. This, then, places a considerable emphasis on self-report. Of interest, it appears that SCD with worry is rather consistently reported over time and is associated with greater increased risk of AD. Wolfsgruber et al. (2016) note that concerns or worries about decline have incremental predictive validity over the report of complaints per se. Other psychological issues may also contribute to psychological factors that influence SCD. Depression elevates the risk of all-cause dementia. Anxiety does the same thing. Worry specifically about cognitive decline is also a potential indicator of SCD and should not be dismissed as evidence of the worried well. There is also accumulating evidence that neuroticism and lack of conscientiousness in particular present concerns that lead to risk for MCI and dementia (Duberstein et al., 2011). Perhaps this applies to SCI also. Physical health, of course, always is an issue in these cases.

In total, clinical assessment of cognitive complaints is still poorly operationalized. Recent qualitative research suggests that certain phenomenological complaint themes may have some specificity for prodromal AD. Miebach et al. (2018) assessed that issue. In a cross-sectional case control study using a mixed-methods approach, 23 memory

clinic patients and 21 psychiatric patients with MDD and 21 health controls, age 55-86, were assessed. A newly developed semi-structured interview assessing 12 complaint themes was used, and transcribed open format responses were coded by qualitative expert rating and compared between the groups. Results revealed that seven complaint themes, for example, sense of problems and progression of problems, were significantly more often endorsed by the cognitive complaint groups, together with a novel theme of distractible speech. Complaint themes in those with depression aligned with the depressive symptoms and appeared to be different from the complaint pattern of the cognitive complaint patients. Specifically, four themes emerge as being typically depressive; MDD patients were found to express more attentional fluctuation and affect influence on memory. In addition, relative absence of contextualization was significantly more present in MDD patients as they complain more often about memory lapses that occur in specific settings. This area is unfolding.

MCI

As problems develop cognitively, MCI has been seen as a key marker in the decline of older adults. There are several types; amnesic MCI (aMCI), 1, 1.5, 1.96 standard deviations low on memory, and non-amnesic MCI (non-amMCI). The neuropathology is largely consistent with AD. Its biomarkers suggest more probable dementia designations (low A-beta, high CSF tau, atrophy of hippocampus), and the progression to dementia continues at a rough rate of 10-15% per year conversion to AD. The DSM reminds us also that there are mostly preserved functional abilities and there is no extant dementia. There are also no FDA approved treatments (Petersen et al., 2001).

Mungas, Brooks, Lowenstein, and Bondi are among the many researchers who have noted significant differences in the conversion to a dementia depending on the criteria used to define MCI. Many can actually revert back to the normal range. Most do not change status. Regardless, clinical interest for MCI is an exciting and fortuitous "problem." By using this designation clinicians argue for continued hope and challenging healthful lifestyle growth. Tasks of concern for MCI patients who are fearful of dementia include: driving (40%), work (12%), relocation (31%), firearms (50%), being treated differently (40%), social limitations (31%), and needing care (50%).

The following table presents the clinical thinking on the predictive problems going from normalcy to SMI and eventually MCI and

dementia. In meta-analyses, episodic memory scores are still more highly predictive in detecting preclinical AD than CSF biomarkers (Schmand et al., 2010).

Decline Markers Leading to MCI or Dementia

- Problem is the abnormal processing of B-amyloid peptide leading to plaques
- Occurs slowly while person is still normal
- Biomarkers of B-amyloid are reductions in CSF AB42 and increased amyloid PET tracer retention.
- After a lag, neuronal dysfunction and neurodegeneration become the dominant biomarkers.
- Biomarkers of neuronal injury and neurodegeneration are increased in CSF tau and structural MRI measures of cerebral atrophy.
- Cognition falters: Neurodegeneration is accompanied by episodic memory problems along with synaptic dysfunction which is marker by uptake on PET.

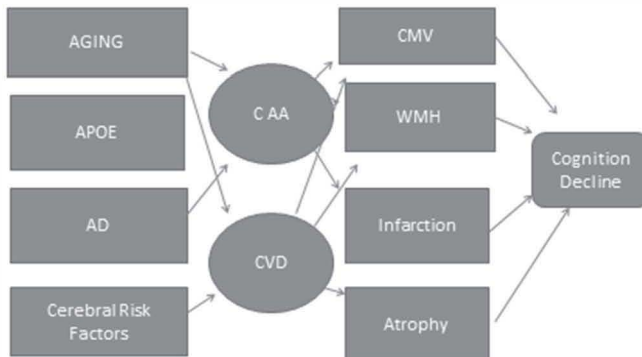
What MCI is and how it is measured are much at issue. Regardless of how it is measured, taking an empirical and actuarial approach to classification results in more reliable diagnoses and more reliable associations to biomarker outcomes and rates of decline. In one study Rentz et al. in 2004 discussed the use of IQ adjustment norms to predict progressive decline in older adults with above-average intelligence. This is but one alteration in the effort to identify cognitively compromised people before a dementia diagnosis. Most important, Jak et al. (2009) identified four clusters that emerged in preclinical work prior to AD. These include dysexecutive problems and amnesic MCI, mixed MCI, and a group that had a single impaired test score on the measure of visual spatial function. That said, he insisted that two measures be applied for a determination to be made of any of these forms of MCI. Finally, Edmonds and Colleagues (2015) discuss the concept of subtle cognitive decline defined in actuarial terms on performance in neuropsychometric tests. They define subtle cognitive decline as impaired scores (greater than one standard deviation below age-corrected norms) on two measures in different cognitive domains, as well as a score of 6-8 on the functional assessment questionnaire (FAQ). They found that this criterion had predictive value in determining who would decline to MCI and AD using data from ADNI in that it provides an operational definition of subtle cognitive decline as referenced in the National Institute of Aging – Alzheimer’s Association (NIA-AA) criterion on preclinical AD (Sperling et al., 2011).

5. Cerebral-vascular Health

Cognitive decline is mimicked or led by several brain features. As noted above, perhaps the most salient factor in neurogenesis is synaptogenesis. Synaptic density is the most accurate biophysical correlate of cognitive impairment. Hippocampal atrophy also counts. Lifetime levels are inversely related to cognitive activity in healthy adults. But part of this, also, is cardiovascular health. This is highly related to cognitive health. Aging, genetics, AD, and vascular factors lead to cerebral amyloid atrophy and cerebral vascular atrophy leading to micro-brain bleeds, white matter hyperintensities, infarcts, and brain atrophy. This results in less cognitive skills.

Below is a model addressing the issues of brain health highlighting vascular problems. Aging, the genetics of APO-e, and AD markers (a-beta and tau), as well as vascular problems, lead the parade. They eventuate in CAA (cerebral amyloid angiopathy) or CVD (cerebral vascular disease), which in turn develop into microbleeds, WMHs, and infarction as well as atrophy. Eventually the behavioral factors of cognitive decline and poor behavior result. The vascular part of the process is unmistakable. It sets the stage, the foothold, for QD and other dementias. It is easier to develop AD if one has Cerebral Small Vessel Disease (CSVD).

Big Picture



CAA is cerebral amyloid angiopathy; CVD is cerebral vascular disease; WMH is white matter hyperintensities.

Let us focus on CSVD for a second. It is the pathological process of subcortical structures, such as lacunar infarcts, white matter lesions, and micro-bleeds. CSVD is responsible for the pathogenesis of ischemic strokes, cerebral hemorrhages, and encephalopathy, which are associated with advanced age. This is led by such chronic diseases as hypertension and diabetes mellitus. CSVD mainly contains a lacunar infarct or lacunar stroke, leukoaraiosis, Binswanger's disease, and cerebral micro-bleeds. As noted, with the development of imaging examination and its clinical usage, it has been determined that cerebrovascular disease, especially CSVD, contributes to the occurrence of AD also. CSVD is responsible for 20-30% of the cases of ischemic stroke, as well as for a considerable portion of the cerebral hemorrhages and encephalopathy. CSVD gradually develops into a decline of cognition, vascular dementia, impairment both of gait and balance and of mood, depression, and urinary incontinence, and often results in greater social and economic burdens (Cai et al., 2015). Gait slowing and cognitive decline are both common in older people. In addition, high fatigability, IL-6 dysfunction, other pro-inflammatory states, and executive dysfunction are also sequelae (Lin et al., 2014).

Vascular health is critical for health, but especially for cognitive health. It influences everything. Brain aging even needs a readjustment. Healthy brain aging is now more confusing and probably less prevalent. As noted before, normal brain aging usually refers to signs of age-related differences in brain structure and function in the absence of clinically significant impairment among the general population. By contrast, healthy brain aging is the apparent structural and functional preservation of the brain with advancing age and is considered less common. Clinically silent brain infarcts increase over the life span at a 75% rate. This includes cerebral microbleeds, small dot-like lesions with low signal intensity. Ten percent of community older adults (50% in stroke patients) have this. Even minor cognitive declines can measurably influence clinical outcomes – rate of hospitalization, medicine adherence.

That said, it is unclear whether effective treatment of vascular risk factors prevents cognitive decline and clinically silent brain injury. It is also unclear whether antihypertensive treatment really reduces incident dementia (Jongstra, Harrison, Quinn, & Richard, 2015). Further, it is unknown whether there is a critical treatment window for the prevention of vascular effects on brain and cognition or whether achieving current treatment targets is adequate to prevent silent brain injury. It is even unclear whether some individuals are at risk and need to be treated differently (family history, genetic markers), and how the time course of vascular brain injury relates to neurodegenerative brain injury is unknown.

Regardless, perhaps the most common contaminate of a degenerative process is the cardiovascular system. Neuropsychologically we will see executive function, attention, and processing speed especially suffer. Affectively, depression is a marker as a reflector and a consequence of vascular problems. Behaviorally, disinhibition and lesser ability to tolerate discomfort and handle stress result. Health-wise, this aspect of living is always relevant and important.

The condition of someone's brain postmortem only partially tells how well it functioned in the years leading up to the person's death. That said, vascular risk factors are ominous. MRI studies show extensive WMHs, subcortical microvascular lesions, lacunes, and microinfarcts in older adults with cognitive problems and depressed older patients. In fact, WMH and executive deficits predict a poor course for depression. Post-mortem studies of elderly persons who have died from suicide reveal lacunes as well as small vessel and Alzheimer-related pathologies. Overall, three mechanisms account for the vascular compromise of depression: focal vascular damage, hypoperfusion, and abnormal inflammatory responses. We also know that APOE-4 carriers with depression were 4.4 times more likely to progress to AD compared to the non-APOE-4 carriers with depression (Irie, Masaki, Petrovitch, et al., 2008). So, vascular markers count and count more with genetics (APOE-4) and depression.

6. Lifestyle

Life value and health in general rest on our daily behavioral routines. Five habits lead to 70% of morbidity and mortality: how much we eat, what we eat, whether we exercise, whether we smoke, and whether we consume alcohol excessively (DeVol & Bedroussian, 2007). If we added sleeping, mating, drug use, and relationship habits, we would account for another significant proportion of the burden of chronic and infectious diseases (Chorpita, Miranda, & Bernstein, 2011). Other noted risk factors include age, lower SES, health status, medication usage, APOE status, unhealthy behaviors, and neuroticism, as well as gene-environment interactions (Hagger-Johnson, Shickle, Deary, & Roberts, 2010). Similarly addressing dementia, Gatz et al. (2005), using a longitudinal view of the Swedish Twin Registry looking at a sample of nearly 12,000 twins now over 65, found that diabetes and obesity are among the most significant NON-genetic risk factors for AD and dementia. Additionally, a sedentary lifestyle leaves older adults at risk for just about everything. Both obesity and

malnutrition increase with age; currently, about 1/3 are obese and 1/5 are malnourished. We appear to be our own worst enemies.

We start with cognitive training (CT). The World Health Organization has argued for an active aging framework that is now widely accepted and that can be readily framed to support initiatives to promote mental activities as well. However, unlike physical activities, there have been no established mechanisms or organized attempts to promote intellectual activities at the population level. This is changing with HABITS (Mayo Clinic) and the FINGER study (see chapter 5). There may be a need to bring cognitive health into policy agendas alongside the traditional focus of physical health and to issue guidelines on this practice. It is not entirely clear whether activities at the MCI stage should simply be a continuation of previous activities from specific efforts to resuscitate cognitive reserve. In any case, the presence of cognitive impairment, declining vitality and physical health may themselves constitute a barrier to this participation.

Recall the Agrigoroaei and Lachman (2011) study where education differences and episodic memory and declining reasoning abilities were significantly attenuated as a function of a number of protective factors. People who engage in frequent cognitive activity can compensate for education differences. The devil is in the detail. The complexity of the program of CT as well as individual factors plays a large role. Definitive statements are hard to assert. In sum, one can say that the data are promising regarding computer-based CT and any side effects are minimal, but any commercial claims of the efficacy of computerized cognitive enhancement systems is perhaps premature (Jak et al., 2013). That said, we need to re-consider the concepts of “rehabilitation” and apply these to CT in general. What might be broadly considered cognitive rehabilitation, in fact, is a blend of restorative activities. Below is a schema that we will pick up on in later chapters.

Simple Cognitive Improvement Taxonomy

Cognitive training itself involves a guided practice-set of standard tasks designed to impact specific cognitive functions with a range of difficulty levels.

Cognitive stimulation is typically applied to persons with diagnosed dementia. It uses simpler tasks and involves reality orientation and the like.

Cognitive rehabilitation has a greater focus on individual needs and goals of the client.

Thinking stuff is followed closely by exercise. There is not a month goes by when the value of exercise is not endorsed, even extolled, in some aging data set. Indications are that physical exercise not only improves cognition, but also moves AD biomarkers in the right direction. Baker (2015) showed that hypometabolic regions of the brain that were associated with Alzheimer's symptoms were re-perfused. Phosphorylated tau, a marker of neuronal injury, fell significantly in the cerebral spinal fluid. This was a six-month exercise trial of 71 sedentary adults who had both MCI and pre-diabetes. The subjects ranging in age from 55 to 89 were randomized to either a controlled program of stretching three times a week or an exercise program of aerobic exercise for 45-60 minutes per week. There were no reductions in CSF tau in the groups with stretching. However, the exercise group experienced significant declines in CSF tau.

●f all lifestyle interventions aimed at preventing dementia, there seems to be more evidence for physical activity, possibly because it more directly affects cerebral blood flow. When you give someone, say, 6 months of exercise, 6 months of moving their legs, you see more effects in their cognitive score than if they did 6 months of cognitive training. The activity does not have to be strenuous. Accumulating evidence suggests that yoga, tai chi, and mindful movement practices may also be helpful. And some studies have found positive effects from combining strength training with aerobics. ●f all the possible lifestyle changes, exercise has been proven to have the biggest impact.

In recent years, the identification of biomarkers for AD has made it possible to compare levels of amyloid-beta ($A\beta$) and tau – both hallmarks of AD – in those who are and those who are not physically active. ●ne new study (Brown et al., 2017) evaluated the relationship between exercise levels and brain amyloid load in carriers of genetic mutations that cause autosomal-dominant AD. The analysis included data from the Dominantly Inherited Alzheimer Network (DIAN) for 139 pre-symptomatic mutation carriers. These patients are destined to develop AD and know approximately when they will start having symptoms. Patients were categorized into those reporting fewer than 150 minutes per week (low exercise) and those reporting 150 minutes or more per week (high exercise). The researchers were able to show that $A\beta$ in those in the high-exercise group accumulated at a slower rate relative to what would be expected. The results suggest that higher levels of exercise may delay the accumulation of Alzheimer's pathology and subsequent symptom onset in Alzheimer's disease

mutation carriers. One big question is, does the lower $A\beta$ mean that the onset of symptoms will actually be delayed?

Exercise programs help in many life quality areas – falls and depression, just to name two. Virtual reality computer-based technology appears effective; it provides the user with opportunities to interact with virtual objects and is used extensively for entertainment. This can also be used for exercise and training (Laufer, Dar, & Kodesh, 2014). Even Wii-based exercise seems effective; compared with no exercise, positive effects were seen on at least one outcome measure related to balance performed by older adults. Studies comparing the Wii-based training with alternative exercise programs generally indicate that the balance improvements achieved by the Wii-based training are comparable to those achieved by other exercise programs. The Wii-based exercise programs may serve as an alternative to the more conventional forms of exercise at improving balance control.

Exercise trumps depression. In the HUNT study (Nord-Trondelag Health Study) Harvey et al. (2018) followed over 33,000 healthy adults in Norway starting around 1985 and following them for at least 11 years. Participants were screened very carefully to ensure that they were healthy, having no pre-existing history of depression. Exercise did not protect against anxiety. However, exercise did have a significant effect on depression. The study investigators estimated that compared with individuals who did not exercise, those who exercised an hour or more a week had a 44% decreased odds ratio of being depressed; quite a significant finding. These researchers also noted that depression was a causal factor: about 12% of cases could be prevented if all adults exercised for a little over an hour a week.

Social relationships fall into the mix in a rather big way. Estimates are that between 20 and 43% of U.S. adults over the age of 60 experience frequent or intense loneliness, higher than the prevalence of merely living alone (Perissinotto, Cenzer, & Covinsky, 2012). Relevant social indicators are regularly collected as part of census data. For example, more than a quarter of the U.S. population (27%) lives alone, over half of the U.S. adult population is unmarried, and 1 in 5 have never married. The divorce rate in the United States continues to hover around 40% for first marriages. Importantly, positive and negative aspects of social relationships appear to be related to cognition throughout adulthood consistent with the hypothesis that social factors have lifelong influences on cognition. Positive and negative aspects of social engagement may thus be important factors to consider in relation to efforts to promote optimal cognitive development and cognitive aging. In later chapters we will see the certain

value of socialization. Being alone is especially troubling after a stressful episode or a loss. Men are at most risk. The World Health Organization (WHO) lists poor social support networks as detrimental to health and the United Kingdom's Ministry of Health has established loneliness as a health priority. Social relationships are therefore a critical issue in healthcare. Although caution must be used in suggesting single, widowed, or divorced adults are less socially connected than those who are married, the structural dimensions provide robust indications of health risk.

Data from another national sample of older adults, aged 65 to 91, revealed that those who experience persistently high levels of negative social interactions (for example, support letdowns, rejection by others) over a two-year period reported poor self-health, more health conditions, and greater fundamental impairment (Newsom, Mahan, Rook, & Krause, 2008). Consistent with this idea, a large study of adults aged 40 to 77 found that negative social interactions decrease with age. The strength of the association between these negative interactions and physical health, however, increased over the adult life course. Remarkably, one study of individuals aged 48 to 77 found that the number of ambivalent social ties, but not the number of aversive social ties, and participant social networks was related to shorter telomere length, an indicator of accelerated aging (Uchino et al., 2012).

And then there is "somatic softening," relaxation. Many studies have shown that eliciting the relaxation response in a psychophysiological state of deep rest induced by practice, such as meditation, yoga, or prayer, not only relieves feelings of stress and anxiety, but also affects physiological factors, such as blood pressure, heart rate, and oxygen consumption. A study out of the Institute of Technology Assessment and the Benson-Henry Institute for Mind/Body Medicine at Mass General Hospital finds that individuals participating in a relaxation-response-focus training program use fewer healthcare services in the year after participation than in the preceding year. Based on the number of healthcare encounters in the study period, which included interactions with healthcare providers in any setting, imaging studies, lab tests, and procedures, the relaxation response participants had an average reduction of 42% in their use of healthcare services in the year after their participation. The controlled group, on the other hand, had an overall but not statistically significant increase in service utilization in that second year. These findings appear also to be related to the practice of mindfulness (Siegel, 2014).

Taking care of one's health matters applies to mental health. Proctor et al. (2003) studied the interaction of comorbid medical conditions and depression. Depression of course is a generalized marker for all mental health. Hospitalized depressed elderly patients evidence high medical comorbidity, with three-fourths having at least one medical illness serious enough to require medical treatment. Vascular, cardiac, musculoskeletal, and neurological conditions are most prevalent, suggesting the need for universal monitoring with larger samples. The rate of heart disease compares with that of older adults hospitalized for reasons other than depression, but the findings of this group indicate higher than expected frequencies of hypertension, Parkinson's disease, osteoarthritis, COPD, and diabetes among the depressed elderly patients. Subgroup analysis focusing on depression within these most common medical conditions, particularly the vascular conditions, project trouble. Models that would predict problems for total functioning would include gender, social economic status, age, the MMSE, and, most importantly, depression. These would interact with medical comorbidity to have a significant impact on total functioning. Depression and cognition would have a direct impact on total functioning, but they would also have an indirect impact on medical comorbidity.

We have already discussed the effects of clinically symptomatic cerebrovascular disease on brain aging. Health here matters. As noted above, these are particularly difficult to separate from normal brain aging, as CVD and cardiovascular risk factors (CVRFs) are very common and CVRF-related brain structure differences such as cerebral atrophy, white matter injury, and MRI infarction are observable as early as middle life. The available treatments and awareness of the health consequences of CVRFs have resulted in significant reductions at the population level with control of common vascular risk factors such as hypertension. This has resulted in a substantial decline in death due to stroke, coronary artery disease, and heart failure. Declines in function occur because of atrophy of gray matter, white matter, and hippocampal volume, reduced white matter integrity and white matter injury, as well as reduced cortical thickness. All of these cause difficulty. As one takes one's health seriously and alters one's lifestyle, to that extent life will be better protected and better quality of life will follow.

Polypharmacy and compliance are two other issues of note related to general health. We briefly address polypharmacy. This occurs with the use of four or more medications. A population-based study showed that most older adults take between five and eight medications daily with 12-39% taking nine or more (Cannon, Shaw & Zuniga, 2006). This trend

seems to be growing as people over the age of 65 take 40% of over-the-counter meds, further complicating polypharmacy (Rolita & Freedman, 2018). The unfortunate consequence of this is a rather high rate of adverse drug reactions (~20%, Routledge, Mahony & Woodhouse, 2004). Of course polypharmacy is a special problem biologically as the aging body processes medications in a less efficient manner. Aging is associated with less absorption, less distribution, poorer drug metabolism, and inefficient excretion (Wooten, 2012). Regarding pharmacokinetics the problems are also considerable. Gastrointestinal bleeding, for example, is common with ANSADs leading also to CHF (Page & Henry, 2000). Opioids have been implicated in many problems including constipation, sleeplessness, and balance. Clearly this needs to be part of any lifestyle evaluation and plan.

There are other factors that make a difference at later life; dietary changes, dietary supplements, and “purpose medications,” such as those currently used for cholesterol or hypertension, as well as the growing number of investigational disease modifying agents. The search is on for a lifestyle marker(s) or therapy that delays the clinical onset of AD by almost five years without also increasing lifespan, having the potential to reduce the number of affected individuals by half. What one does makes a big difference across lifespan. Yaffe et al. (2001) noted that the determinants of cognitive aging in men and women: 30% were maintainers, 53% were decliners, and 16% were major decliners. Not surprisingly, problems tend to compound themselves: 68% of adults with mental health conditions also have medical conditions, and 29% of adults with medical conditions have mental health conditions (Clay, 2013). (We will see more on lifestyles in chapters 4 and 5.)

7. Prevention

We follow up on lifestyle with prevention at a more societal level. Each area noted above can be improved upon by an interactive choice that results in improved health. Higher education level and literacy consistently predict late life health in all its forms, less disability, lower risk of obesity, and less later life diseases, including AD and cardiovascular diseases. Improvements in education may be contributing to decreasing dementia incidence in several countries. The effects of unhealthy lifestyles in childhood and adolescence are manifested in young adulthood as atherosclerosis of the arteries, a

precursor to cardiovascular disease. Prevention or treatment of hypertension and diabetes at every age is associated with decreased risk of heart disease, stroke, and recurrent events. Individuals with fewer social and economic resources manifest chronic diseases and disability earlier in their lives than those with greater resources and access to healthier lifestyles. In middle adulthood, cognitive enrichment and occupational and other exposures are protective against late life dementia. Cognitive activity in late life can improve cognitive reserve and delay or prevent cognitive decline. Exercise and appropriate nutrition can increase muscle mass and strength in frail nursing home patients and decrease the incidence of frailty.

The construct of prevention is worth highlighting. There is strong evidence that primary, secondary, and tertiary prevention are effective in preserving or improving health and function in older adults. Notably, both the primary and secondary prevention of non-communicable diseases, especially hypertension, smoking, obesity, diabetes, cardiovascular diseases, and selective cancers, implemented after age 50 and after age 65 are highly effective, as are interventions to prevent other geriatric conditions, such as falls. In addition, stroke prevention has shown good potential for also preventing cognitive decline and dementia. There is also a strong potential for the prevention of disability and frailty through multiple approaches: Treatment to decrease disease severity, preventions of additional diseases which could interact with preexisting disease, frailty prevention through both the maintenance and strength of exercise tolerance, and nutrition. Tertiary prevention such as rehabilitation after a stroke is critical in improving functioning and reducing disability. Overall, prevention could substantially improve the health of older adults and could achieve such benefits with little or no additional lifetime medical spending.

This needs titration. When only the poorest do not have access to a service, policies probably need to focus on expanding provisions to particular groups that are excluded or marginalized. When almost everyone does not have access to the service, more wide ranging or universal strategies are usually required and can be successful. When policies are aimed at the level of social gradients to improve fairness, the result can be significant health benefits for older adults and improvement in health quality. For example, around 1 in 5 cases of AD worldwide is estimated to be attributed to low education attainment with about 6.5 million attributable cases globally in 2010 alone. This underscores the need for primary prevention strategies. In many settings, the pattern will fall somewhere between the two extremes and a combination of strategies

will be needed, with specific policies to increase access to care by older adults taking into account each national context.

Let's consider depression again, the model for health problems. In 1984, the National Institute of Mental Health Public Information pamphlet entitled "Depression, What We Know" stated that in general the onset of clinical depression cannot be prevented (Munoz et al., 2012). This is no longer the case and it was essential that the healthcare system took notice. In 2009, a report, "Preventing Emotional and Behavioral Disorder Among Young People: Progress and Possibilities," listed several random trials in which the proportion of new cases of major depression were significantly reduced in participants randomly assigned to preventive intervention participation relative to control groups. This was a major scientific advance and requires the reappraisal of the resources allocated to research, practice and training in preventive mental health care. At different stages some factors – for example, parental bereavement, divorce, or having serious medical illness, may increase the likelihood of a child developing difficulties – while others – for example the presence of stable relationships, good schools, or effective vigilant parenting – may promote the development of resilience. Integrative care suggests that causation is determined in part by familial and genetic factors and in part by current life adversities. It also emphasizes that vulnerabilities may be expressed in different ways at different developmental epochs. These results apply in very old people at the subsyndromal level of depression which is prevalent (Ludvigsson, Milberg, Marcusson, & Wressle 2014).

As described by the WHO, success over the life-course requires investment in the prevention of disease, disability, and injury, and the preserving of health and wellbeing at each age and stage of life. The WHO report on healthy aging is built around the concept of functional ability. This is defined by the most recent WHO report as the health-related attributes that enable people to be and to do what they have a reason to value. The report emphasizes that this ability is determined by both the intrinsic capacity as an individual and the influence of the environments that they inhabit. This builds on capability-based approaches used in other areas. The report approaches the changes associated with aging in the context of an entire life-course that focuses on the second half of life: functional gains are dependent on preventative behaviors.

Treatment takes place in a primary care clinic. Physicians are the gatekeepers. Primary care is the first stop to connect patients –

especially those with complex health needs, such as multiple chronic illnesses – with other necessary services, including specialists, after hours or home care, and social services. But a rising number of patients at risk for chronic diseases, an aging population, and life-prolonging medical interventions have added new financial and capacity stresses on primary care systems, with primary care physicians making decisions from an increasingly challenging position.

Primary care is a team sport. Efforts to identify those areas in which physicians have the greatest concerns are a crucial part of recognizing where the greatest challenges and risks in primary care remain. In a summary from the Commonwealth Fund's 2015 International Health Policy Survey, 500 to 2900 physicians from each of ten industrialized countries were surveyed to gauge their perspectives on their preparedness for providing care for patients with complex health needs in the primary care systems in which they work (Osborn et al., 2015). The results show that there is almost universal difficulty in coordinating care for patients with complex health needs. Managing care for patients with mental health issues and substance disorders is a particularly problematic area, with less than 50% of physicians from nearly all the countries surveyed reporting that their practices were prepared for such patients. Interestingly, Canada (49%) and the USA (38%) have the lowest percentage of practices that make after-care arrangements, which is a likely driver in the higher use of emergency departments for chronically ill patients in those countries.

The extent of the opportunities that arise from increased longevity will depend heavily on one key factor, health. If people are experiencing these extra years of life with good physical and mental capacity and if they live in enabling environments, their ability to do the things they value may have few limits. If these added years are instead dominated by declines in capacity and disabling environments, the implications for older people and for society are much more negative. There is now a strong knowledge base that interventions work and matter at every age and stage of life. Some elements apply to every age, such as physical activity, nutrition, and a healthy environment. Others are specific to an age. For societies, this approach is critical to the decreasing of health disparities between individuals of particular social, economic, and age groups in nations, disparities which develop accumulatively and which can be predicted by exposures to each point in the life-course. Further, these investments will form the basis for compressing morbidity into the latest stages. People who arrive at old age healthy are positioned to remain healthier into the older ages. This approach can lead to lower long-term healthcare costs and a healthier and more productive society.

8. Precision Medicine

Niederehe (2013) noted that it is time to move on from a theory-based mode of research and to begin to conduct studies that actively test specific hypotheses about the psychopathology of interest. Ideally the goal would not be simply to replicate in older adults whatever findings have previously been shown about the psychopathology model in younger adults, but to relate aspects of the model to aging process variables – in other words, to examine how the aging process interacts with particular facets of psychopathology. To represent the aging process in such studies, the research design might well incorporate variables drawn from relevant basic science theories of aging (biological, psychological, and social). Moving in this direction will help research become less merely descriptive and more mechanistic by elucidating increasingly specific pathways whereby aging impinges on mental disorders. Concerns about psychopathology as a dimension and longitudinal studies, as well as looking at practical gaps and issues that are not currently being studied, are of interest. Clearly older adults share the complex interplay among multiple interacting forms of disease. If all past, present, and future predictors and processes that contribute to future events were known and quantifiable, algorithms could be constructed that would produce perfect risk estimates of individuals (that is, they would predict with perfect accuracy whether an event would occur or not in every individual), and life would unfold with health precision.

This is currently still a quixotic notion. It is not possible at present because only a fraction of the information that explains the variations and outcomes have been identified. Accordingly, estimates of risk are incomplete and conditional on information included in risk calculation. This, however, is improving. Predictive analytics can improve clinical care by providing general recommendations for populations that can be incorporated in clinical guidelines. Predictive algorithms are an essential component of guideline recommendations. However, because predictive models imperfectly explain clinical outcomes, they do not estimate individual risk very well even when they accurately explain group risks. Consequently, these models cannot replace a physician or healthcare provider in the process of everyday care.

Again according to Niederehe (2013), big data has entered the picture. The NIH has made viable high-resolution imaging procedures which sequence the individual genome, as well as psychosocial components, and so team science has been involved. This includes cognitive aging and depression especially. In addition, there are studies

that involve risk assessment and management involving risk calculators, as well as the science of behavioral change itself. Increasingly there is a personalization of care or precision medicine. Precision medicine is close in some areas (e.g., risk of diabetes).

Some years ago it became obvious that loss of executive function is a problem for adjustment. Mast et al. (2004) examined risk indicators associated with cerebrovascular disease, including diabetes, hypertension, and heart disease. Their relationship to depression and executive function was also assessed. Older adults were initially assessed at the time of admission to a rehabilitation hospital and 18 months thereafter. Persons with below-average executive function at baseline were more likely to develop depressive symptoms as they accumulated cerebrovascular risk factors. Among those with average or above executive function, the addition of risk factors did not lead to greater levels of depression. Thus, the causal risk factors were executive dysfunction with diabetes. Hypertension and heart disease were variable markers. However, the exposure rate of older primary care patients with diabetes, hypertension, and heart disease is substantial. In summary, big data can point to vulnerabilities and prevention should begin when there are depressive symptoms among the population with cognitive and physical disability and vascular diseases.

But, one can drill down even further. One example was with age-related macular degeneration. Rovner and Casten (2008) found a substantial reduction in the incidence of major depression and withdrawal from favored activities among older persons who received problem-solving therapy compared to those who received routine care and depression assessments only. Persons with the diagnosis of macular degeneration in one eye were approached for study participation at the time of diagnosis of macular degeneration in the second eye. Although the groups were nearly free of depressive symptoms at the onset of the study, those that reported depressive mood were more likely to develop a depressive disorder. Presence of any level of depressive mood increased the risk of developing a depressive disorder by 16-fold. The presence of insomnia doubled the risk also. Those randomized to problem-solving therapy received six 45- to 60-minute in-home sessions for 8 weeks. At 8 weeks, 23% of those in the routine care had diagnostic criteria for major depression compared to 11% in the intervention group. This held for up to six months. Although the preventative effect of problem-solving therapy on depressive mood was limited to the 2-month intervention, a reduction in disability carried over up to 6 months and fewer than 1 in 10 patients declined to complete the 8-week intervention.

Smit et al., (2006) followed a population-based cohort of more than 2,000 adults ages 55 to 85 over 3 years to detect the emergence of clinically significant depressive symptoms. Also collected was an array of demographic, functional, biomedical, and psychosocial characteristics thought to predict the incidence of depression among older community residents. This data allowed them to construct a parsimonious predictive model of risk indicators, including female gender, low education, 2 or more chronic conditions, functional limitations, small social network, and depressive symptoms at baseline. This model accounted for 80% of the risk for clinically significant depression. Depressive symptoms at baseline accounted for half the risk. In order to prevent the depressive symptoms from progressing into a clinically significant case, 16 people needed to be treated. However, when the group's baseline depressive symptoms were restricted to include only those with small social networks and functional limitations, the number needing to be treated dropped to 5. This clearly made this a practical intervention.

Precision medicine is alive and well. It needs application at later life. The Scylla of the false positive requires careful titration and the Charybdis of inaction demands to be challenged. The older adult is a panorama of variables related to potential problems; it only needs to be considered, deliberated on and handled. But there are issues: there are three fundamental reasons why precision medicine might not improve the health of populations. First, disease pathogenesis, especially for common non-communicable diseases, is extraordinarily complex. Abundant evidence has demonstrated both this and the association between the multiplicity of specific genes and conditions including obesity, hypertension, and cancers. Second, the central promise of precision medicine is the identification of predictors of disease that can help guide interventions. This may prove to be the case for some diseases, especially cancer, but unlikely to be the case for most complex diseases. Third, the assumed potential benefit of precision medicine is based on accurate and meaningful risk predictions that a disease in the population can be avoided or forestalled by large numbers of individuals who, when provided accurate data on risk prediction, will change their behavior to mitigate their personal risk. Although this may seem intuitively plausible, current data suggests that individuals do not change their behavior much, even when they become aware of being in a high risk group.

There is perhaps no more important area for the practice of precision medicine than drug usage. Recent trends in prescription drug

costs are revealing. After several years of modest growth prescription drug spending rose sharply in 2014. Costly new specialty drugs are the major driver in spending. Nearly one in four people in the United States taking prescription drugs reports difficulty in affording them. In fact, according to the US Census Bureau report, *An Aging World: 2015*, older adults take more over-the-counter meds and are the most likely to experience non-compliance and ER usage. This especially applies to the oldest old (Murabito et al., 2018). There is rampant polypharmacy then in all areas regarding older adults. According to Delafuente (2003), the probability that an older adult taking 5 medications will have an adverse drug interaction is more than 50%. When taking 7 medications, the probability is 100%. Worse, older adults with dementia generally receive more medications and are more likely to be taking potentially inappropriate medications than those without dementia. The judicious application of an algorithm for lifestyle, psychiatric complications, medication management, SES markers, environmental targets, and counseling can make a difference.

9. “Psycho”therapy

First, a lament. Since 1970 psychotherapy has been poorly served. No important fundamentally new developments or techniques in schools of therapy have arisen since the 1970s. In fact, psychotherapy in the generic sense has been largely medicalized. Its targets are now increasingly conceived as functions of individual minds or brains. Psychotherapy has turned into a technology modeled on medicine, which has limited cultural scope and conceptual depth. Any vigorous professional debate now seems to involve guild protections and competency-based tenets. The field has become more humble and intellectually less vigorous. Real innovation has virtually ceased. The field has become fragmented, specialized, and driven by the DSMs. In research, niche investigations are now directed at applications of extant methods to smaller and more trivial questions. What passes for external validation has morphed into clinical trial evaluations where TAU (treatment as usual) succumbs to the “new” therapy at an effect size of less than 0.3-0.7.

The complexity of science then as it applies to psychotherapy reflects a background of elemental human understanding that is prior to and underlies the scientific endeavor. Psychotherapy is an activity that is not theoretically grounded given a proper definition of theory, nor does it emanate from the comprehension of scientifically established underlying causal entities or explanatory mechanisms. What we call psychotherapy outcome research is from pharmaceutical research, which is on a par with

industrial product testing or educational program evaluation (Woolfolk, 2015). The diagnostic classification framework utilized was not predicated on highly developed science. The DSMs were theoretical and entirely based on observed co-occurring clusters of symptoms. They have never understood how the etiology or pathophysiology of anxiety differs from depression in the way that physicians understand diseases. We know very little about how the SSRIs affect the brain, much less than we know about how antibiotics affect bacteria. Research findings that address either the causal mechanisms underlying the disorders or the mechanisms by which drugs produce their effects are not there. For example, there are RCTs showing that a single disorder of major depression is ameliorated not only by SSRIs, but is also treated effectively by such an incredibly wide array of diverse medications that they cannot possibly all operate on the same mechanism. Some newer medications even decrease serotonin in the system. This lack of specificity is true for other psychiatric drugs as well. Some individual SSRIs seem to be somewhat effective in RCTs for a wide variety of disorders; it makes no sense to infer that all the different diagnoses are underlain by a single unitary pathophysiology. Some naturalistic investigations further call into question the common assumption that maintenance on antidepressants prevents relapse (see Woolfolk, 2015).

The question may occur as to why psychotherapy forms a part of the meta-trends. The reason is that the place where the action takes hold is human change. It is interpersonal, where a sense of being human can make a difference. Something has value but we have seen this at a cost to psycho-therapy itself. Something works. We believe that psychotherapy is the method of change for adults, older adults. It is a method of care that optimizes the chances of a better quality of life. We exist in the real “scientific world” of clinical care. We hold our nose and apply care modules. Within psychology there are several projects to codify empirical behavioral treatments for older adults. These have included the *Journals of Clinical Psychology: Science and Practice* (Scogin et al., 2005) and a special edition of *Psychology and Aging* (Ayers et al., 2007; Gallagher-Thompson, & Coon, 2007; Logsdon, McCurry, & Teri, 2007; Scogin, 2007; Yan & Scogin, 2007). These have been updated by Lichtenberg et al. (2014). Additionally, Mieskowski and Scogin (2014) have also updated the empirically supported treatment issues for depression, anxiety, insomnia, and dementia. Mieskowski and Scogin outlined the importance of late life anxiety, depression, insomnia, disruptive behaviors with individuals

with depression, caregiver interventions, and evidence-based strategies for enhancing memory and cognitive function.

There are two psychotherapy assists here. First, the theories of psychotherapy often anchored to CBT suggest several strategies that are connected involving the restructuring of maladaptive cognitive appraisals, changing action tendencies associated with problematic emotional experiences, preventing emotional avoidance, utilizing emotional exposure procedures, as well as emotion-focused interventions, which emphasize the functional nature of emotions, facilitating emotional approach and tolerance, and emotional regulation. The essence is to challenge cognition and to experience the emotion. This has applicability with regard to older adults. This suggests a transdiagnostic model. But modules can be identified and become the bread and butter of psychotherapy for older adults. Their tenets work but require a piecemeal understanding and application.

Second, common factors are the glue of the structure of the therapy itself. Common factors make a difference and perhaps result in change by themselves (Norcross & Rogan, 2015). Additionally, success depends on how the therapy is structured. The extent to which treatment matches shared social constructions about what it means to be remoralized or cured in a specific culture matters. Success depends on the conviction by the patient that the therapist cares and is competent. Makover (1992) long ago noted that continued alteration of the therapy leads to a better “approximation of truth.” Reviews of therapeutic processes and treatment outcomes consistently seem to reveal that the quality of the patient’s participation is most determinate of outcome. What the patient does in this setting and outside of the therapy determines how well the therapy will go. If the patient buys into the model (competent therapist, some change experienced, empathic reactions, and monitoring), change stands a good chance. Change can be acceptance of the status quo and a renewed commitment to living. Perhaps a more accepted view of the change agents that enhance the change process involves the following:

Strategies to Keep Patients on Track

- Educate about their role
- Incorporate patient’s preferences into decision making
- Increase investment in therapy
- Help plan for appropriate end game: Discussing endpoint increases commitment
- Provide education about patterns of change: Watch out for fast gains or temptations to bolt

- Strengthen early hope: Instill optimism
- Enhance motivation for treatment
- Use Motivational Interviewing
- Foster treatment alliance: Be empathic
- Use the team
- Discuss treatment progress: Use objective self reports

Jamie Chamberlin (Monitor on Psychology, April, 2015)

Therapy is people helping people with requisite belief systems, encouraged behaviors, and some rituals. As we will suggest again, techniques may actually be mostly placebo delivery devices. Interpersonal empathy is at the heart of the change in most conditions. It is co-constructed by mutual responses that activate similar cortical and subcortical neural circuits between clinicians and patients (Preston et al., 2002). It is also noteworthy that accurate empathy reflects and creates a synchronizing interpersonal neural biological attunement (Nummermaa et al., 2012). Functional magnetic resonance imaging has demonstrated that when verbal communication is accurately comprehended in speaker/listener dyads, it is correlated with the emergent neural coupling of spatial temporal brain activity (Stephens et al., 2010). Patients in pain who are treated by an empathic physician experience less pain (Jensen et al., 2014).

There is evidence then that psychotherapeutic talent, ability, and skill does exist and that it matters for effective treatment. Being empathic and human in a considered way does something to the professional/patient interaction that helps. Change in psychotherapy depends on empathic care as seen by the patient. Perhaps we need to develop a new intellectual framework for research and to conduct a re-examination of various conceptual levels at which causal mechanisms are conceived. Alternatives emphasize the human side of therapy and an ecological contextual understanding of human beings.

There are then two realities of psychotherapy. One is the real world. The other is the value of the common components of therapy.

Real World of therapy

1. Meds will be used on virtually all patients
2. After a course of medication, patients are as likely to be anxious or depressed as before – even after 3 years on meds.
3. During treatment for depression, anti-depressants work faster than psychotherapy until about 8-10 weeks.
4. During treatment medication plus psychotherapy probably works better than either one alone. This may be different for Panic Disorder.
5. Psychotherapy benefits continue after treatment:
6. Relapse Prevention Power: Data on added values of types of therapy are unknown

Alliance Importance

Research designs have failed to isolate and establish the relationship between which specific ingredient is necessary for therapeutic change.

1. Activate the alliance
2. Activate the patient
3. Activate the placebo
4. Activate the person of the therapist as more empathic

We note that medical necessity is typically divided into a number of aspects, including that the care indicated is appropriate, efficacious, effective, and efficient. These definitions change as a function of the patient and the problem. The work of the Sainsbury Centre is useful in its clear specification of a core set of competencies to improve current practice involving the value of individual planning from a recovery perspective (Shepherd et al., 2010). These include such things as effective communication, effective partnership with individuals, and exchange of information with individuals, the families, and other members, listening to individuals and families, and at the same time maximizing opportunities for them to be heard, and educating individuals and families about the role function and limitation of mental healthcare. These clearly involve developing working relationships and understanding the fundamental importance of relationships to social and psychological wellbeing. They maximize a cooperative alliance and partnership for the use of flexible responses and strategies. Connection matters.

Suggested Therapy Skills

- 1) good communication skills that include the Rogerian qualities of empathy, warmth, and non-judgmentalism,
- 2) the ability to make clients feel that they are understood by the therapist,
- 3) the capacity to form an effective working relationship with the client,
- 4) the ability to provide an explanation of the client's distress that is consistent with the interventions to be provided, acceptable to the client, and useful in the provision of therapeutic benefit,
- 5) the ability to develop and articulate a plan of treatment that is consistent with how the client's problems are explained and with the conduct that promotes health,
- 6) the ability to inspire confidence in the client and to be persuasive in convincing,
- 7) the capacity to faithfully monitor the patient's progress,
- 8) flexibility with regard to treatment options,
- 9) the capacity and willingness to confront difficult material in the therapy, even material that may be especially upsetting to the client,
- 10) the ability to maintain client morale and motivation given the impediments and effective treatment that arise during therapy,
- 11) the broad awareness of the client's and the therapist's personal characteristics' contextual grounds, for example ethnicity, social economic status, physical health, and how these factors directly and interactively affect the capacity of therapeutic change,
- 12) the judicious management of interjecting the therapist's own reactions to the client to ensure that these reactions can be rationally expected to have a beneficial effect,
- 13) being well informed and staying abreast of the latest research on biological, social, and psychological bases of the client's problem, and
- 14) improving one's therapeutic skills, coupled with the ability to obtain usual feedback, to self-reflect, and to modify one's conduct in therapy based on self-reflections.

Finally, the stages of change model was discussed and provides a model for overall care. It involves engagement, persuasion, and then active treatment and relapse prevention protocols. A stages of recovery model is implied, meaning that initially the individual is dependent on the system and disability systems and unaware of the potential for their own recovery. Essentially the individual who is still dependent on the

mental health and disability system becomes more aware of possibilities. Eventually the individual is not dependent on the mental health system or a disability system and is able to make choices about his or her own life and aware of additional opportunities.

One last word about the construct rehabilitation. This is what often occurs to older adults and has several definitions. It is a messy construct that is an over-arching philosophical approach to treatment. Rehabilitation is a more patient-centered collaborative process that is multi-modal, holistic, ecologically relevant, and always situated within the person's own goals. The holistic approach follows the approach of other clinician researchers in both acquired brain injury (Cicerone et al., 2008) and in older adulthood (Huckans et al., 2013). This also means being transparent at every step of the process about how the problem is being conceptualized, what the recommended course of intervention is, and how the intervention is expected to impact on a client's everyday function. We will also address this issue later but want to open the construct to interventions other than pure rehabilitation in the context of brain trauma (Tuokko & Smart, 2018).

10. Purpose in Life

How one assesses one's life counts, and counts a lot for life quality. Considering one's life to be meaningful is associated with a multitude of very positive things. Self-reports on meaningfulness are associated with higher quality of life, especially with age, superior self-reported health, and a decrease in mortality. Meaning in life predicts slower age-related cognitive decline and a decreased risk for AD (Hooker, Masters, & Park, 2018). Meaning in life is associated with a lower incidence of psychological disorders and suicidal ideation. Those who report their lives as meaningful are more likely to rely on adaptive coping techniques. Meaning in life itself has many meanings but seems always to involve the concept of purpose and that meaning in life matters as one possesses a sense of significance (Boyle et al., 2010). Meaning in life is also associated with social connection, positive mood, an environmental pattern that is acceptable, and coherence, as well as several cognitive constructs. Fortunately, from large representative samples and bodies of research, using different definitions of meaning in life, the conclusion that life is rated as meaningful is strongly supported, especially as one gets older. It might even be that meaning in life is essential to survival in the same way as sunlight or calcium.

Almost every problem brought to treatment has an aspect concerning meaning in life. We are always assimilating and accommodating stress and challenges: our baseline sense of meaning is reconfigured often. Therapists often use a form of dereflection, helping patients focus less on themselves and focus on higher level goals such as helping others. Being socially connected is very much a part of this. People are redirected to what is their passion and what nurtures them. In 2010, Wong, a psychologist at Trent University in Ontario, Canada (DeAngelis, 2018), developed a CBT program based on positive psychology and meaning. The acronym is PURE (purpose, understanding, responsibility, and enjoyment form the structure). The full program directs people to assume personal responsibility and embrace positive attitudes about themselves. They develop a personal legacy project to address what is most meaningful to them. It entails a “double vision” where they are in the moment and directed to future ideals.

For aging issues, this is paramount. Subjective age is one part. A younger subjective age has been shown to contribute to positive personality development and more positive expectations of better actual cognitive functioning in older age (Kornadt, Hess, Voss, & Rothermund, 2018). Subjective age also predicts better memory performance, as well as slower decline in performance. Although perceived health has been tied to both subjective age and cognitive functioning, only a few studies have examined the relationship between subjective age and cognitive performance. In one such study, the MIDUS study, participants who reported younger subjective age performed better on two cognitive factors 10 years later (Stephan et al., 2014). In that study subjective age was found to predict cognitive performance above and beyond chronological age, as well as the participants' health as measured by chronic disease burden. How one perceives self matters.

Noted in chapter 1 was the notion of flourishing. This occurs when the individual is optimally happy with positive emotions suffusing meaning in life. An abundance of research in the areas of wellbeing, such as happiness, positive emotions, strength, optimism, hope, flow, mindfulness, love, wisdom, courage, creativity, authenticity, motivation, and goals, has been evaluated. The estimation is that the equation for happiness involves roughly 40% voluntary control. Seligman noted that happiness is involved in pleasure, engagement, and meaning itself, pleasure being a feel-good factor, engagement meaning the depth of involvement, and meaning being the purpose of what is being done.

Psychological wellbeing is entrenched in several constructs that add to life quality. Happiness is only marginally a function of things like wealth, love and connection, education, work, youth, physical wellbeing, beauty, spirituality, living in a better climate, health, and children. Most happiness is internally developed, involving an internal simulator that allows the person to be happy. We create our own happiness internally. Barbara Fredrickson developed a broaden and build theory in which thinking over time accumulates to build multiple resources and support to our wellbeing. Joy leads to being urged to play; interest causes the desire to seek new information; pride makes you think big; elevation inspires you to feel better; and love makes you want to share and explore. A construct of meaning has also been much involved with issues of savoring, gratitude, meditation, learned optimism, resilience, and positive connections, as well as a sense of vitality. The person develops a sense of strength from strength and is able to respond positively to his or her environment. This involves training that is attached to any one of these constructs.

Resilience is another related construct; it allows the person to have a sense of emotional awareness, impulse control, optimism, causal analysis, thinking, empathy, and self-sufficiency, as well as reaching out. Resilience appears to have an important role in shaping whether or not people recover from adversity and sustain healthy growth and functioning at later life. High levels of wellbeing protect against elevated levels of inflammation, decrease the likelihood of disability and early mortality, and help older people manage the negative impact of health changes. Factors such as mastering and self-efficacy are negatively correlated with increased ADL and IADL limitations. Proactive coping has an impact on disability for the better and self-efficacy is associated with increased ability. People with higher levels of resilience appear to suffer from fewer ADL limitations and have improved physical functioning. Perhaps this is partially related to the fact that people who are more resilient also engage in physical activity.

Purpose in Life (PIL) also leads to life satisfaction. Although life satisfaction is typically considered relatively consistent across time, it may change in response to life circumstances, such as divorce or unemployment. Some people may adapt more readily to new situations, and thus appear to have relatively stable life satisfaction. Others may not adapt as quickly. All things considered, how successful one is in life might depend on how one views life satisfaction. Over the course of several studies, researchers seem to have learned that as participants' life satisfaction increases, the risk of mortality is reduced by a considerable amount, as much as 20%. PIL orchestrates these reactions in life.

This un-biological construct seems to benefit in ways not expected. Individuals who report greater PIL in their lives appear to be less likely to develop diseases, especially AD or its precursor, MCI. Participants' PIL can be measured by the level of agreement with such statements as "I see a good life; I can think of what I have done in the past and what I hope to do in the future," or "I have sense of direction and purpose in my life." After an average of four years and a maximum of seven years of annual follow-up clinical evaluations, 155 of 951 participants developed AD. Controlling for other related factors, greater PIL was associated with substantially reduced risk in developing AD, as well as a reduced risk of MCI and a slower rate of cognitive decline (Boyle et al., 2010). This outcome has been duplicated in the context of well-being, with PIL being related to reduced risk of developing AD (Sutin, Stephan, & Terracciano, 2018).

Of course this supports positive aging. Social interaction, being active, independent, having an optimistic outlook, and cognitive health is often the result of a PIL (Sutin et al., 2018; Windsor, Curtis, & Luszcz, 2015). Even holistic wellness is in play. Existential success is a form of PIL; existential despair (good things do not happen to me; physical health has restricted me) is its failure (Foster et al., 2015).

Religion is one way to establish meaning in life. The literature distinguishes between three types of religiosity; organizational, non-organizational, and intrinsic. Organizational religiosity typically involves public or group activities and is most commonly measured by one's religious service attendance. Non-organizational religiosity by contrast is more private and typically occurs on the person's own time alone, encompassing activities such as reading religious text, praying, and meditating. Intrinsic religiosity is concerned with individual subjective meaning of religiosity and how religious beliefs affect everyday life. Research demonstrates that religious involvement of any type benefits clinically depressed individuals. Depressed symptoms have been shown to decrease across time in persons engaged in organizational religiosity. Ronneberg, Miller, Dugan, and Porell (2016) found that organizational and non-organizational forms of religiosity affect depression outcomes in different circumstances. Important strategies to prevent and relieve depression among older adults may include improving access and transportation to places of worship among those interested in attending services and facilitating discussions about religious activities.

Despite its stress-buffering implications, linkages between religious attendance and biological weathering remain unexplored. Bio-

demographic literature suggests that weathering pressures on the three linked physiological subsystems of aging, inflammatory, metabolic, and cardiovascular, can be influenced. Emerging literature in the anthropology and neuropsychology of religious literature also suggest that emotionally charged group activities may also be beneficial. Starting with the work of Pargament (2002), scholars have increasingly conceptualized broad benefits arising from religion and stress mitigating effects. Those studies link a strong sense of divine control with lower psychological distress among older Americans and religious attendance with more self-reported tranquility. Such engagement may also foster stress-buffering network connections with multiple studies indicating that individuals who attend religious services at least once a week receive more social and emotional support. This church-based social capital may also enhance psychological resilience and coping capacity in the face of life challenges. This is especially true in late life when individuals suffer a generalized loss of social and physical assets.

Religion, spirituality, and psychotherapy do intermingle. There are several therapies involving meaning-centered counseling, spiritually sensitive psychotherapy, religious accommodative therapy, value-based psychological treatment, and religiously integrative psychotherapy. There is a sense in positive psychology with the recognition that there is a limit to how much a person can control. It is this area beyond one's control that lends itself to spirituality. This is PIL.

Regardless, there seems to be a feedback loop between social behaviors and personal beliefs (PIL) in which an accurate copying of others' behavior selects for better cognitive skills and bigger empathic reactions. This process leads to enhanced social behaviors and technical skills and better environments. This is a virtuous circle that builds on itself. Our ability to think, learn, and control our lives is natural but our overall PIL is a force that is different from all other animals and becomes reified and even directive in its development. It needs to be accounted for and studied.

We add one other variable, personality. Personality is the scaffold that provides the filter for all of this to happen. This is attached to meaning (PIL) and gives it form. Somewhere below the person coping skills and any Axis I problem personality provides the structure for the form of adaptation. It is the central determinant of dealing with life, of a person's vision, and of meaning in life itself. Importantly, as people age, the trait characteristics are influenced and assist in the later lifespan issues. Personality is the most efficient and economical path to an understandable and human template for both clinical reasons (treatment) and human reasons (meaning making) (McAdams, 2006). Personality may even have

life-long pathways of influence on risk of dementia and MCI. Individual differences in personality traits, such as openness, conscientiousness, neuroticism, and self-efficacy beliefs, impact cognitive intervention effectiveness in both younger and older adults. This applies to depression, anxiety, and health issues. Older adults are less neurotic, less social, and more withdrawn but more altruistic and empathic, for example. We address this in chapter 11.

Conclusion

The biopsychosocial diathesis stress model posits that there are certain interconnected biological, psychological, and social factors that can affect an individual's predisposition to mental health problems, say depression. These include factors that serve either as potential protective factors that can act as buffers against risk factors leading to problems. Some individuals are more vulnerable to problems due to biological concerns, somatic issues, psychological markers, and social influences. They require more coping help in the form of lifestyle markers and structural assists in the form of health, education, and good habits. The emerging picture of our human evolution is that we are creatures largely of our own making. But some must work harder. The distinctive features of humanity are our intelligence, creativity, and language, as well as ecological and environmental success. These are a function of adaptations based on trends, some of which we have discussed here. We are above all culturally and genetically predisposed to grow and behave in better directions (with some noise). Regardless, Ockham's Razor is a misnomer: rarely is their one cause for anything, certainly when psychopathology is concerned.

These metatrends make a difference in the case formulation of the identified patient and his/her milieu and life. We have lamented that, compared to other age groups, older adults have the highest rates of doctor visits, hospital stays, and prescription medication usage. Left unchecked, healthcare expenditures will likely rise from the current level of ~15% to 29% of gross domestic product (GDP) by 2040. We have suggested that we are subject to influences of our behavior and of our culture. Internally and externally we are constantly making sense of our life, our health, and our happiness. Self-management is important in healthcare. Symptoms and changes in function and deviations from the underlying image or self-prototype are the subjective online cues that are the targets of much of self-management.

Intellectual decline of the aged more than a solitary cause - Ockham's razor

"With all things being equal, the simplest explanation tends to be the right one."
(a 14th century philosophical argument for the existence of God as the origin of all causality)

By extension the traditional unitary thought process employed by physicians to explain medical maladies.



William of Ockham (ca. 1287–1347)

In the Common-Sense Model of Leventhal et al. (2015), the meaning assigned to symptoms is a product of a process in which symptoms are matched and linked to one another, underlying the prototype or model of the illness. The meaning created by this match activates expectations as to how a symptom is judged and the intervention will affect the underlying condition. The hypothesis that symptoms and functional changes are mapped or linked to prototypes is a central assumption of the Common-Sense Model. Indeed, mapping is an active process that is both intrapsychic and interpersonal. It constitutes our "prototype checks," checks that we make continually to monitor somatic and functional experience consciously or unconsciously, comparing these experiences with underlying prototypes both of the self and of specific illnesses. This is the essence of being human. Older age is one nice place and time to see this.

The meta-trends then are biopsychosocial in the truest sense. They play out in all cases of older adults. The instances and nuances of problems are unreasonably high in number. Focusing just on depression we see that the ability to reduce it is woefully lacking for current antidepressant treatments because of the virtually infinite person-markers that influence its etiology and expression. Depression is clearly a heterogeneous disease. And, there are significant differences in response to treatments and to side effects among classes of antidepressants, as well as among the individual

drugs of the same class. Older adults are very individual and very complex. They are also understandable and changeable.

References

- Agrigoroaei, S., & Lachman, M. E. (2011). Cognitive functioning in midlife and old age: combined effects of psychosocial and behavioral factors. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 66(suppl_1), i130-i140.
- Alzheimer's Association International Conference (AAIC) 2017. Abstract 14340 (exercise and amyloid in autosomal-dominant AD), presented July 18, 2017; abstract 16680 (exercise and tau), presented July 16, 2017; abstract 19051 (network analysis), presented July 17, 2017.
- American Psychological Association. (2014). Guidelines for psychological practice with older adults. *American Psychologist*, 69(1), 34-65.
- Andrasik, F., Goodie, J.L., & Peterson, A.L. (Eds.). (2015). *Biopsychosocial assessment in clinical health psychology*. New York, NY: Guilford Press.
- Ayers, C.R., Sorrell, J.T., Thorp, S.R., & Wetherell, J.L. (2007). Evidence-based psychological treatments for late-life anxiety. *Psychology and Aging*, 22(1), 8-17.
- Baker, L.D. et al. (2015). Aerobic exercise reduces phosphorylated tau protein in cerebrospinal fluid in older adults with mild cognitive impairment. *Alzheimer's & Dementia*, 11(7 Supplement), P324.
- Belar, C.D. and Deardorff, W.W. (2009). *Clinical health psychology in medical Settings: A practitioner's guidebook* (2nd ed.). Washington, D.C.: American Psychological Association.
- Boyle, P. A., Buchman, A. S., Barnes, L. L., & Bennett, D. A. (2010). Effect of a purpose in life on risk of incident Alzheimer disease and mild cognitive impairment in community-dwelling older persons. *Archives of General Psychiatry*, 67, 304-310.
<http://dx.doi.org/10.1001/archgenpsychiatry.2009.208>
- Brown, B. M., Sohrabi, H. R., Taddei, K., Gardener, S. L., Rainey-Smith, S. R., Peiffer, J. J., ... & Erickson, K. I. (2017). Habitual exercise levels are associated with cerebral amyloid load in presymptomatic autosomal dominant Alzheimer's disease. *Alzheimer's & dementia: the Journal of the Alzheimer's Association*, 13(11), 1197-1206.
- Cannon, K., Shaw, M., & Zuniga, M. (2006). Potentially inappropriate medication use in elderly patients receiving home health care: A retrospective data analysis. *American Journal of Geriatric Pharmacotherapy*, 4, 134-143.

- Cai, Z. et al. (2015). Cerebral small vessel disease and Alzheimer's disease. *Clinical Interventions in Aging*, 10, 1695-1704.
- Chandler, M. & Shandera-Ochsner, A. (2018). Memory compensation in mild cognitive impairment and dementia. In G. Smith & S. Farias (Eds) *APA handbook of Dementia* (pp 455-470). American Psychological Association: Washington, DC.
- Cheng, S. (2014). Double compression: A vision for compressing morbidity and caregiving in dementia. *The Gerontologist*, 54(6), 901-908.
- Cho, J., Martin, P., & Poon, L. W. (2013). Age group differences in positive and negative affect among oldest-old adults: Findings from the Georgia Centenarian Study. *The International Journal of Aging and Human Development*, 77(4), 261-288.
- Chorpita, B. F., Miranda, J., & Bernstein, A. (2011). Creating public health policy: The dissemination of evidencebased psychological interventions. *The Oxford handbook of clinical psychology*, 211-226.
- Cicerone, K. D., Mott, T., Azulay, J., Sharlow-Galella, M. A., Ellmo, W. J., Paradise, S., & Friel, J. C. (2008). A randomized controlled trial of holistic neuropsychologic rehabilitation after traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, 89 (12), 2239-2249.
- Clay, E., Khemiri, A., Ruby, J., Zah, V., & Aballea, S. (2013). Impact of treatment persistence on health care charges among opioid-dependent patients treated with buprenorphine/naloxone: 2006-2012 insurance claims retrospective analysis in the United States. *Value in Health*, 16(3), A68.
- DeAngelis, T. (2018) In search of meaning. *Monitor on Psychology*, October, 2018, 38-45>
- Delafuente, J.C. (2003). Understanding and preventing drug interactions in elderly patients. *Critical Reviews in Oncology/Hematology*, 48, 133-143.
- DeVol, R., & Bedroussian, A. (2007). An Unhealthy America. *The Economic Burden of Chronic Disease*, 2007.
- Foster, T., Galjour, C., & Spengel, S. (2015). Investigating Holistic Wellness Dimensions During Older Adulthood: A Factor Analytic Study. *Journal of Adult Development*, 22(4), 239-247.
- Gatz, M. et al. (2005). Complete ascertainment of dementia in the Swedish Twin Registry: the HARMONY study. *Neurobiology of Aging*, 26(4), 439-447.
- Gallagher-Thompson, D. & Coon, D.W. (2007). Evidence-based psychological treatments for distress in family caregivers of older adults. *Psychology and Aging*, 22(1), 37-51.

- Hagger-Johnson, G.E., Schickle, D.A., Deary, I.J., & Roberts, B.A. (2010). Direct and indirect pathways connecting cognitive ability with cardiovascular disease risk: Socioeconomic status and multiple health behaviors. *Psychosomatic Medicine*, 72(8), 777-785.
- Hammes, J., van Eimeren, T., & Drzezga, A. (2016). Differential contributions of Amyloid and Tau burden to Neurodegeneration in Alzheimer's Disease: A multimodal in vivo PET study. *Journal of Nuclear Medicine*, 57(supplement 2), 124-124.
- Hampstead, B., Briceno, E., & Garcia, S. (2018). Evidence supporting common cognitive rehabilitation techniques in cognitively symptomatic older adults. In G. Smith & S. Farias (Eds) *APA handbook of Dementia* (pp 433-453). American Psychological Association: Washington, DC.
- Hanlon, J. T., Schmader, K. E., Ruby, C. M., & Weinberger, M. (2001). Suboptimal prescribing in older inpatients and outpatients. *Journal of the American Geriatrics Society*, 49(2), 200-209.
- Harvey, S. B., Overland, S., Hatch, S. L., Wessely, S., Mykletun, A., & Hotopf, M. (2018). Exercise and the prevention of depression: Results of the HUNT cohort study. *American Journal of Psychiatry*, 175, 28-36.
- Hooker, S. A., Masters, K. S., & Park, C. L. (2018). A meaningful life is a healthy life: A conceptual model linking meaning and meaning salience to health. *Review of General Psychology*, 22(1), 11-24. <http://doi.org/10.1037/gpr0000115>
- Huckans, M., Hutson, L., Twamley, E., Jak, A., Kaye, J., & Storzbach, D. (2013). Efficacy of cognitive rehabilitation therapies for mild cognitive impairment (MCI) in older adults: Working toward a theoretical model and evidence-based interventions. *Neuropsychological Review*, 23, 63-80.
- Huffman, K. (2012). The Developing, Aging Neocortex: How Genetics and Epigenetics Influence Early Developmental Patterning and Age-Related Change. *Frontiers in Genetics*, 3, 212.
- Hyer, L. (2014). *Psychological treatment of older adults: A holistic model*. Springer Publishing Company.
- Jak, A. J., Seelye, A. M., & Jurick, S. M. (2013). Crosswords to Computers: A Critical Review of Popular Approaches to Cognitive Enhancement. *NEUROPSYCHOLOGY REVIEW*, 23(1), 13-26. <https://doi-org.medlib-proxy.mercer.edu/10.1007/s11065-013-9226-5>
- Jensen, K.B. et al. (2014). Sharing pain and relief: Neural correlates of physicians during treatment of patients. *Molecular Psychiatry*, 19, 392-398.

- Jongstra, S., Harrison, J. K., Quinn, T. J., & Richard, E. (2015). Antihypertensive withdrawal for the prevention of cognitive decline. *Cochrane Database of Systematic Reviews*, 11.
- Katzman, R., Aronson, M., Fuld, P., Kawas, C., Brown, F., Morgenstern, H., Frishman, W., Gidez, L., Eder, H., & Choi Ph, W.L. (1989). Development of dementing illnesses in an 80-year-old volunteer cohort. *Annals of Neurology*, 25(4), 317-324.
- Kirton, J.W., Resnick, S.M., Davatzikos, C., Kraut, M.A., & Dotson, V.M. (2014). Depressive symptoms, symptom dimensions, and white matter lesion volume in older adults: A longitudinal study.
- Kornadt, A. E., Hess, T. M., Voss, P., & Rothermund, K. (n.d.). Subjective Age Across the Life Span: A Differentiated, Longitudinal Approach. *JOURNALS OF GERONTOLOGY SERIES B-PSYCHOLOGICAL SCIENCES AND SOCIAL SCIENCES*, 73(5), 767-777. <https://doi-org.medlib-proxy.mercer.edu/10.1093/geronb/gbw072>
- Laufer, Y., Dar, G., & Kodesh, E. (2014). Does a Wii-based exercise program enhance balance control of independently functioning older adults? A systematic review. *Clinical Interventions in Aging*, 9, 1803-1813.
- Leventhal, H., Phillips, L.A., & Burns, E. (2016). The Common-Sense Model of Self-Regulation (CSM): a dynamic framework for understanding illness self-management. *Journal of Behavioral Medicine*, 39(6), 935-946.
- Lichtenberg, B. T. Mast, B. Carpenter, & Wetherell (2015). *APA handbook of clinical geropsychology, Vol. 2: Assessment, treatment, and issues of later life*. Washington: American Psychological Association. Retrieved from <http://content.apa.org/books/14459-013>
- Lin, F. et al. (2014). Fatigability disrupts cognitive processes' regulation of inflammatory reactivity in old age. *American Journal of Geriatric Psychiatry*, 22(12), 1544-1554.
- Lockhart, S & DeCarli, C. (2014). Structural Imaging Measures of Brain Aging, *Neuropsychol Rev*. 2014 Sep; 24(3): 271-289.
- Logsdon, R.G., McCurry, S.M., & Teri, L. (2007). Evidence-based psychological treatments for disruptive behaviors in individuals with dementia. *Psychology and Aging*, 22(1), 28-36.
- Lojo-Seoane, C., Facal, D., Guàrdia-Blmos, J., & Juncos-Rabadán, J. (2014). Structural model for estimating the influence of cognitive reserve on cognitive performance in adults with subjective memory complaints. *Archives of Clinical Neuropsychology*, 29(3), 245-255.
- Ludvigsson, M., Milberg, A., Marcusson, J., & Wressle, E. (2014). Normal aging or depression? A qualitative study on the differences

- between subsyndromal depression and depression in very old people. *The Gerontologist*, 55(5), 760-769.
- Lyubomirski, S. (2008). *The how of happiness. A scientific approach to getting the life you want.* The Penguin Press, London.
- Makover, R. B. (1992). Training psychotherapists in hierarchical treatment planning. *The Journal of psychotherapy practice and research*, 1(4), 337.
- Marchant, N. L., Reed, B. R., Sanossian, N., Madison, C. M., Kriger, S., Dhada, R., ... & Chui, H. C. (2013). The aging brain and cognition: contribution of vascular injury and β to mild cognitive dysfunction. *JAMA neurology*, 70(4), 488-495.
- Mast, B.T., Yochim, B., MacNeill, S.E., & Lichtenberg, P.A. (2004). Risk factors for geriatric depression: The importance of executive functioning within the vascular depression hypothesis. *Journal of Gerontology Series A: Biological Sciences and Medical Sciences*, 59(12), 1290-1294.
- McAdams, D. P., & Pals, J. L. (2006). A new Big Five: Fundamental principles for an integrative science of personality. *American Psychologist*, 61(3), 204-217. doi:10.1037/0003-066X.61.3.204
- McGinnis, J.M., Williams-Russo, P., & Knickman, J.R. (2002). The case for more active policy attention to health promotion. *Health Affairs*, 21(2), 78-93.
- Miebach, L., Wolfsgruber, S., Frommam, I., Buckley, R., and Wagner, M (2018). Different cognitive complaint profiles in memory clinic and depressive patients. volume 26, issue 4
- Mewton, L., Sachdev, P., Anderson, T., Sunderland, M., & Andrews, G. (2014). Demographic, clinical, and lifestyle correlates of subjective memory complaints in the Australian population. *The American Journal of Geriatric Psychiatry*, 22(11), 1222-1232.
- Mieskowski, L., & Scogin, F. (2014, November). Congruence of experience: Quality of relationship of caregivers and rural care recipients. *Gerontologist*, 54, 174-174.
- Munoz, R.F., Beardslee, W.R., & Leykin, Y. (2012). Major depression can be prevented. *American Psychologist*, 67(4), 258-295.
- Murabito, J., Zhao, Q., Larson, M., Rong, J., Lin, H., Benjamin, E., Levy, D., and Lunetta, K. (2018). Measures of biologic age in a community sample predict mortality and age-related disease: The Framingham Offspring Study. *The Journals of Gerontology: Series A Biological, Social, Medical Science*, 73(6), 757-762.

- Murphy, K. (2018). Multicomponent approaches to secondary prevention of dementia. In G. Smith & S. Farias (Eds) *APA handbook of Dementia* (pp 471-486). American Psychological Association: Washington, DC.
- Newsom, J. T., Mahan, T. L., Rook, K. S., & Krause, N. (2008). Stable negative social exchanges and health. *Health Psychology, 27*(1), 78.
- Niederehe, G. (2013). Personalizing treatment for late-life depression: Research approaches. *The American Journal of Geriatric Psychiatry, 21*(3), S27-S28.
- Norcross, J. C., & Rogan, J. D. (2013). Psychologists conducting psychotherapy in 2012: current practices and historical trends among Division 29 members. *Psychotherapy, 50*(4), 490.
- Nummenmaa, L., et al. (2012). Emotions promote social interaction by synchronizing brain activity across individuals. *Proceedings of the National Academy of Sciences of the United States of America, 109*(24), 9599-9604.
- 'Donnell, R. R., Cummings, N. A., & Cummings, J. L. (2014). Balancing value and cost. In *Handbook of Clinical Psychology in Medical Settings* (pp. 169-198). Springer New York.
- sborn, R., Moulds, D., Schneider, E. C., Doty, M. M., Squires, D., & Samak, D. ●. (2015). Primary care physicians in ten countries report challenges caring for patients with complex health needs. *Health Affairs, 34*(12), 2104-2112.
- Page, J. & Henry, D. (2000). Consumption of NSAIDs and the development of congestive heart failure in elderly patients: An unrecognized public health problem. *Archives of Internal Medicine, 160*, 777-784.
- Pargament, K. I. (2002). Is Religion Nothing But ...? Explaining Religion Versus Explaining Religion Away. *Psychological Inquiry, 13*(3), 239-244.
- Perissinotto, C. M., Cenzer, I. S., & Covinsky, K. E. (2012). Loneliness in older persons: a predictor of functional decline and death. *Archives of internal medicine, 172*(14), 1078-1084.
- Petersen, R.C., et al. (2001). Current concepts in mild cognitive impairment. *Archives of Neurology, 58*(12), 1985-1992.
- Proctor, E.K., Morrow-Howell, N.L., Dore, P., Wentz, J., Rubin, E.H., Thompson, S., Li, H. (2003). Comorbid medical conditions among depressed elderly patients discharged home after acute psychiatric care. *The American Journal of Geriatric Psychiatry, 11*(3), 329-338.
- Raber J, Huang Y, Ashford JW, *ApoE genotype accounts for the vast majority of AD risk and AD pathology* (May-Jun 2004)

- (www.ncbi.nlm.nih.gov/pubmed/15172743).*Id*
- Rajan, K. B., Herbert, L. E., Scherr, P. A., Mendes de Leon, C. F., & Evans, D. A. (2013). Disability in basic and instrumental activities of daily living is associated with faster rate of decline in cognitive function of older adults. *Journals of Gerontology Series A: Biological Science and Medical Science*, 68(5), 624-630.
- Reuter-Lorenz, P.A. & Park, D.C. (2010). Human neuroscience and the aging mind: A new look at old problems. *Journal of Gerontology Series B: Psychological Science and Social Science*, 65B(4): 405-415.
- Rolita, L., & Freedman, M. (2018). Over-the-counter medication use in older adults. *Journal of Gerontological Nursing*, 34, 8-17.
- Ronneberg, C.R., Miller, E.A., Dugan, E., & Porell, F. (2016). The protective effects of religiosity on depression: A 2-year prospective study. *The Gerontologist*, 56(3), 421-431.
- Routledge, P., Mahony, M., & Woodhouse, K. (2004). Adverse drug reactions in elderly patients. *British Journal of Clinical Pharmacology*, 57, 121-126.
- Rovner, B.W. & Casten, R.J. (2008). Preventing late-life depression in age-related macular degeneration. *American Journal of Geriatric Psychiatry*, 16(6), 454-459.
- Saplosky, R. (2018). The health-wealth gap. *Scientific American*, November, 63-67.
- Schmand, B. et al. (2010). Meta-analysis of CSF and MRI biomarkers for detecting preclinical Alzheimer's disease. *Psychological Medicine*, 40, 135-145.
- Schoeni, R., Freedman, Langa, K. (2018). Introduction to a supplement on population level trends in dementia: Causes, disparities, and projections. *J. Gerontol B Psychol Sci Soc*, 73, S1, S1-S9.
- Scogin, F. (2007). Introduction to the special section on evidence-based psychological treatments for older adults. *Psychology and Aging*, 22(1), 1-3.
- Scogin, F., Welsh, D., Hanson, A., Stump, J., & Coates, A. (2005). Evidence-based psychotherapies for depression in older adults. *Clinical Psychology: Science and Practice*, 12(3), 222-237.
- Shepherd, G., Boardman, J., & Burns, M. M. (2010). Implementing recovery: A methodology for organisational change. *Sainsbury Centre for Mental Health*, 1-19.
- Siegel, R. (2014). The science of mindfulness: A research-based path to well-being. Chantilly, VA. The Great Courses.

- Small, G.W. & Greenfield, S. (2015). Current and future treatments for Alzheimer disease. *The American Journal of Geriatric Psychiatry*, 23(11), 1101-1105.
- Smit, F., Ederveen, A., Cuijpers, P., Deeg, D., & Beelman, A. (2006). Opportunities for cost-effective prevention of late-life depression: An epidemiological approach. *Archives of General Psychiatry*, 63(3), 290-296.
- Stephan Y, Sutin AR, Terracciano A. (2014). Subjective age and personality development: A 10-year study. *Journal of Personality*, 83(2),142–154.
- Stephens, G.J., Silbert, L.J., & Hasson, U. (2010). Speaker-listener neural coupling underlies successful communication. *Proceedings of the National Academy of Sciences of the United States of America*, 107(32), 14425-14430.
- Stessman, J., Hammerman-Rozenberg, R., Cohen, A., Ein-Mor, E., & Jacobs, J. M. (2009). Physical activity, function, and longevity among the very old. *Archives of internal medicine*, 169(16), 1476-1483.
- Sutin, A. R., Stephan, Y., & Terracciano, A. (2018). Psychological well-being and risk of dementia. *International journal of Geriatric Psychiatry*, 743–747.
<https://doi-org.medlib-proxy.mercer.edu/10.1002/gps.4849>
- Tuokko, H. A., & Smart, C. M. (2018). *Neuropsychology of cognitive decline: A developmental approach to assessment and intervention*. New York, NY: The Guilford Press.
- Uchino, B. N., Cawthon, R. M., Smith, T. W., Light, K. C., McKenzie, J., Carlisle, M., ... & Bowen, K. (2012). Social relationships and health: Is feeling positive, negative, or both (ambivalent) about your social ties related to telomeres?. *Health Psychology*, 31(6), 789.
- Willis, S. (2018). Cognitive remediation approaches and dementia prevention: Findings from the ACTIVE trial and related research. In G. Smith & S. Farias (Eds) *APA handbook of Dementia* (pp 399-418). American Psychological Association: Washington, DC
- Wilson, R. S., Boyle, P. A., Yu, L., Barnes, L. L., Schneider, J. A., & Bennett, D. A. (2013). Life-span cognitive activity, neuropathologic burden, and cognitive aging. *Neurology*, 81(4), 314-321.
- Wilson, R.S., et al. (2012). The natural history of cognitive decline in Alzheimer's disease. *Psychology and Aging*, 27(4), 1008-1017.
- Windsor, T. D., Curtis, R. G., & Luszcz, M. A. (2015). Sense of Purpose as a Psychological Resource for Aging Well. *DEVELOPMENTAL PSYCHOLOGY*, 51(7), 975–986.
<https://doi-org.medlib-proxy.mercer.edu/10.1037/dev0000023>

- Woolfolk, R. L. (2015). *The value of psychotherapy: The talking cure in an age of clinical science*. Guilford Publications.
- Yaffe, K. et al. (2001). A prospective study of physical activity and cognitive decline in elderly women: Women who walk. *Archives of Internal Medicine*, 161(14), 1703-1708.
- Yon, A., & Scogin, F. (2007). Procedures for identifying evidence-based psychological treatment for older adults. *Psychology and Aging*, 22(1), 4-7.
- Zelinski, E. (2018). Does cognitive training reduce risk for dementia? In G. Smith & S. Farias (Eds) *APA handbook of Dementia* (pp 419-432). American Psychological Association: Washington, DC.
- Zissimopoulos, J., Tysinger, B., St Clair, P., & Crimmins, E. (2018) The impact of changes in population health and mortality of future prevalence in Alzheimer's disease and other dementias in the United States. *Gerontol B Psychol Sci*, 73, S1, S38-S47.

CHAPTER 3

ASSESSMENT

LEE HYER AND KRISTIN WAGNER

The purists echo that if it is not measured, it did not show change: The realists counter yes, but measure what and how. Assessment is the necessary staple of science that makes it a science. We have argued that the “just sufficient” data for older adults involves five areas, and is, well, sufficient. This suggests that each area requires some monitoring as well as a screen for reasonable convenience and time issues. This implies cut-points for each domain. The assessment of depression, anxiety, cognition, and health issues seem straight-forward. Life adjustment, not so much... How much of a low SES warrants an intervention requirement? For our system we have made the decision to divide the problems into tertiles – normal/mild, moderate, problem. Watch and Wait holds to the ideal that the patient needs careful evaluation, that the patient is not broken or a medical anomaly. At some point the patient becomes trapped in their own rigid patterns and of the vagaries of living. This occurs at later life modally. Each requires someone to figure out their unique pattern and address problems.

The Institute of Medicine’s landmark report in 2001, *Crossing the Quality Chasm: A New Health Care System for the 21st Century*, cited person-centeredness and control of health as primary aims of a transformed quality health care system (Wolfe, 2001). Disease management and health promotion are done in the form of a road map, not necessarily sequentially but logically and strategically. Assessment is implied. At late life, the melding of a commitment to person-centered care and the use of evidence-based practice is at issue. The fit between the individual at a particular point in time as judged by the participation and response of that person is critical for care. Again, the core problems of late life, we are arguing, are depression, anxiety, somatic and health issues, as well as cognitive decline and just living. The broadening of the full range of life’s problems is pictured in the context of these domains. These domains embrace a greater range of maintaining mechanisms (e.g., social withdrawal and avoidance,

negative views of self, cognitive distortions) that represent or foster some core psychopathological process baked into current DSM symptom clusters. There is then a broader scope.

Assessment is the only real marker of this process. It is critical and implies precision but its referent in real life is messy: Diagnoses are sloppy. The existence of symptoms alone does not necessarily indicate a disorder. We will all be depressed at one time or other in life. A judgement is made by a clinician that the response is excessive, disproportional to the actual loss or problem. In fact, it has been argued that the subjective meaning of the events and problems is critical; the elaborate set of references (derivatives) to the actual insult determines the designation of a disorder or not. Wakefield (1998) extended this and posited the phrase “harmful dysfunction” as a consideration in the determination of a diagnosis; harmful relating to the social values of the person and dysfunction relating to the failure of some internal mechanism to perform the natural function for which it was designed.

Bobbitt, Cate, Beardsley, Azocar, Sand McCulloch (2012) added something important here. They noted that quality assessment processes and outcomes measurement are now key features of the evolving system. We need to use assessment differently and well. There are now several national organizations that have disseminated standard quality measures, such as the National Quality Forum, The Joint Commission, and the National Committee for Quality Assurance. Importantly, the issue is quality improvement and some demonstration of treatment efficacy and efficiency as a measure of performance. In 2011, Health and Human Services included 10 principles in its strategy for this to occur. Among these were person-centeredness and quality improvement for health care providers to have access to necessary information. This has not been done in traditional clinical programs. It appears optimal for older adults.

Barkley (2014) places perhaps the best perspective on assessment: He reminds us of the vast complexity of the person. We cannot measure all important areas. This applies to all person factors but perhaps especially to cognition. Ardila (2013) also opines that the phenomenology of what we measure has altered over time in just about every cognitive domain (we no longer add and subtract, for example, but use calculators). And as we will show, older adults have problems. The way out of psychiatric and real life problems for older adults is a biopsychosocial assessment and intervention. In Hyer (2014) we articulated the need for the modified assessment model encompassing the five core domains. The fact that an older adult can be subsyndromally anxious and depressed, have a variant

of MCI, and have a poor lifestyle, inadequate sleep, and need for meals on wheels, is bigger than any one diagnosis or typical psychological model.

Assessment Complexity

The Truth must dazzle gradually

●r every man be blind

Emily Dickinson

Assessment needs reasonable anchoring beyond what we have been offered. It is not that we are sending a new call-to-arms. All testing for Mr. X applies to Mr. X and his specific issue; his age, his education, his marriage, his occupation or lack thereof, his place in the country, his comorbidities, his nuanced ethnicity... you get the picture. There are no valid norms then for anyone of us. But, if assessment means anything, it is part of a better person-picture, an improved view of certain complexity, and a series of targets that, with other data, apply to Mr. X in a way that we know more about him than otherwise. This is not science but heuristics. It is part of treatment, part of the unfolding story necessary for care but imperfect.

In this regard and from a broader perspective, Kotov, et al. (2017) assessed the hierarchical taxonomy of psychopathology (HiT●P) as a dimensional alternative to traditional nosologies. The HiT●P consortium brought together a group of clinical researchers who aimed to develop an empirically driven classification system based on advances in quantitative research on the organization of psychopathology. Primary objectives of the consortium are to integrate evidence generated by the research to date and produce a system that reflects a synthesis of existing studies. Importantly, the authors noted the faults of the traditional system. First, traditional systems consider all mental disorders to be categories, whereas the evidence suggests that continuum is a better marker for these entities. There is considerable loss of information. Second, traditional diagnoses generally show limited reliability. This has been shown time and time again. Third, many existing diagnoses are quite heterogeneous and encompass multiple pathological processes. Fourth, co-occurrences among mental disorders, often referred to as comorbidity, is very common in both clinical and community samples. Fifth, many patients fall short of the criteria of any disorder despite manifesting significant distress or impairment, clearly indicating the need for care. The DSM-5 addresses this problem by an “unspecified” category, but it falls short of clarity.

Small changes in diagnostic criteria can have dramatic effects on the number of patients meeting criteria. Reliability and validation for statistical instruments therefore are important. The agreement, for example, between the DSM-5 neurocognitive disorder and the DSM-IV criteria for dementia and international working group criteria for MCI often differ. There is a moderate correspondence between these different definitions across the continuum of cognitive impairment. The size of the Kappa statistics suggests that the correspondence is not anything close to high. The Kappa usually is around 0.6. An often repeated rule of thumb for researchers is that a Kappa of 0.4 to 0.6 may be considered. Perhaps this should be changed to 0.8 or higher. Statistical criteria for an acceptable assessment tool may not translate either. We should probably demand more from assessment instruments and batteries including a very high sensitivity for screening tests. The mistake of not identifying most positive cases is probably more grievous than falsely associating one who is not a true case, however regrettable. Regardless we have what Allen Francis calls a “false positive epidemic.” More on this below.

“DSM is 100% reliable and 0% valid. As long as the research community takes the DSM as the bible, we will never make progress... Biology has never read that book.”

Tom Insel, Head of NIMH

The medical assumption is often that “something is wrong with the person (patient) in front of me.” We make decisions based on failure. This person needs a diagnosis. In turn, for patients who score below norm, determinations are set in motion based on normative scores. The gold standard is to use methods that allow for less false positives and false negatives. Optimally regarding dementia, this is done in most psychological evaluations when the assessment adjusts for core person-centered factors like IQ, uses three or more scores, uses a measure of function, looks at biomarkers, and highlights factors in sleep, pain, inactivity and socialization.

For the typical older adult who has cognitive problems, there are five usual referral questions for cognitive problems: (1) normal aging versus dementia; (2) differentiating among dementias or focal brain impairment; (3) dementia versus cognitive deficit related to stress or depression/anxiety; (4) functional implications of established brain impairment; and (5) enhancing cognitive function in healthy or impaired aging. Once a decision to assess is made, the unfolding of the referral question can begin.

This thinking then is part of the complexity of the case. But these questions only set the table.

The most common method that psychologists use for clinical data collection is the unstructured interview, coupled with informal observation (Mash & Foster, 2001; Mash & Hunsley, 1993; Meyer, et al. 2001). As part of this, clinicians are mandated to follow up symptoms over time. This technique can be important in the diagnosis of more complex cases when it is difficult to determine whether patients have a primary cognitive disorder, primary mood disorder, or both. This method, however, can have significant limitations. Fennig and colleagues (1994) long ago showed that diagnoses derived from clinical interviews alone agreed only about 50% of the time with diagnoses derived from multi-method assessments. Logically, there are a number of shortcomings associated with unstructured, single method approaches to information gathering.

Problems with Single Method Approach

1. Reliance solely on the unstructured interview can mislead clinicians into overlooking potentially important areas of distress or dysfunction while focusing too much on others (e.g., the chief complaint).
2. The client himself/herself, who may be a poor historian, may have issues to hide from the assessor, or who has personality characteristics that bias his/her self-presentation.
3. If the clinician's therapy objectives are at odds with those of the client, or if the client lacks motivation for psychotherapy, the interview will almost invariably be unrevealing.
4. Certain neuropsychiatric conditions, such as anosognosia, amnesia or confabulation, paranoia, or delusions and other subtle psychotic symptoms, interfere with the accurate reporting of information. This is especially true with regard to elders who may present with occult cognitive problems that cloud or distort the history and clinical picture.
5. Using a semi-structured clinical interview alone (e.g., Structured Clinical Interview for DSM-IV Axis I Disorders (SCID); First, Spitzer, & Gibbon, 2004), can mislead the clinician into focusing too much on the yes/no response to the question at hand and ignoring clues that point to less obvious biopsychosocial inputs to the symptom picture.

Multi-method assessments, in contrast, enable the clinician to “deconstruct” the client in terms of cognitive/neuropsychological, personality, and behavioral/functional contributions to the chief complaint using standardized, quantifiable, norm-referenced tests coupled with self-report and input from significant others. There is also the need, however,

for care and accuracy when using multiple measures so that one does not miss the forest for the trees. In the main, tests are often sensitive but not very specific, so sound clinical judgment is vital in “reconstructing” the person from multiple sources of data. One must be especially prudent when assessing older adults because there often can be a serious disconnect between raters on target symptoms or behaviors, even when markers are quite specific. For example, when one compares depression ratings on the Geriatric Depression Scale (GDS) for a client with cognitive decline, clients and caregivers part company on just about every item. Similarly, in dementia, caregivers almost always overrate problems while patients generally underrate them (perhaps anosognosia). Regarding functional assessments, caregivers and clients also are at odds, with the caregiver typically reporting that the client is less functional than what the client perceives to be true.

As noted then, reliance on a single clinician using a single method to obtain information from the patient will lead to generally unreliable and erroneous understanding of the patient. For the treatment of older adults, mental and physical illness are conjoined. Pointedly, treatment for mental problems is less than ideal; for the most part treatment in general is less effective than younger groups. You can even say that current psychological treatment is inadequate. Current medicines for mental health are clearly inadequate. We have noted that current assessment addresses dichotomous problems but the best focus needs to be on continuous and individual symptoms.

Additionally, primary care is now the new psychiatric care. Professional silos are a problem: Teams are complex but necessary. They are “always” sloppy and poorly run! Current newer models of care (e.g., IMPACT, PEARLS) have been helpful, however. Public health models work well also. And, the clinician’s attitude and skills are critical. Given this complexity of input, we would like to suggest that valid assessment can make up for the inherent problems regarding its value. The dynamic interface between the patient, the idiosyncratic features notwithstanding, and the systems involved in care, can be negotiated reasonably with a broader view of assessment.

A word specific to psychological testing: Despite setbacks by critics (see Eisman et al. 2000; Kubiszyn et al., 2000; Turchik et al., 2007), the value of psychological testing has survived and more recently thrived in a context of multi-method assessment for diagnostic and treatment planning (Groth-Mamat, 2009). In fact, psychological assessment is as valid as most medical tests (Meyer, et al., 2001). Psychological assessment can enhance diagnostic musings. Importantly, the process of testing assists

treatment in several ways including: 1) the delineation of clinical symptomatology; 2) hypothesis testing and decision-making regarding differential diagnoses; 3) assisting in case formulation; 4) predicting a client's ability to participate as well as degree of participation in psychotherapy; 5) predicting healthcare utilization; 6) hypothesis testing for therapy impasses or looming therapy failure; 7) monitoring treatment effects over time; 8) the confirmation (or disconfirmation) of perceived psychotherapy outcomes; 9) improving prediction of relapse; and 10) enabling the clinician to respond to managed care and other external pressures (Antony & Barlow, 2011). These have special meaning for older adults.

Neuropsychology especially is noteworthy as it focuses on brain-behavior relationships. This is the province of older adults. Neuropsychological testing provides a snapshot of behavior. Most environmental tasks have multiple cognitive determinants, however. The ecological validity of the assessment is lacking. This includes veridicality (extent to which test results predict real-world performance) and authenticity (the topographical similarity of data collection method to a task in the free environment). In the past, lesion localization and more recently integrated subsystems of brain function have been identified and parsed, or scanned. Additionally, several factors complicate the relationship between neuropsychological assessment and everyday function. Most importantly, neuropsychological tests measure more than one cognitive function (e.g., Trails B). Additionally, the neuropsychological testing environment emphasizes optimal performance which often does not parallel the real world.

Neuropsychology's Lament

Neuropsychology is at a tipping point. It has developed from the position that localization was supreme. Most tests in neuropsychology were not developed for purposes to draw inferences about functioning based on observable behaviors. Now the brain connectome is paramount; functional connectivity outpaces the existence of a single anatomical location. Using modern informatics and aggregating cognitive data alongside biomarkers that underlie brain-behavior relationships seems to result in better outcomes.

In the real world area of testing, clinical neuropsychology has reached a point in its development where it is embracing newer measurement and technological advances. Changes are consistent with other clinical neuroscience areas like computational psychiatry. This may be mean nascent advancements as in NIH initiatives that endorse a process that

integrates neuroscience into behavioral and social sciences, transforms advances in measurement, uses digital intervention platforms, and fosters large scale population cohorts and data integration. In a sense neuropsychology has been limited by the current tools it uses to devise outcomes, outcomes that are not practically based or neurologically titrated (see Parsons & Duffield, 2019).

In 2006 Borsboom noted: “This may be the central problem of psychometrics: psychological theory does not motivate specific psychometric models. It does not say how theoretical attributes are structured, how observables are related to them, or what the functional form of that relation is. It is often silent even on whether that relation is directional and, if so, what its direction is. It only says that certain attributes and certain observables have something to do with each other. But that is not enough to build a measurement model.” (p.435)

Indeed developments in neuroscience and technologies such as fMRI have afforded real-time observations of brain function that are challenging the validity of standard neuropsychological models using traditional paper-and-pencil technologies. There appears to be a lack of specificity as neuropsychological deficits after brain injury rarely are confined to one type of processing. Also the same underlying injury can result in a variety of different symptoms. So, although the integration of imaging and neuropsychological methods have improved our understanding of brain functions, there are multiple ways that the same stimulus can be converted into the same output. Inter-subject variability exists also making firm static conclusions shaky.

In sum, the brain-behavior relationship needs more. Goals will differ depending on the issue, generally functional markers or identification of brain problems. Measurement is not precise. As in our domain areas, the isolation of a domain is quixotic as each domain is fluid and often variable. It is also highly interactive with other variables and is moderated by even more factors. Progress will most be activated when a “bottom up” approach allows data to speak for itself and outcomes seen in the context of validated markers. For now, lower level validation coefficients and adequate reliability markers will have to suffice.

Complexity seems to expand as need for precision rises. It is now recognized that a cognitive pattern (of memory, executive functioning, and information processing deficits) is characteristic of depression and occurs to a greater extent in people with clinical depression. As previously noted, one depressed person is not the same as one other depressed person. The different domains contaminate this. The relative contribution of different

domains of cognitive impairment, their relationship to each other, changes over time and in response to treatment, and what neurobiological changes underpin these cognitive deficits are considered (Thomas, 2012). Depressed people also are less likely to remit and more likely to remain significantly cognitively impaired after a period of antidepressant treatment. The assessment journey is just beginning, replete with faults but as necessary as science itself.

Overall Assessment Problems

“It is better to be roughly right than precisely wrong.”

– Sir Archie Cochrane

Evidence-Based Medicine (EBM) as an information framework, in that we start with what occurs most frequently in clinical settings. The classic EBM approach consists of a five-step process of developing a question using the populations-intervention-comparison-outcome (PICO) format, finding research that may answer the question, evaluating the research for validity, impact, and applicability, applying the information to clinical decision making, and periodically evaluating one’s effectiveness at performing the previous four steps. However, a number of individuals, including the developers of the EBM approach, have outlined its problems. The more common method of information management has been called “satisficing,” whereby busy clinicians will be satisfied with the information they have at hand, sacrificing quality for convenience. For example, internal medicine residents pursued only 30% of their questions during a typical office session and only pursued 70% when specifically given time during their office hours to answer the questions they developed. Instead of striving to find the most rigorous evidence, most full time clinicians report they do very little critical appraisal, instead relying on summaries and practice guidelines, regardless of whether these are evidence based, for information.

It is not the frequency of the clinical problem that decides the approach to the literature, it is several other factors: The clinician’s awareness that new information is available, the feeling that current options are unacceptable or didn’t work, the availability of familiar sources, the perceived likelihood that an answer can be found, the fear of liability if the correct approach is not followed, and the time available to search for the answer. In general, physicians pursue answers to only about a third of questions. Information management focuses on using currently available information tools to remain up to date with new valid information that is

relevant to the care of patients and is accessible while taking care of patients. These information tools can be divided into “foraging tools” that clinicians can use to be alerted to new, relevant, *and* valid information, and “hunting tools” that allow clinicians to find that information again when they need it. Both tools are required for effective practice. The best of these tools provide information that is filtered for relevance to clinical practice, is critically appraised for validity using evidence-based medicine (EBM) techniques, and is presented in a style that is easily grasped by busy clinicians, which greatly reduces the amount of work expended to obtain the best information.

The focus on information management grew out of frustration with the limited ability of EBM to meet the needs of clinicians in active practice. Information management focuses on the usefulness of information to patient care, defined as usefulness of any information source, relevance, validity, work, relevance of information defined in terms of its direct applicability to the care of patients and focuses on three qualifications:

- Does the information focus on outcomes patients care about? That is, will the information help clinicians assist patients to live longer lives, better lives, or both?
- Is the intervention or practice feasible and is the problem addressed common in one’s clinical practice?
- Would the information, if true, require a change in one’s clinical practice? The information should show that an intervention helps patients live longer and better, is feasible to implement, and would require a change in a clinician’s practice.

Fortunately, the issues of assessment are becoming more formalized by Medicare as well as professional organizations. The annual assessment as recommended by the Alzheimer’s Association for operationalizing the detection of cognitive impairment during the Medicare Annual Wellness Visit (AWV) in a primary care setting has been made clear (Cordell et al., 2013). The two-visit approach to evaluate suspected dementia in primary care is consistent with the two-step approach widely used in epidemiologic research on dementia. Regardless of the timing and setting, clinicians are encouraged to counsel patients to include an informant in the diagnostic process. Core evaluation procedures include patient completion and clinician review of a Health Risk Assessment (HRA) and use of a structured assessment tool. Patients with assessments that indicate cognitive impairment during the AWV should be further evaluated to determine appropriate diagnosis (e.g., MCI, Alzheimer’s disease) or to

identify other causes. Initiation of a full dementia evaluation is outside the scope of the AWW, but can occur in a separate visit either on the same day, during a newly scheduled visit, or through referral to a specialist. To date, there are few data to support or endorse this approach.

Decision Making

Our understanding of the components and processes of clinical decision-making is of course still in the earliest stages. We know that effective clinical decision-making requires more than clinical experience in individual therapeutic judgment. When reviewing the relationship between clinical experience and clinical outcomes, the conclusions of the major reviews are mixed at best. As noted, clinicians have not demonstrated high levels of reliability at the tasks of diagnosis, prediction, and case conceptualization (Garb, 2005). Clinical judgment alone seems vulnerable to the same sources of error that often distort ordinary human judgment, including confirmatory biases, self-enhancement bias, the availability bias, and greater emphasis on personal experience than general information. Clinical decision-making requires more than just experience. These include research findings, observation of the patient, assessment of etiology, theories considered credible, authors and trainers found to be compelling, sound education, and conversation with colleagues.

Decision theory is an important development that began as an attempt to deal with making decisions in situations of uncertainty, which has direct relevance to healthcare and mental health practice and training. At the basic person-level, there are two types of thinking that are called upon in making decisions. One is fast, intuitive and emotion-based; excellent when rapid responses are vital to outcome. The other is slow or lazy in that a great deal of cognitive effort is required in order to consider the information and determine probabilities of outcomes from various choices (Kahneman, 2011). Both the fast and slow systems of thinking must flexibly interact and can balance each other to maximize outcome. There are a number of biases/errors that occur without our knowledge when we rely too much on the fast response system. These errors can be avoided by developing knowledge of various types of biases that are common to problem-solving. Intuiting is a process of leading to the recognition or judgment that is arrived at rapidly without deliberative rational thought. This is difficult to articulate verbally and is based on a broad constellation of prior learnings and past experiences.

Tversky and Kahneman (1974) have led the way on dual process thinking highlighting heuristics and biases. Heuristics simplify judgment

and decision-making in that they entail mental shortcuts and rules of thumb. Dan Ariely (2010) indicated that we are all vulnerable to inherent bias generation errors. In fact, he noted that we are not only irrational, but we are predictably irrational. The mark of good decision-making is the ability to match systems 1, which is automatic processing, and system 2, deliberative processing, to their respective optimal context and to consciously blend them into overall decision-making. Explicit theories help but clinical judgment and decision is variable, extensive and profound. The clinician also needs to follow over the course of therapy reasonable tacit beliefs with concepts and propositions of sound explicit theory in guiding clinical judgments and decisions.

Always clinical decision-making involves forecasting outcomes in the face of uncertainty. Effective decision-making involves gathering relevant information, considering and evaluating alternatives, making judgments that are relatively free of biases, and appraising the outcomes of the decisions. What are the best elements in decision-making? These probably involve things like decisions that can be broken down into the possible actions, the possible events, and evaluations of the consequences of the possible actions. Elements of optimal decision-making include good data information, knowledge, and wisdom.

Perhaps there is no fool-proof way of avoiding errors. Accordingly, clinicians should familiarize themselves with the decision analytics and use these to enhance awareness of the decision-making traps. When making clinical decisions, the clinician should be sure to conceptualize problems in multiple ways. Importantly, the clinician should consider using existing algorithms when making clinical decisions by referring to extended literature. Also, decisions are a process and can be changed. Finally, the clinician needs to recognize that personal experience is anecdotal evidence and as substantial research has shown that the anecdotal evidence is not sufficient to ensure optimal decision-making. The principles of probability for complex decision-making are one's friend.

Evidence-based practice in psychology (EBPP) is a strong effort for the integration of the best available research and clinical expertise in the context of patient characteristics, culture, and preferences: What do the data say, what is the evidence, who is the person in front of me, and what are my skills to bring this about? The goal of the therapist's moment-to-moment judgments and decisions arrive from continuous relations between automatic and deliberative processing, drawing on both explicit and implicit theory. Several recommendations are made to reduce confusion. An essential goal is to keep the therapeutic process moving forward in a

constructive fashion. It also involves knowing that over the course of a given session, many clinical judgments and decisions are being made automatically and that, given the multiplicity of stimuli that must be processed and tasks that must be carried out under conditions of uncertainty and limited time, there can be no other way. Deliberative processing then is necessary, messy, and a necessary challenge.

In their book Stewart, Chambless, Stirman, and Shannon (2018) shared the results of a qualitative analysis of interviews with 25 psychologists in independent practice, with a focus on decision making with a selected patient. The authors endeavored to examine how clinical decision making intersected with the principles of evidence-based practice (EBP). Clinicians reported that diagnostic impressions were generally formulated through unstructured assessment rather than validated instruments, and that treatment selection was based on therapists' perceptions of a treatment's match with client characteristics. Therapists viewed CBT as appropriate for addressing symptoms but believed they needed to depart from CBT strategies to address underlying issues. Nonetheless, they often defined successes and failures in treatment in terms of symptoms. Overall, clinicians rarely mentioned utilization of research evidence for assessment or treatment selection and practice. Clinicians like redundancy and simplicity.

A principle-based approach is best served by therapeutic skills and values founded on the commitment to scientific-mindedness, critical thinking, an integrative capacity, and sensitivity to relational context. Therapy choices would be best served by an increased research focus on principles, underlying disorders, and change processes rather than continuing with our current focus on packages or techniques. Recent research that focuses directly on the measurement of underlying principles of change seems more on-point. Preliminary results have been promising showing substantial reductions in re-hospitalization rates, symptomatology, and personality dysfunction among patients with severe and comorbid problems that previously had not responded to standard treatment attempts. There is an observed linkage between the outcome and the use of interpersonal case formulations and interventions that enhance the patient's will to change. A primary rule for clinical decisions in good decision-making is that any intervention is acceptable so long as it matches the case conceptualization. This rule becomes an easy means of reflection, "does what I am doing make sense for this patient in terms of the case conceptualization."

Always, the mandate of professional practice of monitoring case progress is less than adequately validated. Langkass, Wampold, and

Hoffart (2018) noted that patient-focused attempts have been laudatory but fall short of optimal science. Clinical cutoff scores, clinical significance, and expected treatment responses often differ. One value-added movement, however, that improves decision making is multiple feedback systems. There are many (see Langlass et al., 2018). One provided by Bickman, Kelley, and Athay (2012) describes bringing systematic measurement tools and clinically relevant and real-time feedback into decision-making. Measurement feedback systems integrate reliable and valid measurement of relevant client factors, clinical processes, progress, and client improvement with clinically useful feedback that can easily be adopted into short and long-term clinical decision-making. These authors have suggested that there are principles to form the theoretical foundation for the effectiveness of multiple feedback systems. Lambert, Harmon, Slade, Whipple, and Hawkins (2005) also found that the usage of measurement feedback systems, such as the Outcome Questionnaire-45, where clinicians receive feedback, had clients who decreased in total negative outcomes from 21% to 5%. Lambert et al. concluded that clients of clinicians who are not alerted to negative responses have unacceptably high rates of deterioration.

Diagnostic and Statistical Manual of Mental Disorders (DSM): The DSM is a guide and allows the health care provider to approximate truth with one of the better models of care. For this to occur, the worldview of the patient must supersede everything else in assessment and care. Clinicians learn quickly that any person is more complex than their diagnosis. This complex person-based data are beyond the value and help of the DSM. The trilogy of competence in assessment and treatment include empirically supported research, the individual expertise of the clinician, and the values of the patient. The complexity of the person exceeds that trilogy. A value-driven pattern of clinical practice attempts to integrate “best research” derived from the study of populations to inform clinical decisions about individuals within the context of his/her expertise and individual patient values with the goal of maximizing clinical outcomes and quality of life for the patient in a cost-effective manner while addressing the concerns and goals of the patient’s needs and wishes (Chelune, 2010). This is a nice goal.

The word “diagnostic” in DSM is descriptive of its purpose to provide the best guidance currently available to identify those with the disorder. More puzzling is the word “statistical” in the DSM. The DSM-1 and the DSM-2 were proposed for purposes related primarily to counting cases: How many of those in institutions were in a general category rather than in another. DSM then is not designed or intended to further basic science;

that is, the development of scientific theories, research on tissues or animal models, and whether the basic science is medical, psychological, or sociological. It is not designated, for example, to further current medical, psychological, and social theories about cognition, personality, or functioning, although clearly all three are central to the DSM diagnosis. If and when such theories may lead to evidence that would advance understanding of the etiology, identification, treatment course, and prognosis, then those results should and would influence the DSM. Thus, basic science should be expected ultimately to drive the DSM; not the other way around. With such issues in mind, clearly the word “statistical” in DSM now takes on greater meaning, for the goal of the DSM is to facilitate drawing correct statistical inferences from what is observed. In the clinic, this would mean correct inferences about choice of treatment, monitoring, treatment response, and maintaining overall purview of the patient. It has been said that psychiatrists are methodically treating symptoms, not people. The presence of an austere neutrality and the needs of the marketplace allow for this to occur.

● One other issue is that behavioral sciences are not really sciences. They are, in the words of David Brooks, a semi-science. There is often little agreement on constructs like mental disorder as the DSM-5 uses sloppy scientific words like excessive, binge, or unspecified. Labels like schizophrenia are only a description of a set of symptoms, not an explanation of its cause. And, what makes matters worse, is that the treatments of these mental diagnoses are not improvements over remedies formed 30 years ago. In fact then, mental health providers are purveyors of uncertainty, using improvisation and artistry to improve people’s lives. Thankfully, mental health is better in practice than in theory.

● Older adults do not benefit from the diagnostic manual. Traditional medical evaluation includes a chief complaint, history of illness, past medical history, family history, social history, physical exam, diagnostic tests, and assessment and plan. The problems are steeped in biopsychosocial nuances. The paradigm shift of the last decade has proffered that the focus of the healthcare team was shifted, developing a stronger relationship with individual patients in an extensive proactive and personalized healthcare plan. This would involve the profile summary that includes health status, health risk analysis, which is generic, environmental, and lifestyle, a one-year plan and a five-year plan. This involves un-DSM features. It requests that “big data” partake. The DSM is a poor approximation of true help in this process.

This often requires a comprehensive assessment. Ahlund, Back, ●berg, and Ekerstad (2017) assessed comprehensive geriatric assessment on

physical fitness in acute medical settings for frail elderly adults. They noted that frail elderly people are often using emergency care. During hospitalization, physical decline is common, implying an increased risk of adverse health outcomes. Comprehensive geriatric assessment has been shown to be beneficial for these patients in hospital care. Although limited evidence about the effects of physical illness, Ahlund et al. showed that a comprehensive geriatric assessment was most apt and supportive for acute care for frail elderly individuals. Medical care for the acute frail elderly patients and a comprehensive geriatric assessment appears to be superior to conventional care in terms of preserving physical fitness and functional mobility.

In the past theorists and clinicians were more interested in the why of a disorder than the what. Now we have more descriptive and biological models and treatments. Older psychoanalytic models, for example, tended to classify individuals. The DSM now classifies disorders. We use the DSM to compare the signs and symptoms of our clients' presentations through a set of objective criteria. Diagnosing mental health conditions is of course more an art than a science. It always was (and always will be). Diagnosis of general medical conditions is not without an art component, but physical medicine has a major advantage over behavioral medicine or mental health because of its ability to objectify findings and have biomarkers. Practitioners realize that now the concern is with the art of the question. This is not to say that they do not bring an excellent command of signs and symptoms of the mental disorder spectrum to the process. They do.

The interview is the most important part of case formulation. The essence of this skill lies in their ability to frame a series of insightful and intuitive questions around the client's presenting problem. Jointly, assertively, and yet tactfully, they approach assessment with precision and are aware that success with any client means that the language controls the discussion, the discussion controls the relationship, and the relationship controls the quality of outcomes. As such, the highest premium is placed on rapport building. Clinicians, therefore, should always ask their clients what they want from treatment and what they may find themselves without if treatment works. Clients who profess that they want to become more independent and self-reliant might really want to think about this after a period of time.

Complexity: The multitrait-multimethod matrix of Campbell and Fisk (1959) some 50 plus years ago has confirmed the value of assessment from different perspectives. Importantly it is the multiple sources of information – the context of the problem, referral data, behavioral interviews, honesty

of the patient and informant, and the skills of the assessor – that determine “truth.” There is no lab procedure of group or combined procedures that provides a clean-cut, error free discrimination between individuals that have some specific psychopathological diagnosis and those who do not. The reasons for this are legion; powerful influence of state variables, such as motivation, fatigue, attentional fluctuation, intelligence and so forth. Additionally, there are structural problems; faulty taxonomy, inadequate treatment system, fickle and at times dishonest subjects, and poor care and insurance environment. Biological markers are not “trustworthy markers” as is needed in the real world of people. Philosophically, the assessment process allows for a complex slice of nature to be transformed into a symbolic representation to communicate a truth. Rarely is a gold standard present, certainly not in psychiatry. The psychometric dance between the nomothetic and idiographic data is infinitely varied. Indeed we have a better descriptive science than a treatment science.

How this translates into the modal problems of later life is both complex and unknown. Where older adults are concerned, psychological constructs are more complex than thought. Take MCI, for example. Patients with a MCI segue to AD in various ways: reduced learning (Loewenstein, et al., 2004), a decline in delayed recall (Schmand, Huijzen, & van Gool, 2001), a faster rate of forgetting (Greenway, et al., 2006), and overall problems in delayed recall (Jak, et al., 2009), not to mention the build-up of toxic biomarkers (a-beta and tau). Depression also appears to be much more complex than was previously understood. Past research has shown that there are inflammatory mechanisms at work in depression, but in the last 10 years there has been much research on the complexities of how the immune system interacts with brain function both in healthy brains and in people already experiencing depression. This has led to the conclusion that there are now different immune factors at work in depression depending on the clinical phase of the depression and that the genes for this immune response are switched on and off at different times according to the phases of depression. Clinical states of acute depression, response, remission, recovery, and relapse, are highly complex interactive states between the inflammatory processes and other immunological cells. The HPA system also contributes its share of problems in a chronic state. A new model then could help to overcome the simplistic notion that depression is the same kind of disease for everyone, behaving in the same way regardless of the timing of the disease. Professionals now know that depression is a much more neurologically dynamic process and that has many complications for both research and for treatment (Edelstein, et al., 2010).

Or you can check out frontal systems. Executive functioning (EF) depends on the integration of frontal systems. EF impairment may follow disruption of frontal system information processing, regardless of the location of the lesion within the system or direction of the perturbation. In some cases remote lesions can affect processing within the frontal circuits. Functional imaging and EF control are complex; response inhibition with the orbitofrontal region, attentional control with the mesiofrontal region, working memory and rule discovery with dorsolateral region. Neurocognitive functioning is also a marker of treatment resistant depression (TRD). Several domains have been proffered. They include attention, processing speed, memory, visuospatial processing, and executive functioning (Gupta, et al., 2013).

Psychological tests are also all over the place. The Wisconsin Card Sort (WCS) may be the best validated EF measure. It is reasonably affected by frontal lesions and it selectively activates the left dorsolateral cortex in activation studies. Multiple EFs can be ascribed to the various WCS subtests but this is difficult to prove. Neither neuroimaging nor factor analyses have isolated specific and robust WCS-related factors to the frontal lobes. Thus apparently localized tasks while clearly dependent on the frontal functions, may be the heteromodal processes on which truly cybernetic EFs operate. There is also an EF component that subserves inhibition, something in decline at later ages. Unfortunately, EF deteriorates at an exponential rate with age. And, the pattern of age-related cognitive decline in non-EF domains is most consistent with the loss of EF control over intact processes (Royall, et al., 2002).

The goal then is to take a variety of test-derived pieces of information obtained from multiple sources and place them in a context of historical information, referral information, and behavioral observations to obtain a cohesive and comprehensive understanding of the person being evaluated. This process is greater than the individual validity coefficients found in the literature. So, on the one hand, decision rules of a reasoned assessment process are superior to other methods, but on the other, the application of this process in the real world at later life is innately complex.

Know the Identified patient: The modern healthcare system has been driven by a biomedical approach that emphasizes the paradigm in which the treatment typically reduces the symptoms and maybe even removes the underlying pathological process. Dementia of course fits better into a chronic illness and disability model, in which an intervention is focused more on maintaining function, reducing excess disability, and enhancing range of quality of life outcomes. We need to know more about the “person of the patient.”

There are many issues that require our knowing. We need to assess for cognitive reserve, information on premorbid functioning. As discussed, the concept of cognitive reserve emphasizes enhanced adaptation to cognitive loss rather than viewing poorer cognitive ability in young adulthood as an early expression of neuropathology. The Uppsala Cohort Multigenerational Study authors thought to expand on and extend previous research demonstrating that childhood education and work experiences affect subsequent risk of dementia. They reported that higher elementary school grades were associated with a lower risk of late-onset dementia. Although higher levels of schooling and job status added to the earlier protective effects of elementary school grades, the school grades were a stronger predictor of dementia risk. Furthermore, the protective effects of work experience were specific. Employment involving complex work with data was protective, but complex work with people or things did not affect dementia risk. The beneficial impact of early school performance is underscored by the fact that children in the lowest quintile of achieved grades who had highly data-complex occupations were not protected from dementia, whereas children with high grades were conferred protection regardless of their occupation complexity. Elementary school grades, the factor most distal from old age, was the strongest risk predictor of those studied in full incident dementia.

It is not sufficient to understand a person's cognitive status only; clinicians also must understand the way cognitive impairment is experienced by the person on a daily basis. This model includes potential mediators that influence the way the changes of dementia are experienced by the person. These mediators can be aspects of the person or the environment. The advantage of this type of model is that it provides a blueprint for assessing the aspects of the person and his or her life that can provide the basis of living well with dementia and domains that might be fruitful for further development of assessment measures.

A key approach is that assessment of the remaining ability should accomplish two very important goals. First, it should allow for the creation of activity that is personally meaningful to the individual with dementia and second it should encourage the development of meaningful social roles that the individual can fulfill as part of a group. It appears that individuals with dementia can continue to reliably report their values and preferences regarding their life and care. The importance of preferences, values, and activity selection with individuals with dementia is implied. Caregivers tended to perceive that the care receiver would rate each value lower than the care receiver him or herself. In other words, individuals with dementia rated their value as more important generally than

caregivers perceived they would. Moreover, when individuals experience continuity within their life, routine, and activity, they demonstrate both better emotional functioning and fewer behavior and psychological problems (BPSDs) that interfere with wellbeing and quality of life.

That said, assessment provides the necessary components for the needed psychoeducation and later psychotherapy. The profile of problems allows for a plan of action that is a loose model of the person. Take repetitive questioning as an example: This is seen as the most frequent problem and has been reported as being highly prevalent in samples with persons with dementia. It rates as high as 67%. Researchers noted that the presence of themes indicated that such behaviors may serve a function for the person with dementia and should be dealt with sensitively. Executive dysfunction, longer duration of the illness, and higher depression scores were predictive of repetitive motor actions and verbal statements. It is also noteworthy that they serve a functional purpose and are related to the heterogeneity of many causal factors. It is noteworthy too that repetitive questioning could be caused by a variety of factors, including episodic memory deficits, feelings of insecurity or anxiety about one's ability to cope, boredom, and anxiety about future events. Careful assessment provides the base for real understanding of the person and the complete problem.

The following sets the table for necessary background information and concerns about the person of the patient and their context.

Assessment and Care Management Strategies

- Education about problem in later life
- Education about the medications used to treat problem/depression in later life
- Education about good sleep practices
- Review of symptoms
- Review of side effects
- Consideration of system barriers (e.g., transportation, insurance)
- Management of side effects
- Education about suicide and assessment of suicidality
- Encouragement to stay the course long enough to benefit from treatment
- Discussions with family members/caregivers to elicit their support for the treatment plan

This process of evaluation continues over the life of a neurodegenerative process. Assessment continues from before a diagnosis and unfolds after and in the later stages with the identified patient as well as the caregiver. It

never stops, although its value alters with more behavioral concerns over time.

Screens and Psychometrics: Screens are a necessary evil. Yes, we are back to a false positive or a false negative. This is always an issue. A test, for example, that costs \$25 with a sensitivity .9 and a specificity .9 is justified by cost only at age 70; go to .8's and it is age 80. Annual screens might best be targeted to ages beginning at 75, or, if there are several risk factors, age 60. Many screens do have acceptable correct classification ratios including the Min-Cog, Memory Impairment Screen, and the GP-COG. It actually appears that there are rather good tests available and adding tests does always add value. Item response analysis would be of help. In the future it is likely that computer-based tests will add the most value to assessment. In the past decade or so the pick has been global measures like the MMSE but this has lower sensitivity for producing accurate diagnosis (Gonzalez-Palau, et al 2013).

Test data also provides information on more modern test theory that provides probabilities for problems (e.g., dementia severity or AD pathology), and uses ROC analysis based on the continuum of progression from normal through MCI to dementia. Minimal test data on the CCRs (correct classification ratios) of tests, sensitivity and specificity, as well as positive predictive power (PPP) and negative predictive power (NPP) are given. Importantly, these data interact with a combination of information from the patient, a knowledgeable informant, and a clinician's impression. Psychological data then are very much a part of the mix. Often tests are unreliable alone but in combination, provide incremental validation. In this case non-redundant data often the core of psychological information contribute to outcomes.

Reliability, especially test-retest reliability, is the most relevant in repeated measures. Assessing change is always an issue. There are several methods. Simple discrepancy scores, standard deviation index, RCI (reliable change index), and regression based formulas are ones that are typically applied. In fact, it has even been recommended to use a Neurocognitive Composite Score consisting of a z-score composite of several domains to predict MCI and AD connections (Gupta et al., 2013). Practice effects also represent a piece of this as most patients are retested. Additionally, can these be used constructively as markers like training for change (Duff, Beglinger, Moser, Schultz, & Paulsen 2010)? Of course there are issues of retest interval, regression to the mean, and novelty, as well as demographic factors and selected clinical conditions.

Noteworthy is that many methods have evolved for assessing change at the individual level in the past number of years. AS noted above, these are

best represented by RCIs and more sophisticated standardized regression methods. The RCIs have been refined and many variants are available. Some RCIs actually are rather sophisticated and correct for practice effects (Chelune et al., 1993) or for regression to the mean (Hsu, 1999). And, as noted, regression equations can be used to assess cognitive change at the individual level and can control for confounding factors, such as practice effects and regression to the mean or a particular measure (Tuokko & Smart, 2018)

Concepts Associated with Change

Reliability – especially test-retest

Practice effects – present in older adults and can be relevant for 6 months and for a variety of neuropsychological conditions. They may also have clinical utility as they predict longer term outcomes as well as diagnostic information.

Floor and Ceiling effects- important in consideration of tests for change.

Variables Associated with Testing Situation – Retest interval (important but insufficient evidence for recommendations) and Regression to the mean (beware of extreme scores)

Variables Associated with Individual Patient

Demographic variables – similar impact on two testing occasions

Clinical condition -- level of decline expected

Prior experiences – Relatively brief experiences with test affect outcomes

Methods for Assessing Change

Retest interval – Practice effects shown as far out as 2.5 years

Alternate forms – do not guarantee that practice effects will not occur

Appropriate Control norms – This significantly improves the data. But change norms can be very tricky

Reliable Change

There is always more. If you know the mean and standard deviation for any group of individuals, you can transform any patient's observed score to a z-score (assuming the scores are normally distributed; $Z\text{-score} = (X - M) / SD$). Every patient's test data can be viewed as an individual outcome. It is possible to use published research to determine/estimate the specific test operating characteristics of a given patient's specific test scores. By using simple Bayesian methods it is possible to enhance evidence-based practice that is: a) value-driven; b) integrates research derived from the study of groups to inform clinical decisions about individuals; and c) addresses the concerns and needs of our referral sources.

Regardless, it is our belief that this level of test scrutiny is necessary for individual tests and can be applied for research but is often too cumbersome and even inexacting for clinicians. The belief here is that the basics of care for older adults require some change. There is a suggestion to carefully peruse the case, proceeding initially from the real world, targeting issues, and then entering mental health treatment. This requires assessment and monitoring as well as flexibility. Tests then are imperfect tools representing the referent due to the interaction of age with moderating variables, the construct variability of the variable of interest (intelligence for example), and practice effects. Again, Mast (2012) argues for a “Whole Person” approach where the value of “the person” of the diagnostic category becomes as important as the process of the diagnosis and treatment plan. We agree, but add that we need to apply the better known canons of our sciences to the person and formulate real plans that are titrated to tangible outcomes.

Norms, Special Psychometrics: Norms can be considered the standard for the group concerned. In addition, norms provide the context within which the performances of an individual external to the reference group. ● Importance is the need to consider an older adult and the complexity of their life and situation. Base rates are most relevant for norming. This is the proportion of people in the tested population who have the target characteristic. Discrimination is best when the characteristics occur in 50% of the population. This will decline dramatically when the rate falls. Care then should be taken that the base rates of the target population match the sample of interest. With decline and older adults, we do not have the ideal base rate situation and must approximate. ● Older adults are always a problem here.

Descriptive information allows data for the purpose of how this person does in relation to the reference group; the issue is where is the individual in relation to a particular reference group. This must be decided then. ● Or, norming can be concerned with a diagnostic group: Is the functioning of this patient impaired or not? That is, does this person deviate from premorbid expectations? The reference standard is the premorbid status of the person and deviations of interest from this individual comparison standard are unidirectional. The person is then seen as “normal,” “impaired,” “above normal.” A score of 75 on the WAIS-IV can be considered “borderline” then, but not “abnormal” unless the person is low relative to the premorbid status.

Neuropsychology especially is interested in diagnostic or deficit oriented norms and the issue is usually pathology. It is most interested in developing diagnostic norms and issues like an observed versus an

expected score is high. These can be adjusted with education, age gender and ethnicity. As noted above, the concern is the cut-off score, as test operating characteristics like sensitivity, specificity and positive and negative predictive power as well as odds ratios and likelihood ratios as well as receiver operating characteristics are in play. Continuous norming has become popular in recent years. It can be seen too that increased variability occurs at later life as the variance accumulates over time and has an effect: More variability, more problems. Also, most tests are normed on younger groups and the factor structure of many tests like the WAIS-IV is different for younger and older groups. Norms have limits.

Most of the neuropsychological tests then are deficit-oriented rather than population-oriented and therefore patterned after the diagnostic or pathognomonic model. Since the interest is in scores that fall outside of the diagnostic norm, concern is raised regarding **above** cutoff scores. Here test operating characteristics are of interest. Characteristics such as gender, education, and ethnicity assist age in the interpretation of the interested condition. Regardless, as norms are applied to older adults, the random cumulative effects of education, life experiences, illness, medications, and neurological insults lead to progressively greater normal variability in cognitive functioning among individuals, with successfully aging older adults on one end of the continuum and those with problems on the other. **Only** truly random samples are then in order, a rather difficult task at late life replete with virtually impossible decisions for any norming group.

Also, there are very few tests that have norms up to **90**. The oldest old are a special problem. Generally this is the age group **85** and above. But this too is changing. Among the oldest old it is unclear just what constitutes measures of everyday functioning, normal aging, as dementia, sensory impairment, and physical limitations have an extremely high prevalence in this group. If normative data are to be collated in this age group, a decision must be made as to whether to include a random sample of persons in this age group (to characterize cognitive performance in the typical older adult) or whether to exclude persons with physical, sensory, or cognitive limitations. Assessing octogenarians and centenarians makes it clear that these older adults need special care and assessment.

Many of the better norms have been based on the Mayo Cognitive Factor Scores (MCFS) (Smith et al., **1992**, **1994**). The **MOANS** (Ivnik, et al., **1992**) have been applied to older adults up to age **95**. Norming is perhaps most of value when the issue of who was excluded is considered. Exclusion criteria may interact with demographic factors. This is especially a problem for ethnicity as ethnic differences increase with age. The Crum and colleagues norms differ measurably by as much as 11

points for age and education. The cutting score for the MMSE of 24, for example, is quite variable for different education groups. In addition, it is important to compare apples to apples; an older independent adult needs to be compared to a population of similar older adults.

What the parameters of real problems are for old/old adults requires some savvy then. It is clear that decline occurs and can be punishing for even the “normal adults.” What is dementia at late/late life is itself a puzzle? Just the number of neuritic plaques or atrophy can be confusing. Every day needs of IADLs can be cumbersome and prevalence rates of at least one problem at advanced age are greater than 60%. Below are markers for general stasis for older adults: This may not apply to older old adults. Caution is the bi-word for assessing older old adults, especially in tests with no advanced age norms.

Low or No variance Among Normal People

Visual acuity (20/20)

Auditory acuity

Visual Field Attention

Color Perception

Naming

An intriguing and largely unsettled question is how to think about older norms or established psychological tests. If a test was normed 20 years ago and norms include people in their 60s, then do those norms now apply to current 60-year olds or to current 80-year old? The question will depend on whether the test results would be primarily affected by developmental aging or by cohort differences. To our knowledge, the question is not addressed in the current literature in any systematic way. And, it is now becoming very clear that the 80 year old 30-40 years ago is not only not as healthy as the one now but less active. However, it is worth considering the age and representative of the test’s normative data when working with older adults.

Norms then have problems. An older adult who provides 3/10 words on recall is logically impaired. Over 80, such a result produces a low normal scale score. Problems with low base rates or biased raters are similarly compromised as norms can be misleading. It has been estimated that 20% of the variance of cognition and function is all there is. Is that enough? Improvement occurs with domain-specific targets but still.... Marcotte and Grant (2010) argued for a function-led approach to such issues where function leads the parade in determining outcomes. As we have argued, there is simply too much variability from the person

(personality, medical comorbidity, intelligence, SES strengths, support, environmental support, and compliance, to name a few) to the issue itself. PVTs (performance based validity tests) are most helpful but not foolproof.

Environmental Factors

Living situation and supports

Personal Factors

How long with disorder, strengths and weaknesses, mood, stress, sleep,

Compliance issues

Compensatory Factors

What supports exist

Finally norms can also be a problem if not seen over time or in context. Cross-sectional and longitudinal data of a modified version of the MMSE for Chinese adults from the Singapore Longitudinal Study suggested as much. It presented MMSE percentile curves as a smooth function of age and education strata for unconditional and conditional standards based on quantile regression coefficient estimates. They found that the 5th and 10th percentiles were more strongly associated with age and education than were the higher percentiles. Model diagnostics demonstrated the accuracy of the standards. Most standards and their use were unconditional standards in that they only consider performance at one point in time. A person with an MMSE score in the 50th percentile two years ago and the 10th percentile now is considered normal at both points. However, considering the person at a much higher percentile position two years ago, one may suspect that he or she may have been experiencing onset of a cognitive disorder and deserves further attention. Such longitudinal perspectives require the conditional standards, which are exemplified by longitudinal fetal and postnatal growth curves.

Cognitive tests then are best considered imperfect tools. Error is introduced through reliability and less than perfect validation. Practice effects are never eliminated. Lengthening the test time between evaluations does not solve the problem. The standard error of measurement and regression to the mean are two sources of imprecision. Using the Mayo norms, Ivnik et al (1999) showed that practice effects were maximal between test 1 and 2 with the curves flattening after test 2. Reliable change and regression have been proffered as solutions. They are generally not applied to clinical situations.

General Considerations of Test Selection

- Brevity
- Psychometric integrity
- Relevance to intended purpose of testing
- Cost
- Skills of patient
- Content
- Ease of use
- Comprehensibility of results
- Symptom validity tests

Function: In one way or other we address function in each chapter. It is intended as function that devolves from each domain. It is well accepted that function and cognition make up the core components of dementia. In fact, both are noticeable in the later stages of MCI: MCI deficits generally show themselves just prior to a conversion to a dementia but often more complex IADLs reveal problems. Functional measures provide data on any restriction or inability to perform an activity in a manner consistent with the person's stage of development. ADLs and IADLs form a continuum of function and tend to be directly related to cognitive tests.

As we have argued, function matters in the assessment of the nuance between MCI and normal reactions. Lindbergh, Dishman, and Miller (2006) performed a through a systematic review and meta-analysis of functional status of persons with MCI. A total of 151 effect sizes from 106 studies were included in the final analysis. Manual effects models indicated a large overall effect size of functional disability that was significantly more pronounced in persons with MCI versus healthy controls. Persons with multi-domain MCI had significantly worse instrumental ADL performance compared to single-domain MCI. Function may also have an impact on the nuance of SCI also. The new DSM (DSM-5) now recognizes possible problems with function for mild Neurocognitive Disorder.

There are over 40 measures of function noted in the literature. While performance based measures are most reliable, they are often hard to apply and one must depend on patient-ratings (often overestimates) or significant others (often underestimates). Caregiver ratings have been linked to caregiver mood or, less featured, to vacillation in patient's behavior. Functional assessments also may not tap into the ecological merits of the person's life. Would an older adult be found wanting and decisions made about him/her in check writing if that is performed by the spouse?

Remember too that the restrictions in life (driving and finances) has substantial effects on the life of the person.

As noted, deficit in activities of daily living (ADLs) (e.g., eating and dressing) or instrumental activities of daily living (IADLs) (e.g., shopping and cooking) that cannot be attributed to physical limitations should prompt concern, as there is a strong correlation between decline in function and decline in cognitive status across the full spectrum of dementia. In addition to clinically observed concerns, any patient- or informant-reported concerns should trigger further evaluation. Positive responses to conversational queries, such as "Have you noticed any change in your memory or ability to complete routine tasks, such as paying bills or preparing a meal?" should be followed up with a structured assessment of cognition.

Recognizing that there is no single optimal tool to detect cognitive impairment for all patient populations and settings, clinicians may select other brief tools to use in their clinical practice. Best are those that combine function and cognition. The Veterans Administration (VA) listed in a guideline document of the >100 brief cognitive assessment tools that may be suitable for primary care practices. If an informant is present, defined as someone who can attest to a patient's change in memory, language, or function over time, it is suitable to use the eight-item Informant Interview to Differentiate Aging and Dementia (AD-8), the informant component of the GPCOG, or the Short Informant Questionnaire on Cognitive Decline in the Elderly (IQCODE). The AD-8 is also relevant as it requests ratings on changes over the past few years on everyday tasks.

There are difficulties in applying a connection of functional performance in a patient and connecting these ratings to diagnoses. The relationships between cognitive performance and functional behaviors are significant but often moderate at best and frequently explain less than 50% of observed variability in regression models (Loewenstein et al., 2005). In fact, real world outcomes are not frequently employed or depended upon. Prediction is often based on global levels of severity rather than specific cognitive predictors of cognitive tasks. There is typically a lack of sensitivity and specificity analyses (associated with specific base-rates) that guide the clinician in making individualized treatment decisions. That said, it is necessary to assess for function, perhaps not always sufficient for a definitive diagnosis.

The bottom line is that global cognitive functioning is certainly not a proxy for functional change in dementia, because it accounts for only a limited portion of variance. Intact cognition is thus a necessary but not sufficient condition for successful performance of everyday tasks in

normal and abnormal aging. Perhaps then it is advisable to note decisions to limit autonomy in individuals with dementia should not be based solely or even primarily on mental status screens or neuropsychological test performance. Clinicians should rely of capacity based information using diagnostic and neuropsychological test performance as supporting evidence in rendering clinical judgments regarding a person's capacity for limits in everyday living.

The exact cognitive domains that subserve function are relevant. Royall and colleagues (2007) reported that global measures of cognitive status explain on average 11.8% of the variance. Memory predictors explain 2% of the variance of everyday functioning. Measures of EF, as well as processing speed, predict IADL functioning. Gross, Rebok, Unverzagt, Willis, and Brandt (2011) have shown that inductive reasoning is an important determinant of current everyday functioning in community dwelling older adults. This suggests that successful performance in daily tasks is critical, dependent on EF. On the other hand, baseline memory function is more important in determining change over time in everyday functioning (and in the eventual diagnosis of a dementia) suggesting that baseline memory may be a core marker of incipient progressive decline.

Overall, the task of extrapolating to function based on cognitive scores is complicated. The ecological valid question is: "What is needed for this task/issue?" Implied is the notion of real world capability: Does this patient have the ability to do X? Does this patient always have such problems? Does a score of a psychological scale reflect problems mostly, some of the time? And, most importantly, can this older adult have assists or be rehabbed for such a task? No psychological test has the ability to answer these answers. Again, if one is very impaired, this is less an issue for most tests as they have high floors. Perhaps the use of a DRS-2 (Dementia Rating Scale-2) or a FIM (Functional impairment Measure) is warranted. If mildly impaired, some combination of neuropsychological tests and function ratings are more in order. The assumption, however, that neuropsychology and health care providers can make inferences about function is far from settled.

Function interacts with medical comorbidities and behaviors. Dementia is as much a failure of body systems and aberrant behaviors as cognition. In fact, function accounts for more of the variance of dementia problems than cognition or behavior. These should then always be considered in the evaluation.

Two last thoughts: First, moderating variables count. Assessment involving attitudes, interest, and social context as well as the full domain assessment is important. Psychological testing especially provides

information on the patient. Intra-individual variability is a good indicator of cognitive aging. This variability seems to be accentuated in older adults with cognitive disorder, such as dementia, and it may also be predictive of other multiple outcomes over time. Variability may also be due to a variety of acute conditions that can result in transient or more persistent changes in cognitive function of older adults. These include vitamin deficiency, such as B12, hormonal imbalance with such as reduced testosterone, and thyroid disturbances. Many of these conditions of course are treatable and secondarily reduce an older adult's cognitive complaints. In addition, exposure to problems like neurotoxins or medications like benzodiazepine, or AEDs, as well as levels of sex hormone, influence cognition.

Second, adherence (compliance) to a treatment regimen is problematic for as many as 50% of patients. We have addressed this in a previous chapter. It is a big problem and one that is given short shrift in assessment. In one study on cardiac medication compliance for example, adherence was 71% for aspirin, 46% for beta blockers, and 44% for statins (Newby et al., 2006). It is little different for older adults after an MI or bi- (Bosworth, Blalock, Hoyle, Czajkowski, & Voils, 2018). Whatever the cause, multifaceted interventions attached to the real world, good habit format training, and adequate self-regulation, as well as automatic priming, help measurably.

Barriers Associated with Medication Adherence

Patient-related barriers

- Forgetting
- Inadequate instructions
- Health literacy
- Financial challenges
- Chaotic lifestyle

Medication-related barriers

- Number of pills
- Dosing instructions
- Side effects

Clinician-related barriers

- Lack of trust in clinician
- Poor communication

Health system barriers

- Inadequate health coverage
- Transportation and money issues

Effort/Dissimulation/Unawareness: There are always issues of dissimulation, less so for later life problems. The dialogue on this issue is robust, as PVTs and SVTs are important in evaluations. Beware the patient with low performance relative to credible patient groups; ones showing pathognomonic signs (i.e., found only in feigned presentations), ones with non-credible patterns on dedicated measures of response bias or a non-credible pattern on standard neurocognitive tests (“embedded”), people with non-consistency between test scores and ADLs, inconsistency between injury specifics and test scores (improbable outcome); or inconsistency in scores within/across evaluations. Not all individuals feign in the same manner (Boone, 2009) and clearly compensation-seeking shows that response bias is not static across exams.

Current recommendation and practice in the field of clinical neuropsychology is to administer multiple PVTs, interspersed throughout the exam, covering multiple cognitive domains (if not for every task administered) so that performance validity is repeatedly sampled. Multiple failures (≥ 3) on PVTs virtually never occur in credible populations, excepting for individuals with dementia and individuals with very low intellectual scores (FSIQ < 70). Dean et al. (2009) reported that in individuals with diagnosed dementia 36% of PVTs failed in those patients with MMSE > 20 , 47% of PVTs were failed when MMSE scores were 15 to 20, and 83% of PVTs were failed with MMSE < 15 . Tests included the Rey Word Recognition correct false-positive errors, Rey 15-Item recall intrusions and recognition false positive errors, b Test total time, and Dot Counting E-score and grouped dot counting time. Thus, performance on these PVT variables appears relatively robust to cultural/language/educational factors, but may be less so with older adults especially those with cognitive problems. Additionally, PVTs and SVTs are usually less necessary for older adults as they are almost always motivated to do the best that they are able. These scales tend also to be contaminants to populations in cognitive decline: That is, they are a measure of cognition problems more than effort.

One other issue is important here: It has been recently shown that the time of unawareness and rate of progression seem to vary in Alzheimer’s patients. Starting at about three years before dementia onset, episodic memory awareness began to decline sharply at a mean rate of 2.6 units per year, which is nearly half of the baseline standard deviation. Memory awareness began to decline 2-3 years earlier in younger persons compared to older persons and neither sex nor education was related to age and memory awareness. An unexpected finding then is that decline in awareness of memory impairment and dementia begins earlier in young

people compared to older people and older people tend to have a dementia unawareness about 2+ years prior to onset of the diagnosis itself.

Again, caution and perspective are important. But, where performance-based measures are not practically applicable, self and informant reports used together may provide reasonable approximations of actual instrumental activities of daily living. A true analysis of function may take time.

Culture: In 1952 Kroeber and Kluckhohn (1952) addressed the issue of culture. Since their review it is reasonably accepted that the core definition of culture is a collection of ideas and associated values that are reflected in individual and group behavior and that over time have acquired stabilizing power in personality and society. Culture binds people together in ways they are often not aware of. Samuel Johnson called a habit regarding the ties of culture is “too weak to be felt until they are too strong to be broken.” Much later, Ridley and Kelly (2007) identified several characteristics of culture that are relevant to case formulation, noting that culture permeates all human experience and this is present throughout the formulation process, that culture is experienced internally and also has external reference, that culture influences vary among people from similar cultural backgrounds, and culture is a broad and multidimensional term that distinguishes groups of people not only by race and ethnicity, but also by age, socioeconomic status, sexual orientation, gender identity, religion, occupation, and education, among other characteristics.

Virtually all discussions on culture in psychology can be organized according to a continuum with the universalist’s perspective on the one hand and the relativistic or culturalistic at the other. The universalistic perspective holds fundamental psychopathological processes that are shared across all of humanity and that varying expressions of disorders in different cultures are little more than epiphenomenon. Adherence of this perspective site research showing that the core symptoms of depression and schizophrenia, just as two examples, are found in multiple western and non-western cultures. At the other end of the continuum, the culture relativists hold that culture pervades experience so inextricability that the expression of psychopathology can only be understood in the context of the culture in which it manifests itself and therefore cross-cultural comparisons are futile.

Multicultural research has demonstrated that neuropsychological performance among neurologically healthy younger and older adults significantly differs between ethnic minority and non-Hispanic white groups, even after statistically adjusting for other demographic factors, such as age, education, and gender. Emerging literature points to a

significant impact of numerous sociocultural factors on test performance among ethnic minority individuals, including quality of education, acculturation, language, and stereotypic threats. Considerations for psychological testing always include race and ethnicity, country of origin, current U.S. region of origin or neighborhood, immigration history, linguistic background, quality of education, social support, current and childhood SES and nutrition access, and utilization of health and mental health services.

Among older adults, ethnic minority populations, particularly Latinos and African Americans, are growing much faster than the non-Hispanic white population. In 2015, ethnic minority individuals represent a higher percentage of the older adult population in the U.S.A than other age groups. Working from a biosocial cultural theoretical perspective, the sociocultural level of analysis includes consideration of how socioeconomic, institutional, and cultural (that is the shared attitudes, values, goals, and practices that characterize a group from one generation to the next) factors modulate an individual's or group's behavior. Sociocultural issues are critical for the understanding of the neuropsychological tests of performance and neurobehavioral functioning. Ethnic minority individuals also have higher rates of hypertension, diabetes, heart disease, cancer, obesity, and HIV/AIDS.

Five different aspects of culture have been identified that may affect cognitive test performance. These include patterns of abilities, cultural values, familiarity, language, and educational attainment. Unfortunately, it is not possible to create normative information on standard measures for people of all languages and cultures. Special concerns should be considered for caregivers and their reports. Healthcare providers must maintain a sociocultural lens and to be mindful of the potential sociocultural norms throughout any evaluation. In working with informants, for example, of ethnic minority elders, one needs to be cognizant that there may be hesitance to report the cognitive and functional decline of a loved one despite having observed such declines. The clinician must utilize the best available psychological instruments and norms and acknowledge potential limitations and the interpretation section of any report. The literature needs to be constantly consulted. The normative data used also needs to be updated and to be explicit.

There are perhaps five reasons for considering culture in the case formulation. First, failure to do so increases the chance of miscommunication, a lack of understanding, and inadequate empathy, which in turn can lead to poorly suited formulations and ineffective treatment. Poor understanding of the cultural context then can lead to an over-pathologizing, as well as

under-pathologizing. Second, to consider culture in a case formulation is that the language of psychotherapy and thus psychotherapy case formulation is suffused with culture. Psychotherapy is a culturally embedded healing practice (Wampold, 2001): More culture involved in psychotherapy leads to better results. Third, meta-analytic evidence suggests that culturally adaptive psychotherapy may be more effective than therapy that is not culturally adapted. Fourth, related to this point, cultural competence may contribute to improved outcomes. Cultural competence refers to knowledge of those factors that render a particular group distinct from other groups, knowledge of the shared interpersonal and social experiences that characterize a particular group, knowledge of the salience between and within group experiences of a different group member, and knowledge of the relevance of salient group experiences to a therapeutic process. Finally, another reason to consider culture in the case formulation is that cultural factors may directly cause, precipitate, or maintain symptom problems.

This last point is important. This occurs most often through a mechanism, such as acculturation stress and stereotypic stress. Acculturation stress refers to psychological problems associated with adapting to a new culture. These include pressure to alter one's values, attitudes, behavior, and identity and congruence between cultural practices and language difficulties. Acculturation stress can manifest itself in anxiety, depression, feelings of marginality, psychosomatic symptoms, and the like. This is not uncommon at later life among minorities.

Models of culturally informed consideration include the following: First, assess the patient's culture identity. How is his or her symptoms self-defined by the membership in a cultural or ethnic group? Second, consider whether and how culture influences the patient's explanation of his or her problems. Consider culturally relevant interpretations of social stress or supports and levels of functioning. Third, integrate cultural data into the formulation and to the extent to which personality factors versus cultural factors are contributing to the individual's problems. Fourth, consider how cultural factors affect the therapist-client relationship. Credibility of the therapist in a therapeutic relationship may hinge on the extent to which the therapist can convey respect for the individual's cultural values, attitudes, and the like.

Best Evaluation Models

Assessment Issues Related to Older Adults: Careful diagnostic and interview techniques involve some of the following. These are unwavering

regardless of problems or assessment philosophy. Several have special applicability for older adults. Current signs, for example, should have more weight than general past symptoms; the crisis of the recent cognitive problem should not sour the careful clinical considerations; psychometric data are more valuable than most clinical data; value “unspecified” diagnoses as a diagnosis and consider multiple diagnoses; and beware of physical diagnoses and problems. The interface of a collateral is always important, often contentious, and needs planning: The more the disagreement, the more problems there probably are.

Helpful Diagnostic Principles

- History trumps cross sectional data
- Recent history trumps older history
- Obtain collateral data always
- Signs trumps symptoms
- Crises clouds reality of the “person”
- Psychometric data trump clinical information
- Use family history always
- Preference simplicity over nuance
- Preference more frequently encountered diagnosis than elegant new diagnoses
- Medical and Substance abuse diagnoses cause problems for other diagnostic issues
- Watch for contradictory information
- Best data are based on performance and function
- More symptoms increase likelihood of diagnosis
- Typical symptoms more important than non-typical ones
- Always respect previous response to a diagnosis
- Use NOS and Undiagnosed if unsure
- When symptoms do not adequately explain one diagnosis, consider multiple diagnoses
- Avoid personality diagnosis unless it explains behavior or treatment issues
- All diagnoses leave some symptoms unexplained
- Physical illness can exist as cause, comorbid, consequent of mental illness

Psychological assessment again provides a reasonably precise view of the stated problem and its comorbidities. It also allows for cross-checking. Rarely is one method of assessment definitive but, as noted, the added value of a psychological test is palpable. In addition, the assessor can look

at the ancillary features of the person and provide a better summary in the context of the problem. Psychological assessment might not always explain but it allows for a unique understanding of a continuous profile of the problem, its quantity, and place in the life of the person. The following fleshes out the normal steps in the assessment process as well as some unique features related to older adults. Feedback always is a challenge. Providing the “clinical truth,” what actually helps the person and “Medical or DSM truth,” meeting actual criteria, for example, of a mild dementia is an art and not a science. The deferral to the former is generally clinically more apt and helpful than an allegiance to the latter.

Steps in Assessment Process

Understanding the referral

History

Clinical Interview

Testing

Interpretation

Feedback

Treatment

Follow-up Evaluation

Variables involved in Treatment Selection

History of response to Rx

Patient preference

Side effects

Interaction issues

● Older Patient Specific Issues

Expenses for treatment

Readiness to change

Acceptability (what is suitable for this patient)

Availability

Payer approval

Caregiver buy-in

Incongruous recommendations

Prior treatment failures

Intolerable side effects

Case Formulation: We addressed this in Chapter 1. Case formulation has been described for over 100 years. It really is an individualized theory that explains the particular patient’s symptoms and problems. In Cognitive Behavioral Therapy it has most evolved from Jacqueline Persons’ 1989 classic book on *Cognitive Therapy in Practice: A Case Formulation*

Approach. It includes the essentials of care from the cognitive behavioral therapy perspective, which includes a friendly encounter, a problem list or diagnoses, a working hypothesis, strength and assets, as well as a treatment plan. The problem list perhaps is often understated: It is the most central component of the case formulation. It is generally a formulation of the chief complaint and other workable features that deserve the therapist's utmost attention. It can be very expansive involving interpersonal issues of social supports and social conflicts and the full list can include everything that involves the person's issues, notably the domains of depression, anxiety, health, cognition, and life adjustment. The working hypothesis also is formed based on the core issues and components that involve speculation about what is causative for core problems. We argue for the five domains and a drilling down for the core domain(s).

With older adults a chronic care model is often applied. There is a collaborative organized healthcare system of interpersonal providers, which includes physicians, nurses, psychologists, physical therapists, and occupational therapists, but there is also a connection with the new patient's community, connections with the patient, the family, and the other older adults' components of care in the community from senior centers to churches. Research on the chronic care model has shown that the patient with diabetes, for example, will have significant decreases to their risk of cardiovascular disease when the chronic care model is employed. It has been documented that the DPP (Diabetes Prevention Program) is changing this for the better (see Chapter 6). Congestive heart failure patients also are generally more knowledgeable or more often able to recommended therapy and had a considerable percentage of fewer hospital days.

The health domain is in many ways paramount. With anxiety, depression, cognition and life adjustment there can be a more immediate evaluation. This of course will change and the external world will modify its initial plans for these domains. But, with health there almost always is a delay as there is a need to interface with other team members – health care providers, caregivers, home health providers, and others. This may require several weeks/months of slow fact-finding and consults, as needed. ● Of course, we are still talking about a profile of a person: The person is represented by markers in five domains and any intervention of one may and often does impact the others. Health care is just more problematic and encompassing, necessitating wider care and interaction. This may be a special problem for the private practitioner who is often “alone” in practice. Also, we emphasize that the domain profile is likely to change. Again this becomes the “new care model” for that person.

Key interview information involves standard clinical evaluation questions done for years.

- 1) Reason for referral. What types of problems is the patient experiencing and what are the goals of assessment?
- 2) What occurred and when this was first notice as a problem?
- 3) Course of progression. Do you think these changes are getting worse or staying the same or getting better?
- 4) Social situation. What is your current living situation at home, marriage, children, types of activities, etc.?
- 5) Medical history. Describe the medical physical conditions prior to the incident. This includes injuries, psychiatric problems, chronic conditions such as diabetes, heart, and the like. Also describe the treatment that you are receiving for each condition.
- 6) Prior functioning. Describe educational and occupational background. These include education, special problems with schooling, and types of jobs.
- 7) Current functioning. This involves sensory, motor, problems with memory, changes in language skills, changes in mood, and changes in the ability to function with everyday tasks, ADLs and IADLs.

Case Formulation is the ideal format for all CBT therapies. It is cumulative and on-going, subject to change always. With any effort to careful deliberation two things are critical, case formulation and assessment. At later life any symptom can be generated by multiple permutations of multiple causal factors amid multiple causal paths. The health care provider formulates cases based of confirming and disconfirming data to determine whether selected empirically supported causal variables (e.g., cognitive distortions, medically related problems, poor self-control, ineffective problem solving, low rate of positive reinforcement) are relevant, operative, and meaningful to this particular patient. Cognitive restructuring, for example, can be tested by decreasing self-defeating thinking, using behavioral experiments to test the validity of a belief. Bibliotherapy, modeling, mild refutation, didactic explanations, homework assignments, and use of caregivers, to name a few are all implied (Satterfield, 2015).

The case formulation is reasonably seamless. It just needs to be performed. As note previously, the model provided by Barlow is most helpful: The patient is assessed, then monitored, and followed over time. Nomothetic treatment is identified and applied. Problems are noted and, when the empirical input is limiting, person-based characteristics are

entered. Should change not be seen even then, the therapy can be altered, perhaps in the service of a functional analysis. The case formulation then includes ongoing monitoring, formulation, nomothetic EST input, consideration of the patient's characteristics, and on occasion a functional analysis.

Key Features of Case Formulation

- Understanding the relationship among problems
- Choosing treatment modality
- Choosing intervention strategy and point
- Predict behavior
- Understanding treatment compliance
- Understanding working relationship
- Making decisions about extra therapy issues
- Redirecting an unsuccessful treatment

Integrative evidence-based and case formulation-guided psychotherapy are models. In this process, the healthcare provider gathers information and formulates a case, treats, and then looks and monitors over time. The formulation part of this understanding of the case formulation involves creating a problem list, diagnosing, developing explanatory hypotheses, and a plan of treatment itself. Creating a problem list and titrating that to interventions for each specific problem represent perhaps the core of this model. Consideration is given to how one forms a hypothesis. This is dynamic and subject to change. The steps that are involved in this involve identifying precipitants, identifying origins, identifying resources, identifying obstacles, and then stating a core hypothesis regarding this complex procedure.

Case Formulation Issues

1. Just as with generating a problem list, one must consider multiple sources of information, including the comprehensive interview.
2. The diagnosis should flow directly and logically from the problem list. The problem list can be used to assess the presence or absence of specific diagnostic criteria.
3. Pay careful attention to the specific criteria of diagnostic categories. DSM-V explicitly states the specific diagnosis criteria to be used as a guide and not as an absolute requirement for assigning a diagnosis.
4. Be mindful of the potentially harmful and beneficial aspects of a diagnosis. Receiving a diagnosis can have great relief to some, but also can be a hassle and great problems to others.

5. Do not mistake a diagnosis for an explanation. One must be cautious when explaining the person's problems by means of a diagnosis. A simple diagnosis itself is not an explanatory, but merely a category, a noun, a label for interrelated experiences.

Follow-up is implied. It is a lost art. Over the years, service organizations have attempted to apply acronyms to these: PIE model that includes problem identification and evaluation; the SOAP model that involves subjective, objective, assessment, and plan; and the DAP model, which includes description, assessment, and plan in an effort to clarify what is being done and to have a process where there are follow-up and relapse procedures. This is in fact the essence of Watch and Wait: The game continues and continues.

That said, natural tensions permeate case formulation. One tension is between complexity and simplicity. Human behavior is complex and difficult to predict. A therapist cannot hope to fully capture the complexity of a case formulation, nor is there a need to do so since formulation is about a limited range of problems. At the same time, sufficient complexity is needed to serve the goals of treatment. Another tension exists between the therapist's bias and objectivity. No therapist can enter a course of therapy free of bias from personal values, feelings, judgments, and the influence of stereotypes. The therapist's own personal and culture history is in the room. Further, a persuasive body of research has demonstrated that we all are subject to systematic errors and judgment and reasoning.

One other tension is between observation and inference. By observations we mean theory-free descriptive evidence gathered by careful watching and listening. Inference is a conclusion formed on the basis of observation that may logically or reasonably follow from an observation and it may be guided by theory. Therapists may observe tears flowing down a client's face and infers that the patient is sad or, depending on the content, is guilty, feels unlovable, or is histrionic. Finally, a tension forms between the individual and general formulations. A case formulation is always by definition about a specific individual. It should take into account that individual's unique problem, life circumstances, and history stressors, wishes, hopes, goals, and so on. Yet a wealth of information has been generated about causes, characteristics, and the course of specific psychological disorders, problems, and stressors. Prototypic formulations provide useful hypotheses about the causes and maintaining influences in psychological disorders.

Goal Attainment Scale (GAS): This is an individualized approach to measure clinical outcomes that were first introduced by Kiresuk and

Sherman years ago for evaluating community mental health problems (Kiresuk & Sherman, 1968). It is a process of defining individual treatment goals at the outset and monitoring for goal attainment. Goals are comparable as results can be summarized using an individually-developed formula. GAS provides the means for comparing individuals of various stages and presentations of disease and it is adaptable across different populations and interventions as shown in trials of antidepressant drugs and cognitive rehabilitation.

The MINDZ Vital represents another study that applied the GAS. This is a multidisciplinary rehabilitation program comprising of once weekly group therapy sessions, each session lasting 3 hours over 8 consecutive weeks. These sessions involve 45 minutes of a multicomponent physical exercise program of light aerobic exercises varying in range of motion and resistance exercises, as well as balance training. In addition to a one-hour cognitive stimulation and rehabilitation with social and mental activities, such as reminiscence therapy, is 30 minutes of tailored individual activities delivering person-centered care. The use of a GAS in this process is highly effective.

Nonpharmacologic interventions, such as exercise and cognitive rehabilitation programs, have shown considerable promise in reducing the impact of dementia on individuals with multimodal cognitive and physical rehabilitation programs for persons with mild dementia and their caregivers using conventional measures of cognition, behavior, quality of life, and caregiver burden together with the GAS. In a study done by Chew et al. (2015), 34 patients were assessed to have met their GAS goals, mean GAS was improved. Cognition goals were set in only 20%, followed by goals to improve engagement and socialization, reduce caregiver stress, and improve physical functioning behavior and mood. This particular study provides evidence of a multimodal approach combining physical exercise and cognitive rehabilitation that can improve GAS, even assimilating caregiver burden in individuals and caregivers of persons with dementia.

The concept of GAS is relatively important: A person is at Point A and needs to get to Point D. How and what simple stages are needed for this to occur? This is very practical and grounded. As we will argue, lifestyles are often a part of this, as the person flows in a better direction of health. Small (2016) too endorses lifestyle as part of all case formulations. Available data support the integration of healthy lifestyle strategies in the treatment plan to help to stabilize symptoms and potentially delay future cognitive decline. While investigators continue to pursue more effective detection, treatment, and prevention strategies, the scientific data support the use of

symptomatic drug treatments and recommendations for healthy lifestyle behaviors to improve quality of life and potentially stave off future cognitive decline. Success of such healthy lifestyle programs involves educating participants on the connection between lifestyle and disease prevention, offering enjoyable exercises that target the patient's skill level, and providing. Regardless, the plan for a change is necessary.

Special Cases: We start with dementia. Components of a full evaluation can vary depending on the presentation and include tests to rule in or out the various causes of cognitive impairment and establish its severity. Diagnostic evaluations include a complete medical history; assessment of multiple cognitive domains, including episodic memory, executive function, attention, language, and visuospatial skills; a neurologic exam (gait, motor function, reflexes); ADL and IADL functioning; assessment for depression; and review for medications that may adversely affect cognition. Standard laboratory tests include thyroid-stimulating hormone (TSH), complete blood count (CBC), serum B₁₂, folate, complete metabolic panel, and, if the patient is at risk, testing for sexually transmitted diseases (human immunodeficiency virus, syphilis). Structural brain imaging, including magnetic resonance imaging (MRI) or computed tomography (CT), is a supplemental aid in the differential diagnosis of dementia, especially if neurologic physical exam findings are noted. An MRI or CT can be especially informative in the following cases: dementia that is of recent onset and is rapidly progressing; younger onset dementia (<65 years of age); history of head trauma; or neurologic symptoms suggesting focal disease.

In concert with our model, a design could involve a more N-of-1 model. N-of-1 trials consider the individual as the sole unit of observation to study the efficacy and adverse effects of an intervention and are guided by objective data-driven criteria while leveraging the study designs and statistical techniques common to RCTs. To create the platform for such trials, several conditions must be met. Galvin (2017) believes that participants must be deeply phenotyped with characterization of sociodemographic, psychological, clinical, cognitive, functional, biomarker, and genetic traits. Ideally, these individuals would agree to be followed longitudinally, have samples banked for future analyses, and consent to autopsy to provide confirmation of diagnosis and treatment effects on brain pathology. Statistical considerations may take advantage of alternative time-series analyses and within- and between-subject comparisons. In fact, N-of-1 trials are being developed to personalize dementia prevention. In addition to screening for cognitive impairment, broader medical screening for diabetes mellitus, hypertension, vascular risk factors, obesity, mobility,

physical performance, frailty, and depression and anxiety was incorporated into a "healthy body, healthy mind" approach to make the concept of dementia screening more acceptable and to understand the effect of comorbid disease on cognitive performance. We of course add cognition and life adjustment. These collective findings were prospectively applied to develop N-of-1 trials.

Typical Medical Workup

A 70-year-old woman presents with a 1-year history of subjective memory complaints (misplacing car keys, forgetting conversations, defensiveness about memory issues) but with independent functioning in everyday activities. Her relevant past history was significant for hypertension and hypercholesterolemia. Physical examination findings included mild hypertension (blood pressure 140/92 mmHg) but normal cardiac and peripheral vascular examinations. Pertinent neurological findings included mild symmetric weakness and mild postural instability. Quantitative magnetic resonance imaging (MRI) revealed normal hippocampal size and lateral ventricle volume but confluent white matter hyperintensities with frontal lobe predominance. Findings in cognitive testing (executive and working memory deficits with cued episodic memory improvements supporting intact hippocampal circuitry), physical testing (sarcopenia, at-risk nutritional status, poor physical functionality, and early frailty), gait testing (slowed gait speed, impaired dual tasks, postural instability with eyes closed), biomarker testing (lipid profile, inflammation, insulin resistance, ApoE4 genotype suggesting poor response to statins), MRI (preservation of hippocampal and cortical volume, extensive white matter disease), and electroencephalography (executive dysfunction and evidence of vascular injury) that could be treated and supported a diagnosis of vascular cognitive impairment (Calvin, 2017)..

We add the Watch and Wait model. A problem list is developed and treatment unfolds in a Watch and Wait form. Many of the problems are noted but cannot be changed. Clearly, the physical issues (sarcopenia, nutrition, physical functioning), gait training (strength training and falling exercises), biomarker issues (inflammation, statin alteration, and insulin resistance), cognitive training as well as any psychological sequelae, are targets. But, based on neuropsychological and psychological testing, we form a matrix for care – health, cognition, depression, anxiety, and life adjustment. This model suggests care and treatment.

Capacity: Next we briefly address capacity. This can be a unique assessment challenge. This applies to focal brain challenges, idiosyncratic neurodegenerative diseases, and issues like capacity. Capacity especially is a problem and is something that has not been addressed. This term is preferred now to the more traditional competence as there is now a greater focus on specific capacity through abilities; that is, medical decision making, financial decision making, and the like, as opposed to an overall determination of competence. Medical consent capacity or healthcare decision making capacities are defined usually by state statute. The definition of consent capacity found in these statutes are often similar to that of the Uniform Healthcare Decisions Act of 1993 (the American Bar Association Commission on Law and Aging and the American Psychological Association, 2008), which defines capacity as an individual's ability "to understand the significant benefits, risks, and alternatives to proposed healthcare and to make and communicate a healthcare decision." There is general agreement among experts of capacity assessment, that the assessment of medical consent capacity requires formal assessment of cognitive and functional abilities, a consideration of the causes of incapacity, for example dementia, and an analysis of the interaction of these elements with the support or lack thereof of the environment.

●f course, capacity is patient-specific and specific to targets (health, driving, decisions for living and other). It should not be all or none and rests best on the word "limited." It also assumes that the person will thrive in a non-competent status which implies that the environment (caregiver or guardian) will optimize the environment. Capacity should also be reconsidered routinely, especially when limitations are resisted.

In this context the idea of intellectual disabilities is of interest. Here there is often a lack of comparison standard for determining change. There is a large inter-individual variability with cognitive and functional abilities with a population of people that have intellectual disabilities, making use of normative group approach very difficult, if not completely inapplicable. Instead, each individual will present with a lifelong profile of strengths and weaknesses that will provide the basis for identification of change. The Working Group for the Establishment of Criteria for Diagnosis of Dementia in Individuals and Intellectual Disability (Burt & Aylward, 2000) recommends that an objective baseline level of performance be obtained when individuals with intellectual disabilities are cognitively healthy before the age at which risk increases. ●bviously, this is very difficult to do.

We recommend that intelligence be part of a total neuropsychological evaluation (Chapter 7). This is a powerful variable and has been located as the one marker that accounts for a substantial amount of the variance of the decline process. Newer models (e.g, P-FIT) even suggest that the person be assessed according to the brain area that best subserves the function of the person. Overall general intelligence (“G”), however, can be limiting and reflect a poorer summary of the person’s situation. Brain reserve is often equated with intelligence. More on this later.

Watch and Wait Model

Overall: We now consider assessment processes that set the foundation for the Watch and Wait model. As a start, we know that psychiatric comorbidity is the norm rather than the exception in virtually all clinic settings. Therapists increasingly must bring creativity to assessing and treating patient problems. As people in clinical practice can attest, symptom clusters across disorders often are not mutually exclusive and individuals seeking therapy frequently present with co-occurring conditions that pose significant challenges when trying to develop optimal treatment plans. For example, a person seeking treatment for depression might present with accompanying anxiety, panic attacks, and a history of trauma and may resort to self-harm as a means of regulating overwhelming emotions. Identifying which diagnostic hypotheses to consider and which factors, psychological or otherwise, might be driving individuals’ presenting problems can be daunting indeed.

As part of the Watch and Wait model, structured and friendly assessment reigns. In Hyer (2014) and Chapter 1 (and Chapter 4) we argued for the necessary components of this model that included a validation of the problem (“Your problem certainly makes sense”); psychoeducation (“Here’s what you have and what we propose we do”); assessment (“We will ask that you undergo some questions and self-report scales”); monitoring (“And we will follow over time”); case formulation (“Here is the plan”); and alliance (“I want your input on how we are doing”).

We emphasize context. As discussed, any score reflects an actual behavior marker plus measurement error, along with the effects of score bias, error in the prediction of premorbid ability, fatigue, poor motivation, pain, and the vagaries of psychiatric problems. Again, we are interested in the identification of most persons with a reasonable degree of impairment: False positives are acceptable (to a reasonable degree). We are interested in clinical assessment and less actuarial assessment. Low

scores are not to be interpreted without context; education is always important; overgeneralization and confirmatory bias are ever-present; tests over time are more persuasive; absence of evidence is not evidence of absence; and salient data that is dramatic is not persuasive. And, we stress that this is assessment based on the initial evaluation of the person, the screen and its minions. For testing, the rules apply regarding test selection, (reliable and specific to situation), comparison of test scores, the process of identification of test scores that are rare and reflect abnormality, as well as individual patient characteristics.

Recall something more. Luria, the famous Russian neurologist, targeted individuals and used his model to assess an individual clinically. He was interested in a qualitative analysis and description. The emphasis was on behaviors of the person. He developed stages of concern for brain problems and clinical tools to unearth these. We have a system that mimics Luria. It walks a balance between a battery and pure clinical data. It relies to some extent on a formal psychometric approach but emphasizes the need to tailor the assessment to the nature of the particular person's difficulties. Batteries are too inefficient and pure clinical work too undefined. Use of many tests/ratings and multiple sources of information work best. This again falls short of algorithms but seeks reasonable sensitivity and specificity with a population where the false positive is less a problem.

●*Operational Specifics, Screen:* We propose a core battery for older adults that address the components of our model. From that scaffold we branch out and target each area independently with additional tests and interview data. We recommend then a two tier model of assessment. Initially we provide a good-enough short battery, a screen. This is intended to provide information to pursue the target area under evaluation in the context of the other markers. Second, we discuss more in-depth assessment that delves into the target area and hopefully allows for added value and better information on necessary designation and treatment. We do this only on the domains at risk. ●f course it is not necessary to apply all measures of the complete battery. In the ensuing chapters we provide a screen for each domain. The screen then efficiently and with reasonable validation sets the stage for further assessment and the Watch and Wait process.

There has even been a debate as to whether to screen for dementia. Not the least of the issues is that the complexity of the diagnosis, ethnic and cultural issues, issues of brain reserve, and treatment benefits. We note that screens do not diagnose: They Watch and Wait. We ask the logical question: Is this area a problem? Additionally, it asks: What exactly needs

to be tested; is it memory (for AD); is it executive function (for vascular or fronto-temporal dementia); is it a single target or multiple; what is the level of functioning or social interaction. In effect, how do we measure such complexity?

As discussed, we have developed an assessment battery that includes a rating for each domain of Problem, Moderate, or Mild/Normal. The domain(s) labeled Problem requires immediate attention and are causing problems in the person's life now. Moderate ratings require attention also but is less emergent and less intense than Problem. Mild indicates a watch and wait mode but always view this area as an influence on the core problems. Normal is part of Mild. We do not make a distinction here, but allow for its influence in a more positive manner. The problem is rated on the basis of the screen and later may be altered by more intensive evaluations. Again, this rating is arbitrary but based on screen markers anchored in psychological tests, ratings or behaviors. The focus is on identifying a domain(s) that needs attention in the context of other relative domains. We apply a 1-10 metric with $>$ or $= 8$ as a Problem, 5-7 as Moderate, and < 5 as Mild or Normal.

As noted, screens have limits. They do not form a diagnosis. They do not tell us about the quantity of the problem. They also do not reflect the strengths either. From our vantage point, this situation is considered adequate if the cost of a false positive or false negative is minimal— where there are no serious implications or negative treatments. This does occur but less so with older adult where problems related to cognition, depression, anxiety, and life adjustment are more global and tangible. It can be a more serious matter in health where a medication can be very helpful or dangerous. But health interventions are done largely in the context of the PCP and the medicines involved. Clinicians need to be aware of this, perhaps organize this, and always monitor it.

There are always unique problems. We have already mentioned intellectual disability and person variability. This is a specific challenge with older adults. There is a large inter-individual variability in cognitive and functional abilities, especially in a population of people with intellectual disabilities, cognitive and functional abilities. Each individual presents with a lifelong profile of strengths and weaknesses that provide the basis for the identification of change (Krinsky-McHale & Silverman, 2013). Monitoring over time and a good history are two ways to fineness this. This allows for a more ipsative comparison (within the person) over time to assist in a more accurate detection of decline.

Treatment: Treatment follows. From a big picture perspective, change is the reason for treatment. Remission is elusory. Perhaps the best model

is the often used Hamilton Depression Scale (HAM-D). Remission means that the patient no longer meets criteria: In the case of the HAM-D, the post score is < 8 . A response is $\frac{1}{2}$ of baseline, a partial response $< 30\%$ but $< 50\%$, and a relapse and recurrence is between $50-60\%$ depending on previous episodes. Depression and anxiety are relapsing disorders and somatic problems are chronic. Dementia is....well, forever.

This means that the therapist is involved centrally for a long period. Assessment is part of therapy, and we may add, that therapy is part of assessment. Norcross, Hogan and Koocher (2008) recommend that the therapist apply an assessment approach consistently to treatment over time. Perhaps above all the focus is on practical issues; what needs tending to now, what can be accepted and will this intervention foster the longer term Watch and Wait process. Therapeutic questions involve some of the following.

Questions that help patients

- What is the biggest problem?
- What needs to be tackled first?
- What is your primary goal?
- How will you know when you are getting better/solved the problem?
- What will you have to change about yourself?
- What would prevent you from attaining the goal?
- What positive/negative things are you doing?

Watch and Wait opts for a flexible delivery of interventions without compromising effectiveness. We have a multi-step model of treatment (Chapter 4). This involves individualizing interventions while preserving the principles underlying treatment and includes providing training and support for clinicians and monitoring patients' progress. This entails a triage skill with applying interventions and the components that can be implemented or omitted as needed to address presenting problems. This promotes flexibility without violating the treatment integrity. Caregiving of course is a big part of this and we address this issue throughout the book.

Conclusion

We are in a paradigm shift in the care of older adults. Decline represents a teachable moment. We know what needs to be done but how to actually do this and how to integrate it are issues. We also need to bring people in (sensitivity trumps specificity) and get them on board and then

committed. The core question is: What is best for you. The evaluation of the older adult who complains of a problem is the start for clinicians to unpack noise and complexity.

In this chapter we argued for a loose assessment based on the case formulation in the context of Watch and Wait. Assessment needs to be “good enough” and reasonably valid using many forms of assessment, especially psychological testing. The argument was not made but could have been that each area has a flexible parameter that can slide up or down for a “hit.” Importantly, error is built into the system as most problems confess themselves quickly. Accounting for each domain is ambitious and involves time and care. And, we note too that the specific domain model changes routinely. We unfold this now.

References

- Åhlund, K., Bäck, M., Öberg, B., & Ekerstad, N. (2017). Effects of comprehensive geriatric assessment on physical fitness in an acute medical setting for frail elderly patients. *Clinical Interventions in Aging, 12*, 1929–1939. <http://doi.org/10.2147/CIA.S138357>
- Antony, M. M., & Barlow, D. H. (Eds.). (2011). *Handbook of assessment and treatment planning for psychological disorders*. Guilford press.
- Ardila, A. (2013). A new neuropsychology for the XXI century. *Archives of clinical Neuropsychology, 28*(8), 751-762.
- Burt, D. B., & Aylward, E. H. (2000). Test battery for the diagnosis of dementia in individuals with intellectual disability. *Journal of Intellectual Disability Research, 44*(2), 175. Retrieved from <http://medlibproxy.mercer.edu/login?url=https://search.ebscohost.com/login.aspx?direct=true&db=a9h&AN=6132920&site=eds-live&scope=site>
- Ariely, D. (2010). *Predictably irrational: The hidden forces that shape our decisions*. Harper Perennial.
- Barkley, R. A. (Ed.). (2014). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment*. Guilford Publications.
- Bickman, L., Kelley, S. D., & Athay, M. (2012). The technology of measurement feedback systems. *Couple and Family Psychology: Research and Practice, 1*(4), 274.
- Bobbitt, B.L., Cate, R.A., Beardsley, S.D., Azocar, F., & McCulloch, J. (2012). Quality improvement and outcomes in the future of professional psychology: Opportunities and challenges. *Professional Psychology: Research and Practice, 43*(6), 551-559.

- Boone, K. B. (2009). Fixed belief in cognitive dysfunction despite normal neuropsychological scores: Neurocognitive hypochondriasis?. *The Clinical Neuropsychologist*, 23(6), 1016-1036.
- Borsboom, D. (2006). The attack of the psychometricians. *Psychometrika*, 71 (3), 425,
- Bosworth, H., Blalock, D., Hoyle, R., Czajkowski, S., & Voils, C. (2018). The role of psychological science in efforts to improve cardiovascular medication adherence. *American Psychologist*, 73, 8, 968-980.
- Campbell, D., & Fiske, D. (1959). Convergent and discriminant validation by multitrait-multimethod matrix. *Psychological Bulletin*, 56(2). <http://doi.org/10.1037/h0046016>
- Chelune, G. J. (2010). Evidence-based research and practice in clinical neuropsychology. *The Clinical Neuropsychologist*, 24(3), 454-467.
- Chew, J., Chong, M. S., Fong, Y. L., & Tay, L. (2015). Outcomes of a multimodal cognitive and physical rehabilitation program for persons with mild dementia and their caregivers: a goal-oriented approach. *Clinical interventions in aging*, 10, 1687.
- Cordell, C. B., Borson, S., Boustani, M., Chodosh, J., Reuben, D., Verghese, J., ... & Fried, L. B. (2013). Medicare Detection of Cognitive Impairment Workgroup Alzheimer's Association recommendations for operationalizing the detection of cognitive impairment during the Medicare Annual Wellness Visit in a primary care setting. *Alzheimer's Dement*, 9(2), 141-150.
- Dean, A. C., Victor, T. L., Boone, K. B., Philpott, L. M., & Hess, R. A. (2009). Dementia and effort test performance. *The Clinical Neuropsychologist*, 23(1), 133-152.
- Duff, K., Beglinger, L. J., Moser, D. J., Schultz, S. K., & Paulsen, J. S. (2010). Practice effects and outcome of cognitive training: Preliminary evidence from a memory training course. *The American Journal of Geriatric Psychiatry*, 18(1), 91.
- Edelstein, B. A., Drozdick, L. W., & Ciliberti, C. M. (2010). Assessment of depression and bereavement in older adults. In *Handbook of Assessment in Clinical Gerontology (Second Edition)* (pp. 3-43).
- Eisman, E.J., et al. (2000). Problems and limitations in using psychological assessment in the contemporary health care delivery system. *Professional Psychology: Research and Practice*, 31(2), 131-140.
- Fennig, S., et al. (1994). Best-estimate versus structured interview-based diagnosis in first-admission psychosis. *Comprehensive Psychiatry*, 35(5), 341-348.

- First, M. B., Spitzer, R. L., & Gibbon, M. (2004). *Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I), Clinician Version*. Hoboken.
- Garb, H. N. (2005). Clinical judgment and decision making. *Annual Review of Clinical Psychology, 2005, 1*, 67-89.
- González-Palau, F., Franco, M., Toribio, J.M., Losado, R., Parra, E., & Bamadio, P. (2013). Designing a computer-based rehabilitation solution for older adults: the importance of testing usability. *PsychNology Journal, 11*(2), 119-136.
- Greenway, M.C., Lacritz, L.H., Binegar, D., Weiner, M.F., Lipton, A., & Collum, M.C. (2006). Patterns of verbal memory performance in mild cognitive impairment, Alzheimer disease, and normal aging. *Cognitive Behavioral Neurology, 19*(2), 79-84.
- Gross, A. L., Rebok, G. W., Unverzagt, F. W., Willis, S. L., & Brandt, J. (2011). Cognitive predictors of everyday functioning in older adults: Results from the ACTIVE cognitive intervention trial. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 66*(5), 557-566.
- Groth-Marnat, G. (2009). *Handbook of psychological assessment*. John Wiley & Sons.
- Gupta, M. (2013). Comparative impairments across schizophrenia and bipolar disorder. *Cognitive impairment in schizophrenia: Characteristics, assessment and treatment, 50*.
- Ivnik, R.J., et al. (1999). Testing normal older people three or four times at 1- to 2-year intervals: Defining normal variance. *Neuropsychology, 13*(1), 121-127.
- Ivnik, R.J., et al. (1992). Mayo's older Americans normative studies: WAIS-R norms for ages 56-97. *Clinical Neuropsychologist, 6*(sup001), 1-30.
- Jak, A.J. et al. (2009). Quantification of five neuropsychological approaches to defining mild cognitive impairment. *American Journal of Geriatric Psychiatry, 17*(5), 368-375.
- Kahneman, D. (2011). *Thinking, fast and slow*. Macmillan.
- Kiresuk, T. J., & Sherman, R. E. (1968). Goal attainment scaling: A general method for evaluating comprehensive community mental health programs. *Community mental health journal, 4*(6), 443-453.
- Krinsky-McHale, S. J., & Silverman, W. (2013). Dementia and Mild Cognitive Impairment in Adults with Intellectual Disability: Issues of Diagnosis. *Developmental Disabilities Research Reviews, 18*(1), 31-42. Retrieved from

- <http://medlib-proxy.mercer.edu/login?url=https://search.ebscohost.com/login.aspx?direct=true&db=eric&AN=EJ1026292&site=eds-live&scope=site>
- Krober, A., & Kluckhohn, C. (1952). *Culture: A Critical Review of Concepts and Definitions*. Cambridge, MA: The Museum.
- Kubiszyn, T.W. et al. (2000). Empirical support for psychological assessment in clinical health care settings. *Professional Psychology: Research and Practice, 31*(2), 119-130.
- Lambert, M. J., Harmon, C., Slade, K., Whipple, J. L., & Hawkins, E. J. (2005). Providing feedback to psychotherapists on their patients' progress: Clinical results and practice suggestions. *Journal of clinical psychology, 61*(2), 165-174.
- Langkass, T., Wampold, B., & Hoffart, A. (2018). Five types of clinical difference to monitor in practice. *Psychotherapy, 55* 3, 242-254.
- Lindbergh, Dishman, and Miller (2006)
- Loewenstein, D. A., & Acevedo, A. (2005). *Training of cognitive and functionally relevant skills in mild Alzheimer's disease: an integrated approach*. Guilford Press.
- Loewenstein, D. A., Acevedo, A., Czaja, S. J., & Duara, R. (2004). Cognitive rehabilitation of mildly impaired Alzheimer disease patients on cholinesterase inhibitors. *The American Journal of Geriatric Psychiatry, 12*(4), 395-402.
- Mash, E.J. & Foster, S.L. (2001). Exporting analogue behavioral observation from research to clinical practice: Useful or cost-defective? *Psychological Assessment, 13*(1), 86-98.
- Mash, E.J. & Hunsley, J. (1993). Assessment considerations in the identification of failing psychotherapy: Bringing the negatives out of the darkroom. *Psychological Assessment, 5*(3), 292-301.
- Meyer, G.J., et al. (2001). Psychological testing and psychological assessment: A review of evidence and issues. *American Psychologist, 56*(2), 128-165.
- Newby, L., LaPoint, N., Chen, A., Kramer, J., Hammill, B, Delong, E.,... Califf, R. (2006). Longer term adherence to evidence-based secondary prevention therapies in coronary artery disease. *Circulation, 113*, 203-212.
- Norcross, J. C., Hogan, T. P., & Koocher, G. P. (2008). *Clinician's guide to evidence-based practices: Mental health and the addictions*. New York, NY, US: Oxford University Press.
- Parsons, T. & Duffield, T. (2019). National Institutes of Health initiatives for advancing scientific developments in clinical psychology. *The Clinical Neuropsychologist, 33*, 246-271.

- Ridley, C. R., Kelly, S. M., & Eells, T. D. (2007). Multicultural considerations in case formulation. *Handbook of psychotherapy case formulation*, 33-64.
- Royall, D.R., et al. (2002). Executive control function: A review of its promise and challenges for clinical research. *Journal of Neuropsychiatry and Clinical Neurosciences*, 14(4), 377-405.
- Royall, D.R., et al. (2007). The cognitive correlates of functional status: A review from the Committee on Research of the American Neuropsychiatric Association. *Journal of Neuropsychiatry and Clinical Neurosciences*, 19(3), 249-265.
- Satterfield, Jason (2015). *Cognitive Behavioral Therapy: Techniques for Retraining Your Brain*, The Teaching Company, San Francisco, California.
- Schmand, B., Huizenga, H. M., & Van Gool, W. A. (2010). Meta-analysis of CSF and MRI biomarkers for detecting preclinical Alzheimer's disease. *Psychological medicine*, 40(1), 135-145.
- Small, G. W. (2016). Detection and prevention of cognitive decline. *The American Journal of Geriatric Psychiatry*, 24(12), 1142-1150.
- Smith, G.E., et al. (1992). Mayo's Older Americans Normative Studies (MONANS): Factor structure of a core battery. *Psychological Assessment*, 4(3), 382-390.
- Smith, G.E., et al. (1994). Mayo cognitive factors scales: Derivation of a short battery and norms for factor scores. *Neuropsychology*, 8(2), 194-202.
- Stewart, R. E., Chambless, D. L., & Stirman, S. W. (2018). Decision making and the use of evidence-based practice: Is the three-legged stool balanced? *Practice Innovations*, 3(1), 56-67. <https://doi-org.medlib-proxy.mercer.edu/10.1037/pri0000063>
- Thomas, J. A. (2012). *Effect of physical activity on the cognitive performance of middle aged New Zealand workers* (Doctoral dissertation, Auckland University of Technology).
- Tuokko and Smart, (2018) – *Neuropsychology of Cognitive Decline: A Developmental Approach to Assessment and Intervention*; Guilford Press, New York.
- Turchik, J.A., Karpenko, V., Hammers, D., & McNamara, J.R. (2007). Practical and ethical assessment issues in rural, impoverished, and managed care settings. *Professional Psychology: Research and Practice*, 38(2), 158-168.
- Tversky, A., & Kahneman, D. (1974). Judgment under uncertainty: Heuristics and biases. *science*, 185(4157), 1124-1131.

- Wakefield, J.C. (Barron, J.W., Ed) (1998). Making diagnosis meaningful: Enhancing evaluation and treatment of psychological disorders, (pp. 29-72). Washington, DC, US: American Psychological Association.
- Wampold, B. E. (2001). Contextualizing psychotherapy as a healing practice: Culture, history, and methods. *Applied and Preventive Psychology, 10*(2), 69-86.
- Wolfe, A. (2001). Institute of Medicine Report: crossing the quality chasm: a new health care system for the 21st century. *Policy, Politics, & Nursing Practice, 2*(3), 233-235.

CHAPTER 4

WATCH AND WAIT: PERSPECTIVE AND PRACTICAL ISSUES

The preliminaries are over and it's time for therapy. Here we discuss the particulars of psychotherapy from the Watch and Wait perspective. We also explicate the model more fully. In the subsequent chapter we will consider the process of what we will have discussed for each domain.

The species we are attached to is very fickle, but very consistent in making progress regarding longevity and better quality of life. Longevity depends largely on one's commitment to life, largely at middle age but also at later life. In this effort the Watch and Wait model requires the recipient to experience quiet but solid information, reasonable validation, support and a direction. Problems percolate against a backdrop of general degeneration. A cure is not on the horizon. Despite the large variety of compounds and therapies available as candidates for support, their mechanisms of action are few: general neuronal health, conventional receptor agents, tau hyper-phosphorylation, and amyloid. Gary Small indicates that the problem with a drug in development is that it is not specific to the as-yet unidentified primary mechanism that underlies AD neurodegeneration (Small & Greenfield, 2015). The process that accounts for the cell selectivity characterizing the disease is unknown.

So, life unfolds slowly and can be extended. It is likely to persist with debilities. Life spent with debility/cognitive impairment varies by age and can be as much as 10-15 years. The combination of Education/Exercise/Diet/Stress reduction reduces this measurably, more so for women. This is then the timeline of decline. We have seen that dementia is in decline. In the US the prevalence of dementia has dropped from 11.6 percent for people over 65 to 8.8 percent in 2012 (Langa et al., 2017). That said, the number of deaths attributed to dementia is rising. Importantly, this is now a disease that takes time to unfold, and is *psychological in nature*. The brunt of the diagnosis and treatment is behavioral and psychological. Despite inhalers, sprays, vaccines, more targeted monoclonal antibodies, and new elegant molecules, for now real care is best done by prevention. Effective medicaments are anemic or off

in the distance. As if this were not bad enough, we have learned also that dementia is (usually) caused by a mixture of pathologies (Loprinzi & Joyner, 2017). The mess is such that people (as many as one third) who die with no apparent symptoms of impaired cognitive impairment have AD pathology in their brains, enough to meet criteria for AD itself (Jansen et al., 2018). Cognitive reserve and MCI, the two opposing faces in the dementia arena, have actually masked the nuanced diagnosis itself. When the brain alters, however, behavioral change rushes in. In fact, the bottom line is not so much what your biomarkers (plaques/tangles, amyloid positive, APO-E) are, but what your memory, attention, and executive functioning reveal.

Behavioral stuff matters! Exercise matters and alters BDNFs, serotonin levels, vascular health, well-being, and depression, and may even prevent plaques and tangles from formation (Stern, 2012). As noted, this is best done at mid age and done with verve and consistency. Additionally, people who deal with more complex work, who have skilled or professional jobs, have a formal education (at least 9 years), and practice several healthy habits (Med diet, no obesity, not smoking, reasonable health, sleep well, stress free life (good luck!)), and are reasonably “good people” (altruism/gratitude, socialization, practice religion, meditation, volunteer) seem to be protected at least for a while.

This is especially important, given that we seem to be functioning better and better over time. Among older adults with possible dementia, many are still driving, cooking, managing finances, doling out their medications, and attending doctor’s appointments alone (Amjad, 2016). Function interacts with cognition at ~40%. A decline in function is progressive, from IADLs to ADLs. In addition, rarely is it the case that problems or strengths work alone. This is especially so for cognition where you may get away with just memory problems but if you have more than one chronic problem, they multiply. This applies to chronic diseases where greater numbers of problems geometrically accrue over the lesser numbers (2 or 3 are considerably worse than 1).

Thoughts and emotions are mediated by the limbic, immune, endocrine, and nervous systems. This is a biochemical event. The mind and body can create health daily. Unfortunately we are resistant to good lifestyle change. Good diet, moderate exercise, good body fat percentage, and being a non-smoker are four variables that account for the best health: only 2.7% of Americans in one sample achieve all 4; 16% had 3; 37% had 2; and 34% had 1; 11% had 0 (N=4745). Lifestyle habits are comparable to the biomarkers of cardiovascular health (Goeres et al., 2016). A balance of positive and negative genetic factors affects the brain in early/middle

life to determine the degree of cognitive agility or impairment at late life. These factors increase or decrease oxidative stress, inflammation, insulin signaling components, the size and frequency of infarcts, and the concentration of growth factors, cortisol, and other hormones. Optimal heart health in middle age allows one to live up to 14 years longer, free of cardiovascular disease, than peers who have two or more cardiovascular disease risk factors. This especially involves smoking (Nash et al., 2016).

Breakthroughs in the history of aging have been many and impactful. We have gained years of longevity per decade with all sorts of life assists over the last 50 years. This includes the substantial changes in culture and its friendly and empowering role in the psychology and, in turn, the biology of aging. Older people are not only living longer but are smarter, healthier, more mobile, and more positive than BOTH themselves in years before and other groups who reached their age. Notably, when the initial change occurs, the next alterations accrue geometrically and much happens quickly. It has been argued that the norms of 80 year olds in 1980 are outdated for 80 year olds in 2020. In flight, after 1903 the mark of the first flight, we progressed later to the first transatlantic flight, to the onslaught of commercial planes, to supersonic machines, space adventure, and beyond; all this changed in less than 100 years. Change proceeds rapidly after an initial change. This applies also for health. de Grey applies this to age: the “longevity escape velocity” where we can “youthen” at middle age, but cannot do much at 100. Middle age people can remain vibrant at that age for a much longer period. A considerable amount of biology is bearing fruit, from FOXO to rapamycin; much of it is enhanced (or made worse) by lifestyle.

Medical advances have clearly made an impact. Better treatments with new drugs were not available a generation ago. As noted, the average American born today can expect to live years longer than a person born two decades ago. Despite all these new technologies, however, the question is: Given our increased life expectancy, do we really add additional active and healthy lives? The answer to this question is complex. At the phenomenological level, engagement in lost possible selves is negatively related to happiness. Just the possibility of exploring such possible and positive experiences may indeed have important implications for valuable outcomes. Participants in many studies have taught us that the very experiences that marked us may become a source of unexpected strength and play a role in the creation of more differentiated and integrated cells. Heck, just believing in the positive possibilities of age can be an important factor.

In this chapter we discuss therapy, more precisely psychotherapy. We present a biased overview on the merits of the psychotherapies for the various domains. Our goal is to provide a foothold for the second part of the chapter. We pay special attention to lifestyle elements. Personality is also addressed. Next, we discuss our model of psychotherapy for older adults. We end with special thoughts on dementia.

Psychotherapy

We consider psychotherapy in general emphasizing the alliance and common factors, as well as common sense. We extend and elaborate on Chapter 1. We then consider a model that applies this transdiagnostic model. We continue with the importance of lifestyle and its components.

Recall that our position is that treating one domain in isolation detracts from the care of the whole person, especially cognitive and physical limitations, risking a slower or less-effective reduction in depressive symptoms. Targeting psychological problems, in this case depression, anxiety, cognitive impairment, adjustment, and some disability, provides a concerted focus to help the patient adapt and cope with their problems, promoting successful outcomes for the prime target, depression. This requires deliberation (case formulation) and follow-up. Evidence for such multi-faceted approaches to treatment is just not present, and so we must borrow from evidence on each problem and look at the efficacy of treatments for older adults, as well as what works for younger adults. While best care is case-based, with older adults there is more. The reality of older life and the psychological issues herein unfortunately conspire so that there are greater needs for better assessment and treatment.

Psychotherapy meanders in “dark places.” We will not dwell on this but to highlight that there have been several civil wars among the schools of psychotherapy that are not so much resolved as they have accommodated to the realities of the 21st Century. Psychotherapy use is down and medications are efficient. Cynical views of psychotherapy persist.

- “Therapy is what we say it is.” Paul Watzlawick.
- “Psychotherapy may be the only treatment that creates the illness that it treats.” Jerome Frank
- “Sometime in the next century there will be a form of psychotherapy for every adult in the western world.” Sol Garfield

- “Can we make a relatively unhappy person happy.” Marty Seligman

We build, however, on the more common sense models of what therapy is. Jerome Frank (again) called it a rationale that facilitates healing. It is persuasion. Marty Seligman calls it a conversation with power. David Bums labels it as a state of wills -- What is the patient willing to do? The most important thing in therapy is that people are accountable. Psychotherapy is then malleable but seeks connection and change; the processes toward this end are infinite.

More specifically, we seek a common sense model that is even handed and appealing (common factors, technical eclecticism, assimilative integration) in the application of functional care principles. All social- and psycho-therapies can be put on the table but, in addition, the therapist becomes practical and real – patients have real needs, both health and life adjustment. They become (horrible dictum!) nurses and social workers: in a phrase, “holistic therapists.” The challenge of getting older is to strike a balance between fighting disabilities and diseases, and coming to terms with the limitations they impose. This battle will change again and again.

Always, an alliance between and engagement by the patient and reality trump strategies and tactics. Recall that psychotherapy entails a few necessary and often sufficient conditions for change. All forms of therapy address two features at least: a new understanding and a new experience. The therapist must validate the reality of the symptom, develop a functional explanation of the problem, suggest a script (care plan), and emphasize a lack of harm and a vision for change (get commitment to change). As noted before, success depends on the extent to which treatment matches shared social constructions about what it means to get better, to be remoralized or cured in a given culture. In central ways this depends on the conviction of the patient that the therapist cares and is competent.

Every person is an “n of 1” experiment. Each has a unique history involving a set of experiences, genetic makeup, set of functional limits, network of family relationships, response to medications, spirituality, and complex of medical conditions. Interventions are always a combination of some science, some clinical experience, and a moderate amount of inductive reasoning. Often this makes the practice of empirically supported care like climbing a greased pole. Success is hard fought, if achieved at all. There are of course some truths – necessary validation, some science, education, willingness to comply, and a range of therapies that must/will be tried and monitored. Also, other domains like depression

are often problematically at the center of multiple treatment decisions and pitfalls.

The treatment phase of each domain is directed by a loose form of technical eclecticism, strategies being selected from many psychotherapies and social interventions. Symptoms can be addressed with medications, structure and support, specific behavioral tasks, and containment interventions. With most patients, excepting those with a neurodegenerative disorder, the initial phase of therapy is engagement and a fostering of connection to therapy goals. Often the goal is containment, a holding on to skills and a reduction of defeating behaviors. Later life patients are not generally in need of self-identity alterations, but may well need interpersonal help, as well as behavioral activation or extinction. That said, the increasing of immediate awareness of the present condition and “self” is very much in play, to be followed by the pursuit of new experiences, interpersonally and personally. Often there are toxic self-states that, when psychoeducation and behavioral change is initiated, are reduced. Again, with cognitively impaired patients (as well as others), the caregiver is involved.

Watch and Wait opts for a flexible delivery of interventions without compromising effectiveness. This involves individualizing interventions while preserving the principles underlying treatment and includes the providing of training and support for clinicians and the monitoring of patients’ progress. This entails a triage skill with the application of interventions and those components that can be implemented or omitted as needed to address the presenting problems. This promotes flexibility without violating treatment integrity. Again, caregiving is often a big part of this and we address this issue throughout the book.

The issue where outcome is concerned is never just symptom abatement. Lifestyles by themselves account for much of the variance of quality of life (QoL). Therapy should aim at symptom relief and improving QoL. Moreover, although evaluation questions necessarily focus on the reasons why an elder is seeking treatment, such a narrow focus is only helpful for limited targets; for understanding process changes over time or other longer term and broader concerns, more is demanded. In translating ESTs to older adults, and in targeting this to the most researched psychotherapy, CBT, we must concentrate on more general outcomes (not just diagnosis), as well as specific markers associated with the identified problem. With older adults, the therapy also demands a scientific attitude, a skillful and flexible delivery of services, quantitative monitoring of the client’s progress, and an awareness of the personal,

interpersonal, and cultural characteristics of the client as well as core themes.

Unfolding Wisdom and The Relationship

“It is unwise to be too sure of one’s own wisdom. It is healthy to be reminded that the strongest might weaken and the wisest might err.”

Mahatma Gandhi

Not surprisingly, we endorse strongly the importance of the therapist-client relationship. Norcross and Lambert (2018) remind us that the dissemination and implementation of change in therapy come as necessary components of evidence based psychotherapy or medical care. The integration of the technical and relational is key. The Practice Recommendations follow. They heavily endorse the implementation of the fostering of the relationship. We endorse this again and again in this book.

Practice Recommendations

Make the cultivation of the therapy relationship a primary aim of treatment

Assess relationship (alliance, empathy, and cohesion)

Adapt or tailor psychotherapy to transdiagnostic characteristics that support change

Attune psychotherapy to culture specificity

Monitor patients’ satisfaction with relationship and response to treatment

Use EBTs adapted to the whole patient

Transdiagnostic Thinking

As noted in chapter 1, what we are advocating is transdiagnostic in nature. In a unique portrayal of this model, Shaw et al. (2015) identified five areas for technological applications to important life domains. These include physical and mental health, mobility, social connectedness, safety, and everyday activities and leisure. This treatment model applies maximally in situations with comorbidities and is based on distilling principles of treatment rather than developing a new therapy prototype. More importantly, a transdiagnostic perspective really constitutes the basis of a functional approach to treatment that transcends the limits of categorical classification, such as are presented in the DSMs. It helps synthesize comorbidities across theoretical constructs underlying treatment

models, and transforms diagnoses into a coherent, integrated, and parsimonious system. This potentially leads to a more expedient, practical, and efficacious treatment of psychopathology in general. These transdiagnostic perspectives reflect an appreciation that common psychological problems drive different clinical disorders. However (as noted in Chapter 1), they do not offer a truly individualized and comprehensive approach to understanding and treating the person behind the presenting problem. Case formulation models do offer this, although they offer little direction on how to identify specific mechanism hypotheses within the broader range of options that have been established via empirical research and psychological mechanisms and the treatments that target them.

Additionally, Frank and Davidson (2014) specify transdiagnostic mechanisms that are applicable to most diagnoses. This transdiagnostic roadmap is then a case formulation approach rather than a standalone treatment. The clinician can emphasize its strengths in allowing the therapist leeway in incorporating interventions from CBT, DVT, ACT, and other ESTs into treatment plans that are tailored to the individual. This makes a big difference in terms of efficacy, although there are no randomized trials substantiating this effect.

Frank and Davidson also specify treatment interventions that appear optimal for the transdiagnostic approach. These include interventions that enhance understanding and motivation, such as psycho-education, cost benefit analysis, identifying values, and conversations about ambivalence and motivation. It also includes interventions that facilitate a step back from the problem, such as problem solving, monitoring, mindfulness, acceptance and validation, and cognitive diffusion. In addition, core strategies for change are mentioned. These involve interventions noted below. We believe that this model has much to offer. More on this in other chapters.

Transdiagnostic Factors

- Typical interventions
- behavioral activation, behavioral contingencies,
- cognitive restructuring, behavioral experiments,
- attentional training techniques, exposure,
- building of distress tolerance skills, emotional regulation skills,
- interpersonal effectiveness skills.
- Skills training.
- progressive muscle relaxation or relaxation in general,
- guided anger management, problem solving,

- organization and planning, time management,
- sleep management, strategies for eating,
- and strategies for body focus.

Frank & Davidson, 2014

Lifestyle Components

Mental health professionals have significantly underestimated the importance of lifestyle in the etiology and change potential of problems, both of health and quality of life. Importantly, healthy behaviors have a secondary effect, as their value spreads to other behaviors and longer lives. Positive lifestyle markers have few negatives and have substantial secondary benefits. In fact, if one focused only on health, the emphasis would be on good cardiovascular care (blood pressure, cholesterol, small vessel disease), an endocrine watch (thyroid and pancreas especially), liver and kidney health, brain care (no TBIs), and good health practices (vaccines). This would be followed by behaviors, good sleep habits, exercise, a good diet, and joy in life. Now, in an aging world, these need to be followed judiciously.

There are many positive lifestyle factors. The table below represents the factors that are most represented in the literature for lifestyle change. They are highly related to the presence of chronic diseases and can be markers of a positive psychology. Of interest is that lifestyle factors should be shouted from the 40,000 foot level, where there is some specificity but more general research. They are clearly helpful and are touted consistently with regard to cardiovascular disorders, chronic immune disorders (like diabetes), COPD, pain, degenerative problems, and psychiatric problems. They are also highlighted in recommendations from several professional organizations, like the American Psychological Association. We believe then in the importance of lifestyles. This listing is empirically supported and has been identified as helpful, reasonably easy to employ, and in some cases fun.

Specific Good Life Habits

- Practice happiness
- Practice spirituality/religion
- Socialize: Less decline and live longer
- Altruism (increases dopamine and endorphins) and volunteer
- Develop resilience
- Practice sleep hygiene

- Exercise: Even housework
- Diet
- Somatic softening: Meditate and practice mindfulness
- Values and leisure
- Increase cognition: Take a college course or go on-line (lumosity, video games)
- Respect personality: Follow your passion!!!!!!!!!!!!

The first is to practice happiness. For starters there is no universal agreement on what happiness is. It is very much a self-perceived phenomena and often entails an accommodation of the interruption of normal life and suffering. It also seems to have a set point around which the person radiates and feels comfortable. It is a journey, a path, not a destination. Dan Gilbert noted that it took a million years to develop an experience simulator, an internal compass that sets life in the right direction. Humans are pretty good at this. Being upbeat seems to matter, lots.

Happy people synthesize good feelings internally. They make the best of a bad or neutral situation naturally. Pete Best lost his job with the Beatles after he missed a presentation and noted that this was a very good move for him. It has been estimated that one's personality and core traits are 50% genetic, 5% external, and 45% the province of the person. Barbara Fredrickson labeled this the Broaden and Build theory. This view allows one to have more clarity, engagement, creativity, and concentration. It can be learned but needs lots of practice.

In a Norwegian Study, the link between humor and mortality was assessed in 53,000 people over 15 years (Romundstad et al., 2016). Women had higher levels of humor, which was associated with 48% less risk of all-cause death, 73% less risk for heart failure, and 83% lower risk for infection. The argument is to provide the experience of humor, even faking it. The idea is to develop a positive attitude toward life. Between the stimulus and response there are a thousand ways to choose happiness (or sadness). The idea is to label the issue at hand, to expect it, and to tolerate the problem. This may involve a sense of optimism where the person feels confident that the rest of their life will turn out well. The person incorporates the Eastern idea that life is suffering and that that is to be expected. People with enough money, connections to people, safety, and emotional intimacy then better have a sense of happiness and personal growth. They can take realistic risks and can even compare themselves to others. It seems to work best when poor performance occurs. Mindfulness and ACT espouse these ideas.

In a previous chapter we noted the Million Women Study by Liu et al. (2016): During 10 years of follow-up, 4% (31,531) of participants died. Self-rated poor health at baseline was strongly associated with happiness. Happiness means attitude, positive attitude. The APOE gene, as noted, causes substantial problems, especially for those who are at other risks for AD. Not everyone with the gene develops AD, however, or other types of dementia. A number of studies have been done to determine why some people with APOE have AD and others not. While there is no definitive explanation, how people perceive getting older may be the key. The PLOS One study found that among people with an APOE gene, those who view aging with a positive and optimistic outlook have a lower risk of developing dementia than those with negative beliefs about getting older. In fact, those with positive attitudes about aging were nearly 50% less likely to develop dementia (author, Massachusetts General Hospital Mind, Mood, and Memory, 2018). Positive feelings also help reduce the stress that can interfere with concentration and memory and that causes one to engage in bad health habits, like smoking, getting enough exercise, and eating a poor diet. Being happy, therefore, seems to make a difference in your quality of life and mortality.

The solution: Don't worry, be happy! even if feigning it. This is one area where the commitment to "happy behavior" seems to influence the resultant feelings and behaviors. Faking it before making it, then, works. Note that a genuine smile may not reflect an inner state of cheerfulness or amusement. Behavioral Ecology Theory suggests that all smiles are tools used in social interactions; that theory claims that cheerfulness is neither necessary nor sufficient for smiling. Smiling then is not necessarily driven by happiness; it is associated with subjective engagement, which acts like a social fuel for smiling. Behaving happily seems to be better than not.

Number two is spirituality or religiosity. Prayer does many positive things. It has been shown to arrest cancer, to lower blood pressure, and to improve recovery from acute illness. Perhaps some of this is exaggerated but attendance at church by itself reduces risk of mortality in healthy people beyond that conferred by healthy lifestyles. Religiosity may also be linked to physiological processes – neuroendocrine, immune, cardiovascular. It acts as a stress buffer preventing problem linkages (biomarkers) that are helped with attendance or spirituality. The health benefits of the practice of religion or spirituality translate to well-being, hope, and meaning, as well as the reduction of psychiatric problems.

The intervention is relatively easy: Attend a church or develop spirituality. In addition to church attendance the fostering of congregational cohesiveness ("I feel my values are shared by most people in my

congregation”), spiritual support (“Someone in my congregation frequently share religious experiences with me”), emotional support (“Someone in my congregation lets me know they care about you”), as well as connectedness with god (“My personal relationship with god is close”), are important. Of course the devil is in the details. This motivational/emotional force that occurs with the spiritual or social connection is persuasive. Its cause is not fully known. At the least a focus on positivity/good acts/socialization does much for the biology and psychology of the person.

Third, social connection is critical. It has been reported that roughly 20% of older adults report loneliness and another 20% have few enough friends that it makes a difference (Perissinotto et al., 2012). As a group, older adults clearly have less friends than at earlier times in their lives. For starters, it can easily be said that being socially active is associated with well-being. Of interest, being socially active with family members did not count as much as being socially active with others. Loneliness is its own undoing; it feeds on itself and makes things even worse. Lonely people have a 26% greater chance of death. Most lonely people have a faster decline and an increasingly difficult time with ADLs. In fact, it has been estimated that socialization converts to the same magnitude of health effect as cigarettes, cholesterol, obesity, ETOH, and physical inactivity. Feelings of loneliness are also associated with health problems, depression, anxiety, poor functions, vascular problems, HTN, and inflammation. Relationships predict longevity; relationships predict heart attack recovery; relationships predict coronary heart disease; relationships protect against the common cold; relationships predict progression of HIV; and relationships affect HTN, atherosclerosis, immune status, and stress hormone levels.

Loneliness is a perceived state of social and emotional isolation that has been associated with cognitive and functional decline and an increased risk of incident AD dementia (Donovan et al., 2016). This group hypothesized that loneliness might occur in association with elevated cortical amyloid burden, the in vivo research biomarker of AD. To investigate, the team used data from 79 cognitively normal adults (average age, 76) participating in the Harvard Aging Brain Study, an ongoing observational study aimed at defining neurobiological changes in early AD. As part of their four-year assessment, participants were assessed for loneliness, anxiety and social network. Cortical amyloid burden was assessed by Pittsburgh Compound B (PiB)-positron emission tomography, and loneliness by the three-item UCLA Loneliness Scale.

Results showed that, after controlling for age, sex, presence of APOE e4, socioeconomic status, depression, anxiety, and social network characteristics, a significant association emerged between higher amyloid burden and greater loneliness. Individuals in the amyloid-positive group were 7.5-fold more likely to be classified as lonely ($P=0.002$) compared with those in the amyloid-negative group. In addition, the association between high amyloid burden and loneliness was stronger in those carrying the APOE e4 allele compared with non-carriers. For each 0.1 increase in PiB, the average loneliness score increased by an additional 0.5 units in carriers of the allele versus non-carriers.

Social connectedness, for example low social integration, loneliness, and relational distress, is multiply determined. The heritability of loneliness is roughly 40%, suggesting genetics play a large role in the sensitivity to perceptions of social standing. Psychologically there appear to be multiple pathways toward chronic loneliness, including intimate, relational, and collective loneliness, each of which attends to different dimensions of one's social being (Cacioppo et al., 2015). Social isolation may result from interpersonal, behavioral, and environmental factors. During times of stress, close relationships can protect health by buffering individuals from negative effects of stress and by facilitating recovery and resilience (Coan, 2004). Experimental and observational studies show that real or imagined support from a significant other attenuates stress appraisals in cardiovascular reactivity and facilitates emotional recovery from acute stressors (Pietromonaco & At the least a focus on positivity/good acts/socialization does much for the biology and psychology of the person. Collins, 2017).

Stress buffering is the most widely studied form of support, but recent theoretical perspectives emphasize the health benefits of social support during non-stressful times. This focus is on the importance of close relationships in helping people enhance their positive wellbeing by facilitating exploration, goal strivings, and personal growth in the absence of stress or adversity, referred to by attachment theorists as secure base or relational catalyst support (Pietromonaco & Collins, 2017). One longitudinal study of couples found that perceived partner responsiveness predicted healthier diurnal cortisol profiles 10 years later, and declines in negative affect mediated this affect (Slatcher et al., 2015). Studies reviewed by Robles and colleagues (2014) have shown associations with spouse's hostile behaviors (for example, criticizing) and elevated blood pressure, poor immune responses, pro-inflammatory cytokine production, and slower wound healing. Relationship orientations may affect people's willingness or inability to cultivate supportive intimate interactions or their

tendency to experience interpersonal conflict and may lead individuals to construe social situations in more threatening ways and undermine their ability to benefit from protective relationship processes. As a result, individuals with different relationship orientations may be more or less sensitive to the effects of social connection or disconnection on the mediators of outcomes that are noted.

Close ties in later life are often between people who have known each other for many decades. Across adulthood, people accumulate a group of close social ties that form the heart of their social networks, often including a spouse, children, or other family members and close relatives. This relatively stable pattern of social ties buoys health and wellbeing, but these social ties are also influenced by age-related shifts in social motivations and health-related needs (Rook & Charles, 2017). Older adults frequently report greater satisfaction with their social networks than younger adults.

Perhaps a leading reason lies in how older adults structure their social networks. Age-related changes are predicted by socioemotional selectivity theory, which posits that as people grow older and perceive their time left in life to be growing shorter, they shift their priorities toward emotional goals. Older adults report investing more time in maintaining relationships and are less concerned with maintaining strict reciprocity in their social relationships compared to younger adults. Older adults also experience fewer conflicts with their close social partners and appraise these conflicts less negatively than younger adults (Birditt et al., 2005). Older adults lose attachments, however. By their mid-70s, nearly 60% of women and 22% of men are likely to have been widowed. This loss of a spouse can affect other relationships, moreover, as contacts with in-laws and couples with whom the widow previously socialized often wane over time. Marital disruption because of divorce has also become more common among people aged 50 and older in recent years, doubling from 1990 to 2010. Studies on spousal bereavement provide the most extensive evidence of the health effects of losing a close relative at later life. Compared to match controls, widowed individuals experience more physical problems, more acute cardiac events, higher rates of disability and illness, more hospitalizations, and an increased risk of mortality (Moon et al., 2011).

The intervention seems straightforward. Be social! Acts of socialization such as volunteering and forcefully connecting with others are helpful. Only 31% of older adults volunteer. A key here is to involve oneself in pursuits one really enjoys. Even simple acts such as hugging are relevant (they prevent viruses). Notably, socialization can be intimate,

interpersonal, or more congregational. All count and may require some engineering.

Fourth, the practice of gratitude and altruism is important. Volunteering is part of this. Emmons et al. (2004) describe a disposition of gratefulness that includes key people, achievements, strengths, and even wonders of the world. Gratitude looks backward and externally but stimulates optimism. The person asks himself “What went well in my life today?” and establishes a gratitude focus “I am thankful for...” Regarding altruism, over 73 studies on volunteering show that people who volunteer are resilient and happy. This improved mood, increased dopamine and endorphins, and lessened inflammation. Giving advice also seems to work. Even random acts of kindness and forgiveness seem to help. Just the intention to provide help can be helpful.

● One intervention involves a gratitude journal. This can take many forms. The task of Three Blessings – asking yourself what you are thankful for and what you can do in the future that will help – applies. Issues of importance involve: Giving – “How often have you made the effort to be helpful or kind to others”; Relating – “How often do you put effort into relationships that matter to you”; and Acceptance – “How often are you kind to yourself. You are fine as you are.”

Volunteering needs a special emphasis here. It has become an important element in the quality of life of older adults. Formal volunteering is generally defined as unpaid, non-compulsory work done through organizations that provide benefits to people beyond the volunteer’s household. The tasks that older volunteers perform range from collecting and distributing food and donations for local food drives, and ringing up items at a hospital gift shop, to more specialized tasks like tutoring elementary school students, or providing professional, managerial, and fund-raising assistance to a non-profit organization. Volunteering is special because it provides several benefits. Volunteering is linked to physical health (Burr et al., 2018) and function (Carr, Kail, & Rowe, 2018), loneliness (Carr, Kail, Matz-Costa, & Shavit, 2018), and cognitive functioning (Proulx et al., 2018). Using data from all of these sources, the various types of pro-social behavior seem to provide benefit in these areas. Also, it is noteworthy that Guiney and Machado (2018) reviewed the compliments and reviewed findings relating to cognition and volunteering. This review encompassed 15 articles that evaluated the association between volunteering and cognitive functioning and concluded that volunteering has modest benefits on global cognitive functioning, as well as some specific indicators such as intentional control, task switching, and verbal and visual memory, with the magnitude of these associations

varying based on whether the study used longitudinal or cross-sectional data.

These studies underscore that volunteering is beneficial as a life-course process, which roots early in life, especially in high school, and with protective effects that change over the course of time. These results suggested that volunteering may enhance health by promoting physical, social, and cognitive enhancement and that these benefits could spill over into other aspects of life.

Fifth, resilience is a necessary condition of good living. Resilience has been construed as a construct where the person is either immune from trauma and stress or responds in positive ways after brief periods of stress. Regardless, the person responds well to difficult situations. At some point the expression of resilience enters into the positively coping life of the person. The two sides of resilience are played out in the post trauma period. Does the person handle the stress and have problems or does the person improve over time and show growth? The construct of post trauma growth (the latter choice) seems to reign. Life is rest and renewal. It is a series of sprints. Resilience is the expression of ordinary magic. It is optimism (living longer, being less unhealthy, enjoying...), emotional awareness (of what is precious), reaching out (finally being able to be vulnerable), and cognitive positivity (being able to handle this and grow). Who are the resilience heroes (Frankl, Frank, Mandela)?

For many years Lyubomirski (2008) has espoused resilience. This trait is largely voluntary and personal. This is an internal skill and has a personality basis. It is also a trait that is learned and needs practice. Her scale measured how the person constructs happiness, that this is personal, and that the person enjoys this state regardless of the circumstance: After the problem I can better myself, I can depend on others, I will have a greater sense of meaning, life is now not trivial, I will use my toolkit (breathing, physical activity, meditation), and I can apply an adversity journal (as an immune system).

In effect, the practice of resilience is one of centered moments in the now and self-transcendent reactions to any problems unresolved. It consists of having a mind at peace, open to possibilities, and in the moment. It is mindfulness under stress. Importantly, resilience can be developed in later life using many of the positive psychology features noted in this book and lifestyle discipline. It accepts emotions as data, not direction: It accepts difficulty as a moment and growth at the best moment.

● One word about stress (again): Resilience is the ordinary magic that allows stress to be friendly or dissipate. Stress is clearly a problem. Stress continues to act on a person in several ways and continues with an afterlife

that causes trouble. The extent to which a person responds to a stimulus in a stressful situation really depends on how excitable their neurons are in their present state or seconds before they are presented with the stimulus. Probably the most notable finding among all is that, when they are stressed, people become less empathic, less compassionate, and less capable of taking someone else's perspective. The person's belief in their power over stress is critical; things can be predicted by how a person feels control over this, whether they feel a sense of social support, and whether they have outlets for frustration who are important. Managing stress is much more important than the fact that one has stress. Resilience is the coping mechanism for stress.

The intervention for resilience is more complicated if one hasn't a dispositional resilience. That said, the CBT/mindful process of listening for the stress and leaning into it is called for: learning that expectations of having peace and no stress work against the person, and setting up a problem state from which discomfort becomes the enemy instead of the solution. Remember too that the person actively generates the world. People are not passive recipients of data. The simplest study of optical illusions confirms this. As such, an optimistic belief in life choices can make a difference.

Sleep also makes the list (sixth). Sleep has been touted as a major problem correlative to or causative of AD. Bubu et al. (2017) in a systematic review of sleep indicated that 15% of sleep problems account for AD. This is a noteworthy number given that over 5 million people have AD. ●f course if you slept poorly last night this does little to evidence AD: this occurs across the lifetime. The optimal sleep period is 6-8 hours. Sleep hygiene at least allows for the reasonable possibility of improved sleep, as habits often dictate results. In fact, one night of partial sleep activates the gene expression of cell senescence. ●ne poor night of sleep raises amyloid!

Throughout this book we will extoll the importance of sleep. Interventions involve the practice of sleep hygiene. Good sleep involves good slow wave sleep, total sleep, and REM sleep. It leads to a build-up of toxic beta-amyloid, poor glymphatic system clean up, a reduced hippocampus, more stress, and increased memory problems. Additionally, poor sleep (<6 hours) increases catecholamines, increases ghrelin and reduces leptin, increases oxygen consumption and increases C●2 production, and deregulates TNF, CRP, and Il6 (negative cytokine).

●lder adults have problems. So, since the number of older adults with poor sleep continues to rise (>40% with 3 or more nights a week of poor sleep), it is now on the radar of better living books and TV. This is

becoming a target for PCPs and clinics. Sleep apnea is almost modal at later life. It is largely correctable. The application of a medication is a reluctantly offered solution for most sleep problems. It starts with melatonin (a problem in delayed sleep problems or more severe problems). Once prescription hypnotics are in play, they are difficult to stop.

The best solution is sleep hygiene. This takes some time. So one target is to have the person become sensitive to all they can do to maintain sleep hygiene – regularity of getting-up time, sleep habits, sleep environment, caffeine intake, exercise, and reduction of rumination. This depends on lifestyle elements, exercise, structuring the day, and having a reasonable diet (see Hyer, 2014). Regardless, sleep problems are no small matter but can be corrected in part or whole with knowledge and practice.

Seventh, we follow with another critical feature of life, exercise. Again, we have trumpeted the value of exercise: the more, the better. Saint-Maurice and investigators (2018) from the National Cancer Institute analyzed data from 4,840 people of 40 and older who participated in the National Health and Nutrition Examination Survey between 2003 and 2006. Participants wore accelerometers to quantify their physical activity and exertion. Using a national database, the researchers determined that 4,140 participants were still living in 2011. The most dramatic improvements in the overall risk for death and disease can occur with a relatively small amount of effort, and the more you do, the better the benefits. In the study, people who got less than 20 minutes of moderate or vigorous activity each day had the highest risk of death. Those who got 60 minutes per day cut their risk of death by more than half – 57 percent. Getting at least 100 minutes of moderate or vigorous activity per day cut the risk of death by 76 percent, the data showed.

This study has been supported in many ways, with many types of exercise, committed over time, and with all kinds of maladies. Now it seems that even smaller amounts of exercise help. Higher levels of lifestyle physical activity – such as house cleaning, walking a dog, and gardening, as well as exercise – are associated with more gray matter in the brains of older adults, according to a study by researchers at Rush University Medical Center (Halloway et al., 2018). The gray matter in the brain includes regions responsible for controlling muscle movement, experiencing the senses, thinking and feeling, memory and speech, and more. The volume of gray matter is a measure of brain health, but the amount of gray matter in the brain often begins to decrease in late adulthood, even before symptoms of cognitive dysfunction appear. This study used accelerometers to measure the activity of 262 older adults. The study measured the levels of lifestyle physical activity by 262 older adults

in Rush's Memory and Aging Project, an ongoing epidemiological cohort study. Participants are recruited from retirement communities and subsidized housing facilities in and around Chicago to participate in annual clinical evaluations and magnetic resonance imaging (MRI) scans, and to donate their brains and other parts of their bodies for research after their deaths. Lifestyle physical activity is "more realistic for older adults" than a structured exercise program that might require them to go to a gym.

Exercise works especially for diabetes. More than 10 years after a lifestyle intervention program to prevent type 2 diabetes showed dramatic results, researchers found the benefits to be long-lasting. These were lifestyle interventions. In one group in the Diabetes Prevention Program (DPP) landmark trial, 2000 were evaluated in lifestyle and Metformin or placebo groups. The lifestyle intervention had two main goals: a 7% maintained weight loss and 150 minutes per week of moderate to vigorous physical activity (MVPA) at the intensity of a brisk walk. The intervention featured several components including: lifestyle coaches trained through a national network, who kept in frequent contact with participants to help them maintain their goals; supervised physical activity sessions; and adherence strategies tailored to individual participants. After the DPP trial there was a 58% decrease in diabetes incidence among participants in the lifestyle intervention group compared with placebo.

Additionally, participants from all three study groups were offered a group version of the lifestyle intervention. Those who accepted were followed in the DPP Outcomes Study (DPPOS) for more than 10 years from the DPP trial baseline. Rockette-Wagner and colleagues (2016) used accelerometer data to objectively measure whether the increased physical activity seen among DPPOS participants was sustained long-term. These researchers found that MVPA levels were generally higher among DPPOS participants (n = 1574) than among a representative sample from the National Health and Nutrition Examination Survey (2003-2006) matched for sex, diabetes status, and age (n = 1839).

Exercise reflects all features of life. Researchers have learned that older adults with slower walking speeds seem to have a greater risk of dementia than those with faster walking speeds. Recently, researchers from the United Kingdom teamed up to learn more about changes in walking speed, changes in the ability to think and make decisions, and dementia. The researchers examined information collected from the English Longitudinal Study of Aging. The study included adults aged 60 and older who lived in England. In their study, the researchers used information collected from 2002 to 2015. They assessed participants' walking speed on two occasions in 2002-2003 and in 2004-2005, and

whether or not the participants developed dementia after the tests during the period from 2006 to 2015. Then, they compared the people who had developed dementia with those who had not. Researchers discovered that of the nearly 4,000 older adults they studied, those with a slower walking speed had a greater risk of developing dementia. And people who experienced a faster decline in walking speed over a two-year period were also at higher risk for dementia. People who had a poorer ability to think and make decisions when they entered the study – and those whose cognitive (thinking) abilities declined more quickly during the study – were also more likely to be diagnosed with dementia. The researchers concluded that older adults with slower walking speeds, and those who experienced a greater decline in their walking speed over time, were at increased risk for dementia. But, the researchers noted, changes in walking speed and changes in an older adult's ability to think and make decisions do not necessarily work together to affect the risk of developing dementia (Hackett et al., 2018).

● One simple idea is just movement. Several data sets have found a direct relationship between the amount of time exercising and cognitive benefits, cardiovascular benefits, reduced A-Beta, reduced depression, improved well-being, and, well, virtually everything. Cognitive benefits seem to increase as a function of the dosage of exercise (75, 150, 225 minutes). Current guidelines, issued in 2008 by the US Department of Health and Human Services, recommend at least 150 minutes of moderate exercise or 75 minutes of vigorous activity per week, ideally spread out over several days. For decades, Americans have been inundated with a confusing barrage of messages about how best to counteract the health risks of sedentary lifestyles: walk 10,000 steps a day; do a seven-minute workout from a phone app; flip heavy tires in an arduous boot camp class. It turns out that any and all of those tactics – even when done in short bursts throughout the day – could work to reduce Americans' risk of disease and death.

For about 30 years, guidelines have suggested that moderate-to-vigorous activity could provide health benefits, but only if you sustained the activity for 10 minutes or more. This flies in the face of such public health recommendations as taking the stairs instead of the elevator, and parking farther from your destination. Those don't take 10 minutes, so why were they recommended? Even brief trips up and down stairs would count toward accumulated exercise minutes and the reduction of health risks so long as the intensity reached a moderate or vigorous level. Moderate exertion was defined as brisk walking at a pace that makes it hard to carry on a conversation. Boosting that pace to a jog would be

vigorous exercise for most people. And the other side of the coin is that sedentary behavior can be a spoiler and can override exercise. The intervention then is to do something. Even a little housework works.

Diet is number eight. Frailty is common among older people and its prevalence is increasing as the population ages. Frail older adults may often feel low in energy and have weight loss and weak muscle strength. They are more likely to suffer from numerous health concerns, including falls, fractures, hospitalization, nursing home placement, disability, dementia, and premature death. Frailty is also associated with a lower quality of life. Recently the construct “cognitive frailty” has been invoked (Ticinesi et al., 2018). This is defined as the co-existence of MCI symptoms and the physical frailty phenotype of older persons predisposing them to dementia. It is distinct from the frailty syndrome which is a solely physical debility (Fried, 2001).

Nutrition is thought to play a crucial role in the development of frailty. Kojima and colleagues (2018), of University College London, in the UK, looked to see if following a healthy diet might decrease one’s risk of frailty. The researchers analyzed evidence from all published studies examining associations between adherence to a Mediterranean (Med) diet and the development of frailty in older individuals. Their analysis included 5789 people in four studies in France, Spain, Italy, and China. The evidence showed very consistently that older people who follow a Med diet had a lower risk of becoming frail. People who followed a Med diet the most were overall less than half as likely to become frail over a nearly four-year period compared with those who followed it the least. The investigators noted that the Med diet may help older individuals maintain muscle strength, activity, weight, and energy levels, according to their findings. Interestingly, those who follow the Med diet and DASH (Dietary Approaches for Stopping Hypertension) (Dietary Approaches for Stopping Hypertension) diet show longer telomeres, associated with longer life and less morbidity (Science Daily, Leung, 2018)

In a series of articles in *The Journal of Gerontology, Series A: Biological Sciences and Medical Sciences* (2018), new connections between a Med diet and healthy aging outcomes were presented. Among the findings, these articles report on: underlying mechanisms of the diet; the positive relationship between the diet and physical and cognitive function; the value of coenzyme Q10 supplements; and the role of diet in reducing inflammation. Adherence was key to benefits. Additionally, adults over 40 who eat healthy foods most of the time are twice as likely to rate their brain health and mental sharpness as “excellent” or “very good” compared to others who eat a healthy diet (AARP, 2018). This number,

however, is small (<one third). All in all, Med diet patterns seem to involve: lipid lowering protection from oxidative stress and inflammation; the modification of growth factors that can promote cancer; and the inhibition of nutrient sensing pathways by amino acid restriction and gut microbiota-mediated production of metabolites.

One special diet element has recently been proffered as important and relevant. Curcumin appears to provide considerable health benefit for older adults. A UCLA study by Gary Small et al. (2018) is the first long-term double-blind placebo-controlled effort using a bio-available form of curcumin in non-demented patients. The participants, 40 adults between the ages of 51 and 84 years who had mild memory complaints, were randomly assigned to receive either a placebo or 90 mg of curcumin twice daily for 18 months. People who took the curcumin experienced significant improvements in memory, recall, and attention disability, while subjects who received the placebo did not. Those taking curcumin showed mild improvements in mood and their brain PET scans showed significantly less amyloid and tau.

There are many diets promulgated for better health. We have already touted the value of the Med diet and for diabetes, the DPP. The Med diet has been shown to produce a reduction in the incidence of both AD and Parkinson's disease (see Walsh, 2011). The DASH too is noteworthy. This is the bedrock of the American Heart Association American College of Cardiology's efforts to reduce hypertension. The DASH diet alone can lower blood presser as much as taking HTN medication. If one also cuts salt, the effect is clearly superior to medication. Salt free diets and the DASH diet also lower LDL levels. Finally, the addition of probiotics seems to be instrumental in the changing of the microbiome (Liebman, 2018). For a more complete review of nutrition and mental health see *Nutritional Medicine* (Gaby, 2017).

Ninth is relaxation response (RR). This can take many forms; meditation, yoga, deep breathing, repetitive prayer, and mindfulness. The overall message is that RR fights off the fight-flight response. It reduces the cascade of the SNS and the adrenal medulla that results in hyperarousal. In an analysis of 22,000 genes the RR enhanced the expression of genes associated with energy metabolism, insulin secretion, reduced inflammation, and prevention of cell aging: the more, the better. The benefits are many: self-education, relationships, creativity, play, health, altruism, spirituality, and self-transformation.

Mindfulness is the new RR, the new somatic softening. It is paying attention in the moment, on purpose and mindfully. The effort is to bring attention to individual moments in your life without judgment and defense.

In a mindful moment you observe yourself at that time, including the evaluations you make and sensations you feel, and not judging what you are thinking in the moment, being non-judgmental even about your judgments. This is not about relaxing or escaping.

Perhaps a simple philosophy of Buddhist teaching applies best here. Life is suffering and problems arise from desire and the inability to handle universal discomfort. We desire and we pay the price. All things change and “things” require acceptance, reflecting the normal order of living. The operationalization then of Buddhist philosophy has become the new catalyst of the third wave in psychotherapy. Also in the past 20 years Acceptance and Commitment Therapy (ACT) has been empirically supported and espouses acceptance of, and commitment to, problems. ACT also assumes suffering is normal. Most humans suffer even in the context of successful lives: 100% of all individuals will have a suicidal thought within their life and 50% of adult individuals will suffer from some psychiatric disorder at some point. When all major behavioral problems humans face are added up, it is abnormal not to suffer. The solution to suffering is an appreciation of the context of living. The view is accepting that there may not be a solution for suffering or a resolution of problems.

There is a “fix-it” mentality of humans and a tendency to want to rid oneself of problems causing unpleasantness. The very adaptive cognitive constructs that have helped us excel cause suffering. Working harder at alleviating suffering is the antithesis of the ACT point of view. Consequently, less emphasis can be placed on problem-solving (i.e., ridding oneself of depression) and greater emphasis is placed on the role of such problems being barriers to living. The focus of ACT should not be on fighting against a psychological problem (i.e., depression), but on lessening the impact of the problem on the life of the individual. The appearance of distressing thoughts and feelings might not change but its impact can change.

“Vulnerability is not a weakness, a passing indisposition or something we can arrange to do without. Vulnerability is not a choice; vulnerability is the underlying ever present and abiding undercurrent of our natural state. To run from vulnerability is to run from the essence of our nature, the attempt to be invulnerable is to become something we are not and most especially to close off the understanding of the grief of others. . . The only choice we have as we mature is how we inhabit our vulnerability, how we become larger and more courageous and more compassionate through our

intimacy... Our choice is to inhabit vulnerability as generous citizens of loss, robustly and fully, or conversely as misers and complainers, reluctant and fearful, always at the gates of existence, but never bravely and completely attempting to enter, never wanting to risk ourselves, never walking through the door.”

David Whyte

This bespeaks of a sense of flow (being in the zone). The person is requested to slow down and engage in all senses, to luxuriate and bask, and to savor. In fact, plaming a day with savoring is a perfect intervention. Savoring across time (fantasize on a positive experience) can also apply. A concern is not to reach a tipping point where savoring becomes analysis. The feature of savoring is mostly reactive to the moment but can be a planned procedure. It is “joy” in the moment.

The issue of values, tenth, must be included. This is the neon sign of positive movement. It is your goal that comes factory installed; what is important to you, what are you good at, what is the foundation of your nature, who are you, what is the structure of your personality. It is the internal movement that counsels against putting life on hold when caught up in a struggle with psychological problems; an individual postpones living, believing that pain needs to lessen before beginning to live the life one desires.

The intervention is a values exercise: what is of core value. The person is asked to picture the self going through a day with this in mind always. Variants of this include: redefining yourself and allowing the new you to just indulge in living; developing an appreciation of life; relating to others; creating a narrative for a new mission in life; reinforcing personal strength; being different and special; and marching to your drummer. This is also a core feature of ACT.

Leisure is very much a part of this value process. By applying a factor analytic to a depression measure, Radloff and Ray (1997) noted that older adults in a different cultural context tend to express their psychological distress differently. Growing evidence supports the protective effect of leisure activities on depressive symptoms. The relationship between different types of leisure activities and the dimensions of depressive symptoms are likely. In general, engagement in varied ranges of activities benefits mental health among older adults more than participation in any single type of activity alone.

Solutions are plentiful. Have small goals and act. Act also on what is desired. Perhaps a values clarification is in order. This will allow for self-interests to confess themselves. Humans really define themselves with

their expressed desires and passions. We note too that a core feature of lifestyle change is the extent to which the person is committed to change. The person does best at a task if they really enjoy it and appreciate the reason for their efforts. This has a special value for any lifestyle factor: like what you are doing and commit to it. Cognitive flexibility ascends in importance. That is, at times the object is to tackle the problem; at times, the target is acceptance. This is not unlike other problems that are stress-related. Attitude matters!

Eleven is CT (cognitive training). In this context, much has been written on “technopathologies” for younger adults in our society, as well as “digital handicaps” for older adults. In fact, technological innovations and their lifestyle effects are changing more quickly than we know how to change ourselves. What seems to be occurring is that the older adult who can modify habits, especially cognitive ones, ever so slightly finds that positive outcomes are realized. A problem is not the idea of CT but the practice. There is often an uphill battle: even when confronted with a degenerative diagnosis only 10% of patients follow advice.

We then argue for CT. This is one way that this process can occur. The Fountain of Youth is between your ears. The brain has 100 billion neurons amid another trillion support cells. This results to 10 to the millionth power. Neuroplasticity in this sense is always present. It is the survival of the busiest. The mind creates brain creates mind. In fact, it appears that multiple aspects of CT may indeed precipitate changes in other better lifestyles.

Any noticing of, or certainly reduction in, key biomarkers (amyloid), identification of genetic factors or a family history of dementia, noted subjective memory impairment or functional complaints, established low normal cognitive performance, presence of comorbidities (especially CVRFs), poor lifestyle factors (sedentary, diet, low cognitive stimulation, isolation), and present negative attitude toward life (depression or anxiety), have been identified as causing problems.

The Finland national study (FINGER) is especially noteworthy. This involved two groups. These were subjects who had elevated dementia risk (age, education, HTN, cholesterol, obesity, low physical activity), were normal or near normal in cognition, and safe to engage in activity, med stability. Subjects were between 60 and 77. One group was provided nutrition, exercise, CT, and health management. The other received health education. Positive results were seen in all affect, cognitive, and quality of life areas. This was noteworthy for the former (active) group. There were no biomarkers. As noted before, a FINGER-like study adapted is now in progress in the US (n = 2000). In sum, there is an emphasis on the careful

and intense application of lifestyle markers on older, everyday people. It may become the new normal.

A life spent with cognitive impairment increases with age, with impairment projected to be around 1.4 years for men and 2.5 for women. Education level alone reduces this figure by 13% for men and 22% for women. Five and ten year rates of decline do go down but are different for cohorts and intra-individually. Key markers are gait speed and function (IADL, ADL). Protecting cognition is critical. Lifestyle components in general then make a difference. This is especially the case with cognition and its minions.

The challenge of CT is to strike a balance between fighting the disease and coming to terms with the limitations it imposes and to assist patients and their families in adjusting to the changes as the disease progresses. The model of “all hands on deck” is appealing because it subserves many positive features of the lifestyle, in addition to CT. Even people who function poorly on cognitive issues can potentially benefit. The solution is to accommodate strengths and challenges. There are computer-based and non-computer tasks. Both are acceptable and helpful. Each of the cognitive domains – attention, visuospatial, language, new learning (executive functioning), and memory – can have dedicated tasks. We address this in other chapters. Increasingly, older adults are benefiting from the use of the internet and structured programs teaching attention and executive functioning.

Some Suggested Cognitive Strategies

- Focus and attention: Change routes, Dialogue about pictures, Read paper
- Memory Tasks: Learn a poem, memorize US presidents.
- Verbal Skills: Pick 2 vowels, randomly take a sentence and rearrange words
- Motor skills: Move, dance, use of CDs
- Executive Functioning: Play cards and games

Here is a putative listing of the relative value of the lifestyle markers.

Hierarchy of Good Life Habits

- High Value
- Practice sleep hygiene
- Exercise
- Diet

- Increase cognition
- Medium Value
- Socialize
- Practice spirituality/religion
- Somatic softening and meditate/mindfulness
- Lower Value
- Altruism
- Develop resilience
- Practice happiness
- Values and leisure
- Respect personality

Personality

Finally, recall that each person has a personality, a pattern of coping that is central to the person (see chapter 9). This variable is both reasonably enduring and eloquent in providing information on the phenomenology of the domain-problem process. Yes, there is variability and people can change but the construct provides more help than noise. If we can measure it, it helps provide an understanding of the expression of the problem.

With age, personality becomes more of a strength; older adults are less neurotic, more agreeable, more conscientious, less extraverted, more self-transcendent, and more spiritual. Obviously the value system segues and mediates the variables noted above. Cooperation leads a person to become more agreeable and more stable, and to show greater conscientiousness and more inhibition, among other things. In general, older adults become more agreeable, emotionally stable, conscientious, more harm avoidant and socially avoidant, and less neurotic too. They also become more spiritual and self-transcendent. Again, this is the bedrock of the person and is centrally situated in the behavior/patterns of the person to, if known, allow for the health care provider to predict responses to care and stress. Perhaps above all the value of personality is that it is reasonably consistent, assessable, and reflects the vulnerable condition human behavior. More on this in Chapter 10.

Watch and Wait: The Big Picture

We start with the typical types of problems. There are of course many types of domain-related permutations and interventions. Older age patients can present as One and Done, Brief Evaluation, or Usual Watch and Wait. The usual Watch and Wait model involves the majority of treatment options and is a more focused care emphasis on the specific domain. Follow-up is always recommended.

Modal Older Age Problems

One and Done: Quick evaluation and referral

Brief Evaluation: More lengthy Watch and Wait Evaluation (a few sessions). Assessment as Treatment (3-5 sessions in short period of time)

Usual Watch and Wait Models: Assessment and Formal Treatment for moderate period (often 6 months)

Depression and Anxiety: Psychotherapy

Cognitive/Depression/Anxiety: Structural interventions plus psychotherapy

Cognitive/Health/Affective Problems: Multimodal interventions

Life Adjustment: Structural interventions plus needed health/cognitive/affect interventions

Follow-up: Recommended for all types above

This process is harder to convey than to experience. The role of health care provider, the therapist, is undeniable. The therapist is a lifeline, a hope, a constant. Sometimes the role is one of cheerleading or of being a reality checker, a teacher, a leader, a friend, or a director. Often it is as Martin Self and Sally Winston described it, entanglement, the process of fashioning meanings and warning signals out of passing “mental detritus.” Watch and Wait seeks to validate the context of offering hope and direction based on empirically supported interventions. The therapist is making calls in session, consulting with primary care docs in session, calling housing personnel in session, and many other similar tasks. The therapist is in a vertiginous dialectic between a sense of being in control and increasing pressures on the targets where things are less in control. There is in a sense a role-fluidity where one moment the therapist is friend and the next a gadfly.

Remember too that the therapist is always in assessment mode: assessment as treatment. Some of this is formal screening and some is the extended assessment battery for “Watch” domain of Watch and Wait. Some is also follow-up. Kant noted that “Out of the crooked timber of humanity, no straight thing was ever made.”

Of course there are other problems. The problems for older adults can be separated into the patient therapy selection process and specific issues that cause problems. For older adults side effects and preference make a difference. And, for older adults, acceptability, availability and readiness for change are important.

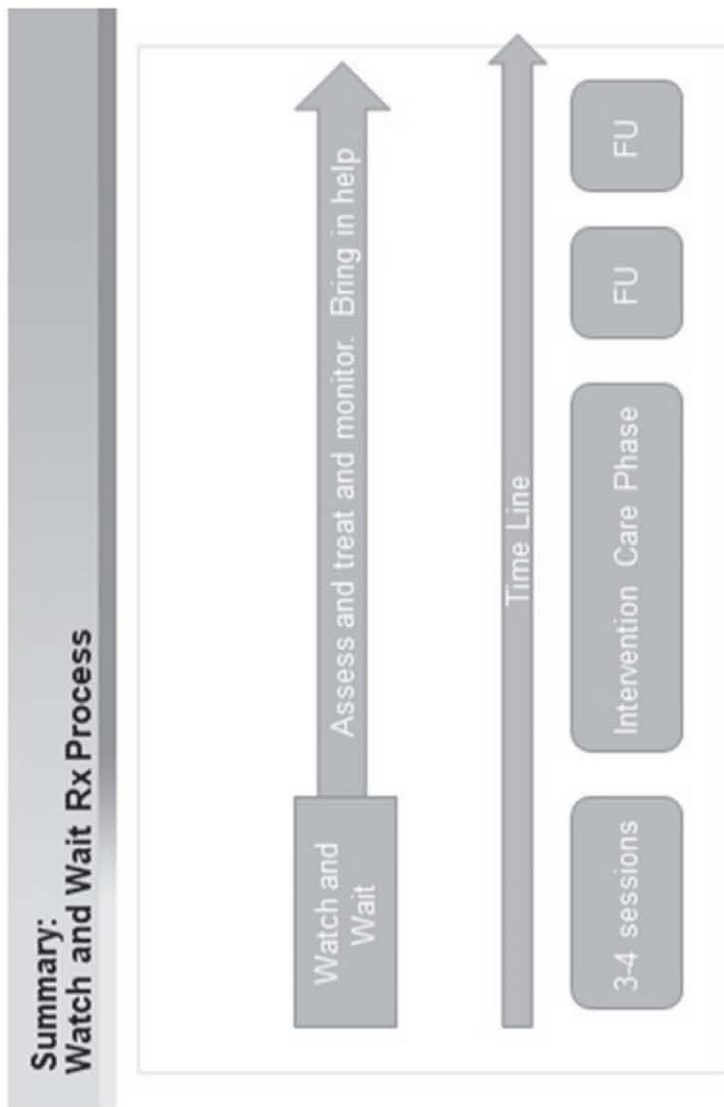
TREATMENT and PATIENT VARIABLES	
Variables in Treatment Selection	Patient Specific Issues
History of rx response	Acceptability
Patient preference	Readiness to change
Side effects	Availability
Expenses for rx	Payer approval
Interaction issues	Caregiver approval
Prior rx failures	Intolerable side effects

Watch and Wait: Nuts and Bolts

Prequel: We build again on Chapter 1. In Chapter 3 we discussed assessment – case formulation and the mechanisms of testing/assessment. This sets the stage for treatment. Assessment is of course part of the treatment. Recall that Watch and Wait is a process. We watch and wait, validate, and build trust. The pursuit of a committed patient in the room with a perceived, competent therapist represents the skeletal structure of psychotherapy. This sets the conditions for an alliance to prosper. Below we explicate the overview model presented in Chapter 1.

Before this, we address three issues. We assess the older person. But the person is complex. He/She is older and the influence of life variables, family and environment, is critical. The initial focus is the wish and autonomy of the identified patient. This is always paramount. Commonly the older adult is accompanied by a spouse or adult child, a caregiver. Or, they are referred by a physician, the referral often representing confusion to the identified patient. In our practice the presence of a caregiver is modal (90%). Often this is amiable but there are times when separation, followed by a reunion, is necessary. This is made a friendly choice and the format is decided after the therapeutic choice. It is most usual to do the cognitive and MINI screen to be applied to the patient alone, but this is not

always possible. Also, the patient will have already completed the anxiety, depression, function, sleep, and personality screens. If not, this can be done between sessions 1 and 2.



The time frame is also worth highlighting. The assessment and validation/psychoeducation takes place in the first 3-4 sessions and then segues into the intervention and follow-up phases. Again, the therapist is always assessing, screening and carrying out more extended evaluations, by domain. The time frame for Watch and Wait is then front-loaded, with the first 3-4 sessions devoted to the initial stages of the model: discernment, validation, and assessment. This can be 3-7 hours. Also modules (more of a treatment focus) can be introduced at any time.

Finally, we should note that, if you were to query mental health professionals, the stock introductory session requires a sensitivity to safety and acute concerns. Foundations of health and severe mental health issues (suicide) are on the evaluator's mind. The focus quickly targets factors that subserve the diagnosis. This allows for a formulation assist. It has limits, as we have seen and will restate. But, you dance with the one that brought ya!

Steps: We start here. It has been reasonably established that the components of change involve the relationship, activation potential and a belief in the therapy and its process, as well as techniques. While this division may be excessive, it points to the need to view therapy as a process where parts need to be emphasized and practiced.

● Overall Components of Change

Therapeutic relationship-30%

Extratherapy change -40%

Placebo-15%

Techniques-15%

Step 1: How does the Watch and Wait model populate the structure noted above? We believe that initially the older adult experiences a sort of "realization moment." This is perhaps best labeled as discernment therapy: "Is this for me?" This can be a quick or slow unfolding from "Why am I here" to "Ah, this feels right and it has been long coming." Or, in the case of a resistant dementia, "At least I will go along with this." The caregiver in this case commits; the patient is dragged along. Patients are often ambivalent or in a forced-compliance situation. At some point the patient is on board, albeit sometimes reluctantly. Commitment is never all or nothing and is never to be taken for granted.

Discernment Process

Patient

Thank you for coming, I would like to get to know you.

What is your reason for being here?

Will you commit to questions and a period of assessment?

Will you allow your caregiver to make input and have access to your information?

Will you commit to some home tasks and challenges?

Your coming here means something important in this new stage of your life.

Caregiver

Will you assist in the assessment process?

Will you allow for some openness in this process, even trying tasks not initially seen as helpful?

Are you open to caregiving help – support, psychoeducation, strategies?

Are you agreeable to the slower Watch and Wait Process?

During the discernment process, person-centered markers are applied. Efforts are made to “motivationally interview” the person and provide constant support and some challenge, allowing for minimal resistance. Motivational interviewing is intended not to confront resistance. A typical phrase is to ask “What is important to you?” This can be delicate but the validation process is itself therapy. Assessment follows suggestions for each domain. The resulting case plan is a work in progress. Importantly a tentative understanding and support process is established. These are the necessary and often sufficient alliance markers for the Watch and Wait model. They are psychotherapy-based and are anchored in interpersonal care.

The focus is on the early discernment of what to do next. Once the interview for Watch and Wait is initiated, the identified patient and others are now in the process of Watch and Wait. This can unfold in many forms. The typical form of the Watch and Wait therapy for older adults includes the following: Validation and assessment process for 2-4 sessions; Explication of the model (Here is what we can do), validation of the person and problem, some assessment, building a connection/alliance, reasonable monitoring, and then a therapist case formulation unfolds. This is basically discernment.

We note also that discernment is a two edged sword. One side is the seduction into the therapy itself. This is a challenge to demoralization, a lack of consensual validation and self-efficacy. Hope is the other side and is implied in the therapy. This starts the process.

Discernment Issues

Demoralization

- Patients seek therapy when they become demoralized from failed change methods. Their efforts at coping is failed.
- Demoralization is a cause, consequence or both of the presenting symptoms that patients bring to therapy.
- Loss of consensual validation: People need to sense validation.
- Positioning is key: Pro-change, Neutral (ambivalent), Don't-Change, Restraining, Normalizing and acceptance, Reframing, Prescribing.

Hope:

- Agency thinking (I can do this) and Pathway thinking (This is what I do).

Step 2: The therapy itself starts the dialogue on what better therapy for older adults is. It starts with the essence of the therapy. This is a connection to be open and to work.

Necessary Validation Process of Watch and Wait	
Validate Problem	X
Psychoeducation of Model	X
Assessment	X
Alliance	X
Monitoring	X
Case formulation	X

As noted, the therapeutic alliance is critical to change. Greenberg (2014) holds that the therapeutic alliance is the treatment in and of itself. It establishes a soothing affect-regulation bond and also functions as a means to an end. The practice of the necessary and sufficient conditions of change, especially genuineness and congruence, is important. The aspect of the therapist to express some control and some affiliation becomes equally important. Getting into the perspective of the patient to where the patient feels like a partner is ideal. Several suggestions are provided below.

Psychotherapy Helpers

Communicate control and care: “We.”

Use comments in treatment (“You have noticed that you can...”)

Reward liberally (“You have been a star in life, now...”)

Reveal: “I respect your struggle. Just yesterday, you...”

Communicate hope: “Yes, your scores are a concern, but you can hold the line or even change this with...”

Get patient stories: “What made you go back and get your GED?”

Use past judiciously: “Yes, you did this, that was the ‘you’ then; now...”

Seduce with the better ideas: Socialization, positivity, altruism, commitment, love of family.

Encourage to envision a self in three months.

Law of little things: Tilt toward the positive.

Social chitchat: “It is really nice to see you”

Reflection in moment: “How are you doing now?”

Express immediate feelings: “I am feeling good about your skills.”

Draw parallel with therapy: “I wonder if you find it easier to be here.”

Make the covert overt: “You are rather quiet today.”

Get on board: “I see you want to take an antidepressant. Great but...”

Make a deal: “Let’s make a deal here. I will... You agree to... at least for one month”

Get early positive treatment outcome expectation/early gains.

Additional Helpers:

1. Use brain pathology as a metaphor: This allows for a shared vulnerability and understanding of what can be addressed from a more medical/physical perspective.
2. Link problems to achievable reality: “This is where we are going and what we can do.”
3. Make haste slowly: “We need a little time to position ourselves for the best chance for change. So, we will take 2-3 weeks and see about our options and make plans for best care. This is a very active period and you will be in the most active stage of treatment, the planning stages. Most treatment fails because this treatment part is missing and you are thrown into an intervention too quickly.”
4. Provide a plan: “Here are the steps for change.” This is based on the case-based model that has been formulated.
5. Solicit feedback: “How am I doing?”

Step 3: Assessment is highlighted even though it is an ongoing process. The initial screen is performed as the validation and alliance is in process. Based on probable domain problems, a more extended evaluation is applied. This is fluid but deliberate as the health issues, neuropsychological targets, depression and anxiety scales, and interviewer-based life adjustment is considered. Typically, this is done pro-forma as part of a natural process of the overall assessment. It continues, then, throughout. Each chapter that follows provides an assessment package for that domain.

It is important to reiterate that the profile of domains is fluid. It changes. As important, there are problem states that remain as issues but becomes less problematic over time (e.g., cognition with dementia that is handled well) and so become less of a "Problem." When this happens, the domain is rated as less than "Problem" and followed. This is incorporated into the new domain profile.

Step 4: Module interventions are applied. This is the eclectic transdiagnostic process of providing processes and modules for change. They include the following plus more depending on the creativity of the therapist. There is no mystery here. These are "quickies." They can be learned quickly and, if practiced, will provide the illusion of control and commitment to change. They set the stage for the possible use of their "parents," Step 4. They activate the patient and provide feedback on change. They are also standard in many therapies.

Mini-Module Targets of Behavior over time

- Activity scheduling
- Mastery-Pleasure activities
- Graded task assignments
- Verbal rehearsal of tasks
- Managing situational contingencies
- Role playing and modeling
- Environmental control
- Contingency plans (If-then)
- Apply progressive relaxation
- Concreteness training
- Imagery exercise for counter-experience
- Compassion training
- Communication and Assertiveness training
- Behavioral experiments: Plan experiment
- Emotional exposure
- Homework
- Worry control

● Quick cognition (autonomic negative thoughts (ANTs))
 Relaxation (breathing, slow deliberate deep breaths at a selected rhythm)
 Simple hypnosis (breathe, focus on a target and close eyes)
 Easy nutraceuticals (e.g., omega 3's)
 Acceptance and willingness (simple targets)
 Flood senses with positivity (each sense with positive experiences)

Step 5: We have the option to apply common sense, empirically valid treatments. These are tried and true processes, CBT, PST (problem solving therapy), ACT, and mindfulness therapies. They are structured in their application and easy to monitor. These are therapies whose modules or philosophy can provide considerable help in the actual therapy.

Treatment Methods that Really Help
 Motivational Interviewing: What do you want for you?
 ACT: Acceptance and Commitment
 CBT: Focus on simple cognitive interference
 Behavioral activation
 Mindfulness: Somatic quieting
 Eastern Philosophy: Being with discomfort, excessive “selfing,” mind creates suffering.

Step 6: Lifestyles are entered. Now this can occur at any time in the Watch and Wait process. We have outlined these above. They are critical to the psychotherapy as applied to older adults with whatever problem, at whatever level.

Overall model

Below we put together the whole process. This is the recommended model of the “therapy” side of the Watch and Wait process. A more extended one is presented in Appendix B. This perhaps quixotic but is a format to apply or model. We do not seek a new psychotherapy. The health care provider formulates cases on the basis of the confirming and disconfirming of data to determine whether selected empirically supported causal variables (e.g., cognitive distortions, medically related problems, poor self-control, ineffective problem solving, low rate of positive reinforcement) are relevant, operative, and meaningful to this particular patient. There are many roads to Rome. Lifestyle needs to be a part of this. Cognitive restructuring, for example, can be tested by decreasing self-

defeating thinking, using behavioral experiments to test the validity of a belief, bibliotherapy, modeling, mild refutation, didactic explanations, homework assignments, visualization, and the use of caregivers, to name a few. The recommended format is as follows:

Extended Watch and Wait Checklist

Discernment

Validation process

Validate Problem	X
Psychoeducation of Model	X
Assessment	X
Alliance	X
Monitoring	X
Case fomulation	X

Assessment

Problem List

Case-Based Plan

Quick Modules:

- Targets of behavior over time
- Activity scheduling
- Mastery-Pleasure activities
- Graded task assignments
- Verbal rehearsal of tasks
- Managing situational contingencies
- Role playing and modeling
- Environmental control
- Contingency plans (If-then)
- Apply progressive relaxation
- Concreteness training
- Imagery exercise for counter-experience
- Compassion training
- Communication and Assertiveness training
- Behavioral experiments: Plan experiments
- Emotional exposure
- Homework

Longer Therapy processes:

- CBT: Possible 3-6 Column Cognitive Evidence Record
- Problem Solving Therapy
- Interpersonal Process Therapy
- Acceptance and Commitment Therapy

Lifestyles (list given above)

Follow-up: Relapse Issues

As the Watch and Wait process proceeds, the problem list is especially important. The need is clear: to have a list of issues from the core domain profile that represents the living document of the case formulation. It is the template from which the treatment unfolds. In a sense IT IS the case formulation. There are early interventions and later ones. The early interventions involve simple tasks for activation and learning. This is the easy part. Later interventions (after discernment) involve easy modules and more formal therapies like CBT/PST/ACT/IPT. The case plan plans out these short modules and longer therapies. The module-specific tasks are then integrated. Follow-up is planned and further assessment planned.

As we have noted, the outcome sought is never just symptom abatement. Lifestyles by themselves account for much of the variance of quality of life (QoL). Therapy should aim at symptom relief and improving QoL. Moreover, although evaluation questions necessarily focus on the reasons why an older adult is seeking treatment, such a narrow focus is only helpful for limited targets; for understanding process changes over time or other longer term and broader concerns, more is demanded. In translating ESTs to older adults, and in targeting this to the most researched psychotherapy, CBT, we must concentrate on more general outcomes (not just diagnosis), as well as specific markers associated with the identified problem. With older adults, the therapy also demands a scientific attitude, a skillful and flexible delivery of services, quantitative monitoring of the client's progress, and an awareness of the personal, interpersonal, and cultural characteristics of the client as well as QoL themes.

Post script: There is a moment in the self-evaluation that transcends the issue at hand. This is beyond "discernment." It is deeper and even existential. The importance of wisdom in the struggle with living is a marker of extreme importance. How does wisdom manifest itself? Wisdom is in some sense expert knowledge that probably involves the fundamental pragmatics of life including mortality and issues of intimacy (Baltes & Smith, 2008). It no doubt involves dialectical thinking and intellectual humility (Grossmann et al., 2017).

Staudinger (Staudinger & Gluck, 2011) argued that the psychological definitions of wisdom can be grouped into general wisdom concerning questions of human life in general and personal wisdom which concerns oneself. Perhaps the notion of wisdom as self-transcendence may have special meaning (Tornstam, 1994) as it implies self-knowledge, detachment (awareness of the provisional nature of the external sources of self) and integration (acceptance of all self-aspects). The person in effect intuits that their sense of self is not easily upset and that they are part of the greater

whole. Again, this comes at moments when it counts: a loss, a moment of self-definition, a trauma, a magic part of everyday life. The person is confronted by the self.

Why is this so important? It is part of the story telling process or reminiscence process of older adults. At moments in life an episode is made real by a reflection, by extended psychological residuals that require self-reflection. This tends to occur with chronological ageing, especially as loss events are plentiful and, even if not so, a sense of generativity is deeply engrained. It is curative by itself. Often, however, it is emotionally negative, as the process of coping and confronting reality is paramount (Weststrate, Ferrari, Fournier, & McLean, 2018). But, it cannot not be responded to. Hopefully resilience and emotional intelligence are part of this. Regardless, the therapist is on notice to listen and respond.

Special Case: Dementia

The above rubrics apply to people with cognitive impairment. That said, there are special concerns for greater cognitive impairment. Broadly, a focus on the whole person has been novel and important. Diehr, Thielke, Newman, Hirsch, and Tracy (2013) showed a five-year change in standardized health that varied as a function of outcome – mental health, hospitalization, bed days, cognition, quality of life, and hospital utilization. This is applicable to dementia. Gait speed and IADLs especially declined precipitously. There can be many targets. Cognitive impairment and physical function especially, such as falls, are related to each other and assistance with the latter may be done with attention to the former (Martin et al., 2013). The shared relationship between neurocognitive and neuropsychiatric symptoms is also understandably related to the phenomena of cognitive aging. Cognitive rehabilitation (CT) then becomes a logical intervention in the treatment of psychiatric symptoms in cognitively impaired adults.

Dementia is always influenced by poor lifestyle habits (across the lifespan). In a recent edition of the *Lancet* (Frisoni et al., 2017) it was shown that tackling risk factors helps protect against dementia. This report brought together 24 international experts to review existing research into dementia. Risk factors extend across the lifespan. Hearing loss, obesity, high blood pressure, smoking, depression, physical inactivity, social isolation, and diabetes were noted. All are modifiable: Addressing them reduces about 35% of dementia cases. Targeting Apo-B4 reduces dementia by 10%.

Overall, it is becoming pretty clear that research carried out over the last 50 years indicates that diseases, especially diseases like dementia, are occurring in a very heterogeneous population and have various pathways. In the future what are needed are biologically relevant tests for mental illness in general, and dementia in particular, to identify biomarkers that indicate what kind of behavior/cognitive problem/depression a person has and consequently the best kind of treatment. Right now it is based on someone going in and talking about symptomatology without being able to have any biological tests.

It has been said that AD, the flagship of neurodegenerative decline, can possibly be prevented. A large number of modifiable (e.g., exposures, lifestyle and social habits) and non-modifiable (e.g., age, sex, genetics) risk factors have been identified. Recent revisions to the clinical criteria for AD and MCI have helped clarify the role of biomarkers in defining the pathological cascade, and the addition of research criteria for pre-symptomatic disease sets the stage for better modeling of the preclinical and prodromal stages of disease (Galvin, 2011). Efforts developing and validating fluid (blood and cerebrospinal fluid) and imaging biomarkers make it possible to explore underlying pathological changes in amyloid, tau, dopamine transport, inflammation, signaling pathways, and in the future, alpha-synuclein and TDP-43 in symptomatic, prodromal, and pre-symptomatic individuals. Advances in genetic, epigenetic, and “omics” (e.g., proteomic, lipidomic, metabolomic) approaches will permit the modeling of transcriptional, translational, and posttranslational changes. Furthermore, precision medicine approaches with demonstrable benefits in oncology are being applied to neurodegenerative disorders. Thus, the platform is in place to begin prevention initiatives.

Alzheimer's Disease Risk and Preventative Factors

Risk Factors (Non-modifiable)

Age
Sex
Family history
Apolipoprotein E ϵ 4 allele*

Risk Factors (Modifiable)

Diabetes mellitus and insulin resistance
●besity
Metabolic syndrome
Hypertension
Hypercholesterolemia
Cerebrovascular disease
Depression
Psychological and physiological stress
Traumatic brain injury
Sleep disordered breathing
Smoking
Alcohol abuse

Protective Factors (Modifiable)

Cognitive reserve and mental activity
Educational attainment and lifelong learning
Cognitive leisure activities
Physical activity and exercise
Social engagement
Mindfulness and wellness activities
●optimism and purpose in life
Diet
Omega-3 intake

Person centered care has arrived. In long term care the competencies for older adults are in play (The Alzheimer's Association and Dementia Care Practice Recommendations, 2018). The fundamentals of person-centered care were outlined, and involve general care, detection and diagnosis, assessment and care planning, medical management, information education and support, ongoing treatment for behavioral and psychological symptoms of dementia, and support for activities of daily living, staffing, supportive and therapeutic environments, transitions, and coordination of services. A person-centered focus involved all of these.

Practice recommendation for person-centered care involves:

- 1) knowing the person living with dementia;
- 2) recognizing and accepting the person's reality;
- 3) identifying and supporting ongoing opportunities for meaningful engagement;
- 4) building and nurturing authentic caring relationships;
- 5) creating and maintaining a supportive community for an individual's family and staff; and
- 6) evaluating care practices regularly and making appropriate changes.

We too will have more to say about this in the context of person-centered care and dementia.

Conclusion

It is rare to have a person who is mentally ill and yet have good sleep habits, a nutritionally abundant diet, consistent exercise habits, no medical comorbidities, no trauma, healthy relationships, no substance abuse, and great energy. Generally, therapeutic interventions for older adults for mental issues include conventional or the "usual" treatments, involving some biomarkers (in addition to genetics), medical comorbidities, lifestyle changes, psychotherapies, and increasingly nutrition. Ideally, a clinical pathway should involve a structured multidisciplinary plan of care, the reasonable translation of guidelines or empirically supported data into real care pragmatics, and detailed steps in the care process.

As we have argued, good mental health is a whole person and whole community approach. In making a real difference there is no mind/body separation. Traditionally, psychotherapy has been delivered through 50-minute face-to-face individual sessions with a therapist and a patient. It has become increasingly clear that for older adults, this is often not a workable or viable solution. This model is also expensive and often does not fit the lifestyles or needs of today's older adults who live much more complicated lives, from the very successful and endowed to those that have few resources. That said, psychotherapy itself has several old and new gifts. For the older process, psychotherapy for the person is a validating and challenging process. This can occur in many formats. This above all set the stage for the Watch and Wait model. The lifestyle addition also makes a difference.

Concepts and the practice of therapy are changing. As noted in chapter 1, Knight and Pachana (2015), using the CALTAP model, noted that the techniques of therapy with older adults differ little from work with

younger clients when the problems and their contexts are similar. Therapy at its best leads the patient in empirically supportive ways and a friendly collaborative process. This approach requires that in this case the health care provider consult more extensively about the patient's own goals, fears, and hopes with respect to what could be achieved by such an assessment.

Additionally, in *American Psychologist* several proponents of aging argue that the core constructs that need to be considered for the reasonable future of aging have more to do with neuroscience (APA, 2014). These promote healthy cognitive aging, and perhaps especially neuropsychological assessment and assistance in protecting older Americans with cognitive losses. In addition, proponents advocated psychological research on decision making and behavioral economics, as much to offer planning for retirement and to reduce formability to financial abuse. Psychological research on self-management and behavior can also contribute importantly to enhancing good health behaviors among older adults. A focus on attitudes and stereotypes gives insight into age bias, which can be detrimental to aging. Adaptive technologies and information technologies are beginning to transform assessment in research and clinical settings. Technology also holds the promise of improving long-term support for older adults in both institutional and community based settings.

All that said, we know that treatments work at best only 50% of the time and most often not permanently: patients continue on as both system and self-change failures; patients often wax and wane as they get a little better before they come to see a therapist and at different times during the treatment; long term treatment is still largely an anomaly; there are few by way of specific effects in therapies – dismantling studies have not provided robust findings and it seems that general effects of care are greater than specific effects. These facts percolate as a back drop to the treatment of older adults. They can be for the better or worse, given the needs and complexity of the care of older adults.

And, regardless of the science of therapy, a newer focus on ethics for older people may assist. Psychotherapy should always be a deliberative ethical process. It has been proposed that geriatric care move away from autonomy as a central ethical principle and toward the realization that as capacity diminishes, older people become more interdependent physically and fiscally. Autonomy may not serve well as a central focus of ethical analysis. Speilman (1986) stressed the principles of sociality (recognizing the need for relationships), temporality (recognizing limitations), and embodiment (respecting the change of aging) as being more important for elder ethics than autonomy. Issues regarding religion, consent, late life

care, advanced directive, as well as competency in various areas will always involve the ethical principles of beneficence and non-maleficence, law and equity, and paternalism and autonomy.

Whatever the value of neuroscience, phenomenology of holistic care, and need for ethical deliberation, we believe that the five domains noted are key in the context of a Watch and Wait system. We address the five domains now.

References

- 2018 AARP Brain Health and Mental Well-Being Survey. (2018). *AARP Research*, (October), 1-46.
- American Psychological Association. (2014). Guidelines for psychological practice with older adults. *American Psychologist*, 69(1), 34-65.
- Amjad, H., Roth, D.L., Samus, Q.M., Yasar, S., & Wolff, J.L. (2016). Potentially unsafe activities and living conditions of older adults with dementia. *Journal of the American Geriatrics Society*, 64(6), 1223-1232.
- Author (2018). Massachusetts General Hospital Mind, Mood, and Memory, volume 14, #4, April.
- Baltes, P. & Smith, J. (2008). The fascination of wisdom: Its nature, ontology, and function. *Perspectives on Psychological Sciences and Social Sciences*, 73, 1374-1383.
- Bennett, D. A., Schneider, J. A., Arvanitakis, Z., Kelly, J. F., Aggarwal, N. T., Shah, R. C., & Wilson, R. S. (2006). Neuropathology of older persons without cognitive impairment from two community-based studies. *Neurology*, 66(12), 1837-1844.
- Birditt, K. S., Fingerman, K. L., & Almeida, D. M. (2005). Age differences in exposure and reactions to interpersonal tensions: A daily diary study. *Psychology and aging*, 20(2), 330.
- Bubu, M., Umasabor-Bubu, M., Shim, A., Hogan, M., Sharma, R. A., Azizi, S., ... & Osorio, R. S. (2017). Sleep problems and changes in AD biomarkers are predictive of persons who ultimately develop AD: Findings from subgroup meta-analysis on sleep and Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 13(7), P1007-P1008.
- Cacioppo, S., Grippo, A. J., London, S., Goossens, L., & Cacioppo, J. T. (2015). Loneliness: Clinical import and interventions. *Perspectives on Psychological Science*, 10(2), 238-249.
- Carr, D. C., Kail, B. L., Matz-Costa, C., & Shavit, Y. Z. (2018). Does Becoming A Volunteer Attenuate Loneliness Among Recently

- Widowed Older Adults? *JOURNALS OF GERONTOLOGY SERIES B-PSYCHOLOGICAL SCIENCES AND SOCIAL SCIENCES*, 73(3), 501–510. <https://doi-org.medlib-proxy.mercer.edu/10.1093/geronb/gbx092>
- Carr, D. C., Kail, B. L., & Rowe, J. W. (2018). The Relation of Volunteering and Subsequent Changes in Physical Disability in Older Adults. *JOURNALS OF GERONTOLOGY SERIES B-PSYCHOLOGICAL SCIENCES AND SOCIAL SCIENCES*, 73(3), 511–521. <https://doi-org.medlib-proxy.mercer.edu/10.1093/geronb/gbx102>
- Coan, J. A., & Beckes, L. (2013). Social baseline theory and the social regulation of emotion. In *The Science of the Couple* (pp. 93-106). Psychology Press.
- Diehr, P. H., Thielke, S. M., Newman, A. B., Hirsch, C., & Tracy, R. (2013). Decline in health for older adults: Five-year change in 13 key measures of standardized health. *The Journals of Gerontology: Series A: Biological Sciences and Medical Sciences*, 68(9), 1059–1067. <https://doi-org.medlib-proxy.mercer.edu/10.1093/gerona/glt038>
- Donovan, N. J., Okereke, O. I., Varmini, P., Amariglio, R. E., Rentz, D. M., Marshall, G. A., ... & Sperling, R. A. (2016). Association of higher cortical amyloid burden with loneliness in cognitively normal older adults. *JAMA psychiatry*, 73(12), 1230-1237.
- Emmons, R. A., Emmons, R. A., & McCullough, M. E. (2004). *The psychology of gratitude*. Oxford: Oxford University Press, 2004.
- Fazio, S., Pace, D., Maslow, K., Zimmerman, S., & Kallmyer, B. (2018). Alzheimer's Association Dementia Care Practice Recommendations. *The Gerontologist*, 58(1), S1–S9. <https://doi-org.medlib-proxy.mercer.edu/10.1093/geront/gnx182>
- Frank, R. I., & Davidson, J. (2014). *The Transdiagnostic Road Map to Case Formulation and Treatment Planning: Practical Guidance for Clinical Decision Making*. Oakland: New Harbinger Publications.
- Fraser, J. S., & Solovey, A. D. (2007). *Second-order change in psychotherapy: The golden thread that unifies effective treatments*. American Psychological Association.
- Fried, L. P., Tangen, C. M., Walston, J., Newman, A. B., Hirsch, C., Gottdiener, J., ... Burke, G. (2001). Frailty in Older Adults: Evidence for a Phenotype, 56(3), 146–157.
- Frisoni, G. B., Boccardi, M., Barkhof, F., Blennow, K., Cappa, S., Chiotis, K., ... & Hansson, O. (2017). Strategic roadmap for an early diagnosis of Alzheimer's disease based on biomarkers. *The Lancet Neurology*, 16(8), 661-676.
- Galvin, J. E. (2011). Dementia screening, biomarkers and protein misfolding: Implications for public health and diagnosis. *Prión*, 5(1),

- 16–21. Retrieved from <http://medlib-proxy.mercer.edu/login?url=https://search.ebscohost.com/login.aspx?direct=true&db=cmedm&AN=21164279&site=eds-live&scope=site>
- Gaby, A. (2017). *Nutritional Medicine (Second Edition)*. Guilford Press, New York.
- Goeres, L. M., Gille, A., Furuno, J. P., Erten-Lyons, D., Hartung, D. M., Calvert, J. F., ... & Lee, D. S. (2016). Rural-Urban Differences in Chronic Disease and Drug Utilization in Older Oregonians. *The Journal of rural health, 32*(3), 269-279.
- Greenberg, L. (2014). The therapeutic relationship in emotion-focused therapy. *Psychotherapy, 51*, 3, 350-358
- Grossmann, I. (2017) Wisdom in context. *Perspectives on Psychological Science: A Journal of the Association for Psychological Sciences, 12*, 233-257.
- Guiney, H., & Machado, L. (n.d.). Volunteering in the Community: Potential Benefits for Cognitive Aging. *JOURNALS OF GERONTOLOGY SERIES B-PSYCHOLOGICAL SCIENCES AND SOCIAL SCIENCES, 73*(3), 399-408.
<https://doi-org.medlib-proxy.mercer.edu/10.1093/geronb/gbx134>
- Hackett, R. A., Davies-Kershaw, H., Cadar, D., Orrell, M., & Steptoe, A. (2018). Walking Speed, Cognitive Function, and Dementia Risk in the English Longitudinal Study of Ageing. *Journal of the American Geriatrics Society, 66*(9), 1670-1675.
- Halloway, S., Arfanakis, K., Wilbur, J., Schoeny, M. E., & Pressler, S. J. (2018). Accelerometer Physical Activity is Associated with Greater Gray Matter Volumes in Older Adults without Dementia or Mild Cognitive Impairment. *The Journals of Gerontology. Series B, Psychological Sciences and Social Sciences*. <https://doi-org.medlib-proxy.mercer.edu/10.1093/geronb/gby010>
- Jansen, W. J., Wilson, R. S., Visser, P. J., Nag, S., Schneider, J. A., James, B. D., ... Boyle, P. A. (2018). Age and the association of dementia-related pathology with trajectories of cognitive decline. *Neurobiology of Aging, 61*, 138–145. <http://doi.org/10.1016/j.neurobiolaging.2017.08.029>
- Knight, B. G., & Pachana, N. A. (2015). *Psychological assessment and therapy with older adults*. New York, NY: Oxford University Press. <https://doi-org.medlib-proxy.mercer.edu/10.1093/med:psych/9780199652532.001.0001>
- Kojima, G., Iliffe, S., & Walters, K. (2018). Frailty index as a predictor of mortality: a systematic review and meta-analysis. *Age and Aging, 47*, 193–200. <http://doi.org/10.1093/ageing/afx162>

- Langa, K. M., Larson, E. B., Crimmins, E. M., Faul, J. D., Levine, D. A., Kabeto, M. U., & Weir, D. R. (2017). A comparison of the prevalence of dementia in the United States in 2000 and 2012. *JAMA Internal Medicine*, 177(1), 51-58.
- Leung, T. (2018). www.sciencedaily.com/release/2018/08/180820164248.htm
- Liebman, Bonnie (2018). Game Changers: 5 studies that make a difference. *Nutrition Action, Health Letter*. December, 2018.
- Loprinzi, P. D., & Joyner, C. (2017). Relationship Between Objectively Measured Physical Activity, Cardiovascular Disease Biomarkers, and Hearing Sensitivity Using Data From the National Health and Nutrition Examination Survey 2003-2006. *AMERICAN JOURNAL OF AUDIOLOGY*, 26(2), 163-169. [https://doi-org.medlib-proxy.mercer.edu/10.1044/2017pass:\[\]AJA-16-0057](https://doi-org.medlib-proxy.mercer.edu/10.1044/2017pass:[]AJA-16-0057)
- Liu, B., Floud, S., Pirie, K., Green, J., Peto, R., & Beral, V. (2016). Articles: Does happiness itself directly affect mortality? The prospective UK Million Women Study. *The Lancet*, 387, 874-881. [https://doi-org.medlib-proxy.mercer.edu/10.1016/S0140-6736\(15\)01087-9](https://doi-org.medlib-proxy.mercer.edu/10.1016/S0140-6736(15)01087-9)
- Loprinzi, P. D., Herod, S. M., Cardinal, B. J., & Noakes, T. D. (2013). Physical activity and the brain: a review of this dynamic, bi-directional relationship. *Brain Research*, 1539, 95-104.
- Lyubomirski, S. (2008). *The how of happiness. A scientific approach to getting the life you want*. The Penguin Press, London.
- Memory Maximizers. (2018). *Mind, Mood & Memory*, 14(8), 5. Retrieved from <http://medlib-proxy.mercer.edu/login?url=https://search.ebscohost.com/login.aspx?direct=true&db=edb&AN=130821397&site=eds-live&scope=site>
- Moon, J. R., Kondo, N., Glymour, M. M., & Subramanian, S. V. (2011). Widowhood and mortality: a meta-analysis. *PLoS One*, 6(8), e23465.
- Nash, S. H., Liao, L. M., Harris, T. B., & Freedman, N. D. (2017). Cigarette smoking and mortality in adults aged 70 years and older: Results from the nih-aarp cohort. *American journal of preventive medicine*, 52(3), 276-283.
- Norcross, J., & Lambert, M. (2018) Psychotherapy relationships that work III. *Psychotherapy*, 55,4, 303-315.
- Perissinotto, C. M., Cenzer, I. S., & Covinsky, K. E. (2012). Loneliness in older persons: a predictor of functional decline and death. *Archives of internal medicine*, 172(14), 1078-1084.

- Pietromonaco, P. R., & Collins, N. L. (2017). Interpersonal mechanisms linking close relationships to health. *American Psychologist*, 72(6), 531.
- Proulx, C. M., Curl, A. L., & Ermer, A. E. (n.d.). Longitudinal Associations Between Formal Volunteering and Cognitive Functioning. *JOURNALS OF GERONTOLOGY SERIES B-PSYCHOLOGICAL SCIENCES AND SOCIAL SCIENCES*, 73(3), 522–531. <https://doi-org.medlib-proxy.mercer.edu/10.1093/geronb/gbx110>
- Radloff, L. S., & Rae, D. S. (1979). Susceptibility and precipitating factors in depression: sex differences and similarities. *Journal of abnormal psychology*, 88(2), 174.
- Robles, T. F., Slatcher, R. B., Trombello, J. M., & McGinn, M. M. (2014). Marital quality and health: A meta-analytic review. *Psychological bulletin*, 140(1), 140.
- Rockette-Wagner, B., Storti, K. L., Dabelea, D., Edelstein, S., Florez, H., Franks, P. W., ... & Kriska, A. M. (2017). Activity and Sedentary Time 10 Years After a Successful Lifestyle Intervention: The Diabetes Prevention Program. *American journal of preventive medicine*, 52(3), 292-299.
- Romundstad, S., Svebak, S., Holen, A., & Holmen, J. (2016). A 15-year follow-up study of sense of humor and causes of mortality: The Nord-Trøndelag Health Study. *Psychosomatic Medicine*, 78(3), 345–353. <https://doi-org.medlib-proxy.mercer.edu/10.1097/PSY.0000000000000275>
- Rook, K.S. & Charles, S.T. (2017). Close social ties and health in later life: Strengths and vulnerabilities. *American Psychologist*, 72(6), 567-577.
- Saint-Maurice, P. F., Troiano, R. P., Berrigan, D., Kraus, W. E., & Matthews, C. E. (2018). Volume of Light Versus Moderate-to-Vigorous Physical Activity: Similar Benefits for All-Cause Mortality? *JOURNAL OF THE AMERICAN HEART ASSOCIATION*, 7(7). <https://doi-org.medlib-proxy.mercer.edu/10.1161/JAHA.118.008815>
- Shaw, E. E., Schultz, A. P., Sperling, R. A., & Hedden, T. (2015). Functional connectivity in multiple cortical networks is associated with performance across cognitive domains in older adults. *Brain connectivity*, 5(8), 505-516.
- Slatcher, R. B., Selcuk, E., & Ong, A. D. (2015). Perceived partner responsiveness predicts diurnal cortisol profiles 10 years later. *Psychological Science*, 26(7), 972-982.

- Small, G. W., & Greenfield, S. (2015). Current and Future Treatments for Alzheimer Disease. *American Journal of Geriatric Psychiatry*, 23(11), 1101–1105. <http://doi.org/10.1016/j.jagp.2015.08.006>
- Small, G. W., Siddarth, P., Zhaoping, L., Miller, K., Ercoli, L., Emerson, N., ... Barrio, J. (2018). Memory and Brain Amyloid and Tau Effects of a Bioavailable Form of Curcumin in Non-Demented Adults: A Double-Blind, Placebo-Controlled 18-Month Trial. *The American Journal of Geriatric Psychiatry*, 26(3), 266–277. <http://doi.org/10.1016/j.jagp.2017.10.010>
- Spielman, A. J. (1986). Assessment of insomnia. *Clinical Psychology Review*, 6(1), 11–25. [https://doi-org.medlib-proxy.mercer.edu/10.1016/0272-7358\(86\)90015-2](https://doi-org.medlib-proxy.mercer.edu/10.1016/0272-7358(86)90015-2)
- Stuadinger, U. & Gluck, J. (2011). Psychological wisdom research: Commonalities and differences in a growing field. *Annual Review of Psychology*, 62, 215-241.
- Stern, Y. (2012). Cognitive reserve in ageing and Alzheimer's disease. *The Lancet Neurology*, 11(11), 1006-1012.
- Ticinesi A, Tana C, Nouvenne A, Prati B, Lauretani F, & Meschi T. (2018). Gut microbiota, cognitive frailty and dementia in older individuals: a systematic review. *Clinical Interventions in Aging, Volume 13, Pp 1497-1511 (2018)*, 1497. Retrieved from <http://medlib-proxy.mercer.edu/login?url=https://search.ebscohost.com/login.aspx?direct=true&db=edsdoj&AN=edsdoj.9543ed2806c4c76a5f56dbc9a696aeb&site=eds-live&scope=site>
- Tornstam, L. (1994). Gero-transcendence: A theoretical and empirical exploration. In L.E. Thomas & A. Eisenhandler (Eds.), *Aging and the Religious Dimension* (pp 203-225). London, Auburn House.
- Walsh, R. (2011). Lifestyle and mental health. *American Psychologist*, 66, 7, 579-592.
- Weststrate, N., Ferrari, M., Fournier, M., & McLean, K. (2018). “It was the worst day of my life”: Narrative content, structure, and process in wisdom-fostering life event memories. *The Journals of Gerontology Series B Psychological Sciences and Social Sciences*, 73, 8, 1359-1373.

CHAPTER 5

SCREEN MEASURES OF FIVE DOMAINS

Previously we noted that the importance of precision medicine. Everyday more information becomes available about factors that predict the risk of the clinical event. Predictive analytics incorporate this information into prognostic models that estimate the likelihood of this event for an individual patient. The Framingham Heart Study pioneered this approach and such estimates have become core elements of clinical care and guideline recommendations, such as recent ACC/AHA guidelines for managing blood cholesterol for primary prevention in cardiovascular disease risk. Advances in predictive analytics and precision medicine have and will continue to change the practice of medicine. Yes, predictive algorithms are an essential component of guideline recommendations. However, because predictive models imperfectly explain clinical outcomes, they do not estimate individual risk very well even when they accurately explain the group risks. Consequently, these models cannot replace a physician or healthcare provider in the process of everyday care. That said, we need reasonable markers for understanding the complexity of problems.

Previously we have also argued that there are five core areas that can be reasonably measured. The areas are more important than the distinct variables that inhabit the area. Being depressed, for example, portends more than a certain amount of symptoms the whole person needs an evaluation of each core domain. We are living longer and that portends more complexity to the older age adult in question. Living to late-late life also has its own demands. Medical advances have clearly made an impact. Better treatments in new drugs were not available even a generation ago. Despite all these new technologies, however, the real issue is whether our increased life expectancy adds an additional active and healthy live.

We have argued for and explicated the Watch and Wait model. ● Over 20% of adults have a current psychiatric diagnosis and 50% experience one in a lifetime. Also over 20% are currently taking a psychiatric medication. Many have multiple diagnoses. Comorbidity itself means that there is a need for more information and precision (splitter's) but does not

mean that there are separate causalities or a need for separate treatments. This of course leads to uncoordinated care and polypharmacy. Unfortunately as noted, there are no clear-cut biomarkers for psychiatric disorders. And, would this help? We know that most older adults suffer from the chronicity of the human condition. In many cases problems are vexing and even life altering but at levels less than pure syndromes. Often also, the less severe the symptoms, the more difficult the diagnosis. Patients at the boundary between psychiatry and medical illness present daunting challenges to both and are often not helped by either. This seems vaguely similar to common problems at later life.

Recall in the Watch and Wait model we treat carefully and slowly. We apply a case management approach. It is a step approach that targets the five domains. In the process there is psychoeducation, validation, normalization, and support. The person is treated with a plan and monitored. The whole person is addressed and the life adjustment domains are given equal weight.

Process of Watch and Wait Evaluation

Stepped diagnosis

Symptoms come and go but the psychological area is often persistent

Assessment, education, normalization, reassurance

Treat and re-evaluate

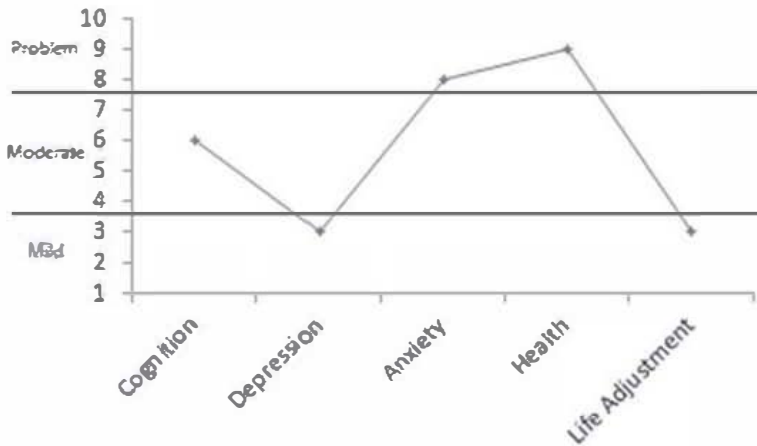
Provide room especially for “universal” medical and life issues

In this chapter we extend on the previous chapters and discuss and outline the “quick and reasonable” measurement of five core domains. We provide a brief introduction into each and the core factors assessed. We address assessment largely but also make suggestions about treatment. We highlight the suggested screen for each area and the importance of each scale. Here we provide a profile of a case. We suggest flexibility with the various scales and rigidity for the five areas. We are most interested in screens. Given problems, a longer evaluation follows. In the chapters that follow we elaborate on each domain and suggest more complete assessment. This chapter then acts as a primer. It is intended to be an introduction to the following five chapters on each of the domains.

Recall that we have an assessment battery that includes a rating for each domain of Mild/Normal, Moderate, or Problem: A Problem requires immediate attention and is causing turmoil in the person’s life now; Moderate designations require attention also but are less emergent and less intense than Problems; and Mild/Normal indicates a watch and wait mode

but is always an influence on the core problems. We apply a 1-10 metric with ≥ 8 as a Problem, 5-7 as Moderate and < 5 as Mild/Normal. Once again, the problem is rated on the basis of the screen and later may be altered by a more intensive evaluation. Again, this rating is arbitrary. It is based on screen markers anchored in psychological tests, ratings or behaviors. The focus is on identifying a domain that needs attention in the context of other relative domains.

Health



This person has presented with health problems along with some anxiety as core issues. Patient is a 64-year-old Caucasian male who was interviewed with his wife. He was admitted to the hospital for post evaluation after an LVAD (heart) procedure. He indicated that in 1998, while he was employed as a principal, he had a massive myocardial infarction that altered his life measurably. Over the years, this heart problem got worse. Sometime after, he had to retire. He has had 4 stents in 1998. In 1999, he had a bypass and he was able to handle things reasonably until more recently. Three weeks ago, he was given the information that he had less than 1 year to live unless he had the LVAD. He lives locally with his wife. We see him here post-LVAD and adjusting to his new life still concerned (anxious) about health. We pick up on this case in the health chapter.

Measures:

HEALTH

Measure	Healthy	Moderate Problem	Problematic
Description	Bodily function is normal. The patient has few complaints of pain and few chronic conditions, which are likely to shorten life or reduce .Good QOL	Minor health concerns which may include lesser chronic conditions. QOL is impacted, but not debilitating so.	ADL/IADLs are compromised. Excessive comorbidities or meds
Number of Chronic Illnesses	≤1	2-3	≥4
Health Rating	Good	Average	Poor
Epworth Sleep	≤10	10-12	≥12
Pain Visual Analogue Scale	≤44 mm	45-74 mm	≥75 mm
Cigarette Use	Never smoked	Smoked 100+ but quit	Current smoker
Alcohol Consumption	≤7 drinks per week if the patient is not on medication	7 drinks per week if the patient is not on medication	>7 drinks per week if the patient is on medication known to interact with alcohol
BMI	18.5 – 24.9	25-29.9	Under 18.5 or over 29.9
Exercise	>30 minutes everyday	<30 minutes/ 1-2 days week	Housework, inactive

Health is most often the initial reason for evaluation. Our position is that health status is the core domain, period. Consequently we spend more time on this variable here leading to Chapter 6. It is the core of expressed problems at later life: One might even say that without health, well you know... Health variables decline on average with advancing age, but at significantly different rates. As we have reported, standardized measures in mental health, cognition, quality of life, and hospital utilization do not decline as fast as gait speed, activities of daily living, and instrumental activities of daily living (Diehr et al., (2013). We chose to measure medical problems (comorbidities), a health rating, sleep, pain, smoking, BMI, alcohol use, and exercise.

Providing medical care for older adults requires a healthy mix of science and art. One needs to respect the unique physiology of aging as well as evidence of the effectiveness of drug and procedural interventions in the elderly. Perhaps one rule is the most obvious: When older adults become sick, always address this but think about the bigger picture. Consider whether the cause could be iatrogenic, that is, caused by a

medication or excessive medical planning/caution. Another unique aspect of geriatric medicine is the atypical presentation of disease. Informed decision-making for frail older adults also requires an honest discussion of the potential risks and benefits of the intervention, in addition to the patient's preferences. Common problems include hypertension, diabetes, coronary artery disease, vitamin deficiency (especially D and B12), osteoarthritis, falls, polypharmacy, and hearing/visual impairment.

Health: The person should be involved. This involves hard data (number of illnesses) and soft data (self-rating of health). Ferraro and Wilkinson (2015) compared the prognostic validity of alternative measures that health rating may assist in over 12,000 Americans ages 50 to 74 in 1995 and vital status tracts 2005 to compare the effect of three types of health ratings on mortality. Conventional indicators of self-rated health implicitly include age comparison of self-rated health and health ratings that incorporate the temporal dimension. Self-rated health has always been a consistent predictor of mortality, but here the respondent's expected health rating 10 years in the future was an independent predictor. Future health expectations were then as or more important than tasks. People with more negative expectations for the future were less likely to survive. People have a sense of their life and future.

Health has many components to target. Just rating one's health is a marker. Blood pressure is perhaps the most studied and visible. It is a chronic condition that causes progressive organ damage. It is well known that the vast majority of cases of AD and related dementias are not due to genetic predisposition but rather to chronic exposure to vascular risk factors. This disorder is the jumping off point for AD. It has becoming increasingly clear that when signs of brain damage are manifest, it may be too late to reverse the neurodegenerative process.

At the Regional Excellence Hypertension Center of the Italian Society of Hypertension in the Department of Angiocardioneurology and Translational Medicine of the I.R.C.C.S. Neuromed, in Italy (Carnevale, et al., 2018), researchers recruited people aged 40 to 65. This work was conducted on patients with no sign of structural damage and no diagnosis of dementia. All patients underwent clinical examination to determine their hypertensive status and the related target organ damage. Additionally, patients were subjected to an MRI scan to identify microstructural damage. Neurocognitive tests were provided. As primary outcome of the study the researchers aimed at finding any specific signature of brain changes in white matter microstructure of hypertensive patients, associated with an impairment of the related cognitive functions. The result indicated that hypertensive patients showed significant alterations in three specific

white matter fiber-tracts. Hypertensive patients also scored significantly worse in the cognitive domains ascribable to brain regions connected through those fiber-tracts, showing decreased performances in executive functions, processing speed, memory and related learning tasks. Overall, white matter fiber-tracking on MRIs showed an early signature of damage in hypertensive patients when otherwise undetectable by conventional neuroimaging. As these changes can be detected before patients show symptoms, these patients could be targeted with medication earlier to prevent further deterioration in brain function (Carnevale, et al., 2018).

Of people 65 and older, most have chronic diseases: 67% are treated for 1 or 2 chronic conditions and 26% had greater than 4 chronic conditions (Machlin & Soni, 2013). The cost for one with 4 or more conditions was twice those of people with 2 or 3 and seven times those with none. Health habits are of course critical. Seventy percent of morbidity and mortality is accounted for by how much we eat, what we eat, do we exercise, do we smoke, and do we consume alcohol excessively (deVol & Bedroussian, 2007). We treat chronic diseases (Scott, et al., 2016) and all that is implied by this. Adverse drug reactions, a typical excessive care feature, are common and account for a substantial amount of emergency room use, hospital admissions, and other healthcare expenditures. In fact, only 50% of medication is taken properly, and there are 1.9 million drug-related injuries (Cogbill, Dinson, & Duthie, 2010).

Mobility is related. Mobility used to be considered as an automatic task. More recent research, however, has found that gait requires an allocation of considerable cognitive abilities. This involves attention and executive functioning. Declines in global cognitive function and executive functioning are associated with increased risks of falling, for example. These changes might be present in cognitively normal older adults. When cognitive impairment is added to the normal age-related decline in cognitive function, however, the impact of function is exacerbated and the risk of falls is almost doubled. Gait disturbances are present in older adults suffering from dementia, whether MCI or other forms of dementia such as AD. These disturbances are often noted by slow gait speed, greater gait variability, shorter step length, balance disturbance, and increased risk of falling, as well as neurological gait signs, often resembling Parkinson's disease. Changes in gait velocity can occur up to 12 years before signs of cognitive impairment are very much related.

Preventing falls is highly relevant here. This is the leading cause of ER visits and hospitalization. Falls also have a cognitive component. Due to physiological impairments in community-dwelling older people, there may need to be an extra focus on cognitive impairment, a key factor in their

inability to compensate for physical decline (Martin, et al., 2013). Use of a cognitive scale with one of several validated fall scales (e.g., Delbaere, et al., 2013) may be a good start. Also, we note that actual falling is not the only problem with this area; fear of falling can be a contributor resulting in isolation and excessive anxiety.

Frailty is especially a problem. It is associated with dementia when adjusting only for demographic variables in many studies. Although frailty was not associated with all-cause dementia in entire samples, an association was found in participants with selective cognitive scores. This was a sample of 2,619 participants aged 65 and older between 1994 and 2010 who were assessed in a Harvard study (Gray et al., 2013). Frailty was assessed and mechanisms underlying their associations were found to be multi-causal. On the other side of the coin, many types of resilience were noted to obviate frailty. They have been mentioned in the context of physical decline. They include health, psychological, emotional, dispositional and physiological (see Resnick, et al., 2011) (See Chapter 11). Cognitive frailty, the frailty phenotype that predisposes one to dementia is often the result (Ticinesi, et al., 2018).

Sleep: Valenti, Bonomi, and Westerterp (2017) assessed the quality of sleep and metabolic rate in older adults. Sleep is a temporary state of altered consciousness that occurs in approximately one-third of our human life. Sleep promotes growth, recovery, and cognitive wellbeing. Reduced total sleep time (TST) affects endocrine and metabolic functions and sleep deprived participants can show confusion, depression, hallucinations, and in extreme cases death. During slow-wave sleep (SWS), it is the stage associated with growth and recovery. Rapid eye movement (REM) sleep is associated with cognitive function and its selective deprivation is associated with executive function, pain threshold, and emotional memory consolidation. Studies that selectively deprive participants of SWS or REM show a rebound effect during a period following the deprivation underlying the importance of both SWS and REM.

As a culture we have lost two hours of sleep per night over 70 years. Sleep is required to rebuild its cache of neurotransmitters, neural growth factors, and cell-building proteins which gradually become depleted during waking hours. The prevalence of people over the age of 65 who have difficulty with distinct sleep features involve the following: Difficulty falling asleep roughly 65%, waking during the night 63%, waking up too early 39%, poor sleep quality 28%, use of concomitant anticholinergic medication 33%, use of alcohol 65%, and 15 days per month using diphenhydramine 37%. This includes people 65-74 year of age. The numbers for adults greater than 75 are somewhat less (Harper, et

al., 2005). With less sleep the amygdala response to emotional images is unchecked and goes into overdrive. In fact, insomniacs have different brains than non-insomniacs, the grey matter has less density and has a different appearance. Cognition suffers to a considerable degree, with 30% loss of motor functioning also. Chronic insomniacs who slept less than six hours are more likely to die during the 14-year follow-up period. Some 45% of individuals between the ages of 65 and 79 years report mild to severe problems falling and staying asleep, and such reports are associated with poor daytime functioning (Harper, et al., 2005).

Short sleep durations, six hours or less per night, are associated with multiple adverse health outcomes, including risk for coronary disease, hypertension, diabetes, obesity, immune system changes, depression, anxiety, and mortality. Short sleep duration is also associated with deficits in memory, processing speed, attention, and language. Other research has shown that individuals residing in urban and/or heavily populated environments may be at increased risk for short sleep duration, perhaps due in part to the stress on neighborhood environments, including crime, noise, limited access to resources, and discrimination. Noteworthy is the fact that sleep duration variability alone causes problems; sleep duration variability by as much as 60 minutes per day influence metabolic syndrome.

In older adults the effects of poor sleep extend over many domains. Documented impacts of poor sleep in older adults include difficulty sustaining attention, slowed response time and impairments in memory and concentration, decreased ability to accomplish daily tasks, increased risk of falls, inability to enjoy social relationships, increased incidence of pain, the reduced quality of life, risk of traffic accidents, increased consumption of healthcare resources, and shorter survival (Albert, et al., 2015). We noted previously that even one night's partial sleep deprivation activates gene expression patterns and peripheral blood mononuclear cells consistent with increasing accumulation of damage that initiates cell-cycle arrest that increases susceptibility to senescence. These findings causally link sleep deprivation to etiology and biological aging and further supports the hypothesis that sleep deprivation may be associated with elevated disease risk because it promotes molecular processes involved in biological aging. Sleep affects emotional regulation also. People who are losing their ability to regulate their emotions may be more likely to suffer from insomnia. If they do, insomnia is more likely to be persistent.

Fatigue needs attention. Lin and colleagues (2014) noted that high fatigability, a dysfunctional adaptation to fatigue, may lead to difficulties performing otherwise regularly encountered cognitive activities and may

be related to pro-inflammatory reactivity. They measured IL-6 of self-reported acute fatigue, and frontally oriented cognitive processes in 55 community-dwelling individuals, age 75 and older. Subjects were classified into groups of low and high fatigability. High fatigue led to pro-inflammatory state in older adults and was related to reduce improvements in cognitive processes. Strikingly there are small things that can be done that seem to make a difference. Sleep hygiene is a good start. Mid-day naps are associated with reduced blood pressure for example. Two influential people, such as Winston Churchill and Margaret Thatcher, did not want to be disturbed around 3:00 in the afternoon. This also seems to apply to older adults at the present time. Average day-night systolic blood pressures in people who take naps are different from those who do not, for the better.

Pain: In the DSM-5, things have changed regarding pain. The Somatic Symptom Disorder is characterized by somatic symptoms that cause significant disruption, excessive thoughts, feelings, and behaviors associated with such symptoms in chronicity. This change in the DSM-5 presents a better recognition that physical and psychological elements of the pain experienced are well integrated. In other words, the DSM-5 appropriately does not encourage separate estimation of the contribution of physical versus psychological factors.

Pain leads to sleep problems. Sleep concerns in chronic pain patients generally have a significant effect on working memory and can now be documented by fMRI BOLD during working memory tasks. This can be independent of pain level and depression even when performance has shown not to be a significant effect. Cognitive complaints are common in patients with chronic pain, as well as objectively measured cognitive deficits. Working memory is often reduced in chronic pain and the reduction is independent of local analgesia. The effect of chronic pain on working memory is moderate and there is considerable discrepancy in studies. Furthermore, working memory is affected by depression and sleep problems, both of which are common in chronic pain patients.

Pain is always a subjective experience. It does the very things that cause problems with depression and anxiety – rumination, catastrophe thinking, and helplessness. Given pain then, usual Axis I problems unfold easily and readily, especially anxiety and depression. Chronic pain often is a core symptom of depression, at least depressive symptoms. Given pain, the site in the brain that is most affected is one subserving depression. Pain also interacts with cognition, as both are equal contributors to ADL and IADL functioning (Shega, Hougham, Stocking, Cox-Hayley, & Sachs, 2008). It is noteworthy also that increasingly there is integration between

psychosocial factors and neurobiology (neurotraits) that reflect patterns of resting state activity in distinct, distributed brain networks. The mind-body in motion.

CBT for chronic pain is typically a time-limited intervention with two primary goals; better management of the person's pain and improved quality of life despite the pain. Recognizing the interconnections among thoughts, feelings, and behaviors, CBT involves largely four broad categories of therapeutic techniques. They include cognitive techniques such as cognitive restructuring and problem solving, behavioral techniques such as relaxation training, pacing, and behavioral activation, supportive techniques such as psycho-education and supportive therapy, and a diverse set of complimentary approaches including, but not limited to biofeedback, relapse prevention, and hypnosis.

Pain is in all domains. Typically, we use standardized pain assessment tools in addition to a physical exam. We apply a 1-10 scale. More specifically, we use the Brief Pain Inventory-short form (BPI-SF: Cleeland & Ryan, 1994), and at times the McGill short form pain questionnaire (SF-MPQ: Melzack, 1987). We also consider the impact of chronic pain on daily functioning on the FAQ and other measures of functioning. In Chapter 6 we noted the importance of attitudes and beliefs about pain, as well as the value of collateral information from family members and caregivers. In this domain we also identify resources that can provide assistance with ADLs/IADLs, as well as reinforce adherence to the treatment plan, as well as comorbidities. We also appreciate the value of the Mayo Clinic's use of the Central Sensitization Syndrome for pain. This sees pain that is "brain-related." More on this in Chapter 6.

Alcohol/Prescriptions: Some of the problems that have been bothering healthcare providers for many years seem solved by the DSM-5 eliminating both substance abuse and substance dependence. Like pain, the DSM-5 includes only one substance-related diagnosis, Substance Use Disorder (SUD; American Psychiatric Association, 2013). To be diagnosed with an SUD, individuals will need to have problematic patterns of substance use leading to impairment or distress, as well as criteria, such as drinking larger amounts over a period of time, unsuccessful efforts to cut down, spending a significant amount of time in activities to obtain a substance, failure to fulfill obligations, continued use in spite of social and interpersonal problems, giving up or reducing activities, recurrent use of hazardous situations, continued use in spite of physical or psychological problems associated with the use, tolerance as indicated by either the need for markedly increased amounts of the substance, or diminished effect

with continued use, withdrawal or craving, as well as a strong desire for the substance.

Alcohol is the most commonly used substance among older adults and drinking may lead to a number of medical, functional, and psychiatric problems. In addition to alcohol, recent studies have shown an increase in prescription drugs as well as cannabis and some other legal and illegal substances. A substantial percentage of older adults seeking mental health services report using both alcohol and drugs (over 50% drink more than 12 drinks per year). A number of negative outcomes are associated with misuse of alcohol or drugs among older adults. Physical systems negatively affected by heavy drinking include the gastrointestinal, cardiovascular, endocrine, hematological, and neurological systems. Assessments for these are always important. Midlife alcohol consumption especially needs to be more carefully examined, especially in relation to dementia incidence. Substance use assessment and intervention are important components of any clinical practice for neuro- or gero-psychology.

Importantly, alcohol use limits have been modified for older adults. The NIAAA has concurrent guidelines that recommend that older adults have no more than 3 drinks in any given day and no more than 7 drinks a week. Some have argued, however, that these guidelines perhaps are too high. Epidemiological studies indicate that substantial numbers of older adults are affected by hazardous or overwhelming drinking. Approximately one-third of older men and one-tenth of older women drink above recommended NIAAA guidelines with the population average of 10-13% overall (Satre, et al., 2018). Drinking patterns in late life may also increasingly affect older women on the basis of cohort-based shifts in drinking patterns and the growth of older women as a sub-group within the U.S. population. Women's drinking and alcohol-related problems may develop later in life. Although older women generally drink less than older men, they potentially experience more related health problems at comparable levels of alcohol consumption.

As part of initial screening then, older adults 65 and older should be asked whether they ever had 4 or more drinks in any one occasion during the prior year. This single item on the screeners tends to be more valid in the general population as a tool for identifying alcohol use, but it has not been examined to any degree with older adults. Of the slightly longer instruments the CAGE (cut down, amoyance, guilt, eye-opener) has been most successfully used to screen for lifetime alcohol use disorders. It has not been well-validated among older adults either. Other measures include the AUDIT or AUDIT-C (Alcohol Use Disorders Identification Test), which is the alcohol use disorders identification test and the MAST

(Michigan Alcohol Screening Test – geriatric version). Both scales appear to be appropriate for older adults. It is noteworthy too that under-recognition among providers is common. Older adults are more likely to deny or minimize substance-related problems than the younger adults because of social stigma associated with the substance use. Of note, moderate drinking among retired older adults tends to increase and can be problematic. Also, there are circumstances when any alcohol consumption in older age is risky, such as with concurrent use of alcohol interacting with prescription drugs.

Prescription drug use is of course an issue. It has been on the rise and in the popular media in the last five years. Scales have been developed to measure opioid use and other general problem use of medications (e.g., S-O-PR). Other problem medications for older adults include sedatives, hypnotics, sleep aides, opioid analgesics and pain killers which have significant potential for abuse and dependence. In addition, in a study of benzodiazepine use among primary care patients, adults age 60 years and older, researchers examined patterns of generally of long-term use. Although ongoing use generally is not recommended, 30% of the participants reported daily use 60 days after their initial prescription, putting them at risk for dependence. Substantial prior month rates of misuse of prescription sedatives have been found in 16% of men and 9% of women aged 60 years and older in depression treatment. Factors associated with prescription drug misuse among older adults are somewhat different than those associated with alcohol misuse.

In the National Survey of Drug Use and Health (NSDUH) data that examine risk factors for non-prescription use of pain medication was presented. Risks for non-prescription use was associated with younger age, Native American, or Alaskan Native ethnicity, and use of cannabis, and adults aged 50-65 are more likely to misuse prescription drugs than adults aged 65 and older. It does seem that use of illicit drugs is increasing among older adults. On the basis of a 2005 NSDUH study, among adults ages 50-59 illicit use in the month prior increased between 2002 and 2005. For those 50-54, the rates increase from 3% to over 5%. Among those from 55-59 the rate was 1 to 4%. (Bishop, et al., 2005).

Interventions are a problem. As much as possible, minimal intervention techniques should be considered first. Many older adults may drink at levels considered risky or problematic, yet they do not meet criteria for substance abuse or dependence and are unlikely to seek out formal alcohol treatment. These adults, however, often come to the attention of health or mental health providers. In the context of brief interventions, motivational interviewing techniques often are incorporated to elicit reasons that the

older patients may have to cut back on drinking. Motivational interviewing can also be used as a therapeutic technique. The Florida Brief Intervention and Treatment for Elders Project was a large three-year, state-funded pilot program of screening and brief intervention for older adults. Brief interventions were conducted among older adults, screening for a range of health and service settings. Prescription medication misuse, that is either using medications faster than prescribed, difficulty remembering how much to take, not taking medications, borrowing someone else's prescription, or saving older medications was the most prevalent substance use problem, followed by alcohol, over-the-counter medications, and illicit substances (Schonfeld, et al. 2010).

Smoking: This is of course an issue: Smoking kills. The percentage of older adults that still smoke is small relative to other ages. Older adults smoke at a lower rate than younger adults, but cessation can be difficult, especially after years of smoking. Choi and DiNitto (2015) examined the influence of a diagnosis of a chronic illness, social isolation, and depression on smoking cessation among the most recent cohort of older smokers. They used data from 2011 and 2012 waves of the U.S. National Health and Aging Trends study. At time one, 8.8%, 47.7%, and 46.5% of the sample respectively were current, former, or never smokers. Current smokers had lower socioeconomic status, were more socially isolated and had higher depressive symptoms than never-smokers. At time two, 88.9% of the T1 smokers continued to smoke and 11.1% no longer smoked. The odds of smoking cessation increased with the new diagnosis of chronic illness since T1 and decreased with the higher number of cigarette smoked at T1. Social isolation at T1 increased the odds of smoking cessation, but depressive symptoms at T1 was not a significant factor.

BMI: This is perhaps the best maker of health. Obesity of course is a problem and is increasingly prevalent at later life. In fact, it may be less an issue after age 70 but, given that people are living longer, it is still an issue. If a person's bodyweight is at least 20% higher than it should be, he or she is considered obese. If your BMI is between 25 and 29.9 you are considered overweight. If your BMI is 30 or over you are considered obese. The BMI is a statistical measurement derived from your height and weight. Although it is considered to be a useful way to estimate healthy body weight, it does not measure the percentage of body fat. The BMI measurement can sometimes be misleading - a muscleman may have a high BMI but have much less fat than an unfit person whose BMI is lower. However, in general, the BMI measurement can be a useful indicator for the 'average person'.

People can become obese for many different reasons. Consuming too many calories is almost always involved. Despite billions of dollars being

spent on public awareness campaigns that attempt to encourage people to eat healthily, the majority of us continue to overeat. In 1980 14% of the adult population of the USA was obese; by 2000 the figure reached 31% (The Obesity Society, 2014). In the USA, the consumption of calories increased from 1,542 per day for women in 1971 to 1,877 per day in 2004.

Several other related factors are also at issue. A sedentary life style is a problem. Physical activity has an effect on how your hormones work, and hormones have an effect on how your body deals with food. Several studies have shown that physical activity has a beneficial effect on your insulin levels - keeping them stable. Unstable insulin levels are closely associated with weight gain. Poor sleep is another problem. Research has suggested that if you do not sleep enough your risk of becoming obese doubles. The 'epidemic' of obesity is paralleled by a 'silent epidemic' of reduced sleep duration, with short sleep duration linked to increased risk of obesity both in adults and in children.

Endocrine disruptors, such as foods that interfere with lipid metabolism, are also in play. Fructose is mainly metabolized in the liver, the target organ of the metabolic alterations caused by the consumption of this sugar. Fructose effect on the brain may promote obesity - researchers from Yale University School of Medicine compared the effects of fructose and glucose on the brain with MRI scans and found that high fructose diets may be behind the current obesity epidemic. Lower rates of smoking suppresses appetite. Among people who do stop smoking, the average weight gain is between 6 and 8 pounds. Roughly 10 percent of people who stop smoking gain a large amount of weight - 30 pounds or more." Finally, medications can make patients put on weight. According to the Annals of Pharmacotherapy, some medications cause weight gain. There is wide inter-individual variation in response and variation of the degree of weight gain within drug classes. The longer a person is overweight, the harder it becomes for them to lose weight: Obesity promotes obesity itself. Obesity seems to become a self-perpetuating state.

Exercise is a central factor. We have discussed it before. Hall et al. in 2017 assessed the physical performance across the adult lifespan and noted correlates with age and physical inactivity. The results of their study emphasized the importance of lifespan approach to studies of function and aging. This work points to the need for a physical performance screener that spans across adulthood as a clinical tool for identifying functional decline. Both rapid and unusual gait were assessed in this study. Physical performance was worse with increasing decades. Although men performed better than women across the ages, the decrement by age group was similar between genders. Worsening physical performance was observed

as early as the 5th decade for chair stands and balance and the 6th decade for gait speed and aerobic endurance. The number and strength of significant associations between physical performance and physical activity increase with greater age. More physical activity was associated with better physical function.

Programs that increase physical exercise among people with MCI have been shown to be feasible and yield cognitive benefits. The randomized trial by Launtenschlager et al. (2008) assigned older adults with and without MCI to a physical activity group or usual care group. The usual care group received educational information about memory loss and healthy lifestyle. The physical activity group received the same educational information, but were also encouraged to get at least 150 minutes of moderate intensity physical exercise (for example walking) per week for 24 weeks with behavioral support provided to increase adherence to the exercise program. On cognitive testing 6, 12, and 18 months later, the physical activity group performed better in areas of memory, language, and visual perceptual skills compared with the usual care group and the participants with MCI benefited as much as those with normal cognition.

A comprehensive evaluation of the effect of exercise interventions on cognitive function in chronic disease patients has not been conducted. In general, reviews generally investigating the effect of exercise intervention on cognitive outcomes in chronic disease patients yield positive overall effects of exercise interventions on cognitive function. Aerobic exercise interventions are usually found to have a positive effect on cognitive function in patients with chronic disease. Low-frequency exercise interventions even seem to have a positive effect on cognitive function in chronic disease patients. It appears that the maintenance of physical activity is associated with increased neural resources in some brain regions and reduced neural resources in other brain regions. Exercise probably affects cognitive function by causing a significant reduction in the peripheral concentrations of IL-6 and TNF- α , as well as a significant increase in peripheral levels of BDNF in individuals with chronic diseases. Exercise no doubt leads to structural changes in the brain, such as increases in dendritic length and branching and hippocampal neurogenesis, as well as maintains the atrophy levels of the whole brain cortex.

One group of studies deserves mention. Teri, Longsdon, and Colleagues (Teri, et al., 2003, 2012) studied older adults with AD and their caregivers. They were randomly assigned to a program that combined physical exercise and behavioral management training or to routine medical care. Twelve in-home sessions conducted by home-health workers over a three-month period instructed the caregivers in safe procedures to

help the person with dementia exercise and methods for a caregiver to use to reduce distressing behaviors. At the end of the training, people with dementia in the active intervention group had higher weekly exercise, better physical functioning, and fewer depressive symptoms than the usual medical care group. The two-year follow-up people with dementia in the active intervention continued to show better physical functioning than those in the usual medical care group and there was a trend for reduced institutionalization for the behavioral institutional group. Quite a finding!

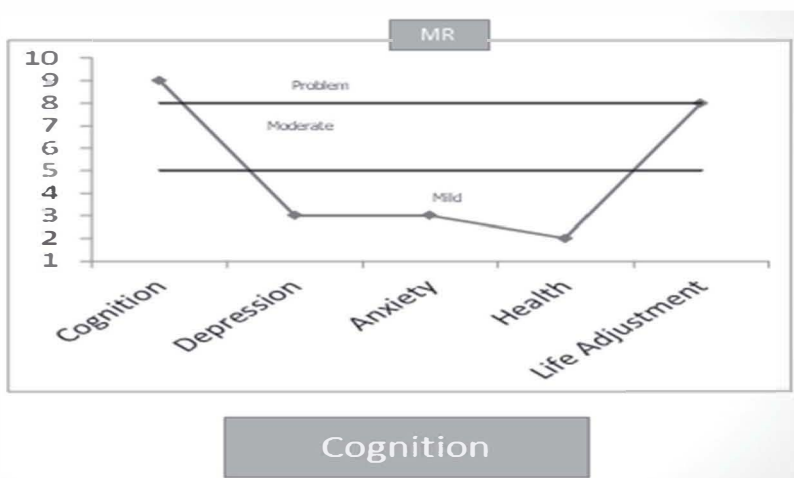
For the first time, a randomized study has demonstrated that vigorous physical exercise not only improves cognition, but also moves AD biomarkers in the right direction. Six months after subjects with MCI began aerobic exercise, scores on composite measure of cognition rose significantly. Not only that, hypermetabolic brain regions associated with AD symptoms were re-perfused and phosphorylated tau, a marker of neuronal injury, fell significantly in the cerebrospinal fluid. After a baseline six months, everyone who completed the cognitive testing, a 400-meter timed walking test, glucose tolerance test, body fat assessment, blood and CSF collection were done. For the participants who underwent the structural and functional MRI, all results were controlled for age and education. There were no reductions in CSF tau in either age group in the stretching cohort. However, in the exercise cohort, both groups experienced significant decline in CSF tau (Baker, 2015).

The benefits of exercise are indisputable and the current perception is that a curvilinear relationship exists between the amount of physical activity and the related health benefits. It is now soft fact that physical activity is associated with reduced risk of cardiovascular diseases, diabetes, cancer, and dementia in a dose-dependent fashion. Increasing from the minimum effectiveness levels of moderate 15 minutes a day, to more vigorous intensity physical activity to the national physical activity guidelines of 30 minutes a day of moderate intensity exercise to 75 minutes a week of vigorous intensity exercise appears to be associated with increased health benefits. Wen et al (2011) showed that each additional 15 minutes of moderate intensity physical exercise was associated with a 4% further reduction in all-cause mortality over 13 years in several populations. These benefits are independent of age, sex, and cardiovascular history. The greatest benefit was obtained by most active individuals (63-88 minutes a day) with higher mortality reductions of vigorous intensity versus moderate intensity exercise (Wen, et al., 2011). Interestingly, the exercise of using DVD can also be applied. It can produce a clinically significant improvement in physical performance and improvement of flexibility and strength demonstrated the effectiveness of

something as simple as low-cost DVD exercise program in improving physical health.

In sum, there is much to like here. There is no known upper limit of moderate intensity physical exercise in healthy individuals, but doses more than 100 minutes a day do not appear to be associated with additional reductions in mortality. Results generally show that aerobic exercise interventions had a positive effect on cognitive function. This result was consistent with the recommendations of the World Health Organization (for a weekly minimum of 150 min of moderate-intensity aerobic or 75 min of vigorous-intensity aerobic activity with additional muscle-strengthening exercises) (WHO, 2010). Aerobic exercises improve the maximum oxygen uptake and increase and redistribute cerebral blood flow, enhance antioxidant action via repair enzymes and proinflammatory cytokines, as well as increase beta-amyloid degradation, levels of neurotrophic factors, neurogenesis, and angiogenesis. There may be insufficient evidence for an effect of exercise intervention on MCI patients, however. Aerobic exercise may have a positive impact on cognitive function in older adults with MCI. The American College of Sports Medicine agrees.

Cognition



CASE: MR

Mrs M was referred by her sister. She was in an accident driving 70 miles in an apparent dissociative state. She was confused as to why this episode took place. Prior to this she had 2 other episodes that took place

over the previous 3 years. She is a retired teacher who lives locally by herself, and has been largely functional in the community. She spent roughly 30 years as being a teacher and the retired a year ago. She never married. She lives alone.

Cognition

Measure	Healthy Intact	Mildly Problematic	Problematic or Syndromic
Description	Normal memory and fully oriented, these patients will display good judgment and problem solving skills.	Memory loss that interferes with everyday activities, difficulty with time and/or place orientation, difficulties with abstraction and problem solving, but social judgments are intact.	Severe memory deficits, inability to retain new information, difficulty with orienting time and place, markedly impaired abstraction and social judgments.
MoCA	27-30	22-26	≤21
MMSE	27-30	21-26	≤20
Trails A	≤ 46s	47-70	≥ 71
Trails B	≤ 115s	116-182	≥ 183

Measures:

Premorbid Functioning: Cognition demands an add-on, premorbid functioning. We have not addressed this as yet. In the best of assessment tools this estimation can be difficult. What is the real baseline intelligence of the person in question? Premorbid intelligence is an estimate of a person's intellectual functioning prior to known or suspected onset of brain disease or dysfunction. More generally, this term is used to describe a process in neuropsychological evaluations in which an individual's level of neuropsychological functioning that existed prior to the onset of known or suspected neurological dysfunction is determined. Establishing a premorbid level of intellectual functioning is necessary for deficit measurement, which is a central tenet for neuropsychological evaluation. Deficit measurement is the process for determining if a change in functioning has occurred that is greater than would be normally observed, or is due to measurement error. This change is typically inferred as a neuropsychological deficit reflecting brain dysfunction.

Again, an accurate estimation of premorbid intelligence is necessary to prevent errors such as under- or overestimation of a patient's level of

cognitive decline. In the absence of objective information about the patient's premorbid level of functioning (i.e., preexisting test results; academic records, employment history, etc.), clinicians often rely on estimation methods such as “hold-don't hold” tests, the “best performance” method, various reading tests, demographic regression formulae, regression formulae that combine demographic variables and current performance levels. Many of these methods demonstrate problems such as under- or overestimation of IQ and range restriction. That said, an improved understanding of which premorbid IQ estimates adequately address these clinically significant limitations continues to be a major challenge within the field of neuropsychology. Interpretation of current neuropsychological performance in the context of estimated premorbid intellectual levels is then a critical component of a clinically useful neuropsychological evaluation. The numerous options of premorbid estimation measures are discussed in the cognition chapter.

Screen Measures: Complete assessment should include patient data, estimation of premorbid functioning, behavioral observations; characterizing normal versus abnormal performance, historical data, medical history including neuroimaging, and clinical presentation (onset; rate and course, relative decline), as well as informant reports. A screen of course needs to be quick and easy to administer. We suggest four measures; two typically used measures, MMSE and MoCA, and two, Trials A and B, for a brief look at speed of processing and executive functioning. All of these are related to cognitive aging and degenerative disease processes. There is nothing sacred about these other than they are frequently applied and sufficiently reliable and reasonably valid. In general, a global measure, like the MMSE, has lower sensitivity for producing accurate diagnosis (Gonzalez-Palau, et al 2013). In the recent past it has been shown that aMCI patients who segue to AD do so in various ways; reduced learning and forgetting (Greenway, et al, 2006), and overall problems in delayed recall (Jak, et al, 2009).

In our judgement the issue of sensitivity should always trump specificity: Identifying possible patients with problems is more important than false positives. Additionally, as noted, needed adjustments involve adjustments for premorbid intelligence, information on function, the application of sound norms, as well as an account for sleep, pain, depression/anxiety, and lifestyle. The use of scores over time, use of an informant, and any other adjustments for any necessary demographics, can be applied. It may also be important to use neuropsychological measures as training tools themselves.

We should add that a basic cognitive evaluation in primary care is provided once a year by Medicare with follow-up if necessary. Tools like the GPCOG (General Practitioner Assessment of Cognition) and Mini-Cog, as well as neuropsychological computer evaluations (e.g., NeuroTrax) that take place in less than a ½ hour are in play.

Background:

We can say that neuropsychological measures serve as biomarkers for illness (i.e. serves as an indicator of biological processes and can detect treatment response); serve as potent predictors for development of AD and other dementias (Bäckman et al., 2005); can capture other mediating influences on disease trajectory; are proxies for important functional deficits (e.g., Dementia Rating Scale and ADLs correlate highly (Fields et al., 2010)); and can suggest treatment targets. Neuropsychological measures capture the competing forces of pathological burden and cognitive reserve. In fact, they may be equal or greater than other biomarkers. As we have discussed, there have been a series of cognitive terms applied to older adults referencing problems. They have involved more benign names as Age-Associated Memory Impairment, Age-Related Cognitive Decline, to more malignant cognitive change as late Life Forgetfulness, Questionable Dementia, Cognitive Impairment No Dementia (CIND), and MCI. MCI has given way to mild Neurocognitive Disorder. This is clearly the most known entity. It is a truism that if you do not suspect the disorder, it will not be diagnosed.

Cognitive decline is based on patient's self-report, often knowledge of a third party informant, or an objective observation, as well as mild deficits on objective cognitive assessment. When serial measures are available as significant, that is 0.5 standard deviations assigned from the patient's own baseline, it would serve as a more definitive evidence of this decline. A particularly powerful approach to dementia severity assessment is the combination of information from cognitive testing, a knowledgeable informant and a clinician's impression. The mix of cognition and function as well as the trajectory of the problem can be persuasive for accurate data on a particular patient. We need to know exactly what needs to be evaluated. For AD, the fundamental focus is memory on a unidimensional continuum.

That said, a dementia syndrome is a heterogeneous disorder. It presents as a disruption of diverse cognitive processes until the underlying vulnerable factor is determined. We know that variables such as recall and orientation are most evaluated and often sufficient for an eventual dementia diagnosis. One other problem is there are individuals who have

dementia and do not test as such; others have the opposite problem as they fail a test and have no AD or other dementia diagnosis. There are ethnic and cultural differences causing norming problems as well.

General Rubrics of Cognition Measurement

- Cognition falls early: memory and EF largely decline first but attention and visuospatial areas also. Memory often plateaus over time.
- Cognitive variables at baseline are comparable or better predictors of progression than other biomarkers. Memory measurement trumps biomarkers for dementia identification.
- MCI counts in the service of cognitive decline but is variably measured.
- Biomarkers are helpful but expensive and not available.
- Nuanced neuropsychology is relevant; category fluency, new learning vs recall, differences between verbal and visuospatial
- Common, normal findings of older adults may be wrongly attributed to disease.

Considerations of Complete Cognitive Assessment:

In the early phases of a dementia workup less and less distinction is often made between normal and pathological conditions, thus creating uncertainty for accurate diagnostic decisions. Perhaps an initial focus on memory function is best but this is not foolproof. In addition, we have no specific test for the staging of a dementia. Probabilities can be estimated where the vector of item responses can be provided as to where on the continuum the subject might be. That said, it is fundamentally important to determine the severity of a dementia as this provides caregivers and treating professionals necessary data on care. As indicated, in the dementia screening process the ideal assessment should involve a preliminary exam by a trained professional that includes a more extensive cognitive evaluation, interview with an informant, appropriate neuropsychological assessment as introduced in the Cognition Chapter, and a summary rating.

The DSM requires one or more cognitive areas to be in decline. DSM-5 (The American Psychiatric Association, 2013) eliminates the antiquated requirement of memory impairment, allowing for multiple cognitive deficits. Major neurocognitive disorders defined by significant cognitive decline from previous level of performance in *one or more* cognitive domains (for example, enumerated as complex attention, executive ability, learning and memory, language, visual, spatial construction, perceptual ability, and social cognition) based on both subjective and objective assessment as evidenced by concerns of the patient or clear deficits in

formal neurocognitive testing. Deficits typically are identified by test performance of more than two or more standard deviations.

DSM-5 Cognitive Factors

- Complex Attention
- Executive Function
- Learning and Memory
- Language
- Perceptual-motor
- Social Cognition

Regardless of the cognitive domain, the central feature of assessment for cognitive problems is memory. We start with memory complaints. Samuel Johnson noted succinctly “the true art of memory is the art of attention.” Subjective memory impairments (SMI) appear to have an impact. Using PET scans, Gandy et al. (2005) showed that those reported having worse memory than their peers were found to have more beta amyloid in their brains than was seen in the comparison group. Forgetting names was the most common complaint among healthy seniors. This is often referred to as a senior moment, when they cannot recall the name of a new acquaintance, a movie, or a book. What may cause considerable concern is the pattern of consistent problems with memory. For example, often forgetting things one normally wouldn’t, being confused by routine tasks, and getting lost in familiar surroundings. An interesting element of the Gandy study is the suggestion that people sense of how sharp their memory is in comparison to that of their friends their age may be an important marker for decline.

Working memory (WM) especially is the cognitive system that actively maintains several pieces of information in mind for immediate use. It is the chief factor underlying cognitive impairment in old age and in dementia. Models now refer to active “online” and short-term maintenance of information in the service of more complex tasks, such as mental arithmetic, language comprehension, planning, or problem solving. WM affects individual differences in fluid intelligence (25%) and executive function (EF), language acquisition, reading comprehension, problem solving, reasoning, cognitive control, and reading comprehension. WM then may serve as a domain-general cognitive resource that modulates ability in a number of seemingly disparate areas of cognitive performance. The greatest activation in WM training comes from parts of the prefrontal cortex and parietal cortex, a standard pattern for WM changes.

Special attention needs to be devoted to frontal systems. Executive control depends on the integration of frontal systems. EF impairment may follow disruption of frontal system information processing, regardless of the location of the lesion within the system or direction of the perturbation. In some cases remote lesions can affect processing within the frontal circuits. Functional imaging and EF control are complex; response inhibition with the orbitofrontal region, attentional control with the mesiofrontal region, working memory and rule discovery with dorsolateral region. Psychological tests are also all over the place. We have already addressed the Wisconsin Card Sort Test (WCST). It may be the best validated EF measure. It is reasonably affected by frontal lesions and it selectively activates the left dorsolateral cortex in activation studies. Multiple domains of the EF system can be ascribed to the various WCST subtests but this is difficult to prove. Neither neuroimaging nor factor analysis has isolated specific and robust WCST-related factors to the frontal lobes. Thus apparently localized tasks, while clearly dependent on the frontal functions, may be merely the heteromodal processes on which truly cybernetic EF operate. Unfortunately EF deteriorates at an exponential rate with age. And, the pattern of age-related cognitive decline in non-EF domains is most consistent with the loss of EF control over intact processes (Royall, et al., 2002).

There are other body systems of note. A Mayo Clinic study recently published in the *Journal of Alzheimer's Disease* found that problems associated with gait can predict a significant decline in memory and thinking. Using the Rochester Epidemiology Project, Mayo Clinic researchers examined medical records of Olmsted County, Minnesota, residents, who were between ages 70 to 89 as of Oct. 1, 2004. The analysis included 3,426 cognitively normal participants enrolled in the Mayo Clinic Study of Aging who had a complete gait and neuropsychological assessment. Alterations in several gait parameters were associated with decline in memory, thinking and language skills, and visual perception of the spatial relationship of objects. The study results also supported the role of computerized analysis because the computer tool detected modifications before impairment was detected with a standard neuropsychological test (Savica, et al., 2017).

It is important to recall here: Dementia is not a single disease. White et al (2016) studied 334 nuns and 774 Japanese American men who had multiple cognitive assessments and were autopsied. Average age was 90. Of the five different brain pathologies: AD (plaques and tangles), Lewy Bodies, hippocampal sclerosis, microinfarcts, and brain weight, the subjects with AD (N=279), three quarters had at least two of the brain

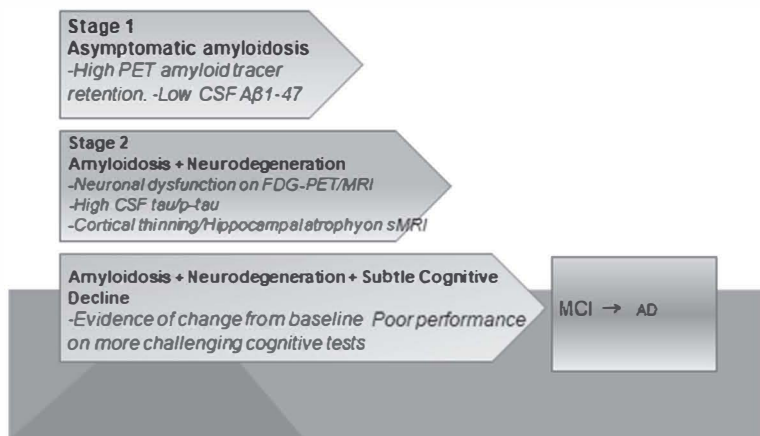
pathologies. Half who had AD in the final years had no AD-type abnormalities. The authors concluded that combination of impairments correlated best with cognitive impairment and most of the impaired had more than one type of pathology. Similarly, during the Rush Memory and Aging Project and the Religious Order Study 3000 older adults were tracked over 2 decades. The authors report that “Autopsies showed that some mentally spry individuals had extensive signs of cellular damage and others with substantial problems of cognitive decline in their later years had few signs of these cellular abnormalities. There was then a disconnect with cellular pathologies and cognitive decline.”

Of course, the search is to turn back the clock on AD. Many researchers are seeking ways to effectively diagnose the neurodegenerative disorder earlier. There are many potential ways to do this. Tracking brainwave activity, for example, can pinpoint brain regions that are likely to be affected by the disease next. This will allow for an early diagnosis. Typically individuals with early stage signs of AD (i.e., MCI of primary progressive aphasia) exhibit sluggish brainwaves and subtle signs of damage in the brain regions responsible for memory and planning. This is then one of many possible methods for ways to diagnose AD early. This in combination with cognition and function allow for the best possible methods of early diagnosis.

Function matters (this is addressed separately). Among older adults with possible dementia, 23% are still driving; 31% prepare hot meals; 22% manage their own finances; 37% manage their medications; and 21% attend doctor’s appointments alone (Amjad, Roth, Samus, Yasar & Wolff, 2016). Assimilation and accommodation are the normative features of older life. Older adults conserve resources and disengage from unattainable goals. A meta-analysis of 11,960 subjects showed a κ value of .88 for the use of function measures to diagnose dementia (Castilla-Rilo, et al., 2007). About 40% of variance of cognition involves function. In the assessment of most cognitive decline adding function to cognition gets best results. In fact, the best markers for dementia are a cognitive and a function variable, Trails B and FA.

Alzheimer’s disease is currently understood to be a pathophysiological process that begins with a long preclinical phase. Research criteria proposed a three-stage model of the preclinical AD in which temporarily ordered biomarker abnormalities proceed and then coincide with newly emerging behavioral and cognitive symptoms in clinically normal individuals. The subtle, but detectable symptoms define stage 3 preclinical AD, a transitional state, that may be evident before the diagnosis of MCI. Subjective cognitive concerns are part of this issue.

GRAPHIC REPRESENTATION OF PROPOSED STAGING OF AD: SPERLING ET AL. 2011



Clearly MCI needs special attention. Luck et al. (2017) was one of the first population-based studies on the prevalence of MCI using the new DSM-5 criteria. They reported a weighted prevalence of 20.3% in a large population without dementia. The age was between 60 and 79 years. There were no differences in sex, but not surprisingly a strong correlation was found with age. Generic criteria for MCI continue to include concern regarding a change in cognition, impairment of one or more cognitive domains, preservation of independence and functional abilities, and not being demented.

MCI of the Alzheimer type is especially noteworthy. If the subject meets criteria, but in addition has one or more topographic (that is structural or function) biomarkers associated with the downstream effects of AD pathology, for example, MRI evidence of medial temporal atrophy or FDG PET, evidence of a decreased temporoparietal metabolism (adjusting for age), then the likelihood is increased the outcome will be AD dementia. The core dementia features continue to include the traditional requirements of disruption of daily function, decline from previously higher state of function, explosion of deliria, and disruption of at least two areas of cognitive behavior. MCI continues to be a concern being defined by amnesic presentation (the most common syndrome associated with AD dementia involving impairment of learning and recall of recently learned information), or non-amnesic presentations, which can have presentations of a language problem where there is a deficit in word

finding or (increasingly) a visual presentation where the most prominent deficits are spatial cognition. There is also an executive dysfunction problem where the most prominent deficits are impaired reasoning.

Composite scores have been created and made into transformed Z scores; the typical criterion of these markers of MCI involve one measure in a domain of greater than 1.5 standard deviations below mean. This is often called typical because it is most common criteria for impairment consistent with the original definition of MCI by Peterson in the 1990s. Comprehensive criteria were developed to better approximate the clinical decision-making process and because the interpretive value of an isolated impaired score is often limited. Also, there is a conservative criterion, which requires at least two measures in a domain to be impaired at the cutoff level of 1.5 standard deviations below the mean. When these are placed and evaluated against each other, differing outcomes occur. In the middle-aged cohort, As previously noted, Jak et al. (2013) showed that the typical and composite 2.5 neuropsychological definitions of MCI and use of hippocampal occupancy measures resulted in the best correspondence of MCI to expected neuroanatomical results. Using the typical composite of 2.5 reduction definition, volume in hippocampus was significantly reduced in the amnesic MCI group as compared with the cognitive normal group.

There are always other considerations. The experienced brain also has a say in adjustment. There has always been an aspect of the person called the experienced brain that it is very individualistic. As it dwells inside of our bodies, the brain is always the result of what the individual has experienced up to that point. This is why when we stimulate the brain or do other things with the brain, we often have to take this experienced brain into account and try and reflect on that. This is a daunting task. A large part of the experience is the brain interaction with the rest of the human body and existing and interacting in a still larger social context. This is unknown at the present time.

Treatment:

We address two medications in progress and cognitive training (CT). Medication works minimally or only partially. We have known for some time that the effect size is $<.3$. Several studies now have suggested that a quarter of AD patients, about 24%, are poor metabolizers of acetylcholinesterase inhibitors due to pharmacogenetic variants or the presence of drug-drug interactions. These findings may partially explain the high rate of adverse events limiting the chronic use of acetylcholinesterase inhibitors. A study by Tak, Zhang, Patel, and Hong in 2015 showed that more anticholinergic medications taken by participants

were significantly associated with a higher percentage of sessions with disengagement.

Recently, the monoclonal antibody, aducanumab, has shown some progress against amyloid plaques in the brain. This is perhaps the best example of a medication that has shown success only to falter. This antibody is able to cross the blood-brain barrier and get into the brain. Between 2012 and 2014, the study authors looked at 163 initial patients with mild Alzheimer disease, with 125 patients available for the final analysis. Patients were recruited by amyloid PET and therefore had to have an amyloid lapse in the brain. This was a dose-finding study with one monthly infusion of 1, 3, 6, or 10 mg of the antibody aducanumab per kilogram of body weight. Outcomes were measured at 26 and 54 weeks; amyloid plaque content using PET imaging. In a time- and dose-dependent manner, the antibody resulted in a significant decrease in amyloid in the treated patients. In patients who received the highest aducanumab dose for up to 1 year, there was a dramatic decrease in the amount of amyloid plaques in the brain. In parallel, there was also a slowing of cognitive deterioration. Patients in the highest-dose group had almost no change in cognition across 1 year, whereas those in the placebo group showed a clear deterioration.

This is, however, not a cure and recent data are suggesting a less robust result. Other agents (e.g., BAN-2401, Biogen), however, are also in play. Potential promising results regarding AD, like intranasal insulin spray, an AD vaccine (CAD106), intravenous infusions, and genetic manipulation to boost nerve growth factor, are in process. At the present time there are as many as 27 agents in the pipeline in phase 2 or 3. The time frame for such major changes in AD, however, is years in the making. Things will change!

CT has had a confusing recent history. We highlight again the two opposing positions on CT. One consensus statement argued that no compelling evidence exists to support the claims of brain-training companies that brain games (CT) enhance cognition or stave off the cognitive consequences of aging (Stanford Group); the other noted extensive support for scientifically grounded brain-training interventions (John Hopkins Group). Jak et al., 2013: "In sum, one can say that the data are promising regarding computer-based CT and any side effects are minimal, but any commercial claims of the efficacy of computerized cognitive enhancement systems is perhaps premature."

That said, the efficacy of CT is more promising but inconclusive. In a recent and highly cited study, Hill and colleagues (2016) conducted a meta-analysis on cognitively impaired patients (MCI or dementia) was

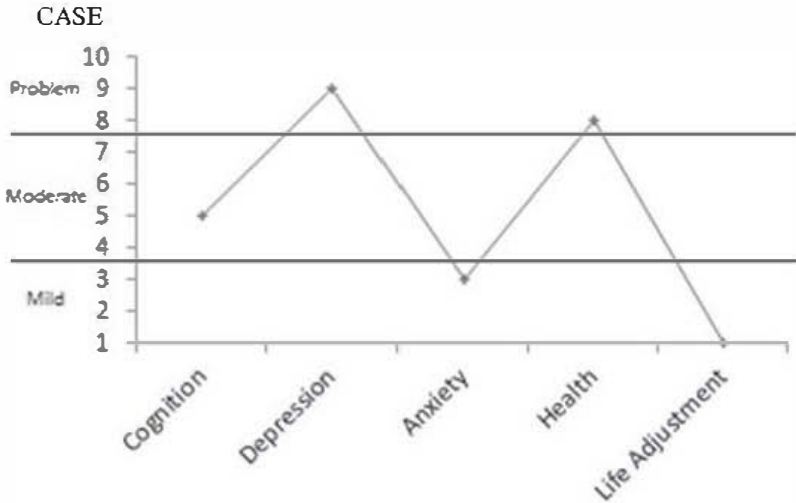
conducted. The key message is that CT, if administered properly in a group format at the right dose and targeting key domains, can have a meaningful effect, potentially even prevent dementia. People in control groups, regardless of whether it was an active or passive control, did not change at all, so we did not see a placebo effect (Hill et al, 2016).

This also entails physical activity as a protector but also as a delayer of degenerative problems. In a review of drug and exercise treatment of AD and MCI patients, Ströhle et al. (2015) showed that demographic changes are increasing the pressure to improve therapeutic strategies against cognitive decline and AD and MCI. They specifically show that besides drug treatments, physical activity seems to be the promising intervention target as epidemiological and clinical studies suggest beneficial effects of exercise, training, and cognition. Using comparable inclusion and exclusion criteria, the authors analyzed the efficacy of drug therapy, cholinesterase inhibitors, lemon tea, and ginkgo biloba and exercise interventions for improving cognition in AD and MCI populations. Looking at studies administrated through 2013, the authors show that discontinuation rates vary substantially and range between 0 and 49% with the median of 18%. Significantly increased discontinuation rates were found for galantamine and rivastigmine as compared to placebo in AD studies. Drug treatments resulted in a small pooled effect on cognition in AD studies and no effect in any of the MCI studies. Exercise interventions had a moderate to strong pooled effect size in AD studies and a small effect size in MCI. The authors concluded that drug treatments have small, but significant impact on cognitive function in AD. Exercise, however, has a potential to improve cognition in AD and MCI.

Cognitive rehabilitation then is a complex target. We have different populations, normal MCI and dementia, and we have different types of training, cognitive training, cognitive stimulation, and cognitive rehabilitation. A blend of restorative activities, attention training and compensatory strategies, memory books, and cognitive stimulation, group socialization can all be applied in different ways. These are very hard to measure for the different populations. Studies assessing cognitive rehabilitation and training are always wanting. It is necessary to take cognitive outcomes as a global indicator rather than being able to ascertain individual effects for specific cognitive domains. It is also not feasible to test for the moderating effects of variables, such as age and gender. Additionally, with greater focus on efficiency, outcome measures tend to focus on cognitive and neuropsychometric outcomes and do not assess ecologically relevant measures. And, like other assessment areas, trials tend to focus on immediate post-test outcomes only so that the primary

preventive effects of these interventions remain unknown. Finally, the stage of decline likely interacts with intervention. In broad terms, the purpose of cognitive and behavioral intervention is to enhance current cognitive function and/or prevent or slow the rate of further cognitive decline. Cognitive training, rehabilitation, or stimulation then are generally always relevant and often helpful and may be more or less emphasized, depending on the stage of the cognitive decline and the goals of the patient.

Depression



This patient’s psychological battery indicated that she was depressed. She has 23 on the **BDI-II**, 13 on the **PHQ-9** and 7 on the **GDS-SF**, all depressed. Her omnibus scales were also a problem (depression). She was also anxious (**GAD-7**). Her **MBMD** suggested a passive and depressive personality who is cooperative. The patient also indicated that she has been having trouble with her memory for the past year. Sometime in the latter part of last year she fell, trying to pick up a newspaper. She had no loss of consciousness, but she did hit her head and was bleeding. There were no broken bones. She is taking multiple medications, and she lives by herself. Her husband died roughly 8 years ago, and she has 1 son who is a long-term care facility for a chronic condition. She is functioning well at home. We will pick this case up in Chapter 8.

Measures:

We rely on several self-report scales. We apply the BDI-2, PHQ-9, and GDS-Short Form. We also use the MINI as an interviewer-rated scale. It often occurs that one or more is in the clinical zone and others not so. This allows for a panorama of depressive symptoms, some more clinical than others. There is of course no fixed rule for standard depression. Like dementia, heterogeneity is the rule. Also many people have subclinical depression or demoralization (lack of motivation and a sense of mild hopelessness) that clearly influences life quality and behavior.

Measure	Healthy	Moderate Problem	Problematic or Syndromic
Description	The patient is not depressed, mood is expressed in a healthy range and not consistently low.	The patient may have a subclinical depression or dysthymia. Mood is slightly depressed without significant distress or impairment.	The patient is likely experiencing depressed mood, anhedonia, and/or suicidal ideation. Many DSM-5 symptoms will be endorsed, some with great severity. Functioning is impaired
MINI	No positive Hits	Borderline MDD or Dysthymia	MDD or Dysthymia
BDI-II	≤10	11-22	≥23
PHQ-9	≤6	7-11	≥12
GDS-SF	≤5	6-10	≥11

Background:

If cognition is the king of assessment problems, depression is the queen. Depression has many forms. It is highlighted by MDD (major depressive disorder), and its minions: minor depression, which consists of two to four symptoms of depression and low mood or anhedonia; subsyndromal depression; mixed depression and anxiety; depression without sadness; bereavement, complicated grief, and traumatic grief; depressive executive dysfunction; depression in dementia; post stroke depression, suicidal depression, which consists of fatigue, hopelessness, and negative outlook (Joiner, et al., 2001); and apathy, which is generally orthogonal to depression. Research in the U.S. and other countries estimates that between 30% and 50% of people have met current psychiatric diagnosis of MDD sometime in their life. Ismail et al. (2017) applied a meta-analysis of 57 studies that reported on the prevalence of depression in a population with MCI, representing 20,892 participants. The overall prevalence of depression was 32%, though prevalence

estimates did differ between community samples (25%) and clinical samples (40%). The prevalence of depression in patients with MCI is high. A contributor to heterogeneity in the reported literature is the source of the sample, with greater depression burden prevalent in clinic-based samples. This staggering high prevalence compared with other mental disorders that affect only about 1 or 2% of the population, such as schizophrenia or obsessive compulsive disorder, seems to pose an evolutionary paradox. We are indeed a necessary consequent of our culture; medical system, media, attribution needs, and secondary rewards. The brain plays crucial roles in promoting survival and reproduction, so that pressures of evolution should have left our brains resistant to such high rates of malfunction. Mental disorders are generally rare. Why isn't this so about depression?

There is another possibility that perhaps in most instances depression should not be thought of as a disorder at all. Depression may in fact be an adaptation: It is a state of the mind that brings real cost, but also brings real benefits. During depression, the mind becomes more analytical and focused, a useful response for solving complex problems that probably triggered the depression in the first place. Clinically, diagnostic criteria required the presence of a clinically significant stressor and impairment for the psychological condition to be considered a mental disorder; but should this be enough given that depression might actually help in certain circumstances.

Perhaps depression is like a fever. This of course is an evolved response to infection. It coordinates the immune responses and it directs infection-fighting cells to tissues that are most likely to be infected and it staggers the production of chemicals that are necessary to the immune response, but could cause tissue damage it produced at the same time. Ultimately it is protective of the system, a warning system.

● One more thing here: Many symptoms of depression make sense in light of the idea that analysis must be uninterrupted. The desire for social isolation, for example, helps the depressed person avoid situations that would require thinking about other things. Certainly the inability to derive pleasure from sex and other activities prevents the depressed person from engaging in activities that could distract him or her from the problem. People in depressed moods, for example, are better at solving social dilemmas and conflicts of interest with a partner on several issues. The over-diagnosis of depression may occur because sometimes people are reluctant to talk about the problem that triggered their depression. Therapies may actually do better if they encourage depressive rumination rather than trying to stop it.

Given this, older adults may be more likely than other age groups to present with subsyndromal symptoms of depression that do not meet criteria for MDD. Subsyndromal depression is associated with increased incidence of physical disability, cognitive impairment, healthcare use, and suicidal ideation. It also is two to three times more prevalent among older adults than major depressive disorder. Although symptoms of depression among older adults may not meet diagnostic criteria for depression, older adults may experience depressive symptoms that are clinically significant and functionally and socially impairing. It may now be time to rearrange the idea of subclinical depression and consider it as depression. The well established relationship between depression and memory complaints in older adults gives credence to his.

Recent studies also suggest that late-life depression presents differently in individuals with cognitive impairment. Among individuals with vascular dementia, depression is associated with less dysphoria, but more of vegetative symptoms like fatigue and weight loss. Because symptoms overlap between depression and cognitive impairment, researchers have recommended that depressive symptoms among individuals with cognitive impairment be diagnosed independent of these overlapping symptoms. ●lin et al. (2002) proposed only three symptoms instead of five that are required to make a diagnosis of depression when dementia is considered. They suggested that symptoms such as increased irritability and social withdrawal be used as additional criteria to diagnosing depression. Depression does affect some degenerative diseases more than others; it affects approximately 58% of the patients with Parkinson's disease, as well as many with a less active version of AD. Regardless, depression in a neurodegenerative disease is resistant to change, especially with medication. Depressed older adults with Parkinson's disease may not exhibit dysphoria or anhedonia, which can result in an under-diagnosis of depression in Parkinson's disease. Also depression with Parkinson's disease is usually mild.

Depression then is more complex than previously understood. The idea that one size fits all simply does not exist. Sundermann, Katz, and Lipton (2017) even noted that only mild depressive symptoms were required in men but moderate/severe symptoms were needed in women as markers of future cognitive impairment. Recent conclusions exist that there are different immune factors at work in depression, depending on the clinical phase of depression and that the genes for this immune response are switched on and off at different times according to phases. What can often be seen in the clinical states of acute depression, relapse, remission, and recovery, is highly a complex interaction between inflammatory and other

immunological cells, brain cells, and systems. This model helps to overcome the simplistic notion that depression is the same kind of disease for everyone, behaving in the same way regardless of the timing of the disease. We can see that depression is a much more neurobiologically dynamic disease and this has many vexing implications for treatment.

Also, brain activity is altered in depression. The phenomenology of depression is distinct in a cognitive decline process. Among the most important effects of depression on brain activity is abnormal function of the HPA, hypothalamic-pituitary-adrenal, axis. This system typically responds to stressors in releasing the stress hormone cortisol, then returning to normal when the crisis has passed. In MDD, however, long-term chronic stress causes the HPA axis to break down and lose its ability to return to normal, resulting in unnaturally high levels of stress hormones that are thought to play a key role in the atrophy of cells in vulnerable regions of the brain. This system, which includes structures such as the amygdala and the nucleus accumbens, is responsible for regulating emotions, physical activities, and stress response. Also atrophy of the hippocampus, a key memory region, is common among people with major depression and may help account for the memory problems associated with depression.

And, do not forget executive functioning and depression. In fact, depression has been linked to several neurocognitive areas. Both white and gray matter regions disrupt the reciprocal interactions between cortical and limbic structures, creating depression executive dysfunction (DED). Also structural neuroimaging studies performed on participants with LLD have found abnormalities in the frontostriatal–limbic pathways (see Bobbitt, et al., 2012). Depression is associated with volumetric changes in prefrontal gray and white matter. Cognitive impaired older adults with LLD report reduced cortical thickness in anterior and posterior cingulate; medial orbitofrontal; dorsolateral prefrontal, superior, and middle temporal cortical areas. In fact, older patients with LLD treated with an antidepressant have lower parahippocampal thinning (Lebedev et al., 2014). The etiopathogenesis of depression in people with AD is also multifactorial. Potential factors include genetic polymorphism and neurochemical alterations of the serotonin system, loss of neurons in the locus cereolus, concurrent subcortical lesions of the WM. In addition there are neuro-immunological and neuro-endocrine changes.

Depressed individuals also have a 45% higher risk for stroke than non-depressed individuals and 25% higher risk for stroke-related mortality. Depressive symptoms are associated with obesity especially among young and middle-aged women. Diabetes also is a problem and has a

bidirectional relationship to depression. Depressive symptoms and stress lead to the greatest elevation of the risk for vascular disease mainly in low income populations. Physiologic changes occurring during depression episodes may increase both risk factor for strokes and directly compromise cerebrovasculature. These include increased sympathetic tone, dysregulation of the HPA-cortical axis, dysfunction of the platelet aggregation process, and increased inflammatory responses.

Reasonable speculation is that recurrent and persistent depression in early or mid-life may lead to cerebrovascular compromise. In turn, cerebrovascular compromise may lead to more frequent depressive episodes with poor treatment response and outcomes. Interrupting the viscous cycle of depression and vascular disease is a critical clinical and public health task. Finally, it is noteworthy that it remains unclear whether treatment of depression can reduce the risk of cerebrovascular pathology and its complications. Given the unclear relationship of antidepressants with vascular disease, a reasonable option is to combine antidepressants with behavioral interventions targeting poor health behavior, such as smoking, inactivity, and the management of vascular disease risk factors such as weight control, blood pressure changes, diabetes, and the like.

Depression, therefore, is a heterogeneous disease. Researchers now believe that what is needed are biologically relevant tests for mental illness in general, and depression and anxiety in particular, to identify biomarkers that will indicate what kind of depression the person has and consequently needed to be treated for. In addition, the brain chemistry for depression is complex. Research has traditionally focused on the relationship between depression and abnormally low levels of two brain chemicals, the neurotransmitters serotonin and norepinephrine. Dopamine is also implicated. These neurotransmitters are a target of widely used antidepressant drugs, which work by increasing the availability of serotonin and norepinephrine in the brain. The level of the transmitter GABA may also be abnormally low in people with depression. Other changes in the brain chemistry associated with depression are a drop in BDNF, brain-derived neurotrophic factor, a chemical that promotes the formation, survival, and repair of brain cells and stimulates brain cells to adapt and forge connectively in response to new experience, an increase in levels of the stress hormone cortisol, and a possible decline in the neurotransmitter dopamine. Glucocorticoids have also been implicated.

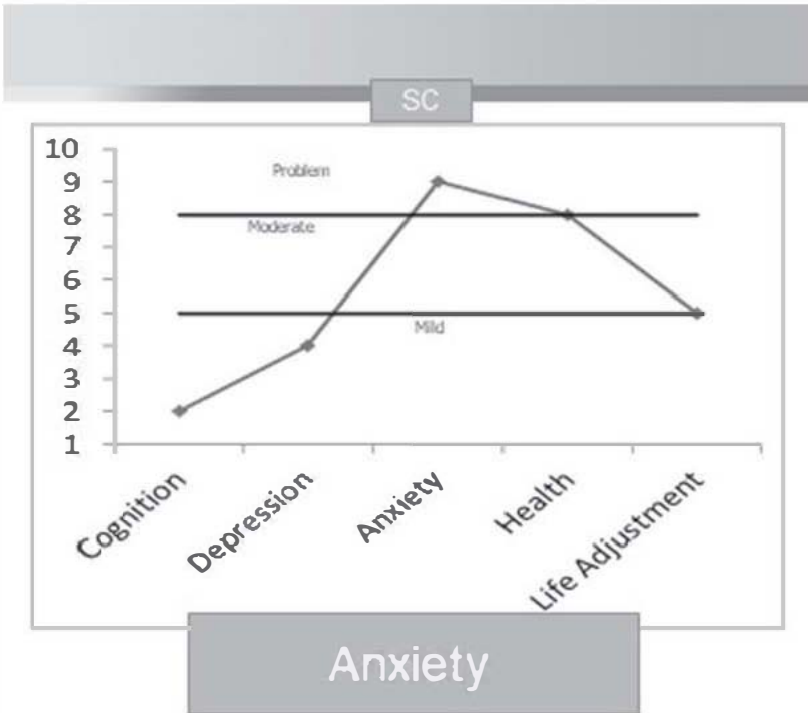
Treatment:

The typical trajectory of MDD is as follows: Roughly 40% will either not respond or poorly respond to medication/psychotherapy. Among those who do respond, roughly 50% will show a relapse within 2 years. Among older adults who developed depression, roughly 25% experience spontaneous remission. For the remainder, several characteristics predict difficulty in achieving remission with any antidepressant therapy. These include a greater number of concurrent physical conditions, depressive symptom severity, higher anxiety, an inadequate prior response to antidepressant treatment, dissatisfaction with social support, suicidal ideation, a history of recurrent episodes, and the use of concomitant psychotropics, such as sleep aids or anxiety agents.

We have reviewed the efficacy studies on depression (Hyer, 2014). In brief, the efficacy for psychotherapies and medications are roughly equal. The psychotherapies that are optimal are CBT, PST, brief psychodynamic, ACT, and supportive therapy. Bibliotherapy also has been reviewed as helpful. In addition, there are several preventative programs that we have identified in previous chapters that are effective.

Similarly, the SSRIs and SNRIs have been effective at levels comparable to the psychotherapies. Research over the past 20 years has provided robust evidence for the efficacy of antidepressants in the acute phase of late-life major depression and, once people are well, for their use in the continuation and maintenance phases. But what do we do when patients do not get well during the acute treatment phase? This problem of treatment-resistant (late-life) depression (TRD), occurring in up to 50–66% of patients depending upon its definition, is a major issue faced by psychiatrists treating people with depression aged older than 60 years. Many forms of TRD are “lite” in that older adults just are not happy (flounder) or “heavy,” those truly unable to get out of a depressive funk that almost seems hard wired. Several newer therapies have been proffered for TRD – new drugs (ketamine or esketamine), new and improved ECT modules, rTMS, and some medications.

Anxiety



This patient scored high on anxiety (and health). On the MINI she was rated as Generalized Anxiety Disorder. Her self-report scales suggested anxiety. She had been living in a Assisted Living Facility for 7 years. She is married, and she attends a CBT group. She is having trouble with anxiety and several medical issues that involve pain and considerable physical problems. She is on several medications and is attempting to deal better with her life in these areas. We pick up on this in Chapter 9.

Anxiety

Measure	Healthy	Moderate Problem	Problematic or Syndromic
Description	Daily functioning is not impaired by worry or fear.	Some activities are impaired by worry or fear, but the person is still functional at work and interpersonally.	Some activities are impaired by worry or fear, but the person is still functional at work and interpersonally.
MINI	No positive hits	Subsyndromal hits	Syndromal hits
GAD-7	≤6	7-14	≥15
BAI	≤9	10-16	≥17
STAI	≤29	30-44	≥45

Measures:

Anxiety follows depression like a fever does a bad flu. They are highly comorbid but also assert problems in their own way. We suggest use of the GAD-7, BAI, and STAI, as well as the MINI. We note here that occult anxiety (subsyndromal) is especially insidious for an older population. Perhaps this is real worry at life issues. Unfortunately “previous” nervousness also becomes robust at later life.

Assessment instruments regarding older adults and anxiety are many. A frequently cited limitation of many commonly used anxiety assessment tools is that they rely heavily on somatic symptoms, which develop with medical symptoms or age-related physiological changes making differential diagnosis difficult. It is often recommended that in primary care settings, use of clinic interviews, rating scales, and laboratory tests are helpful to diagnose anxiety and rule out other medical causes. Therrien and Hunsley (2012) surveyed the literature and identified 91 different measures across 213 articles used to assess anxiety in older adults. There is considerable lack of agreement across studies regarding what is optimal and even what is recommended. As in other areas the majority of scales were not validated adequately for use with older adults. Excepting with the geriatric mental status exam (GMSE; Copeland et al., 1976) virtually all the assessment instruments are developed for use with younger and middle-aged samples. The Hamilton Anxiety Rating Scale (HAM-A; Hamilton, 1959) frequently is used in pharmacological trials. It demonstrates good inter-rater reliability in older adults, but it does not discriminate well from depression.

The influence of anxiety on dementia is a concern. About one-fifth of adults with dementia have clinically significant anxiety and another 22-50% have substantial symptoms of anxiety based on caregiver and clinician ratings. This also occurs in Parkinson's disease. Among older adults with dementia residing in assisted living facilities, rates of clinically significant anxiety range from 11 to 18%, but approximately 50% reporting at least one symptom of anxiety. Prevalence may be lower in those with onset of AD before age 65 compared with late-onset AD, although severity of symptoms may be greater in younger onset patients.

The use of the American Psychiatric Association diagnostic criteria to assess anxiety disorders in patients with dementia is further complicated when the patient is unable to communicate cognitive symptoms that are essential for the diagnosis. Some researchers suggested that anxiety and dementia may be better conceptualized as a subsyndromal depression or disinhibition syndrome based on findings that GAD diagnosis in patients with AD is explained primarily by the presence of dementia-based problems such as delusions, pathological crying, and aggressive behavior (Mah, Binns, & Stephens, 2015). Recent attempts to differentiate GAD from dementia, however, found that those with GAD and dementia endorse more muscle tension, fatigue, and restlessness more frequently than dementia patients without GAD. An excellent review of the available instruments on anxiety and dementia is provided by Seignourel et al. (2008).

● On the basis of patient reports, sources of anxiety include coming to terms with the diagnosis of dementia, loss of skills, environmental stressors, relationships with others, and unknown triggers. For the most part, these factors are modifiable and represent promising targets of treatment development.

Background

We are implicating that anxiety disorders frequently present with other psychiatric problems. Also medical conditions in older adults cohabitate. Somatic symptoms are a hallmark component of anxiety disorders in older adults and older adults frequently present with comorbid medical conditions. Cardiovascular disease, chronic painful conditions like arthritis, migraine, lung disease, and gastrointestinal problems have all been found to be significantly associated with anxiety disorders. Age-related neurobiological changes may contribute to differential emotional experiences in younger and older adults in contrast to the age differences seen in healthy adults. Evidence from imaging studies suggest that older adults with GAD suffer from a deficit in top-down control processes similar to that seen in younger adults with GAD. Almost half of all older

adults with current lifetime GAD experience their first onset after the age of 50. Those who develop GAD later in life report more functional impairment and poor health than those with earlier onset GAD. It is often the case that late-life GAD is associated with less severe worry than early onset GAD. In general, findings suggest that worry content is age dependent. Older adults tend to report worrying more about the health and welfare of loved ones and world concerns, but less about work and school and relationships than younger adults do (Wetherell, Katz, & Craske, 2003).

Anxiety then is a pervasive problem at later life. GAD is the key marker for anxiety in older adults. It is highly prevalent among the elderly, with epidemiologic studies reporting six-month prevalence rates as high as 7.3%. Byers (2010) held that anxiety diagnoses accumulated to 12%. This is again more prevalent than dementias and more prevalent than MDD. In the National Comorbidity Study this number was 5.1% but the construct for GAD was eliminated as it is an exclusionary diagnosis. Furthermore, 20% were seen as having problems, 65% comorbid. GAD actually accounts for at least 50% of anxiety cases; this includes 50% to 97% which are early-onset disorders with late-life exacerbations. Anxiety disorders then are the most common psychiatric illnesses in the United States (Kessler et al., 2005). Only 33% of GAD patients reported using mental health resources (Blazer, 2000). Unfortunately, the prevalence rates may be an underestimate of true population values as older adults tend to minimize anxiety or depressive symptoms or simply not recognize them (Gurian & Minor, 1991).

Somatic symptoms also cause problems. Prevalence is between 10% to 20% among older adults. This is twice as prevalent as dementias and more prevalent than MDD. Problems include physical complications, lower well-being, increased mortality, coronary artery disease, and over-utilized services. As just noted, somatic symptoms are the hallmark component of anxiety disorders in older adults and older adults frequently present with comorbidities and medical conditions. Cardiovascular disease, painful conditions, lung disease, and gastrointestinal problems are all found to be significantly associated with anxiety disorders. Older adults especially with GAD suffer from deficits, largely a top-down control process similar to that seen in younger adults with GAD. Older adults worry and may misattribute their somatic anxiety symptoms to medical causes. As noted, almost half of older adults with current or lifetime GAD experience their first onset after age 50.

Anxiety disorders in older adults are associated with increased disability, diminished wellbeing, and excessive and inappropriate use of medical services (Wetherell, Gatz, & Craske, 2003). Overlapping symptoms then with anxiety, depression and dementia complicate quality of life. If one is depressed, then 38-46% have a comorbid anxiety disorder. This number is less when the pathology dance is started with anxiety. For the most part, 50% to 97% are early-onset disorders with late-life exacerbations. The most common problems of anxiety are avoidance and excessiveness (now “out of proportion of actual danger posed”). That said, the DSM features of impairment and work related anxiety, as well as the absence of fear of falling (upwards of 50%), do not apply.

Anxiety severity and amnesic MCI increased rates of conversion to AD independent of depression or extent memory decline. The hit rate for anxiety was 1.33, indicating AD risk increased by 33%, 78%, and 135% for mild, moderate, and severe anxiety, respectively. The association between anxiety and AD remains significant, even with the inclusion of baseline executive control or other extent atrophy over time. Further anxiety status predicted greater annual rates of decreased EC volume. One-fifth of older adults with dementia have significant anxiety and another 22-52% have significant symptoms of anxiety based on caregiver and clinician ratings (Bower & Wetherell, 2015). Anxiety may indeed be a prodrome for MCI in the sense that it accelerates the progression of decline (Mah, et al., 2015). It also increases the rates of conversion to AD (2015). It is related to the increased rate of atrophy within the entorhinal cortex as well as hippocampus.

Treatment

We address treatment of anxiety in Chapter 9. That said, the first challenge in treating older adults for anxiety disorders is recognizing that treatment is needed. Older adults are less likely than younger adults to recognize symptoms of anxiety and to seek treatment. Byers, Arean, and Yaffe (2012) found that over 72% of older adults who meet DSM-IV criteria for an anxiety disorder did not seek treatment, citing barriers such as feelings of discomfort discussing mental health problems with professionals and a belief that mental health treatment would be ineffective. The authors also found that older adults with mild anxiety or mood disorders were 5 times less likely to seek mental health services than those with severe symptoms.

The most commonly used pharmacological treatment for anxiety includes antidepressants such as SSRIs (for example, citalopram, escitalopram, paroxetine) or SNRIs - serotonin norepinephrine reuptake inhibitors (venlafaxine, duloxetine), and benzodiazepines (lorazepam,

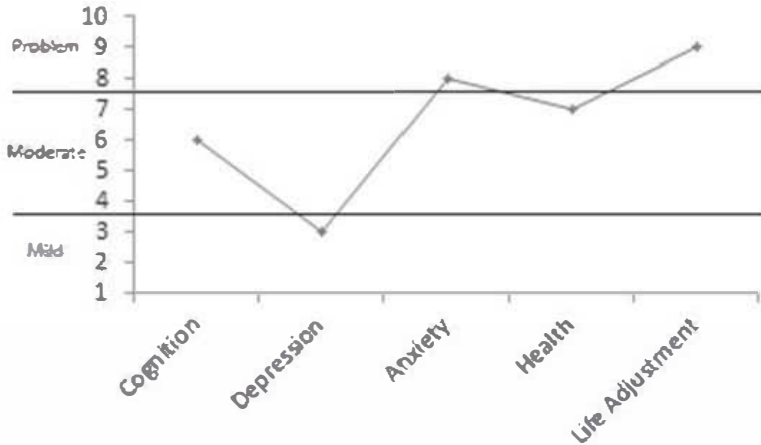
diazepam, alprazolam). Although effective at reducing anxiety symptoms in the short term, benzodiazepines should not be used as a first-line treatment in older adults because of the increased risk of falls and fractures. Recall that hepatic and renal changes in the older adult especially can impact the pharmacokinetics of medication; so doing should be monitored closely.

In general, psychosocial interventions are effective at reducing anxiety symptoms in older adults. However, the effect is more modest than that for younger adults. Adapted CBT programs are being developed to address these issues. Other non-pharmacological treatments include emotion-oriented therapies, brief psychotherapies, sensory stimulation therapies, and person-centered therapies. Modifications such as shortening sessions, presenting material at a slower rate, providing extra psycho-education, and including caregivers and family members in the treatment plan may be necessary with older adults. Group CBT can improve outcome for those who have a limited social support network. Although some studies suggest there are long-term effects of pharmacological treatments, an incomplete response is common.

For patients who do not respond completely to SSRIs, augmenting treatment with CBT may improve treatment response and reduce worry symptoms. Furthermore, those who wish to taper off medications, providing CBT with medication management may improve outcomes over medication taper alone. Treatments for panic disorders, specific phobias, and obsessive compulsive disorders are also present, but largely reflect those provided to younger adults. Post-traumatic stress disorder in older adults is primarily limited to case studies and a few small trials. Preliminary evidence for exposure treatments is suspect, but promising. Compulsive hoarding should be noted to be a distinct compulsive problem with older adults and a concern at later life. Behavioral techniques appear to be the optimal treatment for these.

It is reasonable to speculate that the combined use of CBT, IPT, and PST, along with the judicious application of medication, may provide the best chance of change. Typical CBT protocols seem to do this as they include education about anxiety, self-monitoring, relaxation training, exposure to anxiety-provoking thoughts and situations using systematic desensitization, and cognitive restructuring. Some protocols also included problem solving- skills training, behavioral activation, sleep hygiene, reflective listening, life review, and memory aids. CBT can be conducted in both individual and group formats.

Life Adjustment



This patient has problems in life adjustment. Indications are that roughly 3 years ago after a heart procedure, and then other procedures including stent and pacemaker, the patient started having difficulty with her memory and some behavioral issues. The patient does not see herself as having a problem with cognition. The patient lives locally alone and is on Social Security, and she has been divorced for a number of years. Her SES and education are problems. She has problems additionally with IADLs and is a loner. She has few activities and no leisure activities. Her daughter is concerned. We pick up on this in Chapter 10.

Life Adjustment

Measure	Healthy Intact	Mildly Problematic	Problematic or Syndromic
Description	Overall lifestyle is positive, patient engages in the core components of lifestyle without major problems	Lifestyle choices are likely to contribute to disorder or have begun to do so.	Lifestyle is compromising patient's mental and/or physical health. Self-care is lacking or not present.
Education and SES	College or professional certificate and continuing interests	HS education or extensive interests in reading/leisure	No HS education and little intellectual interests
FAQ (or AD-8)	≤10	11-18	≥19
Safe/supportive environment	Safe	Limited safety	Unsafe
Relationship Status	Married/in a Relationship	Single/divorced	Widowed in the last five years
Meaning in Life	Purpose in life/happy	Limited purpose or happiness	No purpose/unhappy
Stress (1-10)	0-3	4-7	>7

Measures:

This is a fuzzy area. We cannot assess everything in the real world quickly and validly. As such, we have opted to assess several key areas; SES and education, function and overall adjustment with ADLs and IADLs, safe environment, relationships (socialization), meaning in life, and stress, as well as caregiving/LTC placement.

Background:

There are a large number of life adjustment issues. It takes a village to help a patient/family. It is fitting that life adjustment is the last of the domains. It is the “whole person” marker. Bobbitt, Cate, Beardsley, Azocar, and McCulloch (2012) noted that quality-improvement processes and outcome measurement are now key features of the evolving system. There are now several national organizations that have disseminated standard quality measures such as the National Quality Forum, The Joint Commission, and the National Committee for Quality Assurance. Importantly the issue is quality improvement and some demonstration of treatment efficacy and efficiency as a measure of performance. In 2011 HHS included 10 principles in its strategy for this to occur. Among these were person-centeredness and quality improvement for health care providers to have access to necessary information. This has not been done in traditional clinical programs. This then involves the whole person and the whole context of that person. Life adjustment is then now core to this process.

Well-being is complex. Assimilation and accommodation occur daily in the life lived. Older adults conserve resources and disengage from unattainable goals. But decline especially in function is progressive, starting out with more sophisticated IADLs (managing finances, driving, handling medications) and segueing to the faltering of ADLs. Most models distinguish broad categories of relatively independent factors of emotional experience. Residential normalizing theory specifically proposed that when older adults find themselves out of their comfort zones in the environment, they start applying accommodative and assimilative forms of coping. These are adaptive responses referred to as mind strategies or real ones whereby older persons deal with their negative appraisals by lowering their environmental expectations or aspirations, deemphasizing their salience or variously rationalizing that their incongruent residential arrangements are not that important for their self-esteem, self-identity, or unhappiness (Golant, 2015).

Looking at the worst is instructive for the best. When considering patients with dementia, the deficit detection approach leaves the clinician with little data to rely on to make specific recommendations and initiate interventions that might improve the quality of life and wellbeing of the patient with dementia in the family. Indeed, it is difficult to take steps in this direction without a better understanding of the person broadly, including the values and preferences, activity levels and preferred activity, quality of life, quality of caring relationships, and life story. These components provide the clinician with a framework for offering interventions that provide continuity for people and their families during a period of considerable change.

Banerjee et al. (2006) noted the priority of problems. They indicated that neither cognition nor functional abilities were associated significantly with quality of life. Behavioral and psychological symptoms of dementia (BPSD) like agitation, depression, anxiety, disinhibition, and irritability are well-associated with lower quality of life. Similarly, in a longitudinal study of quality of life in individuals with dementia, both depression and anxiety were associated with quality of life at follow-up, but cognition was not. Such findings suggest that factors beyond cognition are most influential in the quality of life and the wellbeing of patients with dementia or in a decline. These studies then suggest that BPSD are important predictors of low wellbeing and quality of life. Again, even in a dementia the idea of a holistic environment evaluation can make a difference.

But overall a presentation of life issues for later life adults is both necessary and massive. Again we have selected the variables most apt for

the older adult who presents with life issues. Any understanding of these comes more from general interviewing than from formal tests.

OVERALL LIFE ISSUES

SES

Adjustment in community

Caregiving

Home care

Community problems

Finances

Competence

Transportation

Medical Issues

Iatrogenic diseases

Housing needs

Meals-on-wheels

Nutrition

Practical functioning

Relapse Issues

Longterm care

SES and Education: There is an SES gradient of health. It is a cascade that undergirds the power of socioeconomic influence. Exposures to poverty risks have a seemingly universal toll. Robert Putnam in his book *Our Kids; The American Dream in Crisis* (2015) argues that children in poverty are at high risk for elevated levels of cortisol, impaired emotional regulation, and potentially decreased ability to concentrate. Affluent children are estimated to be exposed to many million more words by kindergarten than working class or welfare children. This seems to be just the beginning of a negative cascade of effects that poverty sets off in children. Also the opposite effect of a positive cycle is set in motion by stable family structures, intensive parenting style, and a host of resources, both financial and social. The results of all this intermingling was the availability of large amounts of what Putnam called “social capital” in members of the socioeconomic classes. This type of capital is measured by social webs, interpersonal networks, and other types of connections that link one person to another. The more of a social web you weave, the denser the web of social relationships you possess, allowing you to reach out and call someone who can help.

Socio-economic factors, such as low income and poor educational attainment, are associated with comorbid mental disorders and medical conditions. SES contributes to the onset of mental disorders and is a consequence of the “downward drift” associated with a history of psychiatric disorders (Eaton & Muntaner, 1999). NHANES data revealed that poor people in each decade of life (20s through 70s) had higher levels of biological risk than people of similar ages who are not poor, thus supporting the notion of premature aging in those who endure poverty. A study of more than 2,900 people in the Netherlands also found that those participants that resided in neighborhoods with poor economic conditions characterized by vandalism, crime, and high noise levels tended to be biologically 12 years older than participants of the same chronological age who lived in a better neighborhood. Examination of the participants’ cells showed that the protective caps, telomeres, at the end of the DNA strands and genes of participants living in bad neighborhoods were shorter, an indication of aging thought to be related to mental and physical illness (Park, et al., 2015).

Older adults do have more money but are stuck in their social setting. They often have poor social capital. A necessary subsection of SES involves retirement. Often it is forced. Regardless, it changes life and the factors necessary for life satisfaction. The presence of additional stressors, such as those correlated with low SES, are likely to increase the overall levels of stress and the individual’s faith. It may exacerbate the effects of race-based social stressors. Increased exposure to neighborhood stress, such as exposure to violence, pollution, and toxins, disturbances by neighbors, in addition to daily life stressors, such as high levels of financial strain and associated increases in family conflict, may combine additively and synergistically with race-based stressors to influence educational outcomes.

Things pile up. Even changes in stress hormones and sleep processes are important factors to consider in the understanding of ethnic disparities in at least academic achievement. Disparities in stress and sleep compliments previous theoretical work that applies a stress and coping approach to understanding the consequences of social stressors. Specifically, in addition to focusing on coping responses that have been identified previously as important for educational achievement attainment, we should also consider the role of biological response, namely the HPA axis activity and sleep in the emergence of racial ethnic academic achievement gaps.

Education of course is critical. Education leads to leisure. Nimrod and Shrira (2014) explored the relationship over time between participation in

leisure activities and quality of life in later life among almost 8,000 retirees, aged 80 and older. This was done in the SHARE data set, the Survey of Health, Aging, and Retirement in Europe. Although many studies of participation of leisure activity demonstrate positive effects on health and wellbeing, most of these have been cross-sectional rather than longitudinal. In addition, increasing the barriers of pursuing leisure activities with increasing age may impact on the significance of involvement in meaningful leisure activity in later life. The Nimrod and Shrirra study (where they combined cross-sectional and longitudinal analyses) found that the association between leisure activities and quality of life increased over time and this effect remains significant after controlling for the social demographic characteristics, medical conditions, and cognitive functioning.

There are loose measures here. The income level of the person is of course relevant; where they receive money and their monthly budget, what they do for emergencies, and who are their support. Information from significant others is critical.

More specifically, we measure this construct in several ways. First, for the current reality of living, we ask about the objective facts of money and problems at home. Subjectively this is valuable as one's view of their situation tends to be as reliable. Second, we also assess their occupation and education. This involves the level of education, partner's education, and occupational prestige (Kok, et al., 2017). We also ask about the past growth years; mother and father's education and occupation as well as perception of their growth situation.

These are both a current and distal marker. We target then both the economic and living situation as well as education. We especially look for a sense of safety and satisfaction in living. These involve basic living requirements, life quality, and overall satisfaction. Education enhances this. This especially applies to mental health (and low SES). There are significant correlations between female gender, illiteracy, and poverty especially. Depression itself is reliability associated with lower SES, poverty and economic insecurity.

Function: This issue is transparently impactful on life quality. If function is impaired, so is life. This involves basic behaviors and more cognitively based ones. Menne et al. (2002) identified two such activities that are important and meaningful to older adults in the U.S. society, driving and helping others. The choices each participant made regarding driving and helping behavior differed according to their own personal needs. Some made alterations in behavior and their environment to attempt to continue to drive, whereas others decided to stop driving altogether. As

individuals experience a decline and losses of a dementia, the need remains to contribute and to have a sense of purpose and meaning and to have their views heard and considered. Loss of driving in general, however, has a major impact on the person. Recall that about 25% of dementia patients still drive.

There are several other community problems; transportation, meals-on-wheels, and everyday needs, to name a few. We measure with the Functional Activities Questionnaire (FAQ). The FAQ is a marker of function, notably executive function in the adjustment realm. This marker provides a rating of one's instrumental activities of daily living necessary for quality of life. The FAQ had a direct relationship with the level of cognitive decline; better with normal aging, worse with subject memory impairment, and most impaired with MCI and then dementia. Additionally, we often apply the AD-8 (Galvin et al., 2005), as it takes into account trends over time in behavior.

Environment: The element of a safe and desired environment is key. Aging in place also serves a number of issues related to environment. It refers to the ability to live in one's own home and community safely, independently and comfortably regardless of age and common ability level. Promoting aging in place is a policy objective in developed countries. It contributes to the wellbeing of older adults by providing a sense of attachment, connectedness, security, familiarity, identity, independence, and autonomy. Nahemow and Lawton (1973) long ago suggested in their ecological theory of aging that a decision to relocate can be determined by the balance between the environment of the person and the individual's competencies.

Preference of course is central to the concept of aging in place in distinguishing a choice to stay versus being stuck in place. Although it is recognized that older adults generally prefer to stay in their home and local community for as long as possible, the situation is more complicated for elders with low income, who lack financial resources and care support. Their preferred mode of living alters when faced with physical decline. Older adults consistently prefer aging in place, which requires a high level of community support and services that are currently lacking. With a rapidly aging population, however, this priority may change. This will need to play out.

Regardless, the present infrastructure for healthcare will prove even more inadequate to meet seniors' physical and mental health needs. A paradigm shift away from the sole focus on delivery of interventions at an individual level to more prevention-focused, community-based approaches may become essential. Recent initiatives have been proposed to promote

healthy lifestyles and preventive care to enable older adults to age in place. Community support, smart homes, alternative living settings are now plentiful and clearly helpful.

Questions related to the quality, acceptability and tolerability of the overall community are important. Knowledge of what the person's day looks like and how they can find safety and peace in the day. What can the person do and how do they do it. The person is asked if they like their life now where they are. In effect, the issue has to do with the quality of life as lived and rated, as well as where there are openings for improvement.

Social Variables (relationships): Social Connectedness is important to know how older people use their time and what factors drive these choices. Social connectedness, for example, has been a problem for many older adults as it decreases with age, it is related to poorer health, and is even connected to morbidity. Social connectedness refers to the number and quality of social interactions that people have. It is participation in social life. Also there is formal and informal connectedness. Similarly the effect of volunteering has been noted to help with social connectedness but it is complex and what constitutes a volunteer (formal organization or not) is relevant and unclear. In a study by Beckett et al. (2015), living alone was found to be associated with worse care experiences and immunization, especially for men. Health plans, therefore, should target the quality, improvement, and outreach efforts to beneficiaries who live alone, especially men.

Our popular press is replete with weekly reminders that being alone is a health risk. Divorce is at a 50% occurrence, and being older and alone occurs at 30% with another 20% suspect. Our data suggest that focused questions on the quantity and quality of connections, family and friend based, are important and maybe sufficient.

Happiness/Life Satisfaction and Meaning: Aging requires many factors to maintain and to survive in a happy way. Meaning in life is one marker for longevity. If meaning in life is essential to our survival in about the same sense as sunlight or calcium, then it must be available to us. Otherwise, human beings would have long since been rendered extinct. If we take seriously the notion that meaning in life is a human necessity, then we must tolerate and understand that meaning in life is a relatively common but critical experience. Large scale representative surveys and numerous studies of meaning in life suggest that meaning in life is indeed wide-spread and relatively high.

What matters in the meaning of life? These are social relationships, the experience of pleasure, and the detection of a reliable pattern of coherence in the environment. If one believes that what they do has a

purpose and that their life will outlast their physical presence, the person feels connected to others, has a positive mood, and they can deal better with difficult times. In fact, there is good evidence that meaning-constructed existence can be created and is natural to the functions of living. It may be evolutionary and one cause for existence (Heintzelman & King, 2014). A meaningful life is associated with many positive things – better self-reported health, decreased mortality, and higher quality of living. Meaning means something rather simple; one has a purpose and has significance. It involves social relationships, positive mood, environmental coherence, as well as positive illusions, response biases, and more adventurous living.

There are two constructs that support meaning in life. Generativity refers to a concern for establishing, guiding, and contributing to the welfare of future generations. It thrives in supportive exchanges and transmission of values, as well as volunteerism and societal engagement. Also, religion and spirituality are relevant. They certainly create meaning or foster it. There is a sense of positive psychology. This includes a reliance on religion to conserve their sense of significance, or sometimes a radical religious change is required because of the person's meaning and value system.

Life satisfaction then is an important variable. Although life satisfaction is typically considered relatively consistent across time, it may change in response to life circumstances, such as divorce or unemployment. Some people may adapt more readily to new situations, and thus appear to have relatively stable life satisfactions. Others may not adapt as quickly. All things considered, how successful one is in life might depend on how one views life satisfaction. Over the course of several studies, researchers seem to learn that as participants' life satisfaction increased, the risk of mortality is reduced by a considerable amount, as much as 20%.

Stress: We have discussed stress in the context of health. It is also applicable here. Essentially, we are assessing how older persons are frequently exposed to various stressors in their life. It is estimated that about 25% of healthy older adults experience one stressful life event within a 3-month period. This marker (stress) is a life changing phenomena, as it can alter how one feels, sees events, and acts. It is also intrinsic to the biomarkers of the person. **Stress matters.**

Negative affect or neuroticism play a role here overvaluing problems in life and causing problems across the lifespan. This state in combination with an acute stressor can have a profound impact on many people. (The acute stressor alone does this.) Almeida, Piazza, Stawski, and Klein (2011) showed that individual differences in neuroticism were associated

with higher levels of cognitive impairment and accounted for the between-person effect of daily stress. At the within-person level, neuroticism and daily stress were each equally associated with higher levels of cognitive impairment. Covarying for daily physical symptoms did little to draw down the association between daily stress and cognitive impairment at level of analysis.

We assess stress by use of a simple metric – 1-10 on a stress dimension. We will also use the Perceived Stress Scale which is longer and more accurate as it includes many aspects of living.

Caregiving/Longterm Care: This has a special role at later life. We will only address caregiving here (see Hyer & Intrieri, 2006). Based on 1480 family caregivers from the AARP and National Alliance for Caregiving, titled Caregiving in the U.S., it has been estimated that 65.7 million Americans serve as caregivers in 2012. This is 28% of the population. Nearly 1/3 of American households reported at least one person serving in an unpaid caregiving role. Depression is present in 28% and 34% have depressive symptoms. This results in poorer caregiving, more problems with the identified patient, and causes big problems for care receiver (CR). We place this variable here because any household with caregiving, whole or in part, is at risk.

And we note the obvious. Caregiving is harmful to your health. A strong evidence base exists that some behaviors and beliefs make a difference. Maslow (2012) assessed 44 interventions targeting individuals with dementia and/or their family caregivers; Brodaty and Arasaratnam (2012) looked at 23 caregiver interventions demonstrating positive outcomes for families; Gitlin and Hodgson noted over 200 interventions reviewed and 24 meta analyses and systemic reviews conducted between 1996 and 2013. Exemplars of interventions that could address problems include care management (maximizing independence at home, disease education, skills to manage functional independence, strategies to address behavioral symptoms, activities to effectively engage and support). This is often done in a multi-component format.

What causes problems are no psycho-education, poor role clarification, poor self-care, no daily management strategies, belief that behavioral problems are unavoidable, no assertive skills, and no positive feedback to the identified patient. There is also compassion fatigue (when you have cared for others more than yourself; when your sense of responsibility to others has become exaggerated or out of balance). The caregiver needs to acknowledging the disease, make the cognitive shift, develop emotional tolerance, take control, establish a realistic goal, gauge the recipient's capacities, design opportunities for satisfying work, become a sleuth, and

have a sense of perspective. Multiple methods or single targeted method intensely applied work best.

Personality Disorders

At the core of problems is personality. The problem state of this construct is a personality disorder (PDs). It can cause problems for people at all ages. In a sense personality is the bedrock of our model. Everyone has a personality and people with PDs have significant problems at least with relationships. They have difficulty maintaining close ties with family members and they have trouble getting along with coworkers. The interactions of one with a PD can be both predictable and regrettable.

There are several models of personality and PDs that we address. The American Psychiatric Association has defined personality in the DSM-5 with some complexity but also maintaining criteria of an enduring pattern of inter-experience and behavior that deviates markedly from the expectations of the individual's culture. The five-factor model has been used. This explains the patient's variability in both personality disordered and non-disordered populations.

We apply one of the Millon inventories. The assessment of personality or personality pathology needs to reflect the overall behaviors and functioning of the person. From here comes Axis I problems and issues of treatment prognostics and stress moderation. In fact, life of the five domains depends on this construct, whether it involves problems or not. (As noted in Chapter 1, we use the MBMD. Its variables and markers are given in Appendix A.)

Conclusion

We now proceed to each of the five domains. The time involved in the evaluation of the older adult initially is about an hour. We stress again that in the first session only a few formal tests are given. Data can come from any source. The intent is to have a data point that reveals strengths or problems; further testing can then be initiated. Remember this is the initial evaluation for the Watch and Wait model. Tests provided by the screen can be added to, especially as regards the context of these psychiatric problems, personality, history, phenomenological symptom picture. Lichtenberger (2012) noted that an evaluation of older adults needs to be wide – including resistance, life situation and strengths and weaknesses. This can be done in the Watch and Wait model. Let's get specific.

References

- Albert, S. M., Roth, T., Toscani, M., Vitiello, M. V., & Zee, P. (2015). Sleep health and appropriate use of OTC sleep aids in older adults—recommendations of a Gerontological Society of America workgroup. *The Gerontologist*, 57(2), 163-170.
- Almeida, D. M., Piazza, J. R., Stawski, R. S., & Klein, L. C. (2011). The speedometer of life: Stress, health and aging. In *Handbook of the Psychology of Aging (Seventh Edition)* (pp. 191-206).
- Alexopoulos, G. S., Raue, P., & Arean, P. (2003). Problem-solving therapy versus supportive therapy in geriatric major depression with executive dysfunction. *American Journal of Geriatric Psychiatry*, 11(1), 46-52.
- American Psychiatric Association (2013). *Diagnostic and Statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Amjad, H., Roth, D.L., Samus, Q.M., Yasar, S., & Wolff, J.L. (2016). Potentially unsafe activities and living conditions of older adults with dementia. *Journal of the American Geriatrics Society*, 64(6), 1223-1232.
- Andrescu, C., Sheu, L. K., Tudorascu, D., Gross, J. J., Walker, S., Banihashemi, L., & Aizenstein, H. (2015). Emotion reactivity and regulation in late-life generalized anxiety disorder: functional connectivity at baseline and post-treatment. *The American Journal of Geriatric Psychiatry*, 23(2), 200-214.
- Ayers, C. R., Sorrell, J. T., Thorp, S. R., & Wetherell, J. L. (2007). Evidence-based psychological treatments for late-life anxiety. *Psychology and aging*, 22(1), 8.
- Bäckman, L., Jones, S., Berger, A. K., Laukka, E. J., & Small, B. J. (2005). Cognitive impairment in preclinical Alzheimer's disease: a meta-analysis. *Neuropsychology*, 19(4), 520.
- Baker, L. D. et al. (2015). Aerobic exercise reduces phosphorylated tau protein in cerebrospinal fluid in older adults with mild cognitive impairment. *Alzheimer's & Dementia*, 11(7 Supplement), P324.
- Banerjee, S., Smith, S. C., Lamping, D. L., Harwood, R. H., Foley, B., Smith, P., ... & Knapp, M. (2006). Quality of life in dementia: more than just cognition. An analysis of associations with quality of life in dementia. *Journal of Neurology, Neurosurgery & Psychiatry*, 77(2), 146-148.
- Beckett, M. K., Elliott, M. N., Haviland, A. M., Burkhart, Q., Gaillot, S., Montfort, D., & Saliba, D. (2015). Living alone and patient care

- experiences: the role of gender in a National Sample of Medicare beneficiaries. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, 70(10), 1242-1247.
- Bishop, E., Boyle, W. R., Chen, P., Chromy, J. R., Clarke, A., Copello, E., ... Virag, T. G. (2005). *Results from the 2005 National Survey on Drug Use and Health: National Findings*.
- Blazer, D.G. (2000). Psychiatry and the oldest old. *The American Journal of Psychiatry*, 157(12), 1915-1924.
- Bobb, D.S. et al (2012). Neural correlates of successful response inhibition in unmedicated patients with late-life depression. *The American Journal of Geriatric Psychiatry*, 20(12), 1057-1069.
- Bobbitt, B.L., Cate, R.A., Beardsley, S.D., Azocar, F., & McCulloch, J. (2012). Quality improvement and outcomes in the future of professional psychology: Opportunities and challenges. *Professional Psychology: Research and Practice*, 43(6), 551-559.
- Bower, E. S., & Wetherell, J. L. (2015). Late-life anxiety disorders. *APA handbook of clinical geropsychology*, 2, 49-77.
- Brodaty, H., & Arasaramnam, C. (2012). Meta-analysis of nonpharmacological interventions for neuropsychiatric symptoms of dementia. *American Journal of Psychiatry*, 169(9), 946-953.
- Byers, A. L., Arean, P. A., & Yaffe, K. (2012). Low use of mental health services among older Americans with mood and anxiety disorders. *Psychiatric Services*, 63(1), 66-72.
- Byers, A.L., et al. (2010). High occurrence of mood and anxiety disorders among older adults: The National Comorbidity Survey replication. *Archives of General Psychiatry*, 67(5), 489-496.
- Carnevale, L., D'Angelosante, V., Landolfi, A., Grillea, G., Selvetella, G., Storto, M., ... Carnevale, D. (2018). Brain MRI fiber-tracking reveals white matter alterations in hypertensive patients without damage at conventional neuroimaging. *CARDIOVASCULAR RESEARCH*, 114(11), 1536-1546.
<https://doi-org.medlib-proxy.mercer.edu/10.1093/cvr/cvy104>
- Cassidy, K. L., & Rector, N. A. (2008). The silent geriatric giant: Anxiety disorders in late life. *Geriatrics and Aging*, 11(3), 150-156.
- Castilla-Rilo, J., Lopez-Arrieta, J., Bermejo-Pareja, F., Ruiz, M., Sanchez-Sanchez, F., & Trincade, R. (2007). Instrumental activities of daily living in the screening of dementia in population studies: A systematic review and meta-analysis. *International Journal of Geriatric Psychiatry*, 22, 829-836.

- Choi, N. & DiNitto, D.M. (2015). Role of new diagnosis, social isolation, and depression in older adults' smoking cessation. *The Gerontologist*, 55(5), 793-801.
- Choi, N.G., Kim, J., Marti, C.N., & Chen, G.J. (2014). Late-life depression and cardiovascular disease burden: Examination of reciprocal relationship. *The American Journal of Geriatric Psychiatry*, 22(12): 1522-1529.
- Choi, H., Yorgason, J. B., & Johnson, D. R. (2015). Marital quality and health in middle and later adulthood: Dyadic associations. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 71(1), 154-164.
- Cleeland CS & Ryan KM (1994). Pain assessment: global use of the Brief Pain Inventory. *Ann Academy Medicine Singapore*, 23(2):129-38.
- Cogbill, E., Dinson, K., & Duthie, E. (2010). Considerations in prescribing medication to the elderly. In V. Hirth, D. Wieland, & M. Dever-Bumba (Eds.), *Case-based geriatrics: A global approach*. New York, NY: McGraw-Hill.
- Copeland, J. R. M., Kelleher, M. J., Kellett, J. M., Gourlay, A. J., Gurland, B. J., Fleiss, J. L., & Sharpe, L. (1976). A semi-structured clinical interview for the assessment of diagnosis and mental state in the elderly: the Geriatric Mental State Schedule: I. Development and reliability. *Psychological medicine*, 6(3), 439-449.
- Delbaere, K., Closs, J.C.T., Taylor, M., Wesson, J. & Lord, S.R. (2013). Validation of the Iconographical Falls Efficacy Scale in cognitively impaired older people. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 68(9), 1098-1102.
- DeVol, R., & Bedroussian, A. (2007). *An Unhealthy America. The Economic Burden of Chronic Disease*, 2007.
- Diehr, P.H., Thielke, S.M., Newman, A.B., Hirsch, C., & Tracy, R. (2013). Decline in health for older adults: Five-year change in 13 key measures of standardized health. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 68(9), 1059-1067.
- Donovan, N. J., Amariglio, R. E., Zoller, A. S., Rudel, R. K., Gomez-Isla, T., Blacker, D., ... & Marshall, G. A. (2014). Subjective cognitive concerns and neuropsychiatric predictors of progression to the early clinical stages of Alzheimer disease. *The American Journal of Geriatric Psychiatry*, 22(12), 1642-1651.
- D'Zurilla, T. J., & Nezu, A. M. (2010). Problem-solving therapy. *Handbook of cognitive-behavioral therapies*, 3, 197-225.
- Eaton, W. W., & Muntaner, C. (1999). Socioeconomic stratification and mental disorder. Horwitz AV, Sheid TL, ed., "A Handbook for the

- Study of Mental Health-Social Context, Theories and Systems, (Ch. 14).
- Ferraro, K.F. & Wilkinson, L.R. (2015). Alternative measures of self-rated health for predicting mortality among older people: Is past or future orientation more important? *Gerontologist*, 55(5), 836-844.
- Fields, J.A. et al. (2010). Utility of the DRS for predicting problems in day-to-day functioning. *The Clinical Neuropsychologist*, 24(7), 1167-1180.
- Frisch, M. B. (2006). Quality of life therapy. Hoboken: Wiley.
- Galvin, J.E., Roe, C.M., Powlishta, K.K., Coats, M.A., Muich, S.J., Grant, E. Miller, J.P., Storandt, M., & Morris, J.C. (2005). The AD8 a brief informant interview to detect dementia. *Neurology*, 65(4), 559-564.
- Gandy, S. (2005). The role of cerebral amyloid β accumulation in common forms of Alzheimer disease. *The Journal of clinical investigation*, 115(5), 1121-1129.
- Gitlin, L. N., & Hodgson, N. (2015). Caregivers as therapeutic agents in dementia care: the context of caregiving and the evidence base for interventions. In *Family caregiving in the new normal* (pp. 305-353).
- Golant, S.M. (2015). Residential normalcy and the enriched coping repertoires of successfully aging adults. *The Gerontologist*, 55(1), 70-82.
- González-Palau, F., Franco, M., Toribio, J.M., Losado, R., Parra, E., & Bamadio, P. (2013). Designing a computer-based rehabilitation solution for older adults: the importance of testing usability. *PsychNology Journal*, 11(2), 119-136.
- Gray, S.L., et al. (2013). Frailty and incident dementia. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 68(9), 1083-1090.
- Greenway, M.C., Lacritz, L.H., Binegar, D., Weiner, M.F., Lipton, A., & Collum, M.C. (2006). Patterns of verbal memory performance in mild cognitive impairment, Alzheimer disease, and normal aging. *Cognitive Behavioral Neurology*, 19(2), 79-84.
- Gurian, B. S., & Minor, J. H. (1991). Anxiety in the elderly: treatment and research. *Clinical presentation of anxiety in the elderly*. New York: Springer, 191, 31-44.
- Haight, B. K., Michel, Y., & Hendrix, S. (2000). The extended effects of the life review in nursing home residents. *The International Journal of Aging and Human Development*, 50(2), 151-168.
- Hall, K. S., Cohen, H. J., Pieper, C. F., Fillenbaum, G. G., Kraus, W. E., Huffman, K. M., ... & Newby, L. K. (2017). Physical performance

- across the adult life span: correlates with age and physical activity. *The Journals of Gerontology: Series A*, 72(4), 572-578.
- Hallgren, M., Helgadottir, B., Herring, M.P., Zeebari, Z., Lindefors, N., Kaldo, V., Öjehagen, A., & Forsell, Y. (2016). Exercise and internet-based cognitive-behavioural therapy for depression: Multicentre randomized controlled trial with 12-month follow-up. *The British Journal of Psychiatry*, 209(5), 414-420.
- Hamilton, M. (1959). Hamilton anxiety rating scale (HAM-A). *British Journal of Medical Psychology*, 32(1), 50-55.
- Harper, D.G., Volicer, L., Stopa, E.G., McKee, A.C., Nitta, M., & Satlin, A. (2005). Disturbance of endogenous circadian rhythm in aging and Alzheimer disease. *The American Journal of Geriatric Psychiatry*, 13(5), 359-368.
- Heintzelman, S. J., & King, L. A. (2014). Life is pretty meaningful. *American Psychologist*, 69(6), 561.
- Hill, N. T., Mowszowski, L., Naismith, S. L., Chadwick, V. L., Valenzuela, M., & Lampit, A. (2016). Computerized cognitive training in older adults with mild cognitive impairment or dementia: a systematic review and meta-analysis. *American Journal of Psychiatry*, 174(4), 329-340.
- Hybels, C.F., Pieper, C.F., Payne, M.E., & Steffens, D.C. (2016). Late-life depression modifies the association between cerebral white matter hyperintensities and functional decline among older adults. *The American Journal of Geriatric Psychiatry*, 24(1), 42-49.
- Hyer, L., & Intrieri, R. (2006). Perspective on long-term care. *Geropsychological interventions in long-term care*, 3-35.
- Ismail, Z., Elbayoumi, H., Fischer, C. E., Hogan, D. B., Millikin, C. P., Schweizer, T., ... & Fiest, K. M. (2017). Prevalence of depression in patients with mild cognitive impairment: a systematic review and meta-analysis. *Jama psychiatry*, 74(1), 58-67.
- Jak, A., Bondi, M., Delano-Wood, L., Wierenga, C., Corey-Bloom, J., Samon, D., & Delis, D. (2009). Quantification of five neuropsychological approaches to defining mild cognitive impairment. *American Journal of Geriatric Psychiatry*, 17(5), 368-375. <http://doi.org/10.1097/JGP.0b013e31819431d5>. Quantification
- Jak, A., Seelye, A.M., & Jurick, S.M. (2013). Crosswords to computers: A critical review of popular approaches to cognitive enhancement. *Neuropsychology Review*, 23(1), 13-26.
- Jessen, F., Wiese, B., Bachmann, C., Eifflaender-Gorfer, S., Haller, F., Kölsch, H., ... & Wollny, A. (2010). Prediction of dementia by subjective memory impairment: effects of severity and temporal

- association with cognitive impairment. *Archives of general psychiatry*, 67(4), 414-422.
- Joiner Jr, T. E., Pettit, J. W., Perez, M., Burns, A. B., Gencoz, T., Gencoz, F., & Rudd, M. D. (2001). Can positive emotion influence problem-solving attitudes among suicidal adults?. *Professional Psychology: Research and Practice*, 32(5), 507.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of general psychiatry*, 62(6), 593-602.
- Knight, B.G. & Pachana, N.A. (2015). *Psychological assessment and therapy with older adults*. USA: Oxford University Press.
- Kok, M., Coetzee, J. K., & Elliker, F. (2017). Overcoming the Divide: An Interpretive Exploration of Young Black South Africans' Lived Experiences of Upward Mobility in Central South Africa. *Qualitative Sociology Review*, 13(1).
- Ladouceur, R., Léger, É., Dugas, M., & Freeston, M. H. (2004). Cognitive-behavioral treatment of generalized anxiety disorder (GAD) for older adults. *International psychogeriatrics*, 16(2), 195-207.
- Lautenschlager, N.T., Cox, K.L., Flicker, L., Foster, J.K., van Bockxmeer, F.M., Xiao, J., Greenop, K.R., & Almeida, O.P. (2008). Effect of physical activity on cognitive function in older adults at risk for Alzheimer disease: a randomized trial. *JAMA*, 300(9), 1027-1037.
- Lebedev, A.V., Beyer, M.K., Fritze, F., Westman, E., Ballard, C., & Aarsland, D. (2014). Cortical changes associated with depression and antidepressant use in Alzheimer and lewy body dementia: An MRI surface-based morphometric study. *The American Journal of Geriatric Psychiatry*, 22(1), 4-13.e1.
- Lenze, E. J., Mulsant, B. H., Mohlman, J., Shear, M. K., Dew, M. A., Schulz, R., ... & Reynolds, C. F. (2005). Generalized anxiety disorder in late life: lifetime course and comorbidity with major depressive disorder. *The American journal of geriatric psychiatry*, 13(1), 77-80.
- Lichtenberger, E. O., Mather, N., Kaufman, N. L., & Kaufman, A. S. (2012). *Essentials of assessment report writing* (Vol. 22). John Wiley & Sons.
- Lin, F., Roiland, R., Polesskaya, O., Chapman, B., Johnson, M., Brasch, J., Chen, D.G. & Mapstone, M. (2014). Fatigability disrupts cognitive processes' regulation of inflammatory reactivity in old age. *The American Journal of Geriatric Psychiatry*, 22(12), 1544-1554.
- Luck, T., Then, F. S., Schroeter, M. L., Witte, V., Engel, C., Loeffler, M., ... & Riedel-Heller, S. G. (2017). Prevalence of DSM-5 Mild

- Neurocognitive Disorder in Dementia-Free Older Adults: Results of the Population-Based LIFE-Adult-Study. *The American Journal of Geriatric Psychiatry*, 25(4), 328-339.
- Machlin, S. R., & Soni, A. (2013). Peer Reviewed: Health Care Expenditures for Adults with Multiple Treated Chronic Conditions: Estimates from the Medical Expenditure Panel Survey, 2009. *Preventing chronic disease*, 10.
- Mah, L., Binns, M. A., & Steffens, D. C. (2015). Anxiety symptoms in amnesic mild cognitive impairment are associated with medial temporal atrophy and predict conversion to Alzheimer disease. *The American Journal of Geriatric Psychiatry*, 23(5), 466-476.
- Menne, H.L., Kinney, J.M., & Morhardt, D. (2002). 'Trying to continue to do as much as they can do': Theoretical insights regarding continuity and meaning making in the face of dementia. *Dementia: The International Journal of Social Research and Practice*, 1(3), 367-382.
- Martin, K. L., Blizzard, L., Srikanth, V. K., Wood, A., Thomson, R., Sanders, L. M., & Callisaya, M. L. (2013). Cognitive function modifies the effect of physiological function on the risk of multiple falls—a population-based study. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, 68(9), 1091-1097.
- Maslow, K. (2012). Translating innovation to impact: Evidence-based interventions to support people with Alzheimer's disease and their caregivers at home and in the community. *A white paper*.
- Melzack R (1987). The short-form McGill Pain Questionnaire. *Pain*, 30: 191-197.
- zackMerrill, D. A., Siddarth, P., Raji, C. A., Emerson, N. D., Rueda, F., Ercoli, L. M., ... & Bookheimer, S. Y. (2016). Modifiable risk factors and brain positron emission tomography measures of amyloid and tau in nondemented adults with memory complaints. *The American Journal of Geriatric Psychiatry*, 24(9), 729-737.
- Nahemow, L., & Lawton, M. P. (1973). Toward an ecological theory of adaptation and aging. *Environmental design research*, 1, 24-32.
- Nimrod, G., & Shrira, A. (2014). The paradox of leisure in later life. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 71(1), 106-111.
- The Obesity Society (2014). *Obesity Statistics*. Retrieved from <http://www.obesity.org/obesity/resources/facts-about-obesity/statistics>.
- Lin, J. T., Schneider, L. S., Katz, I. R., Meyers, B. S., Alexopoulos, G. S., Breitner, J. C., ... & Krishnan, K. R. R. (2002). Provisional diagnostic criteria for depression of Alzheimer disease. *The American journal of geriatric psychiatry*, 10(2), 125-128.

- Parisi, J. M., Roberts, L., Szanton, S. L., Hodgson, N. A., & Gitlin, L. N. (2015). Valued activities among individuals with and without cognitive impairments: Findings from the National Health and Aging Trends Study. *The Gerontologist, 57*(2), 309-318.
- Park, M., Verhoeven, J. E., Cuijpers, P., Reynolds III, C. F., & Penninx, B. W. J. H. (2015). Where You Live May Make You Old: The Association between Perceived Poor Neighborhood Quality and Leukocyte Telomere Length. *PLoS ONE, 10*(6), 1. Retrieved from <http://medlib-proxy.mercer.edu/login?url=https://search.ebscohost.com/login.aspx?direct=true&db=edb&AN=103566591&site=eds-live&scope=site>
- Pimontel, M.A., Rindskopf, D., Rutherford, B.R., Brown, P.J., Roose, S.P., & Sneed, J.R. (2016). A meta-analysis of executive dysfunction and antidepressant treatment response in late-life depression. *The American Journal of Geriatric Psychiatry, 24*(1), 31-41.
- Pinquart, M. & Duberstein, P.R. (2007). Treatment of anxiety disorders in older adults: A meta-analytic comparison of behavioral and pharmacological interventions. *The American Journal of Geriatric Psychiatry, 15*(8), 639-651.
- Putnam, R. D. (2016). *Our kids: The American dream in crisis*. Simon and Schuster.
- Resnick, B., Galik, E., Dorsey, S., Scheve, A., & Gutkin, S. (2011). Reliability and validity testing of the Physical Resilience measure. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 51*(5), 643-652.
- Royall, D. R., Lauterbach, E. C., Cummings, J. L., Reeve, A., Rummans, T. A., Kaufer, D. I., ... & Coffey, C. E. (2002). Executive control function: a review of its promise and challenges for clinical research. A report from the Committee on Research of the American Neuropsychiatric Association. *The Journal of neuropsychiatry and clinical neurosciences, 14*(4), 377-405.
- Satre, D. D., Bahorik, A. L., & Mackin, R. S. (2018). Alcohol and Drug Use Among Older Adults: Associations with Widowhood, Relationship Quality, and Physical Health. *Journals of Gerontology Series B: Psychological Sciences & Social Sciences, 73*(4), 633-635. <https://doi-org.medlib-proxy.mercer.edu/10.1093/geronb/gbx158>
- Savica, R., Wennberg, A., Hagen, C., Edwards, K., Roberts, R. O., Hollman, J. H., ... & Mielke, M. M. (2017). Comparison of gait parameters for predicting cognitive decline: the mayo clinic study of aging. *Journal of Alzheimer's Disease, 55*(2), 559-567.

- Schonfeld, L., King-Kallimanis, B. L., Duchene, D. M., Etheridge, R. L., Herrera, J. R., Barry, K. L., & Lynn, N. (2010). Screening and brief intervention for substance misuse among older adults: the Florida BRITE project. *American journal of public health, 100*(1), 108-114.
- Schuurmans, J., Comijs, H., Emmelkamp, P. M., Gundy, C. M., Weijnen, I., Van Den Hout, M., & Van Dyck, R. (2006). A randomized, controlled trial of the effectiveness of cognitive-behavioral therapy and sertraline versus a waitlist control group for anxiety disorders in older adults. *The American journal of geriatric psychiatry, 14*(3), 255-263.
- Scott, K. M., Lim, C., Al-Hamzawi, A., Alonso, J., Bruffaerts, R., Caldas-de-Almeida, J. M., ... & Kawakami, N. (2016). Association of mental disorders with subsequent chronic physical conditions: world mental health surveys from 17 countries. *JAMA psychiatry, 73*(2), 150-158.
- Segal, Z. V., Teasdale, J. D., Williams, J. M., & Gemar, M. C. (2002). The mindfulness-based cognitive therapy adherence scale: Inter-rater reliability, adherence to protocol and treatment distinctiveness. *Clinical Psychology & Psychotherapy, 9*(2), 131-138.
- Seignourel, P. J., Kunik, M. E., Snow, L., Wilson, N., & Stanley, M. (2008). Anxiety in dementia: a critical review. *Clinical psychology review, 28*(7), 1071-1082.
- Seligman, M. E. (2002). Positive psychology, positive prevention, and positive therapy. *Handbook of positive psychology, 2*(2002), 3-12.
- Shega, J. W., Hougham, G. W., Stocking, C. B., Cox-Hayley, D., & Sachs, G. A. (2008). Patients dying with dementia: experience at the end of life and impact of hospice care. *Journal of pain and symptom management, 35*(5), 499-507.
- Ströhle, A., Schmidt, D. K., Schultz, F., Fricke, N., Staden, T., Hellweg, R., ... & Rieckmann, N. (2015). Drug and exercise treatment of Alzheimer disease and mild cognitive impairment: a systematic review and meta-analysis of effects on cognition in randomized controlled trials. *The American Journal of Geriatric Psychiatry, 23*(12), 1234-1249.
- Sundermann, E. E., Katz, M. J., & Lipton, R. B. (2017). Sex differences in the relationship between depressive symptoms and risk of amnesic mild cognitive impairment. *The American Journal of Geriatric Psychiatry, 25*(1), 13-22.
- Sundermann, E.E., Maki, P.M., Rubin, L.H., Lipton, R.B., Landau, S. & Biegon, A. (2016). Female advantage in verbal memory: Evidence of sex-specific cognitive reserve. *Neurology, 87*(18), 1916-1924.

- Tak, S.H., Zhang, H., Patel, & Hong, S.E. (2015). Computer activities for persons with dementia. *The Gerontologist*, 55(Suppl 1): S40-S49.
- Teri, L. et al. (2003). Exercise plus behavioral management in patients with Alzheimer disease: A randomized controlled trial. *JAMA*, 290(15), 2015-2022.
- Teri, L., McKenzie, G., Logsdon, R.G., McCurry, S.M., Bollin, S., Mead, J., & Menne, H. (2012). Translation of two evidence-based programs for training families to improve care of persons with dementia. *The Gerontologist*, 52(4), 452-459.
- Therrien, Z., & Hunsley, J. (2012). Assessment of anxiety in older adults: A systematic review of commonly used measures. *Aging & Mental Health*, 16(1), 1-16.
- Ticinesi A, Tana C, Nouvenne A, Prati B, Lauretani F, & Meschi T. (2018). Gut microbiota, cognitive frailty and dementia in older individuals: a systematic review. *Clinical Interventions in Aging*, Vol Volume 13, Pp 1497-1511 (2018), 1497.
- Valenti, G., Bonomi, A. G., & Westerterp, K. R. (2017). Quality sleep is associated with overnight metabolic rate in healthy older adults. *The Journals of Gerontology: Series A*, 72(4), 567-571.
- Wells, A., White, J., & Carter, K. (1997). Attention training: Effects on anxiety and beliefs in panic and social phobia. *Clinical Psychology & Psychotherapy*, 4(4), 226-232.
- Wen, C. P., Pui, J., Wai, M., Tsai, M. K., Yang, Y. C., Yuan, T., ... Tsao, C. K. (2011). Minimum amount of physical activity for reduced mortality and extended life expectancy: a prospective cohort study. *The Lancet*, 378(9798), 1244-1253. [http://doi.org/10.1016/S0140-6736\(11\)60749-6](http://doi.org/10.1016/S0140-6736(11)60749-6)
- Wetherell, J. L., Gatz, M., & Craske, M. G. (2003). Treatment of generalized anxiety disorder in older adults. *Journal of consulting and clinical psychology*, 71(1), 31.
- Wetherell, J. L., Liu, L., Patterson, T. L., Afari, N., Ayers, C. R., Thorp, S. R., ... & Petkus, A. J. (2011). Acceptance and commitment therapy for generalized anxiety disorder in older adults: A preliminary report. *Behavior therapy*, 42(1), 127-134.
- White, L. R., Edland, S. D., Hemmy, L. S., Montine, K. S., Zarow, C., Sonnen, J. A., ... & Masaki, K. H. (2016). Neuropathologic comorbidity and cognitive impairment in the Nun and Honolulu-Asia Aging Studies. *Neurology*, 86(11), 1000-1008.
- Wolff, J. K., Schmiedek, F., Brose, A., & Lindenberger, U. (2013). Physical and emotional well-being and the balance of needed and

received emotional support: Age differences in a daily diary study. *Social Science & Medicine*, 91, 67-75.

World Health Organization. (2010). Global recommendations on physical activity for health. Geneva: World Health Organization. <http://www.who.int/iris/handle/10665/44399>

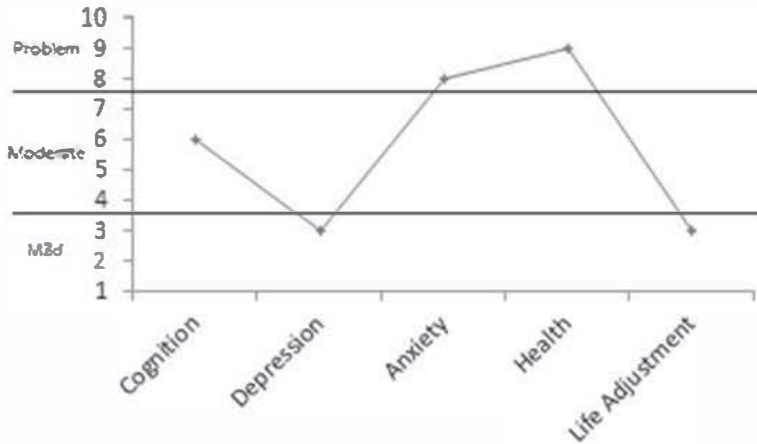
Yeager, D. S., Dahl, R. E., & Dweck, C. S. (2017). Why interventions to influence adolescent behavior often fail but could succeed. *Perspectives on Psychological Science*, 1745691617722620.

CHAPTER 6

APPLICATION OF WATCH AND WAIT TO CHRONIC MEDICAL CONDITIONS AND COMORBID BEHAVIORAL HEALTH PROBLEMS

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We pick up with the case from Chapter 5.



Health Case	
Problems	
Health	
Chronic Diseases:	5
Health Rating:	2
ESS =	14
Pain:	40

Cigarettes: No
 Alcohol: None
 BMI: 41
 Exercise: None, excessive sedentary behavior

Anxiety

MINI: Positive for Generalized Anxiety Disorder (GAD)
 GAD-7 = 11
 BAI = 18
 SAST = 64
 MBMD: Anxiety/Tension

DEPRESSION

MADRS = 5, normal.
 GDS-SF = 4
 BDI-II = 10
 PHQ-9 = 4,

COGNITION

Normal (see below)

LIFE ADJUSTMENT

Good home support and health care team.
 No SES markers and high education

PATIENT: TG

HISTORY OF PRESENT ILLNESS: TG is a 64-year-old Caucasian male who was just admitted to the hospital for post evaluation after a Left Ventricular Assistive Device (LVAD) placement. He reported that in 1998, while he was employed as a principal, he had a massive myocardial infarction that altered his life measurably. Over the years, this heart problem got worse and he eventually had to retire. He had 4 stents in 1998. In 1999, he had a cardiac bypass and he was able to handle things reasonably well until more recently. Three weeks ago, he was given the information that he had less than 1 year to live and agreed to an LVAD.

RELEVANT DEVELOPMENTAL HISTORY: TG is a native of Georgia. He is the second of 4 children. His father was a truck driver and farmer and currently has dementia. His mother raised the family. He indicated that his life on the farm was positive, and he thrived. There were no milestone disruptions. He went to a local college where he was graduated with a bachelor's degree and then went on to receive a master's degree in education. He was employed as a teacher and elementary school principal for 22 years. He retired in the year 2000 and worked part-time for a charity until 2004. He married in 1975, and his wife also is a teacher. They have 2 children. He indicates that these days, he is reasonably active

for one with heart problems. He stays around the house, but does drive, even cuts grass, works in the garden, and keeps mentally sharp by following the newspapers, television, and electronic media.

RELEVANT MEDICAL HISTORY: TG's medical records reflect substantial heart problems (Congestive Heart Failure and cardiomyopathy). He is well known to the cardiac team and is seen weekly. He takes over 10 medications, mostly for heart issues. He also has hypertension (HTN) and Type 2 Diabetes (T2D). It is noteworthy that he does not use alcohol to any degree, but he smoked up until 1998, then quit after his MI. He is not in pain. He sleeps at night relatively well, but also sleeps off and on during the day (perhaps excessively). He is able to do minimal activity, which is frustrating for him. He has recently been placed on an antidepressant, Effexor, which seems to help. He indicated that he has lost his purpose. He denies most depression symptoms and states that his memory is sharp.

MENTAL STATUS: TG is a 64-year-old Caucasian male who was interviewed at the bedside. He was humorous, factual, and provided accurate information regarding his history. He was entertaining with stories about his life. He is obese and uses a cane when ambulatory. He denied having any memory problems, and memory deficits were not evident during the interview. He functions well and is conversant about political events. He indicated that he is searching for a new life but denied being depressed. He is anxious. He notes that he has always been a worrier and now is excessively concerned about his health. He did manifest shortness of breath, understandable for someone with his heart condition. There is no evidence of perceptual anomalies or delusional thinking. He appears to show quite good judgment and have insight into his condition. He is resigned to his fate and is motivated for better living now that his LVAD is placed.

TESTS RESULTS:

PREMORBID FUNCTIONING and **OVERALL COGNITIVE FUNCTIONING:** TG has a Barona Index of Intelligence score of High Average. This is an appropriate marker with which to gage his skills at present. He has been a school principal for many years and 13 years ago. Currently, Mr. G shows no evidence of cognitive impairment. His MoCA is 28/30. He had issues with visuospatial tasks. His RBANS total score is 94, low average. His memory and new learning are in the low average range. His attention was also normal (CPT-2). His EF was also average or above (Trails B). He has a good fund of knowledge. He is oriented fully and has been functioning independently in the community. No further neuropsychological tests were applied.

EMOTIONAL ASSESSMENT: His profile on the MINI is consistent with GAD. He describes himself as not depressed, but he endorses anxiety and worry. There were markers of anxiety on the MBMD and the PAI. He scored in the anxious range on the BAI, SAST, and GAD-7. His MADRS is 5, normal. He also scored in the normal range on several other measures. ● on the GDS-SF he scored 4; 10 on the BDI-II; and 4 on the PHQ-9, indicating no or very mild depressive symptoms.

SLEEP: Patient indicates that he has no problems falling asleep, but sleep is often fragmented. His ESS is 6, indicating that he is not tired during the day.

PAIN: Pain is a mild issue for him. His average pain is 4/10. He rates pain at present as 3/10. As a result of pain, Mr. G has problems with activity (5/10), with mood (4/10), walking (0/10), work (NA), relations with people (3/10), sleep (5/10), and enjoyment with life (4/10).

Watch and Wait: This 64 y/o male presents with anxiousness and serious medical issues. That said, he is well treated medically and is compliant regarding appointments and medications. His medical and behavioral health care needs better integration. Regarding mood, he shows no evidence of depression. His sleep is a minor problem, and pain is also a minor issue. His personality style is cooperative but inhibited. He is also less social than he needs, but he has good support from his wife. He also takes an antidepressant medication. Cognitively he is above average premorbidly, and current testing shows no problems on cognitive tasks, including memory and executive functioning. He is slightly lower visuospatial tasks. He is motivated for help.

Checklist

Discemment: (agrees with mental health plan)

Core markers

Validate Problem
 Psychoeducation of Model
 Assessment
 Alliance
 Monitoring
 Case formulation

Problem List

1. **HEALTH:** Coordinate care with cardiology, help monitor HTN and HbA1c
2. **ANXIETY:** Monitor anxiety daily with SUDS log, CBT group

weekly, teach relaxation, apply worry control, initiate exercise 4x/week via physical therapy, individual counseling focuses on self- image, coping and assertiveness needs, HRV biofeedback

3. DEPRESSION: Despite normal scores, monitor mood, push behavioral activation

Given the importance of health and his positive response to the LVAD procedure, he himself needs to maintain compliance. He is also excessively nervous, fueled in part by his heart problems. Sleep and pain are less of a problem. He was invited to the CBT group and targets of day-to-day coping and behavioral activation are in place. He will be provided HRV biofeedback for anxiety. His wife will also be a part of his therapy and will be seen periodically.

Self or other monitoring: Mini- targets of behavior (check) over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments:

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:

Imagery exercise for counter-experience: X

Compassion training:

Communication and Assertiveness training: X

Behavioral experiments: X

Emotional exposure:

Homework: X

Longer Therapy processes:

CBT: Mild use of 3-6 column evidence record

ACT: use of acceptance interventions

Lifestyle:

- Practice happiness
- Exercise: coordinate with Cardiac Clinic
- Practice spirituality/religion
- Socialize: less decline and live longer
- Develop resilience
- Exercise: even housework

- Improve diet
- Somatic softening: Meditation and Mindfulness
- Values and leisure

Follow-up: Re-evaluate every 6 months

Health (at 6 months post-evaluation)

Chronic Diseases: 5

Health Rating: 4

ESS = 7

Pain: 3/10

BMI: 36

Exercise: Less sedentary behavior, more active socialization

Anxiety

MINI: GAD in remission

GAD-7 =6

BAI = 10

SAST =45

MBMD: Anxiety/Tension

Cognition: MoCA =28/30 (as before)

Depression: Normal

Life Adjustment: Same as before

Relapse Concerns: none noted

Despite some gains, the domain profile did not change (see above).

Measure	Healthy	Moderate Problem	Problematic
Description	Bodily function is normal. Patient has few pain complaints and few chronic conditions that will likely shorten life or reduce QoL	Minor health concerns which may include lesser chronic conditions. Quality of life is impacted, but not debilitating so.	ADL/IADLs are compromised. Excessive comorbidities or meds
Health Rating	Good	Average	Poor
No. Chronic Illnesses	≤1	2-3	≥4

Pain Scale	≤44 mm	45-74 mm	≥75 mm
BMI	18.5-24.9	25-29.9	Under 18.5 or over 29.9
Dietary Screening Tool	>75	60-75	<60
Exercise	>30 minutes every day	<30 min/ 1-2 days week	Housework, inactive
Physical Activity Readiness	0	≥1 discuss with MD	≥1 discuss with MD
Insomnia Severity Index	0-7	8-14	≥15
Epworth Sleep Scale	0-10	11-15	≥16
Stop-BANG (sleep apnea)	0-2	3-4	5-8
Alcohol Consumption	≤7 drinks per week if the patient is not on medication	≥7 drinks per week if patient is not on med	≥7 drinks/ week if patient is on med that interacts with alcohol
Short Michigan Alcohol Screen Test-G	0-1	0-1	≥2
Tobacco Use	Never smoked	Smoked 100+ but quit	Current smoker

Health is an issue that is encompassing. It is also the gateway to behavioral healthcare for older adults. In our Watch and Wait model we always start with assessment. Even in health matters, assessment becomes a mode of treatment. It sets the stage and targets specific behaviors and problems. A number of the screening components listed above are completed in the primary care physician's office (BMI, pain assessment, tobacco use, review of medical problems, and medication reconciliation) and are self-explanatory. That said, unless the behavioral medicine specialist is embedded in the Primary Care Clinic, these data may not be readily available and therefore should be discussed with the patient in the initial

visit or obtained from the medical record. Evidence based condition-specific measures also are recommended to provide the behavioral medicine specialist with important information about a patient's current coping strengths and challenges with the medical problems in question. The most commonly used of these will be noted in the following sections.

Background

More than one in four US adults aged 45 years and above live with two or more chronic medical conditions commonly associated with middle and older ages (US Department of Health and Human Services, 2010). As noted previously, the prevalence of multiple chronic medical illnesses among Americans 65 years and older is 67% with two or more chronic conditions, and 24% with greater than four conditions. At the same time, we are living longer than ever, but clearly with compromised health and often inadequate or splintered medical and behavioral healthcare. The US healthcare delivery system is under pressure to better address this trend under the current requirements of the Patient Protection and Affordable Care Act (2010). So, behavioral medicine specialists (e.g., health psychologists, psychiatric nurse practitioners) are finding themselves in a vital role as partners with physicians in treating the chronic medical and behavioral health problems of their patients.

In the following pages we apply the Watch and Wait Model to the management of common chronic medical conditions seen among older adults in Primary Care Clinics (PCCs). Why consider physical health as a target for Watch and Wait? More than half of older adults who present to their Primary Care Physicians (PCPs) with a chronic medical condition will have comorbid, but often unidentified, behavioral health or life adjustment issues (Mitchell, Rao, & Vaze, 2010; Park & Unützer, 2011; Sinnige et al., 2015). Untreated behavioral health complaints are known to exacerbate and reinforce a number of common, chronic health problems such as hypertension (HTN) and cardiovascular disease (CVD), type 2 diabetes (T2D), chronic obstructive pulmonary diseases (COPD) and asthma, arthritis/chronic pain, and gastrointestinal diseases. How so? Unrecognized or inadequately treated neuropsychiatric and psychosocial problems are associated with ongoing poor self-care, bad lifestyle habits, social isolation, and adoption or maintenance of addictions such as tobacco, alcohol, and prescription drug misuse. Indeed, the relationship between chronic medical conditions and behavioral health problems is a bi-directional one that can lead to premature loss of functional independence and early mortality (Lin EH, et al., 2004; Mezuk et al., 2008; Mitsonis, Potagas, Zervas & Sfagos, 2009; Moussavi et

al., 2008; Rutledge et al., 2006; Schneiderman, Ironson & Siegel, 2005). Fortunately, all of these behavioral risk factors are modifiable and often preventable. Unfortunately, people invariably become trapped in rigid patterns of living that prevent them from achieving better health and wellbeing.

Even when a behavioral health problem is recognized in the PCC, the physician rarely has adequate time or specialty training to address it appropriately in the context of managing the medical condition(s). The best approach to treating the older medically compromised and distressed patient is a collaborative one that includes the PCP, behavioral medicine specialist, case manager, and subspecialty providers in a *Patient Centered Medical Home* (PCMH). The prototypical PCMH focuses on the whole person by addressing medical conditions, behavioral health and functional comorbidities, and life stressors through measured, conservative (i.e., stepped), comprehensive, evidence-based care. Essential to the PCMH model is respect for patients' personal, family, and cultural histories, and attention to care that is safe, of good quality, and has value to the patient and family (see Agency for Healthcare Research and Quality and Institute for Healthcare Improvement websites). The PCMH behavioral medicine specialist intervenes at the crossroads of comorbid physical illness and psychosocial problems, as well as enhances self-care efforts to prevent minor or subthreshold problems from becoming major health concerns (Kazak, Nash, Hiroto & Kaslow, 2017; Yeager, 2014). Watch and Wait thus dovetails nicely with the PCMH model.

In this chapter, we first consider the relationship between stress and the development and exacerbation of chronic disease. We do this from an emerging field of study in medicine and neuropsychiatry: *Psychoneuroimmunology* - the study of the interaction between stress, inflammation, and the development and maintenance of chronic disease. Appreciation of this central connection enables the behavioral medicine specialist to make a more sophisticated case-formulation that attends to important health markers and offers practical education and interventions for the patient. Second, we discuss the importance of addressing counterproductive lifestyle choices that contribute to both disease and behavioral health vulnerabilities, and argue for the inclusion of 'lifestyle monitoring' in the Watch and Wait model. This requires the behavioral medicine specialist to become the patient's *Coach* in the true sense of the word. Last, we apply Watch and Wait to the management of the most common chronic medical conditions in older adults in PCCs: T2D, HTN and CVD, COPD, and chronic pain. Our Watch and Wait teaching case is a 65 year man who presents in the PCC with a rather complex cluster of

medical, neuropsychiatric, and interpersonal conditions; a not uncommon older distressed patient who seeks help from his PCP.

The Problem of Life Stress in Chronic Disease

Aging naturally results in the gradual deterioration of physical, mental, and social capacities, and as such, affects every major organ system in the body, as well as the immune and central nervous systems. Aging is thus naturally associated with a higher risk of infection, prolonged recovery following infection, and a higher frequency of neuropsychiatric complications from infection. The allostatic model assures the chronic unfolding of problematic consequences for older adults especially if they are victims of stress. Chronic or recurrent life stressors take a substantial toll on health and life expectancy. Stress comes in many stripes – often delineated in the aging literature as life adjustment stress such as loss of loved ones, financial strain, neighborhood strain, medical compromise, and ageism – and rarely are any of these experienced singly. Timing of stressors and developmental stage likewise matter: highly stressful early life events (premature birth, childhood serious illness, abuse and neglect) not only prime an individual for a lifetime of internalizing vulnerabilities such as anxiety and depression (Monnat & Chandler, 2015), but they also increase the susceptibility to chronic diseases later in life (Bertagnolli et al., 2016; Taylor et al., 2011). Loneliness, often ignored in medicine, is a profoundly negative moderator of morbidity and mortality at all ages, but especially among the elderly (see Miller, 2011; Perissinotto, Cenzer & Covinsky, 2012).

Stress and Chronic Inflammation: Ongoing stress has been shown to trigger immune and proinflammatory defenses, and increase one's susceptibility to and maintenance of such chronic diseases as cardiovascular disease (CVD), Type 2 diabetes mellitus (T2D), gastrointestinal disorders, neurodegenerative diseases, and autoimmune disorders such as asthma, lupus, and multiple sclerosis (Black & Garbutt, 2002; Cohen et al., 2010; Crimmons et al., 2003; Jeong et al., 2006; DiMatteo et al., 2000; Elenkov and Chrousos, 2002; Surtees et al., 2008; Ziegelstein et al., 2000). Stress is expressed in the body primarily through stimulation of the sympathetic nervous system (SNS) and hypothalamic pituitary adrenal (HPA) axis. SNS and HPA activation cause alterations in neuroendocrine function that, when chronically triggered, dysregulate cortisol and oxytocin production, and keep the inflammatory response system activated. The brain has its own immune components, called microglia, that also release proinflammatory cytokines and prostaglandins in response to systemic inflammation. These molecules bind to specific neuronal receptors that affect neurotransmitter

production, synaptic plasticity, and cortisol concentrations, and appear to induce negative changes in mood/anxiety, cognition, and behavior (Benros et al., 2013; Miller et al., 2009; Smith et al., 2012). Importantly, the pro-inflammatory and immune suppressant effects of chronic stress are linked to, and may underpin in many cases, major depressive disorder and anxiety disorders. In fact, recent research shows that inflammation is specifically linked to the vegetative signs of depression – sleep and appetite dysregulation, fatigue, and anhedonia (Jokela et al., 2015), as well as suicide (Lund-Sørensen, et al., 2016). Hence, it should come as no surprise that depression and anxiety frequently coexist with, and are side effects of, common chronic disease states.

Stress, Inflammation, and the Gut: Also worth considering is emerging evidence of the consequences of stress, poor diet, and chronic antibiotic medication use on the integrity of the gut microbiome. This microbiome plays a critical role in processing nutrients. As gut microbes metabolize food, they produce an astonishing number of small molecules, chemicals and hormones that can influence health. The “brain-gut microbiota axis” is bidirectional and gut integrity appears to play an important role in stress tolerance, mood regulation, cognitive function, obesity, and visceral pain (for a brief review see: Dinan & Cryan, 2016). The gut microbiome affects neuroimmune function, too, including inflammatory cytokine production and production of the neurotransmitters serotonin (95% of which is produced in the gut), dopamine, norepinephrine, and GABA (Bailey et al., 2011; Evrensel & Ceylan, 2015; Mayer et al., 2014). Actually, mood disorders co-occur in over 50% of patients with irritable bowel syndrome (IBS) – and antidepressant medications are commonly used to treat this functional gut disorder (Neufield & Foster, 2009). People suffering from Crohn’s disease and ulcerative colitis similarly manifest depressive symptoms (Ananthakrishnan et al., 2013). This microbiome varies based on gender, age, community setting, dietary patterns, infection, and basal level of inflammation. Older adults have a less diverse microbiome profile than that of young people, which likely shapes the aging process. The term “inflamm-aging” was created to describe the pro-inflammatory environment that exists in older adults and is accompanied by impaired immune responses to pathogens and vaccines (Franceschi, Bonafe, Valensin, et al., 2000; Shaw, Goldstein & Montgomery, 2013). Furthermore, recent pre-clinical and human observational research shows that, in general, the lack of diverse gut microbiota, especially alterations in resident gut flora that are “psychobiotic” in nature, such as *Lactobacillare* and *Bifidobacteria*, are implicated in depression and anxiety (Marin et al., 2017). Altered composition of gut microbiota in the

elderly, especially a decrease in *Bifidobacteria*, has been linked to poor health and frailty (Zapata & Quagliarello, 2015).

Unfortunately, many elders, especially those living alone, do not eat well, relying on microwaveable frozen foods, Meals on Wheels, and fast food eateries as the mainstays of their daily diets, further compromising the integrity of the brain-gut axis. Older adults in long term care are at even higher risk of having inadequate gut microbiota in spite of adequate nutritional care. Claesson and colleagues (2012), in a study of the gut microbiomes of older adults in various settings, showed that the microbiomes of long term care residents were less diverse than those of community dwelling elders and this corresponded to the lack of a diverse diet among long-stay participants. The loss of community type microbiota correlated with increases in pro-inflammatory biomarkers and the Charlson co-morbidity index, as well as lower mini-mental state exam scores and decreased functional independence. Importantly, depressive symptoms correlated with the loss of community-associated microbiota.

There is now intense clinical interest in the treatment of depression and anxiety through probiotics. Although in the early stages of clinical study, treatment with probiotic/psychobiotic concoctions is showing promise in animal models and in humans with depression, anxiety, chronic fatigue syndrome, and IBS (Jiang, et al., 2015; Messaoudi et al., 2011; Ng et al., 2018; Rao et al., 2009; Wallace, 2017). In two recent meta analyses of human RCTs on the efficacy of probiotics for depression (N = 5 studies; Huang, Wang & Hu, 2016) and anxiety (N = 10 studies; Huang, Ning, Yang, et al 2017), probiotics were shown to have a positive effect on depressive and anxiety symptoms, although the included studies had small sample sizes, were underpowered, and focused on younger adult participants. There are insufficient data at this time to determine whether probiotic supplements or actual fermented foods (e.g., kefir, Greek yogurt, kimchi, sauerkraut and other fermented vegetables) are effective dietary interventions for depression and anxiety at later life. It does appear, however, that fecal implants (healthy donor to patient) is not only on the rise but effective for many medical problems (c-Diff) as well as some psychiatric ones (depression).

Lifestyle and Health Behavior Problems

Good health relies on good daily routines. As we have noted, five habits account for 70% of our morbidity and mortality: what we eat, how much we eat, whether we exercise, whether we smoke, and how much we drink alcohol (DeVol & Bedrosian, 2007). If sleep quality and prescription

drug (mis)use are included, it is clear that lifestyle habits go a long way toward enhancing or weakening our health.

The Problem of Obesity and Inflammation: Obesity (BMI \geq 30) is characterized by a chronic low-grade inflammatory state. Obesity has reached epidemic proportions all over the world (The Global Burden of Disease (GBD) 2015 Obesity Collaborators, 2017), even among older adults, and it contributes to the development and exacerbation of metabolic syndrome (i.e., a combination of HTN, elevated blood sugar, hyperlipidemia, and excessive visceral fat), other chronic inflammatory diseases, and cancer. Obesity, or more specifically the adipokines produced by white adipose tissue, also is a risk factor for dementia (Kiliaan, Amoldussen & Gustafson, 2014). Indeed, obesity has outstripped smoking as the number one modifiable behavioral risk factor associated with early premature death (Cleveland Clinic, 2017).

The thinking among many scientists and physicians is that obesity itself is a disease that deserves medical intervention (see Pollack, 2013). NHANES data collected over the past 25 years highlight the gravity of the problem. About 60% of adults 65 years and up were overweight or obese in the 1988-1994 NHANES survey. By 2014, overweight/obesity rates had increased to 70.7% of older adults (CDC, 2016). It is well known that PCPs, for their lack of sufficient face time with patients as well as limited success at motivating patients to change their behaviors, often ignore poor lifestyle habits that contribute to obesity. However, the current obesity epidemic and overall worsening health of the US population led the US Preventive Services Task Force in 2012 to urge screening for overweight/obesity at each doctor's visit (Moyer, 2012). We should note too that under-nutrition or malnutrition have been on the rise among older adults.

The PCMH naturally supports an integrated approach to correcting unhealthful behaviors that lead to obesity. The mainstay of obesity management is lifestyle change in terms of eating habits and exercise, treating comorbid depression, sleep problems, and life adjustment problems that increase stress eating, and considering medical weight loss interventions such as weight loss drugs and bariatric surgery. Newer FDA-approved weight loss drugs include Belviq, Symia, and Contrave. Other drugs that may help with weight loss are orlistat, phentermine, and the diabetes drugs metformin and Liraglutide (remarketed at a higher dose for weight loss as Saxenda). Bariatric surgery, which has a profoundly positive effect on T2D and HTN, is now performed with increasing frequency on adults over age 65 years; older age and health complications are no longer automatic contraindications for the procedure (Abbas, et al., 2015; Giordano & Victorzon, 2015).

The Problem of a Sedentary Lifestyle: Physical activity is among the most important modifiable factors in health and aging, yet few older adults achieve the minimum recommended amount of exercise. Low activity level has emerged as a primary contributing or exacerbating factor in a variety of health problems, including: falls, obesity, T2D, HTN, lung disease, colon cancer, stroke, osteoarthritis, and cognitive decline (see Hoffmann et al., 2016). On the other hand, there is good evidence to show that moderate exercise improves mood, reduces anxiety, builds stress tolerance, and may even stave off dementia. Especially noteworthy is a recently published randomized clinical trial (Blumenthal, et al., 2019) of older adults with cognitive impairment and cardiovascular risk who showed improved executive function with a combination of aerobic exercise and the DASH diet. Increased fitness is associated with a risk reduction of 20%-30% for more than 25 chronic diseases and all-cause mortality – what can be more motivating than the notion that one can keep death at bay by engaging in a minimum of 150 minutes of weekly exercise?

Unfortunately, older American adults are excessively sedentary. A recent synthesis of studies on sedentary behavior found that older adults spend an average of 9.4 hours a day (about 65% - 80% of awake time) inactive (Harvey, Chastin & Skelton, 2015). It is increasingly evident that excessive sedentary behavior puts older adults at risk where dementia is concerned. Modern culture's exuberant embrace of "high tech" ways to apply less effort and more efficiency to daily tasks – think of TV remote controls, cordless telephones and cell phones, ride-on lawn mowers, and smart home technologies – have taken a great toll on general fitness and motivation to be active. Although recent research suggests that retirement can provide a boost to physical activity because more time and resources are available to exercise (McDonald et al., 2015), other research indicates that physical activity after retirement may be dependent on the retiree's former career. For example, a career in the manual occupations is associated with a more sedentary lifestyle post-retirement (Barnett, et al., 2012). A recent analysis of BMI data from the Army Public Health Command supports this argument: approximately 50% of Army retirees are classified as obese (Drum, 2014), a rather stunning finding given that military service members must maintain weight and fitness standards across their careers. Anecdotal information on this group suggests that some retirees are resentful of their years of mandatory exercise and look forward to a sedentary retirement. Many are unaware that, with age and inactivity, calorie intake must be adjusted downward to minimize weight gain, along with maintaining a moderate level of exercise.

Sedentary adults are at much higher risk for falls than their active peers. Poor postural control is a key risk factor for falls, as is sarcopenia, or loss of muscle mass, which naturally worsens as one reaches the early to mid 70's. Exercise programs that improve balance, strength, flexibility, and general fitness can effectively reduce the risk of falls resulting in mild injuries by 37%, falls leading to serious injuries by 43%, and broken bones by 61%. (El-Kourhy et al., 2013; Gillespie et al., 2012). Indeed, a recent meta-analysis of RCTs testing multicomponent long term exercise programs for older individuals showed that 50 minute moderate intensity exercise for 2 – 3 days a week significantly reduces the risk of falling, injuries, and fractures (de Souto Barreto, Rolland, Vellas, Maltais, 2018). Older adults who have sustained a fall may develop significant anxiety about falling again such that they curtail physical and social activities to avoid future risk, but they inadvertently increase the risk of medical complications and depression. Doctors have traditionally recommended against exercise for older adults with chronic conditions and/or increased fall risk, and when exercise has been recommended, doctors often required patients to undergo a medical exam and stress test to be cleared. Such overly cautious prescriptives send the message that exercise is riskier than a sedentary lifestyle. Fortunately, exercise guidelines have been loosened in recent years to encourage patients to be more active without requiring medical clearance (see Hoffman et al., 2016).

Activities that improve strength, balance, and coordination to prevent falls

- Walk, climb stairs or bicycle to strengthen the muscles of the legs and lower body
- Use hand weights and elastic bands to build upper and lower body muscle strength
- Swim and/or use water aerobics to develop whole body muscle strength and coordination
- Improve balance with stretching exercises.
- Practice yoga to strengthen all muscle groups, improve balance, and increase flexibility
- Practice tai chi to improve muscle strength and balance

The Problem of Tobacco Use: Perhaps the most important question a behavioral medicine specialist can ask a patient who smokes is, “Have you thought about quitting?” On average, tobacco users will die 10 years earlier than their non-smoking peers (Jha et al., 2013). The majority of smokers (69%) want to quit, but these are more likely to be adults younger

than 65 years (DHHS, 2014). Although the most recent CDC statistics show that tobacco use has decreased in the U.S. from 2005 to 2015, 8.6% of adults ≥ 65 years still use tobacco and, notably, in a reversal of the trend to be tobacco free, more older men are smoking today than a decade ago (9.7% vs. 8.9%; Jamal et al., 2016). Low education level, low SES, inadequate access to healthcare, physical disability, and high psychological distress are associated with continued tobacco use. Not surprisingly, just the combination of poor diet, sedentary lifestyle, and tobacco use accounts for 35% of morbidity in the U.S. (Moukdad et al., 2004).

Tobacco use is associated with over 20 different cancers, as well as being a primary cause of CVD, T2D, cerebrovascular and pulmonary diseases, and chronic kidney disease. It is worth highlighting that there is hope for those older tobacco users who have resigned themselves to the inevitability of the deleterious effects of the habit and feel hopeless to change. A U.S. population study by Jha and colleagues (2013) shows that quitting by age 64 years can buy back 4 of those 10 years given to early death from tobacco use. And no matter what age one quits, quality of life will assuredly improve even in the face of tobacco-related chronic disease.

The Problem of Poor Sleep: Complaints of poor sleep are common among older adults. As many as 80% of elders report some kind of sleep disturbance such as insomnia, excessive daytime sleepiness, and parasomnias (Harper et al., 2005). That said, sleep problems are not fundamental to aging. Many older adults sleep well until the end of their lives. There are common sleep changes that occur with age: older adults tend to get less sleep overall; they often display an 'advance shift' in circadian rhythm with an earlier bedtime and earlier wake-up time; sleep architecture shifts so that more time is spent in light sleep (stages N1 and N2) and less time in deep, restorative sleep (stage N3), and REM sleep. Insomnia, perhaps the commonest complaint, is defined as a subjective complaint of difficulty initiating or maintaining sleep, or experiencing early morning awakenings, which occur at least 3 times per week for at least 3 months, and are associated with impaired daytime functioning (e.g., poor concentration, mood changes, fatigue). Insomnia is often comorbid with other medical and psychiatric conditions, and is more common in women than in men. The prevalence of people over the age of 65 years who have difficulty with distinct insomnia features involve the following: difficulty falling asleep - roughly 65%, waking during the night - 63%, waking up too early - 39%, poor sleep quality - 28%, using concomitant anticholinergic medication - 33%, using alcohol - 65%, and using diphenhydramine 15 days per month - 37%.

The negative effects of chronic insomnia and poor sleep extend over many domains for older people. Documented impacts of poor sleep in older adults include difficulty sustaining attention, slowed response time, impairments in concentration and memory, decreased ability to accomplish daily tasks, inability to enjoy social relationships, greater risk of falling, increased incidence of pain, reduced quality of life, risk of traffic accidents, increased consumption of healthcare resources and nursing home placement, and greater risk of premature death (Albert, Roth, Toscani, Vitiello & Zee, 2017; Vitiello, Moe & Prinz, 2002). Studies show that even one night's sleep deprivation activates gene expression patterns and peripheral blood mononuclear cells consistent with increasing accumulation of cell damage and increases susceptibility to dementia. These findings support the hypothesis that sleep deprivation may be associated with elevated disease risk because it promotes molecular processes involved in biological aging (Cribbet, et al., 2014).

Sleep apnea also is a common, but underdiagnosed condition that diminishes restorative sleep. Obstructive sleep apnea occurs secondary to obesity, anatomic factors, or drugs that may relax airway muscles (alcohol, sedatives); apneas and hypopneas are present in spite of intact respiratory drive. Central sleep apnea, on the other hand, is caused by reduced respiratory drive secondary to neurologic impairment, heart failure, or CNS depressants such as opioids. Untreated sleep apnea has wide ranging adverse consequences, including advancing HTN and coronary artery disease, insulin resistance, appetite stimulation and weight gain, cognitive impairment and psychopathology, cerebrovascular events, motor vehicle accidents, and death.

Indeed, poor sleep in general has a powerful influence on the immune system, augmenting the risk of infectious disease, cardiovascular and other inflammatory diseases, cancer, and depression. When sleep deprivation is prolonged or severe, such as several days of sleeplessness, the stress response is activated via the SNS and HPA axis (see Rodriguez, Dzierzewski & Alessi, 2015 for review). A focused evaluation of sleep, specifically insomnia, sleep apnea, and excessive movement, as well as daytime functioning, should be performed on every older adult in whom sleep disturbances are suspected. An analog sleep log, in which wake/sleep times are tracked as well as individualized variables such as alcohol and caffeine use, exercise, medication administration, electronics use, and pain, is imperative. Also necessary is an understanding of the role that depression and anxiety play in sleep initiation, maintenance, and early awakening. Not only can depression and anxiety prevent sleep onset, but

the ‘witching hours’ between midnight and 2am are prime time for physiologic arousal, anxiousness, and rumination.

Polysomnography is the gold standard for diagnosing sleep disturbances such as sleep apnea, parasomnias, micro arousals, and movement disorders, but not for diagnosing insomnia. For many people, trying to sleep in a lab environment, decked out with electrodes everywhere, is a recipe for sleeplessness. Insomnia is therefore best assessed at home with sleep actigraphy, easily available now with personal activity trackers or an actigraph from the sleep lab. The Insomnia Severity Index (ISI) is a quick way to quantify insomnia symptoms. This measure has high sensitivity and specificity (Morin et al., 2011). In addition, knowing the patient’s self-perception and expectations for sleep is crucial to formulating the intervention. It is not unusual for older adults to have high expectations for sleep duration based on what they learned decades ago as a child.

Less common sleep disorders in older adults include:

- Periodic limb movement disorder (PLMD) is a repetitive, rhythmic movement of the legs every 20-40 seconds and lasting about 2 seconds. It is usually comorbid with other medical conditions, such as diabetes, kidney disease, sleep apnea, and anemia. PLMD also is associated with a variety of psychotropic medications and may occur when withdrawing from benzodiazepines or barbiturates. PLMD should be part of the differential diagnosis when complaints include poor sleep, excessive daytime sleepiness, and bed partner complaints of the patient’s restless sleeping.
- Restless legs syndrome (RLS) is an uncomfortable sensation in the extremities such as burning, tingling, twitching, aching, or jerking, that generates an irresistible urge to move. RLS and PLMD are often confused with each other; interestingly, 80% of patients with RLS also have PLMD, but not the other way around. RLS can make going to sleep very difficult because of the relentless urge to move and discomfort while lying in bed. Complaints of insomnia and excessive daytime sleepiness are associated with RLS.
- REM Behavior Disorder (RBD) is a parasomnia in which patients physically act out their dreams because of a lack of normal REM muscle atonia. Although generally rare, RBD has been associated with dementia and multiple system atrophy. RBD can be prodromal to Parkinson’s disease as well, especially in males.
- Circadian rhythm sleep disorders may occur as part of aging. The sleep/wake circadian rhythm becomes less synchronized, less robust, and less responsive to external cues, resulting in inconsistent

sleep/wake periods across 24 hours. Typically, older adults will experience a circadian rhythm phase advance in which they fall asleep in the early evening and wake up between 3-5am. Common complaints are of waking in the middle of the night and being unable to return to sleep.

Finally, fatigue needs to be highlighted here. Lin et al. (2014) noted that high fatigability, a dysfunctional adaptation to fatigue, may lead to difficulties performing otherwise normal cognitive activities and may be related to pro-inflammatory reactivity. They measured IL-6 and frontally oriented cognitive processes in 55 community-dwelling individuals, age 75 years and older. Participants were classified into groups of low and high fatigability. High fatigue led to a pro-inflammatory state in older adults and was related to a decline in cognitive processes. Strikingly, there are small things that can be done that seem to make a difference in alleviating fatigue. Mid-day naps, for example, are associated with reduced blood pressure. Average day-night systolic blood pressures in people who take naps are different from those who do not, for the better. Two influential heads of state, Winston Churchill and Margaret Thatcher, must have suspected as much, as they did not want to be disturbed around 3:00pm so they could nap.

The Problem of Alcohol Misuse: The 2012 National Health Interview Survey (National Center for Health Statistics, 2014) highlighted the drinking patterns of adults ages 60 years and older: of those who reported drinking during the year before the survey, 50% of men and 39% of women drank almost daily; and 5.9% of the men and 0.9% of the women reported binge drinking once a month or more. Alcohol misuse or abuse is less common among older adults than in younger cohorts; for example, a SAMHSA survey (Center for Behavioral Health Statistics and Quality, 2016) showed that 2.1% of adults 65 years and older qualified as heavy drinkers (i.e., drinking 5+ drinks at the same occasion on at least 1 of the past 3 days) compared with 5.6% of adults ages 50-54 years. However, as more baby boomers reach older adulthood, with their generally greater acceptance of alcohol and recreational drug use, there likely will be a greater need for alcohol treatment services than in previous generations. Problematic alcohol use is not always obvious in older adults because the usual way of detecting addiction - through time lost from work, legal problems, or decreased participation in important social activities - may not be applicable to older adults who usually have fewer obligations in which aberrant behaviors or absenteeism would be observed.

The National Institute on Alcohol Abuse and Alcoholism (NIAAA) guidelines for adult men and women aged 65 years old and older defines 7 drinks in one week as moderate drinking. Older adults are likely to experience more problems with relatively small amounts of alcohol because of increased CNS sensitivity, slower metabolism, and a smaller volume of distribution. As such, drinking comparable amounts of alcohol produces higher and longer lasting blood alcohol levels in older vs. younger adults. Not surprisingly, people with an earlier onset of alcohol abuse (about 66% of elderly alcohol misusers) will likely have a more severe course of illness. They are mostly male, have more alcohol-related medical problems and psychiatric comorbidities, and are less well adjusted. Older adults with later-onset alcohol abuse usually have fewer medical problems because of their shorter exposure to alcohol. This group includes more women who are likely to have begun misusing alcohol after a stressful event, such as loss of a spouse or financial misfortune. Other risk factors for development of an alcohol use disorder at older ages include a family history of alcohol abuse, chronic pain, predisposition to mood or anxiety disorders, and decreased alcohol metabolism (Liberto & Oslin, 1995). As we suggested in Chapter 5, older adults who recently retire often abuse alcohol for at least a period of time with over several drinks/day.

The Problem of Polypharmacy: Before considering the ways in which Watch and Wait can be applied to the management of comorbid medical and behavioral health conditions, the issue of polypharmacy must be addressed. Polypharmacy, the prescribing of multiple (usually defined as 5 or more) medications at a time, is common in older adults and, although not always a problem, it can cause or contribute to neuropsychiatric symptoms including depression, anxiety, hyperphagia, cognitive fogging, and memory deficits. For example, anticholinergic drugs, some of which are routinely prescribed to older adults for urinary incontinence, nausea, cardiac problems, and irritable bowel syndrome, can cause or contribute to memory deficits and increase fall risk. Multiple drugs with anticholinergic side effects can bring about altered mental status that may look like psychosis or dementia. Also, most classes of psychotropic medications are associated with metabolic and appetite changes that cause weight gain. Stimulants, typically used in older adults for chronic fatigue and hypersomnia, may cause anxiety symptoms and malnutrition from decreased appetite. Anticonvulsants, frequently used for chronic physical pain and headache, are linked to depression. Some antihypertensive drugs (e.g., beta blockers) and statins for hyperlipidemia also produce depressive symptoms. Hormone replacement therapy can produce depressive

symptoms as well. Dopamine agonists that treat Parkinson's disease can cause depressive symptoms over time. When dosing is too high, dopamine agonists may induce paranoid reactions, compulsive behaviors, and mania. Corticosteroids can cause mood dysregulation, including mania. Over-the-counter drugs, such as Benadryl – an antihistamine that can cause mental status and mood changes, restlessness, drowsiness, dizziness, and blurred vision – should not be overlooked as sources of psychiatric side effects. The point is, the behavioral medicine practitioner must learn to routinely review the patient's medication list, including over-the-counter drugs and supplements, for compounds that may be contributing to or producing the behavioral health complaint. There should be a high degree of suspicion of iatrogenic origins when the symptoms in question present after starting a new medication. Chapter 8 includes a table of common medications that can cause or exacerbate depressive symptoms.

Over 75% of prescriptions for psychotropic medications are dispensed by physicians other than psychiatrists or by other prescribers such as nurse practitioners (Mark, Levit & Buck, 2009). It is therefore imperative for the behavioral medicine practitioner to ensure that these medications are being taken as prescribed, are effective, and are still needed. Medication literacy is frequently poor among older adults. It is not unusual, for instance, to learn that a patient is taking her antidepressant medication on an as-needed basis – in the same manner that she has taken benzodiazepines in the past for anxiety. Patients also may stop a medication without discussing it first with their prescribers. Suddenly stopping or skipping doses of certain antidepressant medications, such as venlafaxine, can cause *antidepressant discontinuation syndrome*, the symptoms of which are flu-like malaise, sleep disturbances, headache, anxiety, mood dysregulation, and/or abnormal movements. The unsuspecting acute care physician may start the patient on yet another medication to treat a presumed viral infection or similar physical malady, not knowing that the patient simply forgot to take his/her antidepressant medication.

Timing of medication administration also should be reviewed at each visit. Medications that cause drowsiness, such as some SSRIs and antipsychotics, should be taken at bedtime if possible to prevent daytime sleepiness, sluggishness, or risk of falling. Other psychotropics are activating, such as bupropion, and should be administered in the morning to prevent insomnia. Another common issue to highlight is lack of monitoring for medication effectiveness in PCCs. When PCPs initiate a trial of a psychotropic medication, they may not see the patient again for months, so the patient does not experience symptom relief because titration to a therapeutic level never occurred. Similarly, patients may

remain on a drug for years with no evidence that it is either effective or necessary. Finally, it is important to attend to those medications that have addiction potential, such as opioid pain relievers and benzodiazepines, as well as sedative-hypnotics, to ensure they are used appropriately and only for the short term. For all of these reasons, behavioral medicine practitioners should routinely review the medication list and ask about all psychotropic medications at each patient visit.

Polypharmacy Pearls for Older Adults

- Drugs may cause changes in mental status and mood dysregulation
- Drugs may cause hyperphagia or metabolic alterations that lead to weight gain
- Polypharmacy increases the potential for drug-drug interactions.
- Polypharmacy is independently associated with increased risk of an adverse drug event
- Polypharmacy is an independent risk factor for hip fractures in older adults, especially if one of the drugs is associated with falls risk (e.g., CNS active drugs)
- Polypharmacy increases the possibility of prescribing “cascades” i.e., an adverse drug effect is misinterpreted as a new medical condition and additional drugs are then prescribed to treat it.
- Polypharmacy can lead to medication mismanagement or nonadherence
- Nonadherence can be exacerbated by cognitive deficits resulting from polypharmacy.

Supporting Lifestyle Change and Stress Resilience in Primary Care

As mentioned earlier, convincing patients to change unhealthful habits is difficult for both the medical provider and for the patient. The limbic system’s inner voice (the proverbial “id”) is enormously powerful at convincing one that change isn’t worth the price of deprivation, of psychic or physical pain, of social isolation, of anxiety and worry. The “id” is stubborn – it vigorously defends its right to the pleasurable rush of dopamine and norepinephrine during that first drag off a cigarette, or when eating the first few bites of a juicy triple cheeseburger or a slice of red velvet cake. This has even been attached to the orbitofrontal lobe. Physical inactivity can be as problematic to overcome as smoking and overeating because our brains have evolved over the millennia to conserve energy.

Once obesity has set in, the innate drive to conserve energy through metabolic adaptation is extremely powerful (e.g., Fothergill et al., 2016).

A newer, but similarly stubborn, lifestyle habit is a reliance on electronics for socializing, gaming, or catching up with the day's news at bedtime. The practice staves off sleepiness because the bright light emanating from electronic screens emulates daylight, and the content increases cognitive and emotional engagement. In fact, there have been limited attempts to assess subjective information about light. Sleep diaries do not ask respondents about light in the sleeping environment, despite a growing appreciation of the potent effects of light on circadian rhythms and sleep health. Specifically, a bedroom fashioned with blackout curtains and other modifications may create scotopic conditions (with only rod activation), wherein an individual would only be able to see in shades of gray, if at all. By contrast, natural nighttime light from the moon and stars is typical of mesopic conditions, wherein both rods and cones are stimulated, allowing for the perception of color, even if it is not extremely vivid. Finally, under photopic conditions, as is typical of indoor architectural lighting, cones are stimulated and thus colors should be readily apparent. Each may indeed present its own problems.

Continuing, not only is it a struggle to get patients to initiate change, but helping patients maintain their resolve once they embark on lifestyle improvement is just as challenging. Habit change takes time and is far from a linear process; rather, it moves in fits of resolutions and small successes, mixed in with mistakes, moments of frustration, and floods of guilt (i.e., the proverbial "Superego") when the hand catches itself in the cookie jar. The health benefits of lifestyle change are usually slow to manifest, and Americans are impatient for fast results. Lifestyle change in and of itself can be stressful, especially when one is trying to change longstanding habits to address a serious medical condition before it becomes more debilitating – there is fear that time is running out and regret that change was not started sooner. All that said, often the best motivator for adopting a healthy lifestyle is a new diagnosis of a serious medical condition.

Addressing life adjustment problems and poor health behaviors individually in PC is a daunting task given that the clinic-wide need greatly outweighs the availability of personnel. However, with the PCMH model, stress management and lifestyle interventions such as weight loss, tobacco cessation, diabetes management, and sleep enhancement, can be sustained successfully through small group programs facilitated by the behavioral medicine practitioner, and supported by specialists in nutrition, physical therapy, diabetes education, clinical pharmacology, and other

medical subspecialties. The interventions described below are easily adapted for small groups. For example, The American Diabetes Association's Diabetes Prevention Program, which can be an individual or group intervention, has produced improved HbA1c's, blood pressures, and moderate weight loss in small and large group settings (Ali, et al., 2012; Brokaw, et al., 2014, 2015).

Dietary Issues/Weight loss: People in almost every region of the world could benefit from rebalancing their diets to eat optimal amounts of various healthful foods and nutrients, according to the Global Burden of Disease study that tracked trends in consumption of 15 dietary factors from 1990 to 2017 in 195 countries, published in *The Lancet* (GBD 2017 Diet Collaborators (2019). The study estimates that one in five deaths globally – equivalent to 11 million deaths – are associated with poor diet, and diet contributes to a range of chronic diseases in people around the world. In 2017, more deaths were caused by diets with too few whole grains, fruits, nuts, and seeds than by diets with high levels of trans-fats, sugary drinks, and high levels of red and processed meats. These low quality diets accounted for more than half of all diet-related deaths globally in 2017. Causes of deaths in this study included 10 million deaths from cardiovascular disease, 913,000 cancer deaths, and almost 339,000 deaths from type 2 diabetes. Deaths related to diet have increased from 8 million in 1990. Poor diet and high sodium intake, then, appears responsible for more deaths than other risk factors. The GBD 2017 study also highlights the need for comprehensive interventions to promote the production, distribution, and consumption of healthful foods across all nations.

Excessive weight also contributes to poor health. In general, recommended goals for sustained weight loss can be as little as 3%-5% of body weight to see a reduction in cardiovascular risk factors, but greater weight loss (i.e., 5%-10% of base weight) is likely to produce greater health benefits. Remember that 1 pound of fat has about 1 mile of vasculature supporting it, so the loss of 1 pound of fat can make a positive change to heart health. The preponderance of evidence indicates that the combination of caloric restriction (dietary intake of 1200-1500kcal/day for women and 1500-1800kcal/day for men), less reliance on simple carbohydrates in the diet, and increased physical activity that is of moderate to high intensity, should produce a sustained positive - and sensible - outcome (Kushner & Ryan, 2014). That said, there are recent data to suggest that less emphasis on absolute calorie intake and more focus on food quality (i.e., elimination of processed, energy-dense and “junk” foods/drinks with high glycemic and carbohydrate indices, increase in healthful fats), combined with regular exercise, will lead to sustained

weight loss with fewer cravings and less anguish overall (see Mancini, Filion, Atallah, & Eisenberg, 2016; Tobias et al., 2015 for reviews).

Changing the contents of one's diet as well as meal frequency and portion control are essential components of a *behavioral* weight loss intervention that must be adopted as a permanent lifestyle change and not as a short term 'diet.' Fad diets are doomed to failure and should be discouraged because they are not sustainable. Behavioral interventions are best implemented prior to considering weight loss medications or bariatric surgery because the goal is to help the patient learn new habits to support more healthful, rational eating practices and high quality food choices. As noted above, diet quality matters: dietary patterns that follow the Mediterranean or DASH diets are not only associated with reduction in chronic disease risk, but also with a 30% reduction in risk for depression (Estruch et al., 2013; Psaltopoulou et al., 2013). In fact, judicious practice of the DASH or Mediterranean resulted in longer telomeres, a marker of overall health (Leung et al., 2018). Self-monitoring via smartphone apps, internet sites, or an analog food/activity log also is a vital aspect of any behavioral weight loss intervention.

Best evidence to date indicates that older overweight and obese adults should be prescribed an intervention that reinforces both dietary changes and exercise. Weight loss programs without an exercise component, specifically strength training, have been found to worsen sarcopenia and bone mineral density loss, thus increasing future risk of frailty (Batsis et al., 2017). Strength/resistance training mitigates against sarcopenia and osteopenia/osteoporosis while improving mobility, flexibility, and overall physical functioning.

Exercise: The American Academy of Family Practitioners (Elsawy & Higgins, 2010), the American College of Sports Medicine (Chodzko-Zajko et al., 2009), the American Heart Association/American College of Cardiology (2014), and the US Dept of Health and Human Services (Piercy et al., 2018) have now published physical activity guidelines that include older adults. General recommendations are for 150-180 minutes/week of moderate intensity aerobic exercise, which can be accomplished in as little as 10 minute segments throughout the day to achieve a beneficial effect. Aerobic physical activity should be combined with muscle-strengthening activity twice per week.

Moderate intensity exercise is defined as "an intensity that causes noticeable increases in heart rate and breathing." (Thompson, et al., 2013, pp. 216). Vigorous walking (100-130 steps per minute), jogging, dancing, or playing an active sport such as tennis are good moderate intensity recommendations for older adults. The CDC offers a table for download

that has examples of moderate and vigorous activities, including individual exercise, sports, housework, and occupational activities (see <http://www.cdc.gov/physicalactivity/basics/measuring/index.html>). Also recommended is strength or resistance training that addresses all muscle groups, along with at least 10 minutes of flexibility and balance activities, such as yoga or tai chi, at least two days/week.

A simple health screening such as the Physical Activity Readiness Questionnaire (PAR-Q; Tharrett & Peterson, 2012) can be given prior to recommending exercise to check for the presence of cardiovascular, pulmonary, renal, and metabolic risk factors that might require a medical clearance (see Pescatello, Riebe & Arena, 2013 for exercise risk classification decision tree). Hoffman and colleagues (2016) conducted a comprehensive review of the scientific literature on exercise prescriptions for patients with chronic illnesses, including the common conditions discussed in this chapter. They emphasize the importance of knowing how a particular exercise prescription can help, but also knowing how a patient's fears and beliefs shape motivation and likelihood of adherence. For example, patients who have chronic musculoskeletal pain or osteoarthritis often have the mistaken belief that exercise will add "wear and tear" to the body and so avoid physical activity. The medical term applied here is kinesiophobia, fear of movement usually as a result of pain problems. Evidence indicates no contraindications to exercise in this group and the only adverse events reported have been an occasional increase in pain or pain in other areas. Thus, to promote long term engagement in exercise for such patients, the authors recommend an education component that addresses psychological barriers to activity, such as catastrophizing, poor self-efficacy, and fear of further injury. Goal setting and setting a paced activity schedule are common behavior therapy interventions that work very well with this type of patient. In contrast, patients with COPD will benefit from referral to a pulmonary rehabilitation program where an exercise program can be designed to reduce breathlessness while improving exercise capacity.

Implementing Combined Weight Loss and Exercise Programs in the PCC: As mentioned, obesity in older adults is best tackled with a combination of weight loss and exercise to prevent or treat sarcopenia and osteopenia/osteoporosis. An intensive lifestyle intervention is perhaps best implemented in the PCMH where a close collaborative relationship can be maintained between the behavioral medicine specialist, PCP, nursing staff, and allied health staff. Vitals are easily gotten (especially weights), labs can be ordered, and expertise and creative problem solving is often readily available. There are a number evidence-based programmatic options that address weight loss and exercise in older adults.

Some examples:

- The American Diabetes Association's Diabetes Prevention Program (DPP), which has been running for over 2 decades, targets adults ≥ 65 years, and is designed to delay conversion (or worsening) of T2D (see Diabetes Prevention Program Research Group, 2015, for review). The DPP's components are: 1) a minimum 7% weight loss goal with emphasis on self-monitoring via weigh-ins and calorie tracking; 2) 150 minutes/week physical activity, typically vigorous walking; 3) 16 session core curriculum sequenced over 24 weeks in 30-60 minute sessions; 4) ability to tailor program to meet individual medical and cultural needs; and 5) monthly or telephone or mail/email contact over the maintenance period. The DPP offers a "toolbox" of activity videos, meal plans, enrollment in exercise classes, and vouchers for healthful food samples to assist patients who are struggling in the program. See <http://www.bsc.gwu.edu/dpp/manuals.html#doc>
- Look AHEAD (Action for Health in Diabetes; the Look AHEAD Research Group, 2014) is a modification of the DPP. It is an intensive group lifestyle intervention for people with diabetes, HTN, and other co-morbid conditions. Over the 8 year trial, participants maintained more than half of their weight loss. Nearly 40% of participants who lost $\geq 10\%$ of initial weight at year 1 maintained this loss at year 8. Look AHEAD is designed to be implemented by psychologists, registered dietitians, and exercise specialists, utilizing cognitive behavioral therapy, problem solving, motivational interviewing, and cultural tailoring. Look AHEAD is structured as follows: 1) group and individual sessions with the interventionist for the first program year, and less frequent individual and group sessions (booster sessions) afterward that reinforce accountability; 2) a prescribed diet of 1,200-1,800 kcal/day (depending on initial body weight) with $\leq 30\%$ of calories from fat ($< 10\%$ from saturated fat) and $\geq 15\%$ of calories from protein. Structured meal plans and meal replacements are prescribed to meet these goals; 3) At least 175 minutes/week of moderate intensity physical activity, increasing to ≥ 200 minutes/week once that goal is met. Exercise is commonly unsupervised brisk walking; 4) participants keep daily logs of their food intake, physical activity, and other targeted behaviors. See: <https://www.lookaheadtrial.org/index.cfm>
- The American Heart Association/American College of Cardiology/Obesity Society published a comprehensive document: *2013 Guidelines for the Management of Overweight and Obesity in Adults*. This document reviews and rates a number of evidence-based interventions, including intensive lifestyle modification, pharmacotherapy for obesity, and

bariatric surgery options, and provides an algorithm for decision-making. The intensive lifestyle intervention components should be implemented for ≥ 14 sessions over 6 months and consists of: 1) a moderately reduced-calorie diet; 2) a program of increased physical activity; and 3) use of behavioral strategies to facilitate adherence. The expected 6 month outcome from such an intervention is 5%-10% loss of base weight. After 6 months, the individual is likely to see smaller losses for up to 2 years or some weight regain. Booster sessions, either in the clinic or by telephone, are recommended to help patient maintain new lifestyle habits, smaller portions, and healthful food choices, as well as regular exercise. See: http://circ.ahajournals.org/content/circulationaha/129/25_suppl_2/S102.full.pdf

- Agency for Healthcare Research and Quality's Staying Healthy Through Education and Prevention (STEP) is an exercise program with no diet component. Given that older adults can be particularly resistant to exercise, STEP is designed to improve compliance via a manualized CBT 9 session curriculum to help older adults examine and overcome barriers to physical activity. See <https://www.ahrq.gov/professionals/education/curriculum-tools/stepmanual/stepcoach1.html>

Implementing a Sleep Enhancement Intervention in the PCC: Best practice for the treatment of the most common sleep complaint, insomnia, is psychoeducational and behavioral. Treatment for late-life insomnia may include pharmacologic agents such as hypnotics and sedatives, but it is not a first line intervention, and indeed is no longer seen as safe for even a short period of time. That said, a substantial portion of all hypnotic prescriptions go to elderly patients (American Geriatrics Society, 2015). Older adults are particularly vulnerable to the effects of hypnotics and sedatives, and run the risk of nighttime disorientation if awakened, increased fall risk, daytime altered mental status, and respiratory depression leading to death during sleep. Recent studies on the adverse effects of sedatives and hypnotics are deeply concerning as they show an association between these drugs and early all-cause mortality, cancer, depression, and infection, regardless of age (see Kripkea, 2016, for review). Of note, risk of early mortality is even higher in obese individuals prescribed sleep aids. These patients are more likely to have sleep apnea and sleep aids can prolong an apneic event because of respiratory depression, leading to death by respiratory arrest. As a result of such

strong evidence against sleep aid use, many physicians are now disinclined to prescribe hypnotics or sedatives for insomnia.

Cognitive behavioral therapy for insomnia (CBT-I) is the gold standard intervention for insomnia, whether it is primary insomnia or insomnia secondary to neuropsychiatric causes or pain (Manber et al., 2008; Morin et al., 2006; Geiger-Brown et al., 2015). That said, many older adults with chronic insomnia have been on sleep medications for years, if not decades, and may be quite resistant to their physician's efforts to move them away from medication toward behavior change. In these cases, the behavioral medicine specialist and PCP must collaborate closely to wean the patient off of sleep medication while CBT-I skills are being taught and practiced. Otherwise, the patient is unlikely buy into the intervention and may seek another physician to continue being medicated. CBT-I addresses distorted cognitions and maladaptive behaviors that contribute to and maintain insomnia. The intervention can be administered in individual or group format over 4-6 sessions. Before enrolling patients in CBT-I, however, it is important to ferret out other potential sources of insomnia, such as poorly treated pain, untreated reflux, taking activating medications at night, and disruptive bed partner behaviors. Having the patient keep a sleep log can help identify sleep disruptors beyond the scope of CBT-I.

There are numerous manualized CBT-I protocols to choose from, and all include: 1) stimulus control procedures; 2) sleep hygiene guidelines; 3) relaxation techniques; and 4) a lot of psychoeducation (see <https://sleepfoundation.org>). It is important to determine whether anxiety or worry contribute to insomnia because CBT-I alone is unlikely to address anxiety or worry adequately. Traditional CBT interventions incorporated into CBT-I work well here. There also is emerging evidence that Acceptance and Commitment Therapy (ACT) may be more effective in these cases (Daly-Eichenhardt, et al., 2016).

The most common treatment for sleep apnea is CPAP (continuous positive airway pressure), although treatment noncompliance is high for a variety of reasons. Other options include mandibular advancement and tongue retaining devices, position pillows, and hypoglossal nerve stimulators, which stimulate the nerves to the tongue and related muscles to keep the airway open. For the most severe forms of obstructive sleep apnea, a tracheostomy allows for normal breathing during the day, and an open airway while asleep. The risks of a permanent tracheostomy make this intervention a last consideration for the OSA patient.

Managing Insomnia

Step 1: Evaluation

- Evaluate sleep and daytime symptoms and comorbid conditions.
- Optimize treatment of comorbid conditions (e.g., depression, anxiety, pain).

Step 2: Initial treatment

- Acute insomnia diagnosis: consider short-acting hypnotic (e.g., temazepam or zolpidem 3-4 nights weekly for 3-4 weeks), then taper and discontinue.
- Chronic insomnia disorder diagnosis: implement CBT-I.

Step 3: Evaluate response and continue treatment

- Evaluate sleep and daytime symptom response.
- Address continued symptoms with CBT-I: consider combined treatment using a drug appropriate for sleep onset or sleep maintenance symptoms.
- Address continued symptoms with pharmacotherapy: consider switching class of hypnotic (e.g., benzodiazepine or benzodiazepine receptor agonist to doxepin, trazadone, ramelteon, or suvorexant).

Step 4: Evaluate response and, if symptoms continue, re-evaluate diagnosis

- Reevaluate and treat comorbid disorders.
- Evaluate other contributing factors (e.g., life events, new medical or psychiatric disorder) and address with psychosocial, behavioral, or medical interventions.

Step 5: Treatment-resistant insomnia disorder diagnosis

- Refer to sleep specialist for evaluation of other sleep-wake disorders, including sleep apnea.

Step 6: Monitor

- Monitor for long-term treatment response and sequelae such as depressive or anxiety disorder, substance use disorder, or neurodegenerative disorder.

-- Buysse et al., 2017

Implementing a Tobacco Cessation Program in the PCC: Simply stated, the combination of smoking cessation counseling and pharmacotherapy is the most effective way to help people quit tobacco. Alternative interventions such as acupuncture and hypnosis do not work and should not be encouraged. Counseling is best delivered in multiple sessions; programs offering 4 or more sessions have been shown to improve abstinence rates (Fiore et al., 2008). A group therapy format (which we use) can be as effective as individual counseling, especially in PCCs, and is definitely more effective than self-help approaches (Fiore et al., 2008; Stead & Lancaster, 2005). Group therapy is the treatment of choice in the PCC because it conserves behavioral health resources and makes prescribing and monitoring medications more efficient. Whether the format is individual or group, the most successful counseling approaches combine problem-solving skills training, motivational interviewing, and social support as part of the treatment. An excellent example of a smoking cessation counseling intervention using the 5A's framework (see below) can be found in Hunter, Goodie, Oordt & Dobmeyer (2017). Additionally, the American Lung Association, American Heart Association, CDC, HHS, and various state health agencies all offer guidelines to create an evidence-based tobacco cessation program. To be maximally effective, a tobacco cessation program should be staffed with a behavioral medicine specialist and prescriber to improve delivery and monitoring of pharmacotherapy in the context of the counseling program. We have found that the availability of post-program walk-in counseling or 'booster sessions,' with access to pharmacotherapy after a lapse, is very effective in helping patients remain tobacco free over the long term.

A word about smoking cessation pharmacotherapy or Quit Aids: the evidence is indisputable that pharmacotherapy significantly increases the likelihood of quitting tobacco when combined with a smoking cessation counseling program. FDA-approved Quit Aids include nicotine replacement therapy (NRT: transdermal patch, gum, lozenge, nasal spray, inhaler) and the drugs varenicline (Chantix) and bupropion (Zyban, Wellbutrin), with varenicline achieving the best abstinence results of the Quit Aids (Anthenelli et al., 2016). All of these agents are approved for older adults, but a modicum of caution is advised for individuals with a history of cardiac events (FDA Drug Safety Communication, 12/12/2012), seizures, and serious neuropsychiatric conditions, including alcohol misuse, as there is a potentially increased risk of a new cardiac event and low threshold for intoxication with varenicline, and a potentially increased risk of a new neuropsychiatric event or seizure with bupropion (Anthenelli et al., 2016; FDA Drug Safety Communication, 3-9-2015). ●

note, the black box warning for varenicline and bupropion regarding risk of suicidal ideation was removed by the FDA in 2016 because the risk of a neuropsychiatric adverse event for non-psychiatric patients is not elevated over placebo (FDA Drug Safety Communication, 12-16-2016). Caution also should be exercised when prescribing NRT as people who have unstable cardiovascular disease are at higher risk of experiencing a new cardiovascular event. In sum, the patient and prescriber must weigh the long term benefits of quitting tobacco with pharmacotherapy over the small risk of an adverse event while taking it. Furthermore, patients with cognitive impairment should be assisted by a caregiver to ensure that the Quit Aid is taken as prescribed (i.e., there are scheduled dosing changes with NRT and varenicline) and are actually purposefully cutting back on their tobacco use during their Quit period.

A word should be said about using electronic cigarettes (i.e., e-cigarettes, vaping, juuling) as a Quit Aid. There are insufficient data at this time to recommend e-cigarettes to quit tobacco. That said, NICE has published a tobacco cessation policy update (NICE, 2018) that advises care providers to discuss with their patients who smoke that e-cigarettes are substantially less harmful than cigarettes but are not risk free. Our clinical experience with people who quit tobacco by switching to e-cigarettes suggests that patients must be very strict about titrating down on the nicotine content of the e-cig “juice” or they will find their nicotine use increase. Note that e-cigarette use is not benign healthwise. The CDC recently published a health advisory (CDC Health Alert Network, August 30, 2019), warning of a heightened risk of severe pulmonary disease secondary to the use of e-cigarette devices, liquids, refill pods, and cartridges.

Addressing Alcohol Misuse in the PCC: Screening for an alcohol use problem can be done at any doctor’s visit with the World Health Organization’s Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993) or the Short Michigan Alcohol Screening Test (SMAST-G; Blow et al., 1992), which includes items unique to older problem drinkers. Brief intervention techniques are effective for many problem drinkers (O’Connor et al., 2018; Strobbe, 2014). For example, the clinician can bring up the patient’s drinking with personalized feedback from the AUDIT or SMAST-G, then guide the conversation toward cutting back or quitting alcohol using nonjudgmental language, psychoeducation, and motivational interviewing principles to help the patient gain awareness, insight, and motivation for behavior change. Both the National Institute on Alcohol Abuse and Alcoholism (2005) and SAMHSA (2015) offer brief intervention programs appropriate for PCCs and behavioral health settings.

As part of the initial screening, older adults should be asked whether they have had 4 or more drinks on any one occasion during the past year. This single item on the substance abuse screeners tends to be reliable in the general population when identifying alcohol misuse, but it has not been examined to any degree with older adults. Older adults are more likely to deny or minimize substance-related problems than younger adults because of social stigma associated with substance misuse. Patients identified as needing more assistance than a brief intervention should be referred to specialty treatment. The SAMHSA Substance Abuse Treatment Services Locator (www.findtreatment.samhsa.gov) provides pertinent information about state-licensed substance use treatment facilities throughout the United States.

Interventions short of an inpatient rehabilitation are a special problem. As much as possible, brief, personalized interventions should be considered first. Many older adults may drink at levels considered risky or problematic, yet they do not meet criteria for substance abuse or dependence and are unlikely to seek out formal alcohol treatment. These adults, however, often come to the attention of health or mental health providers. In the context of brief interventions, motivational interviewing techniques often are incorporated to elicit reasons that the older patients may have to cut back on drinking. Motivational interviewing also can be used as a therapeutic technique. A small number of studies have examined brief intervention outcomes among older adults in which interventions were delivered by PCPs. The Florida Brief Intervention and Treatment for Elders Project was a large three-year, state-funded pilot program that delivered brief substance misuse interventions to older adults. Prescription medication misuse, that is either using medications faster than prescribed, difficulty remembering how much to take, not taking medications, borrowing someone else's prescription, or saving older medications was the most prevalent substance use problems, followed by alcohol, over-the-counter medications, and illicit substances (Shonfeld et al., 2010).

Assessment

A standard holistic medical evaluation involves the tasks listed below, and taps other health care providers in a collaborative team approach. As we cited previously, the medical evaluation of Watch and Wait involves a complete medical history, standard labs (TSH, complete blood count (CBC), serum B₁₂, folate, complete metabolic panel, and, if at risk, testing for sexually transmitted diseases (e.g., human immunodeficiency virus, syphilis, chlamydia), assessment of multiple cognitive domains. Include

motor function, a MRI or CT applied (especially for dementia of recent onset and rapid progression; younger onset dementia (<65 years of age); history of head trauma; or neurologic symptoms suggesting focal disease), and our issues (ADL and IADL functioning; assessment for depression; and review of medications for those that can adversely affect cognition)

The 5 A's of Behavioral Health Assessment and Intervention in Primary Care

The beauty of Watch and Wait is that it embraces brief assessments and interventions that can be applied successfully in the PCC. The 5A's model (Goldstein, et al., 2004; ●Donohue et al., 2006) allows for assessment and intervention across a range of medical problems seen in PCC with behavioral health components. The 5A's approach is iterative with each visit and enables the patient to leave with a plan of action that is updated at subsequent visits. Watch and Wait guides the action plan as well as the patient's progress toward his/her goals.

<i>The 5A's of Assessment and Intervention for Behavioral Health in Primary Care</i>	
<i>Assess</i>	Gather information about physical symptoms, emotions, thoughts, behaviors, and environmental context to determine variables associated with patient's symptoms and functioning. ●On the basis of patient's values and degree of control over the environment, determine what is changeable to improve functioning.
<i>Advise</i>	Describe to patient the options for intervention and the anticipated outcomes based on the data gathered during assessment.
<i>Agree</i>	Patient decides on a course of action based on the options discussed. ●or patient rejects the options presented, and generates alternatives. Patient also may defer deciding on an action plan to discuss options with family.
<i>Assist</i>	This is the formal intervention. Help patient implement behavior change through learning new information, developing new skills, and/or overcoming environmental or personal barriers. Reinforcement and occasional retrenchment can occur over several visits – Watch and Wait
<i>Arrange</i>	The follow-up plan is determined here. Consider the need for involvement of other team members such as the PCP, Nutritionist, Clinical Pharmacist, Case Manager, or medical specialist. The focus of the next behavioral health appointment also should be set (e.g., homework).

The 5A's enable the behavioral medicine specialist to be responsive to a patient's needs at any given visit. Therefore, lack of improvement or worsening status indicates that the 5A's need reexamining. Are additional or different medical, psychological, behavioral, and/or social interventions needed? So at each visit, the behavioral medicine specialist will consider whether the degree of progress, or lack thereof, necessitates a readjustment of Watch and Wait to step up care. The action plan that emerges from the 5A's approach should be concise, personalized, and integrated into the patient's overall medical care plan. The action plan must be reviewed with the PCMH team, to include sharing ancillary information that may impact the patient's follow through, so all team members can monitor and coach the patient at subsequent visits.

Watch and Wait Evaluation and Intervention for Modal Chronic Diseases

Prediabetes and Diabetes Management: T2D, a chronic problem of increasing insulin resistance, is currently defined as a glycosylated hemoglobin level (HbA1c) of $\geq 6.5\%$ or fasting blood glucose of $\geq 126\text{mg/DL}$. A diagnosis of prediabetes is the equivalent of a big red flag – attention must be paid by both the patient and healthcare team to prevent conversion to T2D. Prediabetes is diagnosed when an HbA1c is 5.7% - 6.4%. It designates a period in disease progression when lifestyle change through healthful eating, increased physical activity, weight reduction, smoking cessation, and elimination of alcohol can make a huge difference. The prevalence of older adults with prediabetes is about 51%, and T2D is almost 26% (American Diabetes Association, 2016; CDC, 2014). Conversion to T2D is unfortunately common for a number of reasons, including: patient's reluctance to change longstanding lifestyle behaviors and experiencing difficulty when trying to do so; patient's and family's lack of appreciation for the seriousness of the disease often due to cultural beliefs about the inevitability of having what is known in many parts of the US as "the Sugar;" inadequate diabetes education; poor access to ongoing healthcare, absence of a team approach to disease management; and provider bias against using metformin, the most commonly prescribed medication for early management of T2D, to treat prediabetes (Moin, et al., 2015).

Current standards for the evaluation and management of T2D recommend a collaborative care model that includes a physician, dietician, pharmacist, and behavioral medicine specialist. In developing a management plan, patient and family preferences should be taken into

account along with consideration of the patient's age, school/work/living situation, cultural factors, eating patterns, physical activity level, medical comorbidities, health priorities, and presence of diabetes complications (ADA, 2016). The American College of Physicians (ACP) has issued an updated guidance statement recommending an HbA1c level between 7% and 8% in most patients with T2D. This new guidance can be found in the *Annals of Internal Medicine* (Qaseem et al., 2018). The authors evaluated six sets of current guidelines promulgated by other organizations and reviewed five clinical trials on which those guidelines are based (ACCORD, ADVANCE, VADT, and both UKPDS trials). The resulting four key statements were published:

ACP 2018 Guidelines for Management of T2D

- Clinicians should personalize goals for glycemic control in patients with T2D based on a discussion of the benefits and harms of pharmacotherapy, patient preferences, patient general health and life adjustment.
- Clinicians should aim to achieve an HbA1c level between 7% and 8% in most patients with T2D.
- Clinicians should consider de-intensifying pharmacologic therapy in patients with T2D who achieve HbA1c levels less than 6.5%.
- Clinicians should treat patients with T2D to minimize symptoms related to hyperglycemia and avoid targeting tight HbA1c levels in patients with a life expectancy less than 10 years due to advanced age (80 years or older), residence in a nursing home, or chronic medical conditions (i.e., dementia, cancer, end-stage kidney disease, or severe COPD/heart failure) because the harms of overly tight glycemic levels outweigh the benefits.

The American Academy of Family Physicians (Koenigsberg & Corliss, 2017) recommends that lifestyle modifications for diabetes management should aim to achieve 7% weight loss and moderate-intensity physical activity of at least 150 minutes per week. The ADA has put out the following guidance for behavioral health involvement in diabetes care:

The ADA Behavioral Health Guidelines for T2D

- The patient's psychological and social situation should be addressed in the medical management of diabetes.
- Behavioral health screening and follow-up should include: attitudes about the illness, expectations for medical management and outcomes, affect/mood, general and diabetes-related quality of life, resources (financial, social, and emotional), and psychiatric history/status.
- Routinely screening for depression, diabetes-related distress, anxiety, eating disorders or disordered eating behaviors.¹
- Older adults (aged ≥ 65 years) with diabetes should be evaluated cognitively and screened for depression screening, and treat appropriately.
- Patients with comorbid diabetes and depression should receive a stepwise collaborative care approach for the management of depression.

Opportunities for behavioral health screenings occur at the time of prediabetes or diabetes diagnosis, during medical follow-up appointments, with new onset of complications, or when problems with glucose control or medical self-management arise. Patients are likely to exhibit increased distress at diagnosis, when their medical status changes, when the need for intensified treatment is identified, and when complications are discovered. Diabetes-related distress is a significant negative reaction to having to manage a complicated and demanding chronic disease. High levels of distress are linked to medication nonadherence, higher HbA1C, lower self-efficacy, and poor eating behaviors and exercise. In a recent multi-national study of the psychosocial outcomes of individuals with diabetes (Nicolucci et al., 2013), diabetes distress was reported by 45% of the 8500 participants, but only 24% reported that their healthcare team asked them how diabetes affected their lives. Note that diabetes distress is different from depression but can co-occur with depression, so both conditions must be assessed.

¹ Disordered eating behaviors represent a spectrum of cognitions and actions that can include distorted body image, bingeing, late night eating, excessive calorie restriction, purging for weight loss via laxatives, excessive exercise, vomiting, etc. The prevalence of disordered eating behaviors in older adults is unknown.

<i>Watch and Wait Assessment for Type 2 Diabetes</i>			
<i>Psychopathology Screen</i>	<i>Cognitive Screen</i>	<i>Life Adjustment Screen</i>	<i>Lifestyle Problems</i>
PHQ-9 or GDS	MoCA	SDSCA-6 ²	AUDIT/SMAST-G
PAD-5 ³	MMSE	PROMIS ⁴	Eating habits review
GAD-7 or GAI ⁵	Mini-cog	FAQ	Activity habits review
BES ⁶			Diabetes Eating Problems Survey ⁷
MBMD as needed			ISI/ESS/SBQ ⁸ Tobacco use

To summarize, the behavioral medicine specialist's role in the case formulation and management plan for prediabetes and diabetes is to provide the healthcare team information on: psychiatric history and current status; psychosocial challenges and life adjustment problems; presence of lifestyle problems such as poor eating habits (e.g., consumption of energy dense foods, high carbohydrate intake, reliance on fast foods), eating disorders/disordered eating behaviors, sedentary behavior, tobacco/alcohol/drug use; patient's and family's degree of understanding of the disease and identification of preventable and modifiable risk factors; cognitive status; psychotropic medication concerns, especially highlighting medications associated with weight gain/metabolic syndrome; and proposed behavioral health interventions, including targets for psychoeducation, brief problem-focused interventions, and specialty consultations. Much of this information can be gathered in the clinical interview. It is very important to include a knowledgeable family member such as the spouse/partner or adult child in the intake to not only gain collateral input about life adjustment, psychopathology and lifestyle concerns, but also to assess degree of social

² Summary of Diabetes Self-Care Activities measure (Toobert et al., 2000)

³ Problem Areas in Diabetes-5 (Diabetes Distress) (Nicolucci et al., 2013)

⁴ Patient Reported Outcome Measurement Information System (Cook et al., 2016)

⁵ Geriatric Anxiety Inventory (Pachana, 2007)

⁶ Binge Eating Scale (Gormally et al., 1982)

⁷ Diabetes Eating Problems Survey (Antisdel et al., 2001)

⁸ Insomnia Severity Index (Bastien et al., 2001), Epworth Sleepiness Scale (Johns, 1991), Stop-BANG Quest. for OSA (Chiu et al., 2016)

support and the ability and motivation of the support group to participate positively in the patient's care.

Cardiovascular Disease: Hypertension (HTN) is the most important risk factor for death secondary to cardiovascular disease (CVD: coronary artery disease, cardiac arrhythmias, heart failure) and myocardial infarction (MI). Almost 65% of adults 60 years old or greater have HTN (Yoon, Fryar & Carroll, 2015). The progression of CVD is often related to HTN, pre-existing diabetes, and hyperlipidemia, so treatment must be in the context of staying on top of these other chronic diseases. New guidelines for the diagnosis of HTN in adults, including community dwelling older adults, are a persistent systolic BP of ≥ 130 mm Hg or diastolic BP of ≥ 80 mm Hg, and goal of less than 150 mm Hg. (Qaseem et al., 2017; Welton et al., 2017). Although BP targets can differ if HTN is complicated by other medical conditions, in most cases the adverse effects of anti-hypertensive medications (hypokalemia, hyperkalemia, hyponatremia, hypotension, dizziness, depression, headache, edema, erectile dysfunction, and cough) outweigh the benefits of systolic pressures of 120 mm Hg or lower. Behavioral management is a primary intervention for both HTN and CVD. Opportunities for behavioral health screenings occur at the time of HTN/CVD diagnosis, during medical follow-up appointments, with new onset of complications, and after a major acute coronary event occurs.

Behavior change recommendations for HTN/CVD encompass eight areas⁹

1. Weight reduction for all overweight and obese individuals
2. Exercise that includes both strength training and 30-60 minutes of moderate intensity aerobic activity 4-7 days/week
3. Dietary change to DASH, Mediterranean, or similar diet that emphasizes fruits, vegetables, low fat dairy, lean meats and fish, soluble fiber, and plant-based proteins that are lower in saturated fat and cholesterol
4. Increase potassium in patients not at risk for hyperkalemia
5. Alcohol use reduction to ≤ 2 drinks/day
6. Tobacco cessation
7. Sodium intake reduction to < 2000 mg per day
8. Maintain medication compliance
9. Stress management interventions as needed and should include relaxation techniques

⁹ Leung et al., 2016; USPSTF, 2012

Psychiatric conditions are known to increase CVD risk as well as complicate CVD. Depression, in particular, is linked to CVD. This relationship is bidirectional and forecasts a poor prognosis. Depression early in life is an independent risk factor for the development of CVD later on. Further, approximately 20% of cardiac patients develop major depression for the first time after a major adverse cardiac event (Thombs, et al., 2006). Depression overlaid onto CVD is the strongest predictor of death in the first decade after cardiac diagnosis and therefore should be continually assessed in the clinic (May, 2017). The underpinnings of the relationship between depression and CVD are not entirely clear, but scientists highlight both behavioral factors in depression such as low motivation for self-care (e.g., medication nonadherence, tobacco use, poor diet, lack of exercise) and biologic factors (autonomic nervous system dysfunction, elevated cortisol levels, and elevated markers of inflammation) as reciprocal causes (Carney et al., 2016).

Anxiety similarly affects about 20% of CVD patients and is especially problematic for about 80% of patients after a major adverse cardiac event (Trumper & Appleby, 2001). Anxiety is common in atrial fibrillation, a disorder of cardiac electrical conduction. Both conditions share a number of symptoms (palpitations, shortness of breath, chest pain or discomfort, irregular or accelerated heartbeat, lightheadedness or dizziness, and fatigue) and the ambiguity of symptom source can produce heightened distress and reduced engagement in healthful activities. Patients who receive implantable cardioverter defibrillators (ICD) to control ventricular fibrillation are at especially high risk of developing event-related anxiety, PTSD, avoidance behaviors, and depression secondary to an ICD firing.

Research to date has shown mixed results regarding the efficacy of psychotherapy for cardiovascular disease, in part because of the variety of interventions tested (CBT, ACT, psychodynamic therapies, etc.) as well as the variety of delivery systems used (e.g., in-person vs. telephone counseling vs. tele-monitoring, individual therapy vs. group counseling). The beneficial effects of cognitive-behavioral interventions are mainly psychological with reductions in depression and anxiety symptoms, whereas cardiovascular morbidity and mortality are reduced slightly at best (Domelas, 2012; Pedersen et al., 2017). Psychodynamic therapies have not been found to be effective in relieving psychiatric symptoms associated with CVD, and there are little data to date on interventions that use ACT (Tan & Morgan, 2015). On the other hand, comprehensive cardiac rehabilitation programs that focus on changing health behaviors are impactful; mortality can be reduced by an estimated 35% with smoking cessation, by 25% with increased exercise, by 45% with positive

dietary changes, and by 20% with moderate alcohol use (Iestra et al., 2005). Stress management with relaxation training, medical family therapy, individual psychotherapy, substance abuse treatment, and cardiac rehab support groups should be part of a comprehensive cardiac rehabilitation program. There must be good communication between the cardiac rehab program and PCMH so that behavioral changes made while rehabbing can be supported and monitored by the behavioral medicine specialist in the PCC. The behavioral medicine specialist should be comfortable with Motivational Interviewing in cases where interest in healthful routines begins to wane as the cardiac scare dims in memory.

<i>Watch and Wait Assessment for HTN/Cardiovascular Disease</i>			
<i>Psychopathology Screen</i>	<i>Cognitive Screen</i>	<i>Life Adjustment Screen</i>	<i>Lifestyle Problems</i>
PHQ-9 or GDS	MoCA	PROMIS	AUDIT/SMAST-G
GAD-7 or GAI	MMSE	FAQ	Eating habits review
CAQ ¹⁰	Mini-cog		Activity habits review
MBMD as needed			Tobacco use ISI/ESS/SBQ

To summarize, the behavioral medicine specialist’s role in the case formulation and management plan for HTN/CVD is to acquire information on: psychiatric history and current status; psychosocial challenges and life adjustment problems; specific fears associated with a-fib, ICD firing, and pacemakers; presence of lifestyle problems; motivation for change; patient’s and family’s degree of understanding of the disease and identification of preventable and modifiable risk factors; cognitive status; psychotropic medication concerns; proposed behavioral health interventions; and specialty consultations. As noted earlier, inclusion of family members is an important part of data gathering and treatment planning. Remember that the family’s motivation to participate in lifestyle change is as important as the patient’s motivation to change.

Chronic Obstructive Pulmonary Disease: COPD is a chronic inflammatory lower respiratory disease that encompasses emphysema and chronic bronchitis. Prevalence of the disease in the U.S. is 6.4%, and is

¹⁰ Cardiac Anxiety Questionnaire (Eifert et al. 2000)

responsible for more than 125,000 deaths per year. Indeed, COPD is expected to become the 3rd leading cause of disease mortality worldwide by 2020. Eight of 10 deaths from COPD are caused by smoking. Over one half of Americans with poor pulmonary output don't know they have COPD. The disease is most frequently seen in adults ≥ 65 years; women; former and current smokers; Native Americans, Alaska Natives, and multiracial non-Latinos; the unemployed or disabled; those with less than a high school education; and people who are not married (Wheaton et al., 2015). COPD is characterized by dyspnea, chronic cough, and chronic sputum production.

It is increasingly recognized as a systemic inflammatory disease that negatively impacts cardiovascular function and the autonomic nervous system (increased sympathetic output at rest and low parasympathetic tone). By far, the commonest cause of COPD is smoking. In a smaller number of cases, COPD is caused by chronically inhaled noxious gases or particles, causing airway obstruction. A history of asthma also increases the risk for COPD. Diagnosis can be made in the PCC with spirometry, and patients are classified as mild, moderate, severe, or very severe based on spirometry findings, patient self-assessment (CAT: COPD Assessment Test or mMRC: modified Medical Research Council Dyspnea Scale), and number of exacerbations per year (Hobart, Kim & Tagmazyan, 2013).

COPD cannot be reversed, but medications and lifestyle change, especially tobacco cessation, can slow its progression and improve day to day functioning. The medication regimen can be complicated, relying on both short and long acting anticholinergics, β_2 -agonists, inhaled corticosteroids, and phosphodiesterase-4 inhibitors. For individuals with moderate to severe disease and hypoxemia, continuous oxygen therapy is used to ensure oxygen saturation of at least 90%. Lung volume reduction surgery may be helpful for those with severe COPD characterized by heterogeneous emphysema with upper lobe predominance. Lung transplantation is an option for patients with severe COPD, but may not be available to individuals over 65 years of age, to obese individuals, or to individuals with psychiatric conditions or neurologic/cognitive impairments. Finally, new treatments are in the early stages of development that use stem cell therapies and lung-mirroring devices to improve lung function (Schilders et al., 2016).

Functional problems associated with worsening COPD are: difficulty walking or climbing stairs and low motivation to exercise; inability to work; inability to keep up with family routines; inability to participate in social activities; and increased emergency room visits and overnight hospital stays. Comorbidities such as T2D, CVD, congestive heart failure,

asthma, and arthritis are common. Cognitive deficits are associated with severe COPD, and evidence suggests that the deficits worsen over the course of the disease (Hung et al 2009). In a large population-based study by Martinez and colleagues (2014), 17.5% of older participants reporting COPD showed mild cognitive impairment when tested and impaired cognition had an additive effect on disability. Impaired cognition in COPD compromises self-management and adherence to medical treatment, and is associated with physical deconditioning, poor overall health status, more frequent hospital admissions for acute exacerbations, and longer hospital stays (Dodd et al., 2013).

Both depression and anxiety are associated with poorer COPD prognosis. In a meta-analysis by Yohannes and colleagues (2000a), 40% of older adults with COPD were shown to have depression and 36% had symptoms of anxiety. When these three conditions present together, the source of symptoms can be confusing because of their remarkable similarities. COPD, depression, and anxiety share fatigue, lethargy, disrupted sleep, anhedonia, pessimism, poor concentration, anxious rumination, decreased appetite, social withdrawal, frequent somatic complaints, shortness of breath, rapid shallow breathing, and rapid heart rate. The unique relationship between COPD and anxiety is worth noting because of their similar pathophysiologies. Both are experienced as shortness of breath and excessive sympathetic nervous system arousal - an exacerbation in one condition aggravates the other. Hunter and colleagues (2017) refer to this as the anxiety-dyspnea or “shortness of breath” vicious cycle (pp. 125) in which shortness of breath increases health-related worry, cognitive errors (overpredicting dyspnea effects), and anxiety. These cognitive distortions and anticipatory anxiety, in turn, cause further deconditioning from a fear to move and exacerbation of dyspnea during mild exertion. There is good evidence to suggest that treating comorbid depression and anxiety with individual or group CBT not only reduces the psychiatric symptoms, but also may improve some aspects of COPD (Smith et al., 2014). Mindfulness based techniques such as meditation, progressive relaxation, yoga, and tai chi also hold promise for improving respiratory function and quality of life (Tselebis, et al., 2016).

Shortness of Breath Cycle for COPD/Asthma

Shortness of breath leads to:

- Worry about breathing, passing out, dying, etc, leading to...
- Anxiety or panic physical reaction, leading to...
- Increased breathing rate, leading to...
- Less effective (shallow, rapid) breathing, leading to...

- Increased oxygen use by, and less oxygen available for, muscles, leading to...
- More shortness of breath...
- And the cycle continues

This cycle can be stopped by following these steps:

1. **STOP** activity when shortness of breath is noticed
2. Rest by sitting or lying down
3. Relax with diaphragmatic or 'pursed lips' breathing
4. Focus on reassuring thoughts about symptoms
5. Follow the Action Plan, if in place*
6. Take medication as prescribed (e.g., rescue inhaler)
7. When breathing improves, gradually resume activity in a paced manner

*The Action Plan is commonly used in asthma treatment to codify steps to take depending on the seriousness of symptoms during an exacerbation.

Multi-disciplinary pulmonary rehabilitation programs are a mainstay of COPD treatment at initial diagnosis as well as after an acute exacerbation requiring hospitalization. These programs typically run 8-12 weeks. Target areas for rehabilitation are: tobacco cessation; increasing activity level; improving physical fitness through aerobic exercise and upper body strengthening; learning breathing strategies to reduce dyspnea and fatigue; developing self-management skills to increase self-efficacy and reduce healthcare overutilization; improving ADLs/IADLs through occupational therapy; learning good nutrition; encouraging social support; and addressing behavioral health symptoms (Global Initiative for Chronic Obstructive Lung Disease, 2014). A recent Cochrane Review of the effectiveness of pulmonary rehabilitation programs for COPD showed strong evidence for relief of dyspnea and fatigue, better exercise capacity, and improved emotional functioning, self-mastery over the disease, and health-related quality of life (McCarthy, et al., 2015). Unfortunately, pulmonary rehabilitation programs often exclude patients with cognitive deficits, even though they can benefit from tailored interventions that accommodate their difficulty in monitoring symptoms, adhering to medication instructions, and remembering daily routines for good pulmonary function.

Given the strong physiologic relationship between COPD and anxiety, Heart Rate Variability (HRV) biofeedback may be a useful nonpharmacological treatment for COPD with or without comorbid anxiety. The slow (approximately 6 breaths per minute) pursed breathing technique used in HRV biofeedback training increases parasympathetic

tone while reducing excessive sympathetic arousal. Asthma studies by Lehrer and colleagues (e.g., Lehrer et al., 2004) showed that HRV biofeedback training improves the sympathovagal imbalance while producing a decrease in respiratory resistance and better spirometry performance. While there are too few studies to date of HRV biofeedback in the treatment of COPD to build a good evidence base, the therapy does show promise for reduction in dyspnea with mild exertion (i.e., distance walked in 6 minutes) and improved overall health quality (see Gevirtz, 2013 for review). Although biofeedback is a specialty behavioral health intervention, the breathing technique can be taught in PC with the help of a personal adjustable breathing pacer such as the smartphone apps Breath2Relax or Paced Breathing. Slow (not deep) inhalation, by expanding the diaphragm/belly, through the nose for 4 seconds and pursed lips exhalation for 6 seconds accomplishes the 6 breath per minute pace with good effect.

Opportunities for behavioral health screenings in the PCC occur at the time of COPD diagnosis, after discharge from the hospital or pulmonary rehabilitation, during medical follow-up appointments, and with new onset of complications.

Watch and Wait Assessment for COPD

<i>Psychopathology Screen</i>	<i>Cognitive Screen</i>	<i>Life Adjustment Screen</i>	<i>Lifestyle Problems</i>
PHQ-9 or GDS	MoCA	PROMIS	Tobacco Use
GAD-7 or GAI	MMSE	FAQ	Activity habits review
MBMD as needed	Mini-cog	CAT or mMRC ¹¹ or Manchester Respiratory Activities of Daily Living Questionnaire ¹²	AUDIT/SMAS-T-G
			ISI/ESS/SBQ

¹¹ COPD Assessment Test (Jones et al., 2009); modified Medical Research Council Dyspnea Scale (Stenton, 2008)

¹² Manchester Respiratory Activities of Daily Living Questionnaire (Yohannes et al., 2000b)

To summarize, behavioral medicine should be integral to the workup of a patient with COPD given the high rates of comorbid anxiety and depression in this population. Data to be gathered include: psychiatric history and current status, including medications; psychosocial challenges and life adjustment problems in the presence of reduced respiratory function; lifestyle misbehaviors (smoking, poor diet, low physical activity); patient's and family's degree of understanding of the disease, its progression, and identification of preventable and modifiable risk factors; cognitive status; motivation to change; and proposed behavioral health interventions. Family members and/or caregivers must be included in the case formulation and care plan because they are often doing the patient's ADLs when fatigue or shortness of breath is too severe to function independently. Remember that the family's motivation to participate in lifestyle change is as important as the patient's motivation to change. Finally, don't forget to always ask the COPD patient who still smokes: "Would you mind if we talk about quitting smoking at this visit?"

Chronic Pain: Persistent pain problems affect more than 50% of older adults living at home; its prevalence is even higher in long term care settings. Pain is one of the most frequent reasons for a visit to the doctor and can lead to suboptimal treatments such as bedrest or dangerous treatments such as prescriptions of opioids for chronic pain and sedatives for sleep. Pain is a common comorbidity of other chronic medical conditions and is associated with substantial disability from reduced activity and mobility, falls, depression and anxiety, insomnia, social isolation, and, importantly, disruption of close relationships. Risk factors include advancing age, female gender, lower SES, low educational level, obesity, tobacco use, previous injury, history of a physically strenuous job, childhood trauma, and depression or anxiety (Patel et al., 2013) – risk factors that are now quite familiar to the reader. What is more, having a history of depression or anxiety, maladaptive coping skills such as worry and avoidance, and low social support are predictive of poor outcome. The good news is that all of the behavioral medicine interventions identified in this chapter – dietary improvements, weight loss, exercise, smoking cessation, HRV breathing practice, especially when coupled with cognitive behavioral therapy for chronic pain - can have a positive impact on quality of life and functionality, while reducing reliance on high doses of analgesics.

Diseases associated with chronic pain in later life, by system or specialty

- Dermatology—pressure or ischemic ulcers, bums, scleroderma
- Gastrointestinal—constipation, irritable/inflammatory bowel disease, diverticulitis
- Cardiovascular—advanced heart disease, peripheral vascular disease
- Pulmonary—advanced COPD, pleurisy
- Rheumatology—osteoarthritis, rheumatoid arthritis, gout, pseudogout, polymyalgia, lupus, fibromyalgia
- Orthopedics—spinal stenosis and other low back syndromes, myofascial syndromes, osteoporotic related fractures
- Endocrine—diabetic neuropathy, Paget’s disease
- Nephrology—chronic cystitis, end stage renal disease
- Immune—herpes zoster, post-herpetic neuralgia, HIV/AIDS neuropathy
- Neurology—headache, peripheral neuropathies, compressive neuropathies, radiculopathies, Parkinson’s disease, post-stroke pain
- Oncology—cancer
- Miscellaneous—depression, tendonitis, bursitis, joint repair/replacement

From: Reid et al., 2015

The diagnosis of chronic pain is made when pain lasts longer than 3 months or beyond the usual course of the disease or injury. It is important to note that a specific cause of chronic pain cannot always be identified despite a thorough evaluation. Persistent pain problems in older adults are often an accumulation of impairments in multiple systems. Chronic pain is more than a sensory event; it also includes emotional, cognitive and behavioral experiences. A good chronic pain evaluation will include assessments of these factors as well as family/caregiver attitudes toward the pain patient and overall level of social support. Reid and colleagues (2015) recommend the following components in a comprehensive pain evaluation:

Comprehensive Pain Evaluation

- ✓ Use standardized pain assessment tools in addition to a physical exam. The Brief Pain Inventory-short form (BPI-SF: Cleeland & Ryan, 1994), Geriatric Pain Measure (GPM: Ferrell, Stein & Beck, 2000), and McGill short form pain questionnaire (SF-MPQ: Melzack, 1987) are commonly used self-report measures for older adults.
- ✓ Ascertain the impact of chronic pain on daily functioning (Patient Reported Outcome Measurement Information System (PROMIS):

Cook et al., 2016) and sleep (Insomnia Severity Index (ISI); Bastien et al., 2001). If addiction potential is a concern, the Screener and Opioid Assessment for People with Pain-Revised (SOAPP-R; Butler et al., 2008) and Opioid Risk Tool (ORT; Webster & Webster, 2005) are useful measures for guiding prescribing decisions.

- ✓ Identify attitudes and beliefs about pain. The West Haven-Yale Multidimensional Pain Inventory (WHYMPI; Kerns, Turk & Rudy, 1985) assesses pain beliefs as well as perceptions regarding family members' support or lack thereof.
- ✓ Elicit expectations and goals for treatment so that the degree of alignment between patient and provider expectations and goals can be ascertained.
- ✓ Gather collateral information from family members and caregivers to better understand the patient's response to pain and impact on functioning. This is an especially important task when the patient has difficulty communicating secondary to neurologic impairment.
- ✓ Identify resources that can provide assistance with ADLs/IADLs, as well as reinforce adherence to the treatment plan.
- ✓ Review comorbidities (including psychiatric) and medication list for potential adverse effects of adding analgesics – pain medications can worsen other medical conditions as well as cause adverse drug-drug interactions.

In line with the Watch and Wait model, repeat assessments of the chronic pain patient's quality of life, mood, gait, and overall functioning are advised at each visit (Makris et al., 2014). Also, be aware that the five most frequent psychiatric illnesses found in persistent pain patients are depression, anxiety, somatic symptom disorders, PTSD, and substance abuse disorders. Undiagnosed or inadequately treated psychiatric conditions tend to exacerbate subjective pain, thereby putting patients at risk for misusing prescription drugs, alcohol, and other substances in an attempt to alleviate their physical and psychological discomfort. It is therefore essential to evaluate for comorbid psychiatric symptoms and substance misuse when assessing persistent pain.

Chronic pain in older adults is often inadequately treated, yet pain relief is one of the most commonly endorsed treatment goals at late life (Fried et al., 2011). It is well known now that long term treatment of persistent pain with opioid drugs is associated with addiction and, for some, tragic outcomes. This is believed to be less likely for older patients. The 2015 National Survey of Drug Use and Health reported that those who misused/abused opioid medications tended to be younger and male (Hughes et al., 2016). However, a CDC Morbidity and Mortality Weekly Report (Shah et al., 2017) noted that "Patients who continued opioid therapy for ≥ 1 year were more likely to be older, female, have a pain

diagnosis before opioid initiation, and [were] initiated on higher doses of opioids, compared with patients who discontinued opioid use in <365 days.” (p. 266). Although little data are available to recommend therapeutic long term use of opioids in older adults with non-cancer pain, there is good evidence to show a high risk of adverse events in elders, such as: falls; fall-related fractures and other injuries; hospitalizations; and death (Buckeridge et al., 2010; Solomon et al., 2010). As baby boomers reach older ages, the question arises whether they will be at higher risk for abusing pain medications than earlier cohorts because of their greater acceptance and use of recreational drugs. Indeed, a strong determinant as to whether a patient will misuse opioid medication is a history of substance abuse.

As in other chronic medical conditions with complex comorbidities, chronic pain management is best addressed both pharmacologically and nonpharmacologically within the PCMH or similar collaborative care model. A Functional Restoration Program (FRP), typically found in large medical centers and university medical schools, is an example of a collaborative approach to pain treatment. FRP includes a physician/nurse team specializing in pain management; a psychologist to help patients manage treatment expectations, address psychiatric and interpersonal issues, develop coping skills, and maintain a healthy lifestyle; an occupational therapist to help patients enhance everyday functioning to attain predetermined goals; a physical therapist to improve body mechanics with joint movement and body awareness, as well as introduce patients to stepped exercise, and a nutritionist to educate on the impact of food choices on inflammation and pain as well as counsel patients on weight management (Gatchel et al., 2014). Patients undergo a comprehensive multidisciplinary evaluation in which problems are identified and treatment goals are mutually agreed upon in the following broad areas:

Goals of a Functional Recovery Program for Chronic Pain
<ul style="list-style-type: none">• Improve sleep, mood, and cognitive functioning• Increase tolerance for daily activities• Rebuild family and social support systems• Resume work and family roles as appropriate• Manage pain through a variety of non-medication approaches and conservative pharmacotherapeutics

Collaborative care models enable the physician to use medications judiciously to minimize adverse events and promote maximal functioning. Guidelines by the American Geriatric Society (2017) recommend acetaminophen as a first line agent in the initial and ongoing treatment of

mild to moderate pain because of its effectiveness and good safety profile. NSAIDs and COX-2 inhibitors should be avoided except for certain select patients because of the side effect/adverse events profile in older adults. Opioid pharmacotherapy can be considered for patients with moderate to severe pain, functional impairment due to pain, or poor quality of life due to pain. Adjuvant medications, such as some tricyclic antidepressants, SNRIs, anti-convulsants, muscle relaxants, cannabinoids, corticosteroids, and topical medications, are indicated for neuropathic pain, fibromyalgia, headaches, low back pain, and diffuse bone pain.

In the realm of nonpharmacologic interventions for chronic pain, exercise may be the most important and helpful treatment for chronic pain. It increases strength, endurance, and balance, reduces social isolation, and as a bonus, improves cognitive sharpness. Any type of exercise can be beneficial. The most commonly cited types are resistance training with weights, Tai Chi for balance and strength, and aerobic exercise for stamina and cardiac health (Naugle, 2016). For older adults who have not been exposed to exercise at younger ages, convincing them that exercise will help their pain can be met with deep skepticism and reluctance to engage (Makris et al., 2014), so a slow approach that titrates exercise through a concrete paced activity-rest plan is recommended. For obese patients with chronic musculoskeletal pain, intensive weight loss can substantially reduce pain while improving mobility and functionality. Of course, a combination of diet and exercise is best for all the reasons stated in this chapter. Indeed, the IDEA randomized clinical trial (Messier et al., 2013), a large study of the effect of diet and/or exercise on pain and functionality in overweight and obese older adults with osteoarthritis, showed that a combination of intensive weight loss of at least 10% of body weight, combined with moderate exercise, significantly reduced knee pain, lowered IL-6 and improved quality of life, compared with diet alone or exercise alone.

A potent part of any pain management program is a psychotherapy intervention that teaches acceptance of chronic pain, self-efficacy in spite of it, and hope through positive coping. Cognitive behavioral therapies for chronic pain are designed to address catastrophizing thoughts, cognitive errors, and aberrant core beliefs about pain and self, as well as behavioral activation and distraction through exercise and pleasant events-scheduling. Turk and Winter (2006) have written an engaging book for chronic pain patients that is full of self-management strategies and is easy to implement in the PCC. An important adjunct to CBT for chronic pain are mindfulness practices, including HRV breathing, described earlier, progressive muscle relaxation, and visual imagery, all of which cultivate a nonjudgmental awareness of the self in the present moment. ACT for chronic pain,

Mindfulness-based Stress Reduction (MBSR) and Mindfulness-based Cognitive Therapy (MBCT) are structured, mindfulness-based psychotherapy programs that also have shown effectiveness in chronic pain conditions (see Carlson, 2012 for review). MBSR is especially useful in the PCC because it is delivered in group format.

It needs to be said, too, that the approach to pain intervention is important. Most pain problems do not completely extinguish. How ones approaches pain and learns their role in its treatment is critical. The position espoused by the fear-avoidance model of chronic pain holds that there exists secondary learned behaviors of catastrophizing, excessive threat perception and escape or avoidance leading to depression or disuse/disability (Vlaeyen & Linton, 2000). Building on this the Mayo Clinic has promulgated a Central Sensitization Syndrome. This is pain that starts with nociceptors leading to the thalamus and then, in turn, stimulates a series of brain connectors resulting in the pain experience (amygdala, hippocampus, somatosensory nerve pain, limbic system (emotion) and prefrontal cortex (rational thinking). In effect, the patient learns that pain is always only organic, is intractable, and disuse is the best answer. The brain connection is left out and the learned helplessness results (Henschke, Kramer, & Maher, 2015). How one responds to the natural element of pain becomes central to care. Focusing only on the physical pain or only on the psychological aspects of pain is therefore ineffectual.

<i>Watch and Wait Assessment for Chronic Pain</i>				
<i>Psychopathology Screen</i>	<i>Pain</i>	<i>Cognitive Screen</i>	<i>Life Adjustment Screen</i>	<i>Lifestyle Problems</i>
PHQ-9 or GDS	BPI-SF ¹³ •r...	MoCA	PROMIS	Tobacco Use
GAD-7 or GAI	GPM ¹⁴ •r...	MMSE	FAQ	Activity habits review
MBMD as needed	SF-MP ¹⁵	Mini-cog	WHYMPI ¹⁶	AUDIT/SMAS-T-G
SOAPP-R17/ORT ¹⁸				ISI/ESS/SBQ

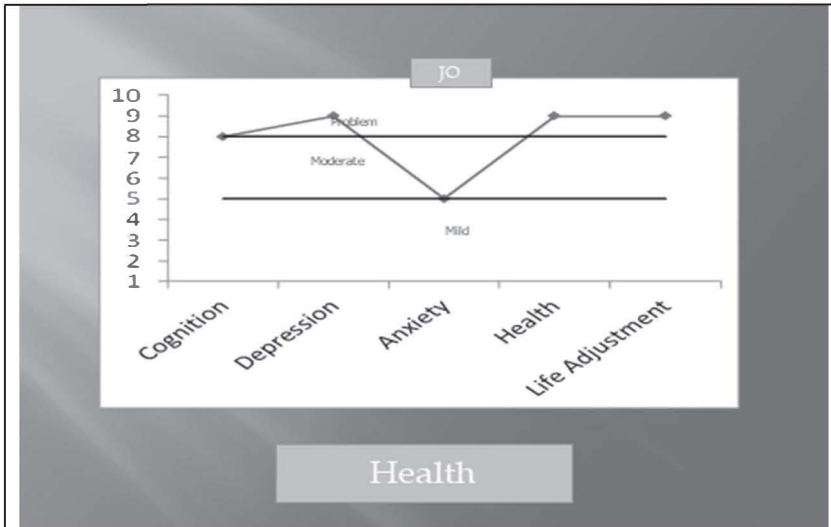
¹³ Brief Pain Inventory- short form (Cleeland & Ryan, 1994)
¹⁴ Geriatric Pain Measure (Ferrell, Stein & Beck, 2000)
¹⁵ Short form-McGill Pain Questionnaire (Melzack, 1987).
¹⁶ West Haven-Yale Multidimensional Pain Inventory (Kerns, Turk & Rudy, 1985)
¹⁷ Screener and Opioid Assessment for Patients with Pain-Revised (Butler et al., 2008)
¹⁸ Opioid Risk Tool (Webster & Webster, 2005)

To summarize, opportunities for behavioral health screenings in the PCC can occur any time staff are concerned about a patient's pain complaints. Close attention should be paid if a new patient comes to clinic with a history of long term pain medication use – specific pain measures, and opioid risk screeners can be immensely informative. It is also vital to obtain a psychiatric history and current status to understand the interaction of pain and psychopathology. Both the patient's and family's knowledge of the pain causes, and their beliefs about what helps and what hurts the patient's functionality are important targets for education and support. As with other chronic medical conditions, the team needs to be aware of lifestyle misbehaviors (smoking, poor diet, low physical activity), especially history of, and risk for, substance abuse, and create a treatment plan that includes steps to improve these behaviors. Remember that family members and/or caregivers must be included in the case formulation and care plan, not only because they can be excellent collaborators, but because they may inadvertently enable a patient's poor coping or sabotage progress made because of their own maladaptive cognitions.

Case: Mr. O

This case highlights the need for healthcare professionals to appreciate the complexity of an older adult with comorbid medical and psychiatric/psychological conditions. Psychologists and other behavioral health providers will do their patients a disservice when they focus only on the psychosocial needs of the individual and ignore their physical health. In

Mr. ●'s case, below, the index issue was ultimately not cognitive decline as the chief complaint suggests, but chronic, debilitating headache pain that was poorly treated by a medication known to cause cognitive deficits in a subset of patients. With no relief from the migraines and increasing stress from financial and caregiver obligations, poor sleep, and apparent functional decline, a cascade of medical and psychiatric signs and symptoms developed: depression, anxiety, stress eating and overeating, weight gain, unstable blood pressure, and the beginnings of insulin resistance. By collaborating with the PCP to aggressively treat this patient's chronic severe migraines first (i.e., getting the biggest bang for the buck), Mr. ● achieved remarkable pain relief and so was motivated to continue working with the behavioral medicine specialist and the PCMH team to get his other health issues under tow.



PATIENT: J O
DOB: 1948
GENDER: Male
ETHNICITY: Puerto Rican

Case-Based Plan:

Problems

Health

Migraine headaches that are frequent and debilitating

Poorly controlled hypertension

Restless Legs Syndrome, insomnia (ISI: 16)

Weight gain and at risk for Metabolic Syndrome:

BMI = 29.5 with increased abdominal adiposity

BP = 166/91 HbA1c = 5.9 (pre-diabetes)

Total cholesterol = 212 (H); triglycerides = 100; HDL = 72 (H);

LDL = 130 (H)

Pain

BPI-SF

Pain Severity mean score = 8.5/10

Pain Interference mean score = 10/10

Depression

First depressive episode, ongoing for 6 months with passive suicidal ideation. PHQ-9: 22 – severe symptoms

Cognition

Recent cognitive decline, especially memory for new information and slowed processing speed

MoCA: 17 – with deficits in attention, delayed recall, executive tasks, verbal fluency (slow).

Life Adjustment

Education: Bachelor's degree

FAQ: 12/30

Zarit Caregiver Burden Scale: 57/88 (high burden)

Environment: Good – safe neighborhood, financially stable

Relationships: supportive wife, primary caregiver for both parents with dementia

Meaning in life: fair

Stress: Substantial

Anxiety

GAD7: 7 – mild-moderate symptoms

Case Formulation

REFERRAL: Patient asks a nurse in the PCC to help him get tested for dementia. Nurse refers patient to the PCMH behavioral medicine specialist.

CHIEF COMPLAINT: “I can’t seem to remember anything; it’s terrible. I can’t work. I feel overwhelmed by my parents’ needs, but I have a duty to take care of them. I promised that I would not put them in a home.”

HISTORY OF PRESENT ILLNESS: The patient is a 65yo Latino married man who, until recently, had a career in information systems. Over the past year, he noticed increasing difficulty remembering medication dosages, financial obligations, to-do lists, and the day/date. He is unable to maintain his hobby of furniture building because of trouble thinking through details of measurement and construction. He retired from computing because he could no longer recall commonly used algorithms. He is severely depressed with passive suicidal ideation on “bad migraine days.” While recently cleaning his gun, he thought about how easy it would be use the gun on himself. Mr. ● has trouble falling asleep, experiences middle of night awakenings, and is fatigued all day. He worries that he is developing dementia; both parents have dementia. He also complains of chronic distress from taking care of his parents: they still live in their home, across from Mr. ●. They need help with all IADLS and some ADLs. All care is provided by Mr. ● and his wife. Father is

increasingly disinhibited, aggressive, and wanders off the property in spite of several safety measures put in place.

RELEVANT DEVELOPMENTAL HISTORY: Mr. ● is a native of Puerto Rico. He is the elder of two children. His brother, who is not local, is marginally involved with the family. His father was a laborer in Puerto Rico and mother was a homemaker. Wanting to leave Puerto Rico as a teen, Mr. ● joined the Army after obtaining his high school diploma. He received a BS in information science while in the Army, and worked in communications for 25 years before retiring in a senior technical position. Mr. ● began a second career in information systems technology and retired earlier than he would have liked because memory deficits made him feel incompetent. He is now financially unstable from having to maintain two households. Mr. ● has been married for 40 years and enjoys a warm, supportive relationship with his wife. They have one child who is not local. His wife has taken over the finances because Mr. ● doesn't trust himself. He has lost interest in many activities because of poor sustained attention and confused thinking. He recently got lost twice while driving.

RELEVANT MEDICAL HISTORY: Mr. ● worries that the stress of caring for his parents at home has caused his health to decline. Medical records reveal a 10 year history of migraine headaches without aura, the frequency and intensity of which have become more debilitating in the past 1.5 years. He experiences migraines as much as 5 times/week; a sustained migraine can last several days. Headaches are treated by a neurologist, historically with sumatriptan, but as breakthrough headaches became more frequent, topiramate was added. He has put on weight from stress eating and lack of exercise, and developed HTN. He is prescribed metoprolol by his PCP, but BP is poorly controlled. He recently was started on simvastatin for hyperlipidemia. He has restless legs syndrome (RLS), is prescribed a dopamine agonist (ropinirole Hcl), but symptoms persist and he has difficulty settling down for sleep. He experiences frequent middle of the night awakenings, sometimes because his father has set off the alarm, sometimes because of anxiety and worry about the future. He does not drink alcohol or use recreational drugs, and has a 25 pack year smoking history. Mr. ● has been quit for 22 years and is proud that he has not started smoking again given his high stress level. His religion, Catholicism, provides some emotional support.

MENTAL STATUS: The patient is a 65-year-old Latino male who drove to this appointment alone. He was casually dressed and well groomed. He was pleasant, but clearly distressed about his health and feelings of helplessness regarding his parents' care. He had a dull headache at this visit. His thoughts as expressed were goal directed, linear,

and logical. He has been bilingual since childhood and the assessment was done in English. He provided historical information that was generally consistent with his medical record. He exhibited some word finding deficits and processing speed appeared slow. Handwriting was clear and precise. He admitted to feeling depressed and lately struggled with suicidal ideation during bad migraines. He stated that he is a worrier: he worries about his future given that both parents have dementia, about his ability to continue caring for his parents as he promised (i.e., he would never place them in a nursing home), and about his subjective inability to be a “good husband.” There is no evidence of perceptual anomalies or delusional thinking. His insight and judgment are intact.

TEST RESULTS

Cognitive Functioning: As noted previously, baseline MoCA was 17/30. In a test of premorbid intelligence, Mr. ● scored at the 45th percentile, in the average range. The Shipley Verbal and Abstract scores were similarly in the average range. His WRAT-4-Reading score was consistent with average reading ability. Mr. ●’s RBANS composite index was 91, placing him at the 50th percentile as compared to same age peers. He scored high average on visuospatial and language, average immediate memory, low average on attention, and impaired on delayed memory. Trails B showed slow processing speed, but no errors, suggesting intact cognitive flexibility. He was below average on tests of verbal/category fluency secondary to slow processing speed.

Emotional Functioning: Emotional self-report scales indicated significant depression and mild-moderate anxiety. His degree of insomnia, as measured by the Insomnia Severity Index, is moderately severe.

Life Adjustment and Health Behaviors: Mr. ●’s life adjustment is poor. He does not feel up to the task of caring for his parents, yet he promised that he would not put them in long term care. He worries about his parents constantly, especially his father, whose disinhibited rants and wanderings have brought police to the house. The Zarit Caregiver Burden score shows that Mr. ● experiences a high degree of burden. He worries that he will not be able to maintain two households for much longer because of financial strain. The FA●, a measure of executive functions in activities of daily living, revealed areas of impaired functioning. He described needing his wife to take over finances and taxes. He needs reminding for appointments, events, and medications. He has had some recent scares finding himself in an unfamiliar place while driving. His health behaviors are also poor. He stress eats and craves energy dense, highly processed salty foods and fast foods. He eats late at night when he

cannot sleep. He no longer exercises or plays a sport. He has stopped building furniture because he finds it mentally taxing and finds little pleasure in other activities that he's enjoyed in the past.

WATCH AND WAIT

Summary: This is a 65 y/o male who was referred for an evaluation of his cognitive status. Health issues are prepotent and worsening. He is heavy and his weight gain is related to stress eating and a sedentary lifestyle. He is prescribed several medications, none of which seem to adequately address his medical conditions of HTN, pre-diabetes, migraine headaches, Restless Legs Syndrome, and hyperlipidemia. Psychiatric comorbidities include depression and occasional SI, moderately severe insomnia and anxiety/worry. Cognition is relevant. While arguable, he would seem to be MCI or a mild dementia. Migraine medication is confusing this diagnosis. Depression is significant and asserts an influence over physical problems. Life Adjustment is also important. The degree of functional independence is not age typical.

Intervention:

Decernment: X (minimally compliant)

Core Markers

Validate Problem X

Psychoeducation of Model X

Assessment X

Alliance X

Monitoring X

Case formulation X

Treatment Summary

We applied a stepped, collaborative approach in which the PCP and the behavioral medicine specialist work closely together to develop the initial treatment plan that includes combined medical and psychiatric/psychological interventions. Mr. ● has assets. He shows good insight, good judgment, and has a supportive, collaborative relationship with his wife. He is motivated to improve his quality of life short of placing his parents in a nursing home. He led an active life prior to becoming his parents' caregiver. He is in a PCMH and the PCP and psychologist are frequent collaborators; case management also is available.

Most striking in this case is Mr. ●'s inadequately treated, debilitating migraines and his current medication regimen. Migraine headaches are associated with many of Mr. ●'s complaints: impaired attention and concentration, impaired ability to retain information when in pain,

decreased functional ability, depression and anxiety symptoms, fatigue, and impaired sleep. Furthermore, of the medications Mr. ● is prescribed, topiramate is known to produce transitory cognitive deficits in some patients, including changes in memory, problem-solving ability, processing speed, language, and reading that are reversed when the drug is stopped.

A different treatment approach clearly is needed to ameliorate Mr. ●'s migraines, which also could improve his cognition, depression, anxiety, and sleep. Another consideration: given the degree of depressive symptoms with suicidal/morbid ideation and concomitant anxious worry, Mr. ● may benefit from a trial of an SNRI to address both clusters of psychiatric symptoms. Mr. ●'s PCP also needs to reflect if it is time to change the current antihypertensive medication and add metformin to treat pre-diabetes, or to Watch and Wait to see if lifestyle interventions can successfully lower Mr. ●'s numbers and his weight. Mr. ●'s poorly addressed restless leg symptoms, which often prevents him from staying in bed no matter how tired, also must be dealt with if we are to effectively treat his insomnia. He also needs help at home with IADLs.

Life Adjustment

Finances and taxes are problematic, but he also needs reminding for appointments, events, and medications. He has had some recent scares finding himself in an unfamiliar place while driving. He cannot care adequately for his parents. His health behaviors are poor. He stress eats and craves energy dense, highly processed salty foods and fast foods. He eats late at night when he cannot sleep.

Treatment Specifics

Health/Cognition/Depression/Life Adjustment

1. Address the migraine headaches ASAP. Discontinue topiramate and refer Mr. ● to a PCMH-affiliated headache specialist for a trial of botulinum toxin (Botox) injections. Look for improvements in cognition and daily functioning, sleep, more stable blood pressure readings
2. Because of frequent thoughts of death and suicide, address depressive and anxiety symptoms with a combination of: a) SNRI that causes little to no weight gain, b) behavioral activation (e.g., a walking Rx), and c) heart rate variability (HRV) breathing biofeedback to reduce state anxiety
3. Recommend a 30 minute exercise routine in the evening to decrease restless leg symptoms. Look for improvements in sleep latency, mood, anxiety, and late night eating. Blood sugar may improve.

4. Case management services to educate Mr. ● on Medicare supported in-home services for his parents that may reduce caregiver burden
5. Watch and Wait for 1 month: HTN, pre-diabetes, sleep, diet/eating habits before applying a targeted intervention to those problems.

Mr. ● agrees to interventions #1, #2, and #3. He is not ready to talk to a case manager, but agrees to visit the local Department on Aging for information on local services for senior citizens. Behavioral medicine specialist conferred with the PCP and Mr. ● was referred to headache specialist and starts venlafaxine 75mg for anxiety and depression, and botox injections were initiated for migraine. The behavioral medicine specialist meets with Mr. ● three times in the following month to teach HRV breathing biofeedback and relaxation techniques. Mr. ● is asked to keep a sleep log for the next month that documents sleep and wakefulness, daily exercise, bedtime stretching/walking for RLS, RLS episodes, migraine episodes, and caffeine. Mr. ● is given contact information for the local Department on Aging.

Self or other monitoring: Mini-targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments:

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:

Imagery exercise for counter-experience:

Compassion training:

Communication and Assertiveness training: X

Behavioral experiments: X

Emotional exposure:

Homework: X

Longer Therapy processes:

ACT: use of acceptance interventions

Biofeedback: HRV

Lifestyles:

- Practice Spirituality/Religion

- Socialize: Less decline and live longer
- Exercise: Even housework
- Diet
- Somatic softening: Meditate and Mindfulness
- Values and Leisure

Follow-up:

The follow-up plan was tweaked based on feedback at each visit. After the first month of treatment, Mr. ● experienced a remarkable improvement in his migraines with a combination of a botox injection and PRN dosing of sumatriptan for breakthrough headaches, which now occurs only once a week. He noticed that his thinking was no longer “foggy” and attention was much improved. Mr. ●’s blood pressure was more stable, probably due to reduced chronic headache pain. Restless legs symptoms showed mild inconsistent improvement, and sleep latency continued to be prolonged. Mood was still low, but Mr. ● no longer had suicidal thoughts now that his headaches were under good control. The physiological symptoms of anxiety improved with HRV breathing, but Mr. ● still found himself worrying into the night about his finances and his ability to care for his parents at home.

Over the next two months, Mr. ● agreed to the following interventions:

1. PCP changed medication for restless legs to a different dopamine agonist (pamipexole), which eliminated the symptoms.
2. Behavioral medicine specialist implemented cognitive strategies to manage worry, especially at night. Sleep hygiene strategies were added, especially creating a bedtime routine and turning off electronics. Late evening eating was limited to a small portion of cottage cheese with fruit, eaten slowly.
3. Mr. ● became more active and felt better with regular exercise (45 minutes daily walking with his wife) and started to work with his hands again as his depression lifted.
4. Mr. ● and his wife were referred to a nutritionist so that they could improve their food choices and reduce portion size. Mr. ● lost 15 lbs after 4 months with improved HbA1c.
5. Mr. ●’s parents’ needs were becoming too complicated for independent living. The behavioral medicine specialist addressed Mr. ●’s guilt through cognitive therapy and subsequently referred him to an elder care counselor. He ultimately placed dad in a good quality long term care facility, and mom moved in with Mr. ● and his wife with in-home ADL support services. His sleep improved significantly afterward and he was no longer fatigued during the day.

Screening measures after 4 months showed good progress: MoCA: refused, but believes his cognitive status has improved substantially. Other scores were: PHQ-9: 2 GAD-7: 1 ISI: 3 FAQ: 0 BP: 119/80 HbA1c: 5.7: BMI: 27.1.

His domain profile was lower (below Problem) in all areas but need constant monitoring.

Conclusion

Recall that quality of life is anchored in health. Older adults go for yearly physical exams; not yearly depression, anxiety, cognition or life adjustment exams. It is problematic too to note that virtually all older adults have a chronic disease. Quality of life depends on one's evaluation of health. Of course, this can and is often mediated by the other core domains.

The Watch and Wait model applied throughout this book means that we treat carefully and with sensitivity, using a stepped approach that avoids overtreating or undertreating the patient. In this chapter we highlighted the necessity of attending to maladaptive lifestyle behaviors and stress when addressing the psychological aspects of chronic medical conditions. The behavioral medicine specialist who is integrated into the PCC is in a unique position to educate, assist, coach, and collaborate with patients and their families to enhance quality of life, extend functionality, and reduce medical burden because the whole person is taken into account. The time spent targeting health misbehaviors for change while other members of the PCMH or collaborative care team address the disease itself will pay itself forward by easing the management of, and perhaps avoiding further complications from, chronic medical conditions.

References

- Abbas M, Cumella L, Zhang Y, Choi J, Vemulapalli P (2015). Outcomes of Laparoscopic Sleeve Gastrectomy and Roux-en-Y Gastric Bypass in Patients Older than 60. Obesity Surgery, 25(12): 2251-2256.
- Albert SM, Roth T, Toscani M, Vitiello MV, Zee P (2017). Sleep Health and Appropriate Use of OTC Sleep Aids in Older Adults-Recommendations of a Gerontological Society of America Workgroup. Gerontologist, 57:163-170.
- Ali MK, Echouffo-Tcheugui J, Williamson DF (2012). How effective were lifestyle interventions in real-world settings that were modeled on the Diabetes Prevention Program? Health Affairs, 31: 67-75.

- American Diabetes Association (2016). <http://www.diabetes.org/diabetes-basics/statistics/>
- American Diabetes Association (2016). Foundations of Care and Comprehensive Medical Evaluation. *Diabetes Care*, 39(Supplement 1): S23-S35. <https://doi.org/10.2337/dc16-S006>
- American Geriatrics Society (2015). Beers Criteria Update Expert Panel: American Geriatrics Society 2015 Updated Beers Criteria for Potentially Inappropriate Medication Use in Older Adults. *Jl American Geriatrics Society*, 63:2227-46.
- American Geriatrics Society (2017). Pain Management GUIDELINES Pocket Card. https://www.guidelinecentral.com/shop/pain-management-guidelines-pocket-card/?attribute_pa_format=digital
- American Heart Association/American College of Cardiology Task Force on Practice Guidelines and the Obesity Society (2014). 2013 AHA/ACC/TOS Guideline for the Management of Overweigh and Obesity in Adults. *Circulation*, 129:S102-S138.
- American Psychological Association (2012). Stress in America: Our Health at Risk. American Psychological Association, Washington DC, released January 11, 2012
- Ananthakrishnan AN, Khalili H, Pan A, Higuchi LM, de Silva P, et al. (2013). Association between depressive symptoms and incidence of Crohn's disease and ulcerative colitis: results from the Nurses' Health Study. *Clinical Gastroenterology and Hepatology: the official clinical practice journal of the American Gastroenterological Association*. 11:57-62.
- Anthenelli RM, et al. (2016). Neuropsychiatric Safety and Efficacy of Varenicline, Bupropion, and Nicotine Patch in Smokers With and Without Psychiatric Disorders (EAGLES): A Double-Blind, Randomized, Placebo-Controlled Clinical Trial. *The Lancet*, 387(10037): 2507-2520
- Antisdel JE, Laffel LM, Anderson BJ (2001). Improved detection of eating problems in women with type 1 diabetes using a newly developed survey. *Diabetes*, 50(Suppl 2):A47.
- Bailey MT, Dowd SE, Galley JD, Hufnagle AR, Allen RG, Lyte M. (2011). Exposure to a social stressor alters the structure of the intestinal microbiota: implications for stressor-induced immunomodulation. *Brain, behavior, and immunity*, 25:397-407.
- Barnett I, van Sluijs EM, Gilvie D (2012). Physical activity and transitioning to retirement: a systematic review. *American Journal of Preventive Medicine*, 43:329-36.

- Bastien CH, Vallières A, Morin CM (2001) Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine*, 2:297–307.
- Batsis JA, Gill LE, Masutani RK, Adach-Mejia AM, Blunt HB, et al. (2017). Weight loss interventions in older adults with obesity: A systematic review of randomized controlled trials since 2005. *Jl American Gerontological Society*, 65:257-268.
- Benros ME, Waltoft BL, Nordentoft M, Østergaard SD, Eaton WW, Krogh J, Mortensen PB (2013). Autoimmune diseases and severe infections as risk factors for mood disorders: a nationwide study. *JAMA Psychiatry*, 70:812-20.
- Berk M, Williams LJ, Jacka FN, Neil A, Pasco JA, Moylan S, et al. (2013). So depression is an inflammatory disease, but where does the inflammation come from? *BMC Med J*, 11:200.
- Bertagnolli M, Luu TM, Lewandowski AJ, Leeson P, Nuyt AM (2016). Preterm Birth and Hypertension: Is There a Link? *Current Hypertension Reports*, 18:28.
- Black PH, Garbutt LD (2002). Stress, inflammation and cardiovascular disease. *Journal of Psychosomatic Research*, 52:1–23.
- Blow FC, Brower KJ, Schulenberg JE, Demo-Dananberg LM, Young JP, Beresford TP (1992). The Michigan Alcoholism Screening Test-geriatric version (MAST-G): a new elderly-specific screening instrument. *Alcohol Clin Exp Research*, 16:372–374.
- Blumenthal JA, Smith PJ, Mabe S, Hinderliter A, et al., (2019). Lifestyle and neurocognition in older adults with cognitive impairments. *Neurology*, 92 (3): e212-e223; DOI: 10.1212/WNL.0000000000006784
- Boeckxstaens P, Peersman W, Goubin G, Ghali S, De Maeseneer J, Brusselle G, De Sutter A (2014). A practice-based analysis of combinations of diseases in patients aged 65 or older in primary care. *BMC Family Practice*, 15:159:15-159.
- Boyd CM, Darer J, Boult C, Fried LP, Boult L, et al. (2005). Clinical practice guidelines and quality of care for older patients with multiple comorbid diseases: Implications for pay for performance. *JAMA*, 294(6):716-724.
- Brokaw SM, Arave D, Emerson DN, Butcher MK, Helgeson SD, et al. (2014). Intensive lifestyle intervention goals can be achieved as effectively with large groups as with small groups. *Primary Care Diabetes*, 8: 295-300.
- Brokaw SM, Carpenedo D, Campbell P, Butcher MK, Furshong G (2015). Effectiveness of an Adapted Diabetes Prevention Program Lifestyle

- Intervention in Older and Younger Adults. *Journal of American Geriatric Society*, 63: 1067-1074.
- Buckeridge D, Huang A, Hanley J et al. (2010). Risk of injury associated with opioid use in older adults. *Journal of American Geriatric Society*, 58(9):1664-1670.
- Butler SF, Fernandez K, Benoit C, et al., (2008). Validation of the Revised Screener and Opioid Assessment for Patients in Pain (SOAPP-R). *Journal of Pain*. 9:360-372.
- Byusse DJ, Rush AJ, Reynolds CF (2017). Clinical Management of Insomnia Disorder. *JAMA*. Published online October 23, 2017. doi:10.1001/jama.2017.15683
- Carlson LE (2012). Mindfulness-based interventions for physical conditions: A narrative review evaluating levels of evidence. *ISRN Psychiatry*, Volume 2012, Article ID 651583, 21 pages. <http://dx.doi.org/10.5402/2012/651583>
- Carney RM, Freedland KE, Steinmeyer B, Rubin EH, Marm DL, Rich MW (2016). Cardiac risk markers and response to depression treatment in patients with coronary heart disease. *Psychosomatic Medicine*, 78:49-59.
- Center for Behavioral Health Statistics and Quality. (2016). *Key substance use and mental health indicators in the United States: Results from the 2015 National Survey on Drug Use and Health* (HHS Publication No. SMA 16-4984, NSDUH Series H-51). Retrieved from <http://www.samhsa.gov/data/>
- Centers for Disease Control and Prevention (2014). National Diabetes Statistics Report: Estimates of Diabetes and Its Burden in the United States, 2014. Atlanta, GA: US Department of Health and Human Services.
- Centers for Disease Control and Prevention (2016). Prevalence of overweight, obesity, and extreme obesity among adults aged 20 and over: United States, 1960–1962 through 2013–2014 National Center for Health Statistics. National Health and Nutrition Examination Survey Data (NHANES). Hyattsville, MD: U.S. Department of Health and Human Services, https://www.cdc.gov/nchs/data/hestat/obesity_adult_13_14/obesity_adult_13_14.htm.
- Centers for Disease Control Health Alert Network (August 30, 2019). Severe Pulmonary Disease Associated with Using E-Cigarette Products. <https://emergency.cdc.gov/han/han00421.asp>
- Chiu HY, Chen P-Y, Chuang L-P, Chen N-H, Tu, Y-K, et al. (2016). Diagnostic accuracy of the Berlin questionnaire, STOP-BANG, STOP,

- and Epworth sleepiness scale in detecting obstructive sleep apnea: A bivariate meta-analysis. *Sleep Medicine Reviews*, online 5 Nov 2016.
- Chodsko-Zajko WJ, Proctor DN, Fiatarone Singh MA, Minson CT, Nigg CR, et al. (2009). Exercise and physical activity for older adults. *Medicine and Science in Sports & Exercise*. 41:1510-1530.
- Claesson MJ, Jeffery B, Conde S, Power SE, O'Connor EM, et al. (2012). Gut microbiota composition correlates with diet and health in the elderly. *Nature*, 488: 178-184.
- Cleeland CS & Ryan KM (1994). Pain assessment: global use of the Brief Pain Inventory. *Ann Academy Medicine Singapore*, 23(2):129-38.
- Cleveland Clinic. (2017). Obesity is top cause of preventable life-years lost, study shows. *Science Daily*. Retrieved May 28, 2017 from www.sciencedaily.com/releases/2017/04/170422101614.htm
- Cohen BE, Panguluri P, Na B, Whooley MA (2010). Psychological risk factors and the metabolic syndrome in patients with coronary heart disease: findings from the Heart and Soul Study. *Psychiatry Research*, 175:133-7.
- Cook KF, Jensen SE, Schalet BD, Beaumont JL, Amtmann D, et al. (2016). PROMIS measures of pain, fatigue, negative affect, physical function, and social function demonstrate clinical validity across a range of chronic conditions. *Journal of Clinical Epidemiology*, 73: 89-102.
- Cribbet MR, Carlisle M, Cawthon RM, Uchino BN, et al. (2014). Cellular Aging and Restorative Processes: Subjective Sleep Quality and Duration Moderate the Association between Age and Telomere Length in a Sample of Middle-Aged and Older Adults. *Sleep*, 37(1): 65-70. <http://doi.org/10.5665/sleep.3308>
- Crimmons E, Johnston M, Haywaerd M & Seeman T (2003). Age differences in allostatic load: An index of physiological dysregulation. *Experimental Gerontology*, 38:731-734.
- Daly-Eichenhardt, A., Scott, W., Howard-Jones, M., Nicolaou, T., & McCracken, L. M. (2016). Changes in Sleep Problems and Psychological Flexibility following Interdisciplinary Acceptance and Commitment Therapy for Chronic Pain: An Observational Cohort Study. *Frontiers in Psychology*, 7, 1326. <http://doi.org/10.3389/fpsyg.2016.01326>
- Dantzer R, O'Connor JC, Freund GG, Johnson RW, Kelley KW (2008). From inflammation to sickness and depression: when the immune system subjugates the brain. *National Review of Neuroscience*, 9:46-56.

- de Souto Barreto P, Rolland Y, Vellas B, Maltais M. (2018). Association of Long-term Exercise Training with Risk of Falls, Fractures, Hospitalizations, and Mortality in Older Adults: A Systematic Review and Meta-analysis. *JAMA Intern Med.* published online December 28, 2018. doi:10.1001/jamainternmed.2018.5406
- DeVol R & Bedrosian A (2007). *An Unhealthy America: The Economic Burden of Chronic Disease – Charting a New Course to Save Lives and Increase Productivity and Economic Growth.* Los Angeles: the Milken Institute.
- DHHS: U.S. Department of Health and Human Services (2014). *The Health Consequences of Smoking—50 Years of Progress: A Report of the Surgeon General.* Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health. [accessed May 20, 2017].
- Diabetes Prevention Program Research Group (2015). Long-term effects of lifestyle intervention or metformin on diabetes development and microvascular complications over 15-year follow-up: the Diabetes Prevention Program Outcomes Study. *Lancet Diabetes Endocrinology*, 3(11):866-75.
- DiMatteo MR, Lepper HS, Croghan TW (2000). Depression is a risk factor for noncompliance with medical treatment. Meta-analysis of the effects of anxiety and depression on patient adherence. *Arch Intern Med*, 160:2101–2107. doi: 10.1001/archinte.160.14.2101.
- Dinan TG, Cryan JF (2016). Mood by microbe: Towards clinical translation. *Genome Medicine*, 8:36 DOI10.1186/s13073-01600292-1
- Dodd JW, Charlton RA, van den Broek MD, Jones PW (2013). Cognitive dysfunction in patients hospitalized with acute exacerbation of COPD. *Chest*, 144:119–127.
- Dornelas EA (2012). *Stress Proof the Heart.* New York: Springer Publishing.
- Drum C. (2014). Post-retirement weight gain – fact or fiction? US Army Public Health Command. Retrieved Feb 17, 2017 from: https://www.army.mil/article/127085/Post_retirement_weight_gain_fact_or_fiction/
- Eifert GH, Thompson RN, Zvolensky NJ, Edwards K, Frazer NL, et al. (2000). The cardiac anxiety questionnaire: development and preliminary validity. *Behavioral Research and Therapy*, 38:1039-1053.
- Elenkov IJ, Chrousos GP (2002). Stress hormones, pro-inflammatory and anti-inflammatory cytokines, and autoimmunity. *Annals of the New York Academy of Sciences*, 966:290–303.

- El-Kourhy F, Cassou B, Charles M-A, Dargent-Molina P (2013). The effect of fall prevention exercise programmes on fall induced injuries in community dwelling older adults: systematic review and meta-analysis of randomised controlled trials. *BMJ*, 347:f6234.
- Elsawy B & Higgins KE (2010). Physical Activity Guidelines for Older Adults. *American Family Physician*, 81:55-59.
- Estruch R, Ros E, Salas-Salvado J, Covas MI, Corella D, et al. (2013). Primary prevention of cardiovascular disease with a Mediterranean diet. *New England JI Medicine*, 368:1279-1290.
- Evrensel A, Ceylan ME (2015). The Gut-Brain Axis: The Missing Link in Depression. *Clin Psychopharmacol Neurosci*, 13(3): 239-244.
- GBD 2017 Diet Collaborators. Health effects of dietary risks in 195 countries, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. *The Lancet*, April 3, 2019; DOI: 10.1016/S0140-6736(19)30041-8
- Gatchel RJ, McGeary DD, McGeary CA, Lippe B (2014). Interdisciplinary chronic pain management: past, present, and future. *Am Psychol*. 69(2):119-30.
- Forouhi N & Unwin N (2019). Global diet and health: old questions, fresh evidence, and new horizons. *The Lancet*, DOI: 10.1016/S0140-6736(19)30500-8
- Fakhouri TH, Ogden CL, Carroll MD, et al. (2012). Prevalence of obesity among older adults in the United States, 2007-2010. NCHS data brief, no 106. Hyattsville, MD: National Center for Health Statistics.
- FDA: Food and Drug Administration (2012). FDA Drug Safety Communication: Safety review update of Chantix (varenicline) and risk of cardiovascular adverse events. 12-12-2012. <https://www.fda.gov/Drugs/DrugSafety/ucm330367.htm>
- FDA: Food and Drug Administration (2016). FDA Drug Safety Communication: FDA revises description of mental health side effects of the stop-smoking medicines Chantix (varenicline) and Zyban (bupropion) to reflect clinical trial findings. Dec. 16, 2016. <https://www.fda.gov/downloads/Drugs/DrugSafety/UCM532262.pdf>
- FDA: Food and Drug Administration (2015). FDA Drug Safety Communication: FDA updates label for stop smoking drug Chantix (varenicline) to include potential alcohol interaction, rare risk of seizures, and studies of side effects on mood, behavior, or thinking. Mar 9 2015. <https://www.fda.gov/downloads/Drugs/DrugSafety/UCM436960.pdf>

- Ferrell BA, Stein WM & Beck JC (2000). The Geriatric Pain Measure: validity, reliability and factor analysis. *Jl American Geriatric Society*, 48(12):1669-73.
- Fiore MC, Jaen CR, Baker TB (2008). Clinical Practice Guideline—Treating Tobacco Use and Dependence: 2008 Update. Rockville, MD: U.S. Department of Health and Human Services.
- Fothergill E, Guo J, Howard L, Kerns JC, Knuth N, et al. (2016). Persistent metabolic adaptation 6 years after “The Biggest Loser” competition. *Obesity*, 24:1612–1619.
- Forouhi, N & Unwin, N. (2019). Global diet and health: old questions, fresh evidence, and new horizons. *The Lancet*, April 3, 2019; DOI: 10.1016/S0140-6736(19)30500-8
- Franceschi C, Bonafe M, Valensin S, et al. (2000). Inflamm-aging. An evolutionary perspective on immunosenescence. *Annals of NY Academy of Science*, 908: 244–254.
- Fried TR, Tinetti ME, Iamone L, Leary JR, Towle V et al. (2011). Health outcome prioritization as a tool for decision making among older persons with multiple chronic conditions. *Archives Internal Medicine*, 171(20):1854-1856.
- Fryar CD, Carroll MD, and Ogden CL, (updated 2016). Prevalence of Overweight, Obesity, and Extreme Obesity among Adults Aged 20 and Over: United States, 1960-1962 Through 2013-2014. Division of Health and Nutrition Examination Surveys, CDC
http://www.cdc.gov/nchs/data/hestat/obesity_adult_13_14/obesity_adult_13_14.htm
- GBD 2017 Diet Collaborators (2019). Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *The Lancet*, 393(10184): P1958-1972,
- Geiger-Brown JM, Rogers VE, Liu W, Ludeman EM, Downton KD (2015). Cognitive behavioral therapy in persons with comorbid insomnia: A meta-analysis. *Sleep Medicine Reviews*, 23: 54-67.
- Gevirtz, R (2013). The promise of heart rate variability biofeedback: evidence-based applications. *Biofeedback* 41, 110–120
- Giardino ND, Chan L, Borson S (2004). Combined heart rate variability and pulse oximetry biofeedback for chronic obstructive pulmonary disease: preliminary findings. *Applied Psychophysiology and Biofeedback*, 29:121-33.
- Gillespie LD, Robertson MC, Gillespie WJ et al. (2012). Interventions for preventing falls in older people living in the community. *Cochrane Database Systematic Reviews*, 9:CD007146.

- Giordano S, Victorzon M (2015). Bariatric surgery in elderly patients: a systematic review. *Clinical Interventions in Aging*, 10:1627-1635.
- Global Initiative for Chronic Obstructive Lung Disease (2014). Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease. Online at: <http://www.goldcopd.org>.
- Goldstein MG, Whitlock EP, DePue J (2004). Multiple behavioral risk factor interventions in primary care: Summary of research evidence. *American Journal of Preventive Medicine*, 27(Suppl.2),61-69.
- Gormally J, Black S, Daston S Rardin D (1982). The assessment of binge eating severity among obese persons. *Addictive behaviors*, 7: 47-55.
- Harper DG, Volicer L, Stopa EG, McKee AC, Nikita M, Satlin A (2005). Disturbance of endogenous circadian rhythm in aging and Alzheimers disease. *American JI of Geriatric Psychiatry*, 13:359-368.
- Harvey JA, Chastin SF & Skelton DA (2015). How Sedentary are Older People? A Systematic Review of the Amount of Sedentary Behavior. *Jl Aging Physical Activity*, 23:471-87.
- Henschke, N, Kramer, S, & Maher, C. (2015). The Epidemiology and Economic Consequences of Pain. *Mayo Clinic Proceedings*, 90 (1), 139-147.
- Hobart L, Kim J, Tagmazyan K (2013). Treatment of stable chronic obstructive pulmonary disease: the GOLD guidelines. *American Family Physician*, 88:655-663.
- Hoffmam TC, Maher CG, Briffa T, Sherrington C, Bennell K, et al. (2016). Prescribing exercise interventions for patients with chronic conditions. *Canadian Medical Association Jl*, 188(7):510-518.
- Huang R, Ning H, Yang L, Jia C, Yang F, et al. (2017). Efficacy of Probiotics on Anxiety: A Meta-analysis of Randomized Controlled Trials. *Neuropsychiatry*, 7:862-871.
- Huang, R., Wang, K., & Hu, J. (2016). Effect of Probiotics on Depression: A Systematic Review and Meta-Analysis of Randomized Controlled Trials. *Nutrients*, 8(8): 483. <http://doi.org/10.3390/nu8080483>
- Hung WW, Wisnivesky JP, Siu AL, Ross JS (2009). Cognitive Decline among Patients with Chronic Obstructive Pulmonary Disease. *American JI of Respiratory and Critical Care Medicine*, 180:134-137.
- Hunter CL, Goodie JL, Oordt MS, Dobbmeyer AC (2017). *Integrated Behavioral Health in Primary Care*, 2nd Ed, pp. 5-108. Washington, DC: American Psychological Association.
- Hughes A, Williams MR, Lipari RN, Bose J, Copello EAP, et al. (2016). Prescription Drug Use and Misuse in the United States: Results from the 2015 National Survey on Drug Use and Health. SAMHSA: National Survey on Drug Use and Health, Bethesda, MD.

- <https://www.samhsa.gov/data/sites/default/files/NSDUH-FFR2-2015/NSDUH-FFR2-2015.htm#topofpage> Accessed October 10, 2017.
- Iestra JA, Kromhout D, van der Schouw YT, Grobbee DE, Boshuizen HC, et al. (2005). Effect size estimates of lifestyle and dietary changes on all-cause mortality in coronary artery disease patients: A systematic review. *Circulation*, 112:924-934.
- Institute for Healthcare Improvement (website). The IHI Triple Aim Initiative. Retrieved from: <http://www.ihl.org/engage/initiatives/TripleAim/Pages/default.aspx>
- Jamal A, King BA, Neff LJ, Whitmill J, Babb SD, Graffunder CM (2016). Current Cigarette Smoking Among Adults — United States, 2005–2015. *MMWR Morbidity Mortality Weekly Report*, 65:1205–1211. DOI: <http://dx.doi.org/10.15585/mmwr.mm6544a2>
- Jeong YH, Park CH, Yoo J, Shin KY, Ahn SM, et al. (2006). Chronic stress accelerates learning and memory impairments and increases amyloid deposition in APPV717I-CT100 transgenic mice, an Alzheimer's disease model. *Federation of American Societies for Experimental Biology Journal*, 20(6):729-731.
- Jha P, Ramasundarahettige C, Landsman V, Rostron B, Thun M, et al. (2013). 21st-Century Hazards of Smoking and Benefits of Cessation in the United States. *N Engl J Medicine*, 368:341-350.
- Jiang H, Ling L, Zhang Y, Mao H, Ma Z, et al. (2015). Altered fecal microbiota composition in patients with major depressive disorder. *Brain, Behavior, and Immunity*, 48:186-194. <https://doi.org/10.1016/j.bbi.2015.03.016>
- Jones PW, Harding G, Berry P, et al (2009). Development and first validation of the COPD Assessment Test. *European Respiratory J*, 34: 648-654.
- Johns MW (1991). A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep*, 14: 540–5.
- Jokela M, Virtanen M, Batty GD, (2015). Inflammation and Specific Symptoms of Depression. *JAMA Psychiatry*. Published online November 18, 2015.
- Kazak AE, Nash JM, Hiroto K, Kaslow NJ (2017). Psychologists in patient-centered medical homes (PCMHs): Roles, evidence, opportunities, and challenges. *American Psychologist*, 72: 1-12.
- Kerns, R.D., Turk, D.C., & Rudy, T.E. (1985). The West Haven-Yale Multidimensional Pain Inventory (WHYMPI). *Pain*, 23, 345-356.
- Kilian AJ, Arnoldussen AC, Gustafson DR (2014). Adipokines: a link between obesity and dementia? *Lancet Neurology*, 13: 913–923.

- Kim Y, Na K, Shin K, Jung H, Choi S & Kim J (2007). Cytokine imbalance in the pathophysiology of major depressive disorder. *Progress in Neuropsychopharmacology & Biological Psychiatry*, 31: 1044-1053.
- Koenigsberg MR, Corliss J (2017). Diabetes Self-Management: Facilitating Lifestyle Change. *Am Fam Physician*, 96(6):362-370.
- Kripke DF (2016). Hypnotic drug risks of mortality, infection, depression, and cancer: but lack of benefit. *FLORIDA Research*, online May 2016 doi: 10.12688/floridaresearch.8729.1
- Kushner RF, Ryan DH (2014). Assessment and lifestyle management of patients with obesity: Clinical recommendations from systematic reviews. *JAMA*, 312:943-952.
- Lehrer PM, Vaschillo E, Vaschillo B, Lu SE, Scardella A, et al. (2004). Biofeedback Treatment for Asthma. *Chest*;126;352-361.
- Leung (2016). Hypertension Canada's 2016 Canadian Hypertension Education Program Guidelines for Blood Pressure measurement, Diagnosis, Assessment of Risk, Prevention, and Treatment of Hypertension. *Canadian JI Cardiology*, 32:569-588.
- Leung CW, Fung TF, McEvoy CT, Lin J, Epel ES (2018). Diet Quality Indices and Leukocyte Telomere Length among Healthy US Adults: Data from the National Health and Nutrition Examination Surveys (NHANES) 1999-2002. *American Journal of Epidemiology*, DOI: 10.1093/aje/kwy124.
- Liberto JG, & Oslin DW (1995). Early versus late onset of alcoholism in the elderly. *International Journal of the Addictions*, 30(13-14):1799-1818.
- Lin EH, Katon W, Von Korff M, Rutter C, Simon GE (2004). Relationship of depression and diabetes self-care, medication adherence and preventative care. *Diabetes Care*, 27(9): 2154-2160
- Lin F, Roiland R, Heffner K, Johnson M, et al. (2014). Evaluation of Objective and Perceived Mental Fatigability in Older Adults with Vascular Risk. *Journal of Psychosomatic Research*, 76(6), 458-464. <http://doi.org/10.1016/j.jpsychores.2014.04.001>
- Lund-Sorensen H, Bemos ME, Madsen T, Sorensen HJ, Eaton WW, et al. (2016). A nationwide cohort study of the association between hospitalization with infection and risk of death by suicide. *JAMA Psychiatry*, 73: 212-219.
- Makris UE, Abrams RC, Gurland B, Carrington Reid M (2014). Management of persistent pain in the older patient: A clinical review. *JAMA*, 312(8):825-836.

- Manber R, Edinger JD, Gress JL, San Pedro-Salcedo MG, Kuo TF, Kalista T (2008). Cognitive behavioral therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. *Sleep*, 31: 489–95.
- Mancini JG, Filion KB Atallah R, Eisenberg MJ (2016). Systematic Review of the Mediterranean Diet for Long-Term Weight Loss. *Am J Medicine*, 129:407-415
- Marin IA, Goertz JE, Ren T, Rich SS, Onengut-Gumusc S, et al. (2017). Microbiota alteration is associated with the development of stress induced despair behavior. *Nature/Scientific Reports*, 7, 43859; doi: 10.1038/srep43859.
- Mark TL, Levit KR, Buck JA (2009). Datapoints: psychotropic drug prescriptions by medical specialty. *Psychiatric Services*, 60(9):1167.
- Martinez C, Richardson C, Han M, Cigolle C, Martinez CH, et al (2014). Chronic obstructive pulmonary disease, cognitive impairment, and development of disability: The Health and Retirement Study. *Annals American Thoracic Society*, 11:1362–1370.
- May H (2017). Depression doubles long-term risk of death after heart disease diagnosis. *American College of Cardiology (ACR) 66th Annual Scientific Session & Expo*, Mar 17-19.
- Mayer EA, Knight R, Mazmanian SK, Cryan JF, Tillisch K. (2014). Gut microbes and the brain: paradigm shift in neuroscience. *The Journal of neuroscience: the official journal of the Society for Neuroscience*, 34:15490–6.
- McCarthy B, Casey D, Devane D, Murphy K, Murphy E, Lacasse Y (2015). Pulmonary rehabilitation for chronic obstructive pulmonary disease. *Cochrane Database of Systematic Reviews 2015, Issue 2. Art. No.: CD003793. DOI: 10.1002/14651858.CD003793.pub3*
- McDonald S, O'Brien N, White M, Sniehotta FF (2015). Changes in physical activity during the retirement transition: a theory-based, qualitative interview study. *International Journal of Behavioral Nutrition and Physical Activity*, 12(25):1-12. DOI: 10.1186/s12966-015-0186-4
- Melzack R (1987). The short-form McGill Pain Questionnaire. *Pain*, 30: 191-197.
- Messaoudi M, Lalonde R, Violle N, Javelot H, Desor D, Nejdj A, Bisson JF, Rougeot C, Pichelin M, Cazaubiel M, Cazaubiel JM (2011). Assessment of psychotropic-like properties of a probiotic formulation (*Lactobacillus helveticus* R0052 and *Bifidobacterium longum* R0175) in rats and human subjects. *British Journal of Nutrition*, 105:755-764.

- Messier SP, Mihalko SL, Legault C, Miller GD, Nicklas BJ, et al. (2013). Effects of intensive diet and exercise on knee joint loads, inflammation, and clinical outcomes among overweight and obese adults with knee osteoarthritis: The IDEA randomized clinical trial. *JAMA*, 310(12): 1263-1273.
- Mezuk B, Eaton WW, Albrecht S, Golden SH (2008). Depression and type 2 diabetes over the lifespan: a meta-analysis. *Diabetes Care*. 31:2383–2390.
- Miller AH, Maletic V, Raison CL (2009). Inflammation and its discontents: The role of cytokines in the pathophysiology of major depression. *Biol Psychiatry*, 65:732-741.
- Miller, G (2011). Why Loneliness Is Hazardous to Your Health. *Science*, 331: 138–140.
- Millon T, Antoni MH, Millon C, Meagher S, Grossman S. (2001). Test manual for the Millon Behavioral Medicine Diagnostic (MBMD) Minneapolis, MN: National Computer Services.
- Mitchell AJ, Rao S, Vaze A (2010). Do primary care physicians have particular difficulty identifying late-life depression? A meta-analysis stratified by age. *Psychother Psychosom*, 79(5):285-94
- Mitsonis CI, Potagas C, Zervas I & Sfagos K (2009). The effects of stressful life events on the course of multiple sclerosis: A review. *International JI of Neuroscience*, 119(3): 315-335.
- Moin T, Li J, Duru K, Ettner S, Turk N, et al. (2015). Metformin Prescription for Insured Adults With Prediabetes From 2010 to 2012: A Retrospective Cohort Study. *Annals of Internal Medicine*, 162:542-548.
- Monnat SM, Chandler RF (2015). Long Term Physical Health Consequences of Adverse Childhood Experiences. *The Sociological Quarterly*, 56(4): 723–752.
- Morin CM, Belleville G, Belanger L, Ivers H (2011). The insomnia severity index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep*, 34:601-608.
- Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, Lichstein KL (2006). Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004). *Sleep*. 29: 1398–414.
- Moukdad AH, Marks JS, Stroup DF, Gerberding JL (2004). Actual causes of death in the United States, 2000. *JAMA*, 291:1238-1245.
- Moussavi S, Chatterji S, Verdes E, Tandon A, Patel V, Ustun B (2007). Depression, chronic diseases, and decrements in health: results from the World Health Surveys. *Lancet*, 370 (9590):851-8.

- Moyer VA (2012). Screening for and management of obesity in Adults: U.S. Preventive Services Task Force Recommendation. *Annals of Internal Medicine*, 157:373-378.
- National Center for Health Statistics. Survey Description, National Health Interview Survey, 2012. Hyattsville, Maryland. 2014.
- Naugle KM (2016). The role of regular physical activity and exercise in the prevention of chronic pain in older adults. *Practical Pain Management*, 16(9):34-39.
- Neufeld KA, Foster JA (2009). Effects of gut microbiota on the brain: Implications for psychiatry. *J Psychiatry and Neuroscience*, 34:230-231.
- Ng Q, Peters C, Ho C, Lim D, Yeo WS (2018). A meta-analysis of the use of probiotics to alleviate depressive symptoms. *L Affective Disorders*, 228:13–19. DOI: <https://doi.org/10.1016/j.jad.2017.11.063>
- NICE (2018). Smoking cessation interventions and services [C] Evidence reviews for advice on e-cigarettes on general sale. NICE guideline NG92 (March) Evidence reviews, Public Health Internal Guideline Development Team: UK.
- Nicolucci A, Kovacs Burns K, Holt RI, Comaschi M, Hermanns N, et al. (2013). Diabetes Attitudes, Wishes and Needs second study (DAWN2): cross-national benchmarking of diabetes-related psychosocial outcomes for people with diabetes. *Diabetes Medicine*, 30:767–777.
- Connor EA, Perdue LA, Senger CA, Rushkin M, et al., (2018). Screening and Behavioral Counseling Interventions to Reduce Unhealthy Alcohol Use in Adolescents and Adults: Updated Evidence Report and Systematic Review for the US Preventive Services Task Force. *JAMA*, 320(18):1910-1928.
- Donohue WT, Cummings NA, Cucciare MA, Runyan CN, Cummings JL (2006). *Integrated behavioral healthcare: A guide to effective intervention*. Amherst, NY: Humanities Books.
- Pachana N, Byrne G, Siddle H, Koloski N, Harley E, Arnold E (2007). Development and validation of the Geriatric Anxiety Inventory. *International Psychogeriatrics*, 19, 103-114.
- Park M, Unützer J (2011). Geriatric Depression in Primary Care. *Psychiatric Clin North Am*. 34(2): 469-487.
- Patel KV, Guralnik JM, Dansie EJ, Turk DC (2013). Prevalence and impact of pain among older adults in the United States: findings from the 2011 National Health and Aging Trends Study. *Pain*, 154:2649-57.
- Patient Protection and Affordable Care Act, 42 U.S.C. § 18001 (2010).

- Pedersen SS, von Känel R, Tully PJ, Denollet J (2017). Psychosocial perspectives in cardiovascular disease. *European JI Preventive Cardiology*, 24_suppl: 108-115.
- Perissinotto CM, Cenzer IS, Covinsky KE (2012). Loneliness in Older Persons: A Predictor of Functional Decline and Death. *Archives of Internal Medicine*, 172:1078-1084.
- Pescatello LS, Riebe D, Arena R (2013). *ACSM's Guidelines for Exercise Testing and Prescription*, 9th ed. Baltimore: Lippincott Williams & Wilkins.
- Piercy KL, Troiano RP, Ballard RM, et al. (2018). The Physical Activity Guidelines for Americans [published online November 12, 2018]. *JAMA*. doi:10.1001/jama.2018.14854
- Pollack A. (2013). A.M.A. Recognizes obesity as a disease. June 18, 2013. Available from: <http://www.nytimes.com/2013/06/19/business/ama-recognizes-obesity-as-a-disease.html>.
- Pothirat C, Phetsuk N, Liwsrisakun C, Deesomchok A (2015). Real-world comparative study of behavioral group therapy program vs education program implemented for smoking cessation in community-dwelling elderly smokers. *Clinical Interventions in Aging*, 10: 725-730.
- Psaltopoulou T, Sergentanis TN, Panagiotakos DB, Sergentanis IN, Kosti R, et al. (2013). Mediterranean diet, stroke, cognitive impairment, and depression: A meta-analysis. *Annals Neurology*, 74:580-591.
- Qaseem A, Wilt TJ, Rich R, Humphrey LL, Frost J, et al. (2017). Pharmacologic Treatment of Hypertension in Adults Aged 60 Years or Older to Higher Versus Lower Blood Pressure Targets: A Clinical Practice Guideline From the American College of Physicians and the American Academy of Family Physicians. *Ann Intern Medicine*, 166: 430-437
- Qaseem A, Wilt TJ, Kansagara D, Horwitch C, Barry MJ, Forciea MA, et al. (2018). Hemoglobin A1c Targets for Glycemic Control With Pharmacologic Therapy for Nonpregnant Adults With Type 2 Diabetes Mellitus: A Guidance Statement Update From the American College of Physicians. *Ann Intern Medicine*, 168:569-576. doi: 10.7326/M17-0939
- Rao AV, Bested AC, Beaulne TM, Katzman MA, Iorio C, Berardi JM, Logan AC (2009). A randomized, double-blind, placebo-controlled pilot study of a probiotic in emotional symptoms of chronic fatigue syndrome. *Gut Pathogens*. 1:6.
- Reid MC, Eccleston C & Pillemer K (2015). Management of chronic pain in older adults. *The BMJ*, 350, h532. <http://doi.org/10.1136/bmj.h532>

- Rodriguez JC, Dzierzewski JM, Alessi CA (2015). Sleep problems in the elderly. *Medical Clinics of North America*, **99**:431-439.
- Rutledge T, Reis SE, Olson M, Owens J, Kelsey SF, Pepine CJ (2006). Depression is associated with cardiac symptoms, mortality risk, and hospitalization among women with suspected coronary disease: The NHLBI-sponsored WISE study. *Psychosomatic Medicine*, **68**; 217-223.
- Saunders JB, Aasland OG, Babor TF, de la Fuente JR, Grant M (1993). Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption--II. *Addiction*, **88**(6):791-804.
- Schilders KA, Eenjes E, van Riet S, Poot AA, Stamatialis D, et al. (2016). Regeneration of the lung: Lung stem cells and the development of lung mimicking devices. *Respiratory Research*, **17**:44.
- Schneiderman N, Ironson G & Siegel SD (2005). Stress and health: Psychological, behavioral, and biological determinants. *Annual Review of Clinical Psychology*, **1**: 607-628.
- Shah A, Hayes CJ & Martin BC (2017). Characteristics of Initial Prescription Episodes and Likelihood of Long-Term Opioid Use — United States, 2006–2015. *Morbidity and Mortality Weekly Report* Vol 66:10, September 2017.
- Shaw AC, Goldstein DR, Montgomery RR (2013). Age-dependent dysregulation of innate immunity. *Nature Reviews: Immunology*, **13**: 875-87.
- Schonfeld, L., King-Kallimanis, B. L., Duchene, D. M., Etheridge, R. L., Herrera, J. R., Barry, K. L., & Lynn, N. (2010). Screening and brief intervention for substance misuse among older adults: the Florida BRITE project. *American journal of public health*, **100**(1):108–114.
- Sinnige J, Korevaar JC, Westert GP, Spreeuwenberg P, Schellevis FG, Braspenning JC (2015). Multimorbidity patterns in a primary care population aged 55 years and over. *Family Practice*, **32**(5):505-13.
- Smith SMS, Sonogo S, Ketcheson L (2014). A review of the effectiveness of psychological interventions used for anxiety and depression in chronic obstructive pulmonary disease. *BMJ Open Respiratory Research*, **1**:e000042
- Smith JA, Das A, Ray SK, Banik NL. (2012). Role of pro-inflammatory cytokines released from microglia in neurodegenerative diseases. *Brain Res Bulletin*, **4**;87:10-20
- Solomon DH, Rassen JA, Glynn RJ et al. (2010). The comparative safety of opioids for nonmalignant pain in older adults. *Archives Internal Medicine*, **170**(22):1979-1986.

- Stenton C (2008). The MRC breathlessness scale. *Occupational Medicine*, 58(3): 226–227.
- Stead LF, Lancaster T (2005). Group behavior therapy programmes for smoking cessation. *Cochrane Database of Systematic Reviews*, 2:CD001007.
- Strobbe S (2014). Prevention and screening, brief intervention, and referral to treatment for substance use in primary care. *Primary Care*, 41(2):185-213.
- Surtees PG, Wainwright NWJ, Bockholdt, Lubren RN Warcham N, & Khaw KT (2008). Major depression, C-reactive protein, and incident ischemic heart disease in healthy men and women. *Psychosomatic Medicine*, 70: 850-855.
- Tan MP, Morgan K (2015). Psychological interventions in cardiovascular disease: An update. *Current Opinion in Psychiatry*, 28:371-377.
- Taylor SE, Way BM, Seeman TE (2011). Early adversity and adult health outcomes. *Development and Psychopathology*, 23: 939-954.
- Tharrett SJ and Peterson JA (2012). *ACSM's Health/Fitness Facility Standards and Guidelines 4th ed.* Champaign, IL: Human Kinetics.
- The GBD 2015 Obesity Collaborators (2017). Health Effects of Overweight and Obesity in 195 Countries over 25 Years. *New England Journal of Medicine*, June 12, 2017; DOI: 10.1056/NEJMoa1614362
- The Look AHEAD Research Group. (2014). Eight-Year Weight Losses with an Intensive Lifestyle Intervention: The Look AHEAD Study. *Obesity* (Silver Spring, Md.), 22(1), 5–13.
<http://doi.org/10.1002/oby.20662>
- Thombs BD, Bass EB, Ford DE, Stewart KJ, Tsilidis KK, et al. (2006). Prevalence of depression in survivors of acute myocardial infarction. *Journal of General Internal Medicine* 21: 30–38.
- Thompson H, Peters JC, Hill J (2013). *ACSM's Guidelines for Exercise Testing and Prescription*, 9th ed. Baltimore: Lippincott Williams & Wilkins.
- Tobias DK, Chen M, Manson JE, Ludwig DS, Willett W, Hu FB (2015). Effect of low-fat diet interventions versus other diet interventions on long-term weight change in adults: a systematic review and meta-analysis. *Lancet Diabetes Endocrinology*, 3:968-979.
- Toobert DJ, Hampson SE, Glasgow RE (2000). The Summary of Diabetes Self-Care Activities Measure. *Diabetes Care*, Volume 23, Number 7.
- Trumper A, Appleby L (2001). Psychiatric morbidity in patients undergoing heart, heart and lung, or lung transplantation. *Jl Psychosomatic Research*, 50:103-105.

- Tselebis A, Pachi A, Ilias I, Kosmas E, Bratis D, et al. (2016). Strategies to improve anxiety and depression in patients with COPD: a mental health perspective. *Neuropsychiatric Disease Treatment*, 12: 297–328.
- Turk DC, Winter F (2006). *The Pain Survival Guide: How to Reclaim Your Life*. Washington DC: American Psychological Association,
- US Department of Health and Human Services. (2010). *Multiple chronic conditions: A strategic framework—Optimum health and quality of life for individuals with multiple chronic conditions*. Washington, DC: US Department of Health and Human Services.
- USPSTF (2012). Behavioral counseling interventions to promote a healthful diet and physical activity for cardiovascular disease prevention in adults. <https://www.uspreventiveservicestaskforce.org/Home/GetFileByID/1863>
- Vitiello MV, Moe KE, Prinz PN (2002). Sleep complaints co-segregate with illness in older adults: clinical research informed by and informing epidemiological studies of sleep. *Journal of Psychosomatic Research*, 53:555-559
- Vlaeyen, J & Linton, S. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain*, 85 (3), 317-332.
- Wallace, C. J. K., & Milev, R. (2017). The effects of probiotics on depressive symptoms in humans: a systematic review. *Annals of General Psychiatry*, 16, 14. <http://doi.org/10.1186/s12991-017-0138-2>
- Webster LR, Webster RM (2005). Predicting aberrant behaviors in opioid-treated patients: Preliminary validation of the Opioid Risk Tool. *Pain Medicine*, 6(6): 432-442.
- Welton PK, Carey RM, Aronow WS, et al. (2017). 2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the prevention, detection, evaluation, and management of high blood pressure in adults. *Hypertension*, DOI:2017;HYP.0000000000000065.
- Wheaton AG, Cunningham, TJ, Ford ES, Croft JB (2015). Employment and activity limitations among adults with chronic obstructive pulmonary disease—United States, 2013. *MMWR*. 64:290-295.
- Yeager CA (2014). Behavioral Health Treatment of Older Adults in Primary Care. In: L Hyer (Ed.) *Psychological Treatment of Older Adults*, pp. 249-282. NY: Springer Pub.
- Yohannes AM, Baldwin RC, Connelly MJ (2000a). Mood disorders in elderly patients with chronic obstructive pulmonary disease. *Reviews in Clinical Gerontology*, 10:193-202.

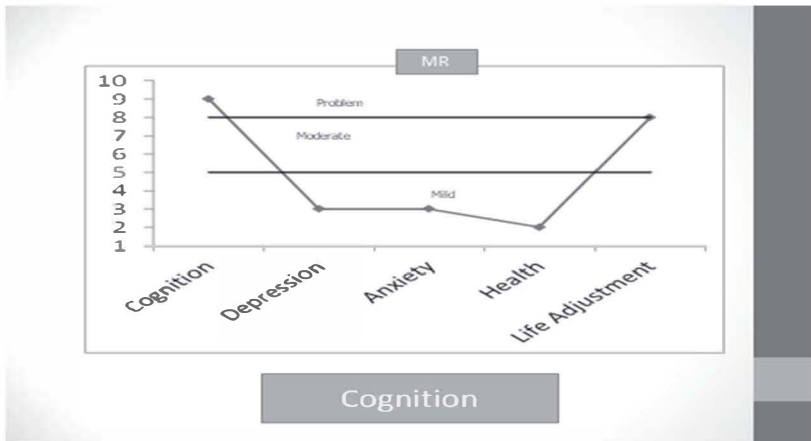
- Yohannes AM, Roomi J, Winn S, Connolly MJ (2000b). The Manchester Respiratory Activities of Daily Living Questionnaire: Development, reliability, validity and responsiveness to pulmonary rehabilitation. *Jl American Geriatric Society*, 48:1496-1500.
- Yoon SS, Fryar CD, Carroll MD (2015). Hypertension Prevalence and Control among Adults: United States, 2011-2014. NCHS Data Brief no. 220. Hyattsville, MD: National Center for Health Statistics.
- Zapata HJ, Quagliarello VJ (2015). The Microbiota and Microbiome in Aging: Potential Implications in Health and Age-related Diseases. *Journal of the American Geriatric Society*, 63: 776-781.
- Zhou L, Foster JA (2015). Psychobiotics and the gut-brain axis: in the pursuit of happiness. *Neuropsychiatric Disease Treatment*, 16: 715-723.
- Ziegelstein RC, Fauerbach JA, Stevens SS, Romanelli J, et al. (2000). Patients with depression are less likely to follow recommendations to reduce cardiac risk during recovery from a myocardial infarction. *Arch Intern Med*, 160:1818-1823.

CHAPTER 7

COGNITION

CHRISTINE MULLEN AND LEE HYER

CASE: MR



This case builds from Chapter 5.

Case MR

PATIENT: MR

DOB: 1948

Screen Problems

Cognition

MMSE = 25/30 raw

MoCA = 23/30 raw

TMT-A = 10%ile

TMT-B = <10%ile

RBANS: RBANS Total = Low memory and new learning

Life Adjustment:

Normal and average SES.

Lives alone but has social contacts. Three episodes of “dissociation” on her own.

Health

Medications: 4 medications

Chronic conditions: HTN, cholesterol, hypothyroidism

Health Rating: 7/10

ESS = 4

Pain: 2/10

Cigarettes: No

Alcohol: 1 drink/day

BMI: 27

Exercise: None, excessive sedentary behavior

Depression: Effexor (10 years)

MINI: Negative for Depression

BDI = 10

PHQ-9 = 5

GDS-SF = 5

MBMD: Normal

Anxiety

GAD-7 = 2

STAI = 12

CHIEF COMPLAINT: Evaluation for cognitive problems.

RECENT INFORMATION: MR was referred by her sister. The patient was driven to this appointment by her sister. Several months prior to the evaluation she was in an accident on the interstate, having driven 70 miles in an apparent dissociative state. She was confused as to why this episode took place. Prior to this event, she had two other episodes that took place over the previous three years. She is a retired teacher who lives locally by herself, and has been functioning well in the community. She is socially active.

RELEVANT DEVELOPMENTAL HISTORY: The patient is a native of rural Georgia. She is the second of four children. Her father was a physician, and her mother had a master’s degree in bioscience. She indicated that her upbringing was positive and there were no milestone disruptions. She completed her bachelor’s degree and received a teaching certificate. She worked as a hotel evaluator in New York for a few years, and then in Georgia for several years. She spent roughly 30 years as a French teacher and retired a year ago. She never married. She lives alone

in a city in Georgia.

RELEVANT MEDICAL HISTORY: Her primary care physician's note indicates that there were no outstanding problems. Her blood chemistries were normal. She did however have hypothyroidism and fatigue. Ms. MR indicated that she had not eaten on the day of the "dissociative event" and it was possible that she had a low blood sugar on the day of the episode. She takes a statin medication, lovastatin, and lisinopril for blood pressure. She has been on Effexor for 10 years. She consumes one glass of wine nightly and denied tobacco use. She denied pain. She had two knee replacements. She indicated that she sleeps well, and that she does not have an exercise program. It is also noteworthy that she has a number of pets in the house, nine cats and several dogs. Indications are that she had two other "episodes." Roughly three years ago she had a syncope episode when she was with students. A year ago she had an accident where she hit her head falling out of a bed and was unconscious for a very short period of time. Her ER visit was uneventful and her MRI was normal. The current incident occurred after she placed her aunt in a nursing facility. She was visiting and attempted to drive home at night, 50 miles away. She drove an hour beyond her home and had an accident where she was on the opposite side of an interstate. She sustained minor injuries and did not lose consciousness. She was brought to the emergency room. Her MRI was normal and she was released the same day. The event was deemed to be an aberrant episode.

MENTAL STATUS: The patient is a 66-year-old Caucasian female who was asked to come to this appointment as a result of her sister's insistence. She was pleasant and answered questions with some specificity. Her speech was normal. She was oriented. She was casually dressed and appeared to have a normal weight. She indicated that she is a messy person, and this has been a pattern in her life for a long period of time. She reported age related memory changes over the past several years. Her mood was euthymic and affect was consistent. There was no evidence of perceptual anomalies or delusional thinking. Her judgment appeared to be reasonably good, and her insight appears to be adequate.

Watch and Wait Summary: This is a 66-year-old Caucasian female who was referred for an evaluation for her cognitive status. She suffered a unique experience where she drove over many miles away from her destination and was in an accident. There was no loss of consciousness and her ER visit was uneventful. She has no explanation but two similar events occurred within the past three years. There were no medical reasons provided. She has a college degree, has taught school for many years, and is retired. She has never married. She lives alone and is independent for all

ADLs and IADLs.

Cognitively, she was high average premorbidly. Her Wechsler Abbreviated Scale of Intelligence-II (WASI-II) Full Scale IQ was in the higher end of the average range (FSIQ SS = 109). Comparatively, her screen showed deficit performance in several areas. Her MoCA was below normal (23/30) with memory at 1/5. Trail Making Test Part A (TMT-A) and Part B (TMT-B) were in the 10th percentile or less for her age and education (no errors). Her overall RBANS was low average (RBANS Total Index SS = 94) but presented with problems in attention (Attention Index = 80) and memory (Delayed Memory Index = 72). Her Continuous Performance Test-2 (CPT-2) performance was deficient (80% Confidence Index for clinical Attention Deficit/Hyperactive Disorder symptoms) and showed problems in processing speed, sustained attention, and heightened omission and commission errors.

It is noteworthy that she had considerable variability in her profile – often a marker of problems. From all indications, she was responding less accurately and sharply than her premorbid state. She indicated she is experiencing age related memory changes but denied other cognitive difficulties. Her level of functioning was reported to be intact (FAQ and IADLs were normal).

Her endorsement of symptoms on psychological/psychiatric assessments was non-significant. She had no Axis I problems noted on the Millon Behavioral Medicine Diagnostic (MBMD) or the Personality Assessment Inventory (PAI). She endorsed a confident personality pattern and a high penchant to be independent and be in control. She is on an antidepressant and does not want therapy.

MR

Psychiatric Indications	AA	3	50				ANXIETY-TENSION
	BB	1	15				DEPRESSION
	CC	5	50				COGNITIVE DYSFUNCTION
	DD	8	70				EMOTIONAL LABILITY
	EE	5	40				GUARDEDNESS
Coping Styles	1	5	60				INTROVERSIVE
	2A	2	60				INHIBITED
	2B	1	15				DEJECTED
	3	4	45				COOPERATIVE
	4	8	50				SOCIABLE
	5	18	88				CONFIDENT
	6A	6	45				NONCONFORMING
	6B	8	45				FORCEFUL
	7	19	60				RESPECTFUL
	8A	6	60				OPPOSITIONAL
	8B	8	75				DENIGRATED

Summary: Ms. MR has been having minor problems over the past several years. With impairments in attention and memory compared to premorbid functioning with reported intact IADLs, she meets criteria for Mild Cognitive Impairment (MCI). Her “dissociative episode” is still unexplained. She defended this by noting that she had not eaten that day. Her brain imaging is unremarkable. She has hypothyroidism, hypertension, and mild cholesterol problems. Her health problems are less of an issue. She is on an anti-depressant. She indicates that does not have an exercise program. Sleep and pain are normal. She lives alone.

Regarding her domains, she is in the Problem area in cognition and with life adjustment as her behavior alone is unclear.

Recommendations were that she attend the Memory Clinic with structured cognitive training (CT) classes. This will optimize her struggling cognition through development and effective implementation of compensatory strategies. It will also assist with socialization. Her family needs to become more active with her care. She also needs to have her health monitored more frequently given these unexplained episodes. She is rather independent and compliance needs monitoring (MBMD). We will also follow her over time with retesting.

Watch and Wait Process

Checklist

Discernment: X (agrees with mental health plan; sister agrees)

Core markers

Validate Problem X

Psychoeducation of Model X

Assessment X

Alliance X

Monitoring X

Case formulation X

Problem List

1. **COGNITION**: Special problems with attention and memory: MCI profile.
2. **LIFE ADJUSTMENT**: Monitor over time and suggest assisted living facility (ALF) if needed. Have her sister assist in mild caregiving.
3. **HEALTH**: This is less an issue but we will monitor with PCP and sister

Self or other monitoring: Mini-targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments:

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Environmental control:

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training: X

Imagery exercise for counter-experience: X

Compassion training: X

Communication and Assertiveness training: X

Behavioral experiments: X

Emotional exposure:

Homework: X

Longer Therapy processes:

CBT: Mild use of 3-6 column evidence record

CT: Computer and clinic

Lifestyles:

- Practice happiness
- Cognitive tasks: Training in group
- Socialize: Less decline and live longer
- Develop resilience
- Exercise: Gym membership
- Diet: Consult nutritionist
- Somatic softening: Meditate and Mindfulness
- Values and Leisure

Re-Test 1 year later:

Post Cognitive Scores:

MoCA: 24/30 (1/5 memory), impaired

MMSE: 25/30, low normal

TMT-B: 20%ile level

RBANS: Total at pre-levels.

Memory: Poor

New Learning: Poor

Depression: Scores normal

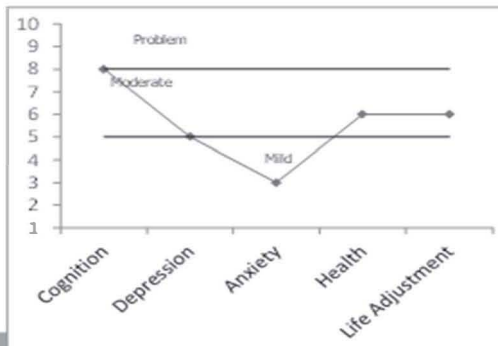
Anxiety: Scores normal

Health: ALF manages problems

Life adjustment: Admitted to an ALF

Post Profile:

Her post-profile is now near normal as she is in an ALF. Her cognition scores were not obtained but she is still having problems. She is active in the facility, attends groups, and is social, but this bears watching. .



Cognition

In this chapter, we begin with our model. We start with a brief background of cognitive issues as these relate to the societal picture. We break down the cognitive domains and highlight Apolipoprotein E (APOE), as well as early onset dementias. We consider general assessment problems of older adults. We then discuss our recommended screening battery and the extended battery, later focusing on more standard neuropsychological tests. We then consider treatment and the lifestyle markers as previously presented in prior chapters. Cognitive training is especially discussed.

Cognitive Model

Measure	Healthy Intact	Mildly Problematic	Problematic or Syndromic
Scale Description	Normal memory and fully oriented, these patients will display good judgment and problem solving skills.	Memory loss that interferes with everyday activities, difficulty with abstraction and problem solving, but social judgments are intact.	Severe memory deficits, inability to retain new information, difficulty with orienting time and place, markedly impaired abstraction and social judgments.
MoCA	27-30	22-26	≤21
RBANS	≥90	75-85	≤74
TMT-A	≤ 46s	47-70	≥ 71
TMT-B	≤ 115s	116-182	≥ 183

As we have discussed, the cognitive aging process can be complex and convoluted. Cognitive decline in late adulthood is becoming one of the nation's primary health concerns. Approximately 5.2 million Americans suffer from AD; 200,000 who are younger than 65 years of age. By 2050, it is believed that the number of people will almost triple to 16 million (Alzheimer's Disease Facts and Figures, 2014). The disease has become the nation's sixth leading cause of death for all ages and the fifth for those 65 and older. The prevalence rate has increased to 68% from 2000 to

2010, and has become the most expensive condition in health care; costing an estimated \$214 billion for the year 2014. It is estimated to cost \$2 out of every \$5 spent on health care for older adults. Additionally, prevalence rates of MCI are estimated to be between 10% and 20% of people 65 years and older (2014 Alzheimer's Disease Facts and Figures, 2014).

The normal aging process for adults includes several structural changes of the brain: decreased volume size of 10% to 15%, less efficient cognitive networks, and potential irreversible metabolic or cellular damage (Sattler & Ryan, 2009). Hence, a main concern for health care providers is what can be done in regard to cognitive decline, whether due to normal aging or a progressive degenerative disorder (Le Couteur & Sinclair, 2010). The NIH Alzheimer's and Cognitive Decline Prevention (Sperling et al., 2011) provided cautious recommendations of how to impede neurodegenerative processes including: exercise, leisure, and cognitive activity (Chapter 5). Systematic review of the literature (Barnes & Yaffe, 2011) found seven potentially modifiable AD risk factors: diabetes, depression, low level of education, mid-life hypertension, mid-life obesity, physical inactivity and smoking. Reduction of these factors was found to reduce prevalence by 10-25%. The updated CDC Healthy Brain Initiative (2011) provided a guideline to maintain a healthy brain that emphasized physical activity and vascular factors, which will be introduced in the treatment section of the chapter. The report also identified cerebral risk factors that include: diabetes, smoking, depression/anxiety, metabolic syndrome, and the presence of the APOE ε4 allele. Smith and Farias (2018) recently published an extensive review of the disease process, assessment, as well as intervention strategies that is beyond the scope of this publication.

It should be noted that a taxonomy of dementia that changes the assessment from a syndromal to a more biological level has been proposed. This updates the guidelines of the National Institute on Aging (Sperling, 2011) for preclinical, MCI and dementia stages of AD. Over time, because many AD patients did not have amyloid plaques or tau tangles, the constructs, 'possible' and 'probable' AD, were simply shortened to AD. In the new framework, AD will be defined by beta amyloid and pathological tau (Jack et al., 2017). A patient who has both markers will be considered to have AD. One with amyloid pathology but not tau would be labeled "Alzheimer pathological change." AD and Alzheimer's pathological change, then, would be phases on a continuum. Neurodegenerative biomarkers and cognitive symptoms would be used only to stage severity, and not for presence on the continuum. Depression would presumably fit into the severity staging and not continuum presence. However, this is a research proposal.

Cognitive decline is not limited to the presentation of plaques and tangles. Self-report surveys conducted in 21 states by the CDC showed 12.7% of Americans 60 years of age and older reported memory loss or increased confusion and functional difficulties. Eighty-one percent of these individuals did not consult a health care provider [Centers for Disease Control and Prevention (CDC), 2013]. These results could be attributed to an imbalance of negative over positive health habits that affect brain health in early/middle life (e.g., Barnes & Yaffe, 2011; Kivipelto, 2001). The normal aging process can lead to cognitive difficulties; subjective memory impairment (SMI) causes even more problems.

Background

Cognitive assessment is essential to fully understand the dynamic presentation of elderly adults, regardless of reason for referral. This is especially true within the elderly population in the differentiation between affect and cognitive changes (e.g., in years past labeled “pseudo dementia”), functional changes, progression of cognitive dysfunction, capacity, and prognosis. There is a plethora of cognitive assessment measures that cover the gamut of functioning. As competent clinical practitioners, it is pertinent to have a thorough understanding of the appropriateness of the tests and their merits and weaknesses. Meyer et al., (2001) maintain that psychological assessments are as valid as most medical tests; therefore, it is essential to have a working knowledge of the nuances of test features and potential implications regarding different populations. Specific to the elderly population, cognitive assessment is a key marker for adjustment in almost all areas of life (Hyer, 2014). Neuropsychological outcomes are strongly correlated with activities of daily living, especially instrumental activities of daily living (Richardson, Nadler, & Malloy, 1995). Thus, cognitive assessment within the elderly population should be routine despite the referral question; the reduction of cognitive functioning may account for presentation of difficulties within other areas of functioning.

We now re-address the continuum of cognitive decline as a progression from normal cognition to age associated memory impairment (AAMI) followed by SMI, MCI, then dementia. As we previously suggested, AAMI is considered to be a normal progression of age that presents with little conversion to dementia. We address here the more pathological side of the continuum starting with SMI. One challenge faced by practitioners is determining appropriateness of assessment tools to answer the referral

question. It is important to be mindful of cognitive domains being assessed and to include different aspects of those domains (e.g., auditory and visual memory). Another area of difficulty for clinicians is access to appropriate well-validated and reliable measures.

Subjective Memory Impairment

SMI is another “pre” marker in the armamentarium of incipient disease states. The relationship between subjective cognitive concerns and neuropsychiatric symptoms to each other and their progression in preclinical AD or any dementia remain undefined. Efforts are being made to develop criteria and measures for SMI, which examines several features in addition to cognitive factors. For example, The Subjective Cognitive Decline–Questionnaire (Rami, et al., 2014) assesses problems of SMI, including worry about potential cognitive decline, self-belief of cognition is worse than others of similar age, and subjective memory decline. The measure also considers collateral confirmation and the presentation of APOE ε4 and other biomarkers. Comparatively, the Cognitive Difficulties Scale (McNair & Kahn, 1983) assesses subjective complaints on a Likert scale pertaining to orientation, attention, memory, language, and psychomotor functioning. Other assessment methods include observation of patient behaviors in primary care clinics. These can include inability to list current medications, being a poor historian, denial of obvious depression, and inability to recall recommendations. None of these are validated but the interest and appeal is in their face validity, a real state prior to MCI that has markers.

SMI seems to be a real phenomenon. Jessen and colleagues (2010) asked adults 75 and older ($N=2415$) if memory was a problem and if they worry. Over time participants with SMI and no worry converted to a dementia two times over baseline, those with SMI and worry conversion rate was six fold, while those with MCI were ten times more likely to convert. Additionally, SMI is associated with depression, anxiety, and neuroticism in cross-sectional studies of community-dwelling older adults with and without objective cognitive impairment and that being considered by many to be an indication of poor mental health and unrelated to cognitive decline and AD. Donovan et al., (2014) attempted to identify neuropsychiatric and cognitive predictors of clinical progression across early stages of AD within the cohort of older adults initially classified as MCI. They found that the greater symptoms of depression, irritability, and agitation, as well as lower memory and somatic processing at baseline predicted more rapid progression to a worse prognosis across all three

groups. These findings support the model of AD in which cognitive and neuropsychiatric alterations are measurable before the stage of MCI and offer the potential to enhance early detection and intervention. As discussed before, Merrill et al., (2016) added to this. MCI and SMI patients with levels of physical exercise, adherence to the Mediterranean diet, and BMI were compared with levels of plaques and tangles. The results were intriguing: MCI with higher BMI or lower physical exercise showed more amyloid binding; for both MCI and SMI, lower adherence to the diet had more amyloid binding. The lesson is to live a good lifestyle and SMI seems to matter. Perhaps also the lesson is that SMI is a problem.

Finally, function is a competitor for the influence of problems with life. Many cases where there are cognitive problems are really functional in origin. This can present as Functional Memory Disorder (FMD). This has been suggested as a candidate in the continuum of decline where the person holds the line cognitively. According to patients' account, nonorganic memory disorder encompasses credible, frequent, and significant anamnestic and attentional deficits in daily living. The proposed diagnostic criteria by Schmidtke, Pohlmann, and Mettemich (2003) is provided below. Another construct is Subjective Cognitive Decline (SCD) and emphasizes a subjective decline in memory, as well as affect and biomarkers. The below proposed criteria were developed from the SCD-Initiative (Miebach et al., 2019).

Criteria

FMD

- Complaint of acquired dysfunction of memory for at least 6 months that significantly impacts functioning
- Presence of external and/or subjective factors addressed as psychosocial burden causing significant distress
- Verbal memory and attentional capacity above -1.5 SD on standardized cognitive tests
- Absence of recognizable organic cause of cognitive impairment
- Absence of a major psychiatric disease

SCD (SCD-I)

- Subjective decline in memory
- Onset of SCD in last 5 years
- Age of onset >60
- "Worry" about SCD
- Feeling worse performance compared to peers
- Informant confirmation of perceived decline
- APOE ε4

Along with the upstream variable SMI, there is at least one other variable of note. Donahue and colleagues (2015) examined longitudinal cognitive and biomarker information from 445 cognitively normal adults from the United States and Canada, as part of the ADNI study. Of these individuals, 243 presented with normal amounts of amyloid while 202 had elevated amyloid. Individuals were followed for a median of 3.1 years. Mean cognitive scores as assessed with the MMSE and a contextual memory test decreased over time that was attributed to the function of amyloidosis. The group with elevated levels of amyloid had worse cognitive scores on multiple measures at approximately 4 years. Edmonds et al. (2015) also examined pre-clinical patients within the ADNI database. Results showed neuro-degeneration was 2.5 times more common at baseline than solely amyloidosis. Further, neuro-degeneration was the most common single biomarker at baseline in those who later progressed to MCI or AD. Thus, authors propose that characterization of preclinical AD may be more well-defined than previously considered.

Mild Cognitive Impairment

MCI, now labeled as Mild Neurocognitive Disorder (mild NCD) in the 5th edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5, American Psychiatric Association, 2013); has several definitions but collectively presents as either an amnesic or non-amnesic decline with lack of significant interference of independent performance of IADLs. Specifically, DSM-5 criteria of mild NCD is characterized as modest cognitive decline from a previous level of performance in one or more cognitive domains (complex attention, executive function, learning and memory, language, perceptual motor, or social cognition) with potential requirement of “greater effort, compensatory strategies, or accommodation” for independent performance of IADLs (pp. 611, American Psychiatric Association, 2013). A modest level of decline is defined as “typically 1-2 standard deviation range (between the 3rd and 16th percentiles)” (pp. 607, American Psychiatric Association, 2013). In regard to subtypes, the field in general has adopted the classifications developed by Petersen (2004) of distinguishing deficits as either single versus multidomain and amnesic versus non-amnesic.

Comparatively, dementia, now defined as major NCD (American Psychiatric Association, 2013) presents with problems in cognition that typically involves multiple domains and deficits in functional abilities (i.e., IADLs and ADLs). Areas that are most impacted from degeneration subserve fluid intelligence that is subject to mediators, such as social

economic status, education, and life habits. Neuropsychological testing performance is “typically 2 or more standard deviations below appropriate norms (3rd percentile or below)” (pp. 607, American Psychiatric Association, 2013). However, it is typical for the average person to have reduced performance in at least one area of assessment, without the presentation of organic impairment. Thus, the mediation process between these levels of functioning is often difficult, requiring clinical judgment.

Mild NCD is a strategic construct that allows clinicians to closely monitor the change in presentation as well as intervene early in the process. Individuals typically present as less efficient and make more errors when performing complex functional tasks such as preparing a meal, shopping, or financial management. Areas of cognitive functioning that can be impacted include memory, executive function, attention, language, and visuospatial skills (Albert et al., 2011). Studies have demonstrated protective brain factors pertaining to the degenerative process. These factors include life skills such as intelligence, occupational complexity, active engagement of leisure, computer games, stress reduction, and healthy lifestyles (Hyer, Mullen, & McKenzie, 2015). Given the potential complexity of a cognitive profile, it is important for clinicians to examine all of these areas of functioning.

Conversion rates from MCI to AD are highly variable in the literature. A systematic review of longitudinal studies found an overall rate of 10% per year; high variability was present in the studies reviewed (Bruscoli & Lovestone, 2004). This variability observed in the literature can be attributed to differences in samples, measures, and interrater reliability of diagnosis. For example, Morris et al., (2001) found that 100% of MCI subjects in Memory Disorders Clinic progressed to a diagnosis of dementia over a period of 9.5 years, 84% met criteria for AD type. Comparatively, a systematic review of the epidemiological studies found an average reversion rate from MCI to normal functioning of 20% (Roberts & Knopman, 2013). However, those who have met criteria for MCI and reverted back to “normal” have a greater likelihood of progressing to MCI or dementia than those who never developed MCI. These findings further support the importance of awareness of symptoms and health provider intervention. The progression of cognitive changes needs to be monitored in order to enhance functioning and the development of compensatory strategies.

Dementia

Alzheimer's disease is the most prevalent neurodegenerative dementing disorder. Approximately 95% of AD is sporadic or late onset (Bird, 2015). The most studied biomarkers to date include histopathological counts of plaques and tangles, oxidative stress, genetic markers (e.g., APOE ε4), white matter hyperintensities, global cortical atrophy, dopamine receptor binding, and amyloid loadings. Additionally, several cerebrospinal fluid biomarkers have demonstrated some efficacy in identifying the disease: 42 amino acid *B*-amyloid peptide, total tau protein, phosphorylated tau (Henry et al., 2013). Ferreira et al., (2014) postulate that the combination of cerebral spinal fluid biomarkers can be used to diagnose MCI, more accurately for amnesic MCI. However, these biomarkers are not able to distinguish AD from other forms of dementia. Consequently, cognitive assessment is pertinent in the diagnosis of dementia and the differentiation of the several classifications of the disease (Smith & Farias, 2018; Tuokko and Smart, 2016).

The modal presentations of the dementias include but are not limited to: AD, vascular dementia, frontotemporal dementia, dementia with Lewy bodies, Parkinson's dementia, and normal pressure hydrocephalus. Each of these dementia types presents with varying cognitive and affective profiles. Thorough descriptions and research relating to these dementias can be found in Smith & Farias (2018) and Tuokko and Smart (2016), as well as Lichtenberg (2010) and Parsons and Hammeke (2014). Generically, domains affected by these variations of the disease include attention, memory, language, executive functioning, visuo-perceptual skills, motor dexterity, and social cognition. Decline in these areas leads to difficult decisions for care providers and family members.

●f note, it is relatively common for AD to overlap with cerebrovascular disease (vascular dementia), as much as up to 84% in recent epidemiological data. Diabetes is especially a problem. Specifically, the most frequently observed co-occurring anomalies are cerebral amyloid angiopathy and small vessel disease. However, severity of co-occurrence of prominent presentation can significantly vary (Attems & Jellinger, 2014).

Diagnosis	Typical Cognitive Presentation
Alzheimer's Disease	<p>●<i>onset</i>: Insidious with gradual progression; early onset (i.e., prior to 65 years old) has more rapid progression.</p> <p><i>Early difficulties</i>: Deficits in memory (e.g., consolidation of information, rapid loss of new information, recognition); subtle personality changes. High comorbidity with cerebrovascular disease.</p> <p><i>Progressive difficulties</i>: Deficits in orientation, simple attention, confrontational naming, semantic verbal fluency worse than phonemic fluency; agitation, confusion, apathy, emotional blunting, agnosia, potential delusions and hallucinations; eventual global decline.</p>
Vascular Dementia	<p>●<i>onset</i>: Similar to vascular event(s) or evolution of cerebrovascular disease that can present as fluctuating or step-wise progression.</p> <p><i>Difficulties</i>: Typically involves diminished attention, processing speed, phonemic verbal fluency worse than semantic fluency, visuoconstruction, executive function, psychomotor speed, and fine motor dexterity; depression, anxiety, apathy, and disinhibition. Deficits vary and can be patchy or focal dependent on infarct/disease process.</p>
Frontotemporal Dementia	<p>●<i>onset</i>: Insidious with gradual progression.</p> <p><i>Early difficulties</i>: Deficits in executive function and language; behavioral disinhibition, emotional blunting, changes in personality - dependent on variant of disease (i.e., behavioral, primary progressive aphasia, semantic).</p> <p><i>Progressive difficulties</i>: Deficits in memory, sustained attention, visuospatial abilities; apraxia, apathy, extreme agitation, akinesia, rigidity, tremor - dependent on variant of disease.</p>
Dementia with LewyBodies	<p>●<i>onset</i>: Insidious with a generally more rapid decline compared to AD. Cognitive changes typically present prior to motor dysfunction but they can co-occur. Can be comorbid with AD.</p> <p><i>Early difficulties</i>: Deficits in attention, visuoperceptual/construction, and executive function,</p>

	<p>daily fluctuation in cognition; hallucinations and delusions, syncope, falls, transient loss of consciousness, autonomic dysfunction, spontaneous parkinsonism.</p> <p><i>Progressive difficulties:</i> Deficits in verbal fluency and memory.</p>
Parkinson's Dementia	<p><i>Onset:</i> Insidious with slow progression. Motor dysfunction presents before cognitive difficulties by at least one year. Can be comorbid with AD.</p> <p><i>Early difficulties:</i> Executive dysfunction and attention deficits; micrographia.</p> <p><i>Progressive difficulties:</i> Deficits in visuospatial processing/construction, confrontation naming, verbal fluency, learning and memory; apathy, changes in personality, potential hallucinations and delusions, depression, or anxiety.</p>

Accounting for the identification of dementia also needs consideration. Gender may make a difference. When it comes to verbal memory skills women have the edge over men, yet in AD this cognitive reserve may not confer an advantage but rather mask early signs of the disease. For example, a recent analysis of more than 900 participants in the Alzheimer's Disease Neuroimaging Initiative (ADNI), all of whom had an amnesic MCI (aMCI) or AD dementia, and almost 400 healthy controls showed that women in all three groups had better verbal memory scores at baseline than men (Sundermann, Maki, Rubin, Lipton, Landau, & Biegon, 2016). High temporal lobe glucose metabolic rates (TLGluMR) were higher in the full group of women than in men, and were linked to higher scores on the Rey Auditory Verbal Learning Test (RAVLT). After stratification for diagnosis, only the women with aMCI had a significant association between high TLGluMR and high verbal memory skills, suggesting an accelerated decline in those with AD dementia. The female advantage in verbal memory varied by TLGluMR, such that the advantage was greater among individuals with moderate to high TLGluMR and minimal or absent among individuals with lower rates.

Right now, memory test norms or cutoff scores are used to determine whether someone is impaired, regardless of sex. This may need further examination. The "cognitive reserve" theory proposes that persons with favorable premorbid factors, such as higher education or IQ, maintain normal cognitive function longer as AD-related brain pathology accumulates. For these individuals, cognitive decline is delayed until time

points closer to dementia diagnosis; however, once decline begins, persons with high reserve have more rapid decline because pathology is more advanced.

There are other measures in the assessment of older adults. Difficulties associated with gait have shown to predict significant decline. Savica and colleagues (2017) assessed 3,426 cognitively normal participants between ages 70 to 89 years at baseline and every 15 months. Examination included neurological evaluation, neuropsychological assessment, and gait performance. Diminishment in spatial, temporal, and spatiotemporal components of gait, as well as greater intraindividual variability was significantly associated with future decline in global cognition and domain performance in visuospatial, language, memory, and executive functioning. Thus, computerized analysis of gait may be able to assist in early detection of cognitive changes before impairment is detected with a standard neuropsychological test.

Early Onset Dementia/Early Onset Alzheimer's Disease

Early onset dementia (EOD) or early onset Alzheimer's disease (EOAD) present in those who are diagnosed with AD before the age of 65 years old; typically presenting with initial symptoms between 45 and 60, but as early as 35 years old. In a meta-analysis conducted by Zhu and colleagues (2015) across 13 studies, a prevalence rate of approximately 5.5% was found for EOAD. Importantly, results indicated rates in developed countries were relatively higher compared to developing countries.

Compared with late-onset AD, EOAD demonstrates to have greater neocortical pathology particularly in the parietal cortex, greater tau compared with amyloid burden, and less hippocampal disease (Menendez 2017). There are several variants of EOAD that have been identified, with ranging presentations. These include logopenic progressive aphasia and posterior cortical atrophy (PCA). The literature also suggests bipartial phenotype including progressive ideomotor apraxia, and a dysexecutive variant.

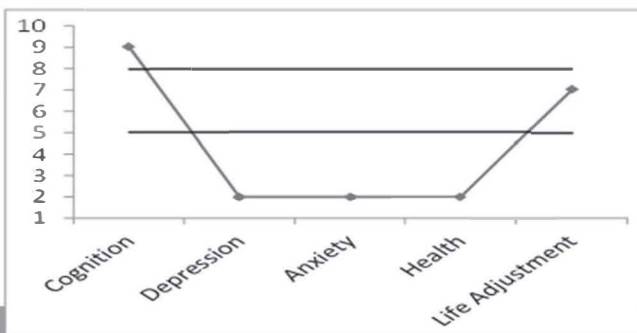
Menendez et al., (2017) provides a review of the differences between AD and EOAD presentation. In general, these include reduced cognitive disorder, greater insight, greater depression, increased progression in course of disease, relatively larger deficits in attention, praxis, visuospatial functioning, and executive functioning. Importantly, EOAD typically takes longer to diagnose, given its low prevalence rate and behavioral/psychiatric presentation is initially the focus of treatment.

Case 1: EOAD

Below is a brief case of a 60 year-old African American female who had a chief complaint of memory problems. She has recently been placed on Aricept by her primary care physician. She is married and has a small child. Her husband is quite successful and very supportive. He is now most concerned. She is a college graduate and had been a teacher until she was let go in the last few months due to incompetence. She was also an officer in the army for 20 years. She takes only the one medication, has a normal MRI, and is content in life but for her memory. She was also active in the community. She has no psychiatric history.

JJ's MoCA was impaired (16/30), with memory at 0/5. Her WASI-II FSIQ was low average (FSIQ SS = 84). Her CPT-2 was very poor (99% Confidence Index for clinical Attention Deficit/Hyperactive Disorder symptoms), with high commissions and omission errors, and high variability and perseveration. Her RBANS is borderline (RBANS Total Index SS = 75) with poor memory and immediate memory, as well as visuospatial skills. Her executive functioning is also very low (TMT-B, Matrix Reasoning, Block Design, WCS). Her IADL skills are also in decline: driving, cooking, math and simple problem solving. She is normal on all psychological scales for anxiety, depression, pain, sleep, and somatic issues. Her modal domain problems include cognition and her life adjustment. She appeared to have an early onset dementia.

JJ



APOE ϵ 4

The APOE ϵ 4 allele is the strongest genetic risk factor for late onset AD and is associated with earlier age of onset. This allele is associated with an increased risk for other types of dementia and MCI. Individuals with MCI who also have an ϵ 4 allele are at greater risk of progressing to AD than those without the allele. The APOE is associated with the transport of lipids and cholesterol, neuroplasticity, neurogenesis, and inflammation response in the brain, all of which may contribute to the manifestation of disease symptoms. Individuals with an APOE ϵ 4 allele who do not have cognitive impairment exhibit faster age-related loss of the hippocampal volume than those without the allele. Hippocampal atrophy is one of the biomarkers identified by the NIA Alzheimer's Association Work Group that reflects neuronal injury in individuals with Alzheimer's disease (Poey, Burr, and Roberts, 2017). Corsentino and colleagues (2009) found that although both depression and the APOE ϵ 4 allele were associated with greater risk of cognitive difficulty, the relationship between the ϵ 4 allele and cognition was greater than those with depressive symptoms. So, depression itself as well as anxiety are predictors of dementia but this is more the case with an APOE ϵ 4 positive state (Mackin et al., 2104).

Several studies have also examined the interaction of the social context with the APOE ϵ 4 allele. Perceived isolation to feelings of loneliness and perception of social support has shown to be related to the ApoE-4 allele (Zhao et al., 2005). Poey et al. (2017) found that the ϵ 4 allele and being less socially engaged were independently associated with a greater risk of AD. Specifically, not married and living alone were associated with greater risk of non-Alzheimer's dementia when compared with persons who are married. Self-respondents who reported to feel lonely were also at greater risk of cognitive difficulty compared to those not reporting loneliness. Increased social engagement levels were associated with less risk of any type of cognitive difficulty. These findings supported the many effects of the hypothesis that the richer social environment is associated with less risk of cognitive decline and the presence of APOE ϵ 4 allele was related to poor cognitive health.

Overall, the genetic risk is stronger for AD than for other types of dementia or cognitive difficulty. These findings have implications for genetic testing and counseling for AD. Although predictive testing for AD using the APOE ϵ 4 genotyping is not currently recommended clinically, it is of considerable interest to family members of AD disease patients and has been successfully implemented in controlled research settings.

We note here that dementing disorders organized by their molecular classification of abnormal protein deposition reveals a complex taxonomy of origins for degenerative diseases. The major dementias (AD, PD and FTD) can be organized by amyloidosis, tauopathies, a-synucleinopathies and the transactive response DNA-binding protein 43 kilodation (TDP-43) proteinopathies (Murray & Detrué, 2018). The concordance between clinical phenotype and molecular-level pathology is rather imperfect. It is then not uncommon for a neuropathological entity to target a network not associated with a typical clinical phenotype. There may be very few pure dementias as we have conceived them. In addition, clearly there are vascular contributions to brain degeneration.

General Assessment Issues

There are several issues to consider when assessing cognition with this population. Many factors can contribute to their presentation such as medication side-effects, depression, and pain. Progression of cognitive decline is typically slow and the patient might not be aware of any changes. Therefore, it is important to secure a reliable collateral during the clinical interview in order to develop a thorough and reliable history of the presenting problem as well as any potential contributing factors from the patient's history. A semi-structured interview to assist with this process is presented in subsequent sections.

Another area of concern that many practitioners do not consider is potential ageism that could bias perceptions of the patient. Ageism has been associated with numerous health outcomes and impacts day-to-day functional health, including cognitive performance, psychosocial well-being, and longevity (Levy, Slade, Murphy, & Gill, 2012; Levy, Zonderman, Slade, & Ferrucci, 2009; B. R. Levy, Zonderman, Slade, & Ferrucci, 2012). Thus, it is critical for practitioners to be mindful that the patient should be the center of the assessment and interview, while maintaining a balance of interactions with family members or friends that may be present. Elderly patients may not be receptive to psychological services, especially if the patient does not have insight to their deficits. It is important for practitioners to provide education to patients and their families about mental health assessment and treatment in order to establish and maintain rapport and for patients to buy-in to the process (Molinari, 2011).

Assessment fidelity can be a large concern for this population. Patients may present with potential sensory changes that may require practitioners to adjust volume, rate of speech, and size of visual materials (Molinari,

2011). Even though it is important to be mindful of these considerations, it is critical for practitioners to maintain the integrity of the assessments. For example, on measures that require the patient to repeat numbers in order, patients may request to have the information repeated because they did not hear the information. Therefore, it is imperative for practitioners to provide the standardized instructions to the patient and ensure they are understood before proceeding onto test materials. If the patient requires additional directions, practitioners should explain the task in a way that does not invalidate the measure and does not confuse the patient by providing too much information about the measure. This should especially be considered on measures that are typically more complex or challenging for the patient (e.g., TMT-B).

Additionally, repeated exposure to these measures can result in practice effects by the patient, which can artificially inflate test scores. This can occur even at annual visits since patients may be primed based on expectations of the established protocol of the facility (e.g., “you want those three words, again? Apple, Table, Penny”). The screening measures that are recommended have alternative versions in order to avoid this prevalent issue. It may also be instructive that “teaching the client” to the tests may represent cognitive training; however, this can artificially inflate testing scores and render that type of testing useless in determination of current functioning.

Elder patients with cognitive impairment can also rationalize their deficits, which can impede testing. On a task such as Serial 7’s, a patient may state that he/she would never do such a thing in real life and it should not have any bearing on the test or interpretation. Patients should be encouraged empathically to complete the task as best as possible. Practitioners can present cognitive testing similarly to the instructions of the Wechsler Adult Intelligence Scale-4; “I will be asking you to complete several tasks today. Some may be easy, and others may be more difficult. Even if some tasks are frustrating, it is important for you to try your best.” This set of instructions does take extra time and might raise some questions from the patient, but it is an important step in order for the patient to understand the varying challenges of the tasks and that the practitioner is transparent in what is being requested of the patient. Depending on the setting, this length of introduction may not be necessary, especially if only a brief screening measure is being utilized for a quick assessment of need for a referral for further testing.

Recall that cognitive decline is based on patient’s self-report, a knowledge of a third party informant, or an objective observation, as well as mild deficits on objective cognitive assessment. When serial measures

are available as significant, that is 0.5 standard deviations assigned from the patient's own baseline, it would serve as a more definitive evidence of this decline. The mix of cognition and function as well as the trajectory of the problem can be persuasive for accurate data on a particular patient. Cognition is variable and there needs to be a focus or target. For AD, the fundamental focus is memory on a unidimensional continuum. A dementia syndrome may appear heterogeneous due to disruption of diverse cognitive processes until the underlying vulnerable factor is determined. We know that variables such as recall and orientation are often sufficient for an eventual dementia diagnosis. Ethnic and cultural differences result in norming difficulties as well.

Intraindividual differences are also important to consider in assessment. Multiple factors can impact patient presentation that can vary across time. However, variation across shorter periods of time is considered an indicator of cognitive aging (Tuokko & Smart, 2016). Thus, it is critical to assess ongoing stressors or other contextual factors that can significantly impact testing results, which can include attitudes, social context, and interest.

Another area of concern that can be easily overlooked in a high-paced medical setting is the patient's confidentiality. The patient maintains the right about what information can be shared to other family members, as long as the patient does not have a family member who has power of attorney over his/her medical information. This can become a complicated situation if test results indicate cognitive impairment. In order to avoid situations like these, clinicians should address the issue prior to initiating assessment, clearly document patient intent, and obtain appropriate release of information requests.

Neuroimaging

Neuroimaging can be incrementally beneficial in the differentiation of cognitive impairment diagnosis and monitoring of progression. There are several types of imaging studies that can assist with this process: computerized tomography (CT scan), magnetic resonance imaging (MRI), positron emission tomography (PET), and single photon emission computed tomography (SPECT). Each of these types of scans has its own strengths and weaknesses in regard to differentiating diagnoses; these nuances are outside the scope of this chapter. Neuroimaging as applied to the detection and progression of Mild and Major NCD will be addressed. Additionally, PET can be extended to where PET allows now for tau (Tau PET, in addition to amyloid) binding. This is certainly a newer area and

one with considerable promise. Functional MRI (fMRI) allows for a real time representation of the processing of brain function. Fluorodeoxyglucose PET (FDG-PET) can also be applied for glucose monitoring. Diffusion Tensor Imaging (DTI) has been available for more in-depth assessment of white matter.

Finally, we note that over the past 20 years tests for selected proteins excreted into the fluid by brain cells have been available. Cerebral Spinal Fluid (CSF) now plays a critical role in the diagnosis and prognosis of dementia (especially AD). This allows for B42, total tau and phosphorylated tau (p-tau). We add that increasingly there are less sensitive but promising tests for smell and sight to identify AD especially. There have even been measures using polygenic hazard sums for AD and prediction equations for risk factors out of the Karolinska Institute and other sites (Susman, 2019).

More specifically regarding AD, neurofibrillary tangles appear early in the progression and accumulate in the entorhinal cortex, hippocampus, and other temporal regions (Duyckaerts & Boller, 2003). As the disease advances, tangles present both cortically and subcortically, with relative sparing of primary sensory and motor cortices (Lezak et al., 2011). Another core feature of neurodegeneration of AD is neuronal loss, involving temporal lobes and the brain stem. This is typically observed in neuroimaging studies as early atrophy in the hippocampus lobe and the entorhinal cortex (Scheltens & Korf, 2000). Other cortical and subcortical areas may also demonstrate atrophy.

However, the most obvious changes are enlarged ventricles and cortical thinning on MRI studies. Synaptic dysfunction is also at issue. Dysfunction of the midfrontal and lower parietal areas around the temporal lobes attributes to the disconnection of the temporal structures with are areas of the cortex, contributing to memory disorders (Lezak et al. 2011). Further, cortical degeneration results in disconnection of prefrontal and parietal structures (Braak & Braak 1991). Varying neurofibrillary changes were found to be related to different stages of the disease progression and later studies identified stages of tau pathology (Braak & Del Tredici, 2011). In addition to reduced activity detectable by fMRI (medial temporal lobe), metabolic changes also occur within the hippocampus and other associated areas as indicated through PET (Lezak et al., 2011). Finally, the BBB (blood brain barrier) has been implicated in the problem cascade of AD starting with leakage, foreign agents in the brain and eventual amyloid and tau.

Despite the progress made in identifying different neuropathological processes and their detection through different neuroimaging techniques,

there is significant overlap of neuroimaging findings with AD and other dementing processes, mild NCD, mixed dementias, and non-dementing adults (Lezak et al., 2011; Petersen et al., 2001). For an expansive review of different techniques with varying dementia, please refer to O'Brien (2014).

Value of Cognitive Testing

Cognitive measures have demonstrated to be as or more sensitive in predicting progression of cognitive impairment than neuroimaging (e.g., Silbert, Howieson, Dodge, & Kaye, 2009). Jedynack and colleagues (2012) found performance on the Rey Auditory Verbal Learning Test delayed recall to better predict progression to AD than amyloid beta and tau biomarkers. Comparatively, conventional methods of assessing mild NCD (e.g., using a cutoff of 1.5 or more *SDs* below normative means on at least one measure) (Petersen & Morris, 2005; Winblad et al., 2004), were shown to increase false positive rates due to reliance on single impaired scores, rating scales, or subjective memory complaints (Bondi et al., 2014). Authors caution use of MMSE due to its lack of sensitivity in detecting and tracking progression of cognitive impairment (e.g., Aksu, Miller, Kesidis, Bigler, & Yang, 2011; Bondi et al., 2014).

Longitudinal and cross-sectional neuroimaging studies have especially revealed the value of cognitive testing. Pertaining to mild NCD, a study examining 129 amnesic MCI patients established baseline MRI measures and compared to imaging studies conducted 36 months post. Predictive accuracy of MCI conversion to AD was 60.4% examining hippocampal volumes. However, subtests of the Alzheimer's Disease Assessment Scale-cognitive subscale better predicted conversion rate at 78.8%. The addition of neuroimaging did not enhance predictive ability (Fleischer et al., 2008). The value of memory is especially robust. Regardless, utilization of more than one screening measure is important, including more sensitive measures than the MMSE (e.g., MoCA and RBANS). Incorporating the RBANS in the screening process of cognitive impairment may be especially helpful. Bondi and colleagues (2014) cautioned however that under-reliance of neuropsychological performance contributes to false positive errors. The subtests included on the RBANS reflect varying neuropsychological assessment measures, while remaining a screening measure.

Brief Battery

We recommend utilizing four brief measures in order to screen for potential cognitive deficit within this population, as indicated below. These measures are selected because they are easy to administer, reasonably valid and can be done quickly. There are many other measures recommended and we will pay notice to some of these. The cut-off scores provided not only effectively work within our clinic, but are also supported within the literature (e.g., Julayanont, Brousseau, Chertkow, Phillips, & Nasreddine, 2014).

Further, it is critical for clinicians to utilize more than one screening measure in identifying potential cognitive impairment, the typical criteria in diagnosing MCI. Previous research has indicated those diagnosed with mild NCD have revision rates as high as 40% (e.g., Luis, Loewenstein, Acevedo, Barker, & Duara, 2003). However, Loewenstein, et al. (2009) investigated the longitudinal stability and progression of the different subtypes of mild NCD in older adults. The variability in diagnosis is significant; therefore, using a more comprehensive criterion based on more than 2 measures, will almost always provide a more definitive outcome. Recall that there is considerable intraindividual variability for normal older adult, as much as several standard deviations for 66% of older adults (Schretlen, Munro, Anthony, & Pearlson, 2003); however, many measures have normative variability data to minimize this issue.

Practitioners may not be able to administer, score, and interpret lengthy neuropsychological assessments. Therefore, it is important to be aware of the strengths and weaknesses of the highly used screening measures. The measures utilized in our clinic are reviewed below. Based on our model, we specifically recommend the combination of both the Mini-Mental Status Examination and the Montreal Cognitive Assessment, which will be applied to the case examples presented.

The Mini Mental State Examination (MMSE) (Folstein, Folstein, & McHugh, 1975) is the most used scale in the world (Arevalo-Rodriguez et al., 2015). This assessment is comprised of several sections: Orientation, Registration, Attention and Calculation, Recall, and Language. The measure takes approximately 5 to 10 minutes to complete and is easy to administer and score (Lezak et al., 2012). Age and education can influence scores such that stratified norms have been developed to provide a better normative structure (Crum, Anthony, Bassett, & Folstein, 1993; Tombaugh, McDowell, Kristjansson, & Hubley, 1996). The measure is most effective in assessing moderate or severe deficits (Lezak, Howieson, & Loring, 2004). Therefore, despite its popularity, the MMSE it is not

recommended as a stand-alone screening measure to assess cognitive functioning when there are few or no problems, or MCI is suspect. This recommendation echoes the findings of the most recent Cochrane Review in utilizing the MMSE in detecting mild NCD (Arevalo-Rodriguez et al., 2015). Further, meta-analysis conducted by Mitchell (2009) demonstrated the MMSE offers modest accuracy for ruling-out dementia presentation in community and primary care settings; suggesting the measure should be combined with other measures.

The Montreal Cognitive Assessment (MoCA) (Nasreddine et al., 2005) serves as another screening tool that utilizes other popular neuropsychological assessment components including a shortened version of TMT-B, clock drawing task, construction of a cube, serial 7's, letter fluency and similarities. The measure assesses several areas of cognitive functioning: Visuospatial/Executive, Naming, Memory, Attention, Language, Abstraction, Delayed Recall, and Orientation. Three English versions are freely available online for retesting purposes. Since education was found to be a factor in performance, the assessment adds one point to the score for individuals that have 12 years of education or less when comparing scores to the MMSE. These scales highly correlate with each other ($r = 0.87$) (Lezak et al., 2012). According to Nasreddine (2019) the most common errors made by clinicians with the MoCA are provided below.

Errors of the MoCA

1. Informing patients of words they missed
2. Not applying the second learning trial for word recall
3. Scoring clock incorrectly
4. Over explaining tasks/modules

Virtually every study has found the MoCA to have better correct classification ratios than the MMSE (Hyer, 2014). The measure has less of a ceiling effect and stronger association with functional status post-discharge of inpatient stroke patients (Toglia, Fitzgerald, Dell, Mastrogiovanni, & Lin, 2011).

Utilization of both the MoCA and the MMSE does not necessarily improve sensitivity when detecting suspected cognitive impairment. Lamer (2012) investigated this within a memory clinic sample of 150 participants to assess potential mild NCD. Results demonstrated sensitivity of the MoCA as 0.97 whereas the MMSE a mere 0.65, with cut-off scores set at $\geq 26/30$. Reduction of cut-off score to $\geq 20/30$ revealed sensitivity of 0.97 and 0.63, respectively. Thus, the addition of the MMSE to the MoCA

did not improve detection. We recommend use of the MoCA and, given time or needs of the clinic, then the MMSE.

Comparatively, the combination of the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS, Randolph, Tierney, Mohr, & Chase, 1998) and the MoCA encapsulate many areas of cognitive functioning would enhance the specificity of the diagnosis. However, if time is an issue, it is recommended to utilize the RBANS, due to the segments from other neuropsychological measures and the memory subtests that assess both verbal and non-verbal components. Importantly, there is no executive function component included on the RBANS. Thus, it is critical for practitioners to be mindful of assessment choice in regard to diagnostic considerations.

There are several other briefer scales that have additional value, especially for executive functioning. One of the most widely used assessments within this population is the TMT-B. TMT-B was also found to be the single best neuropsychological predictor of the conversion from MCI to AD with a 64.6% prediction accuracy (Ewers et al., 2012). The meta-analysis conducted by Martyr and Clare (2012) demonstrated that commonly used executive function assessments; including Clock Drawing Test, TMT-B, and Letter Fluency, had a significant moderate association with activities of daily living as well as driving ability. Importantly, portions of these executive measures are included measures are included in the MoCA.

Extended Battery

Premorbid Functioning:

An important factor in the discrimination of MCI and dementia from normal functioning is the estimation of premorbid functioning. Reading tests can be utilized as a quick measure; e.g., Wechsler Test of Adult Reading (WTAR), Wide Range Achievement Test-4 (WRAT-4) Reading subtest, and the American version of the National Adult Reading Test (AMNART). These tests require the patient to read aloud phonetically irregular words to assess premorbid intellectual ability, which has demonstrated to be resistant to brain insult (Strauss, Sherman, & Spreen, 2006). These reading tests have been found to be effective in the estimation of premorbid function in elderly adults (Ferraro & Sturgill, 1998; Law & Carroll, 1998; Maddrey, Cullum, Weiner, & Filley, 1996; Ryan & Paolo, 1992). Given that the NART is a good predictor of WAIS FSIQ and that the WTAR is co-normed with the WAIS-III (Strauss et al., 2006), and the WTAR and WRAT-4 are highly correlated (.75-.78)

(Mullen & Fouty, 2014), it can be generalized that all three of these measures would produce similar outcomes.

Additionally, the NART in combination with the Barona Index of Intelligence; a premorbid functioning measure that is solely based on background factors (i.e., age, sex, race, education, occupation), accurately predicted IQ for individuals with mild to moderate AD; therefore, is sensitive to AD severity (Paolo, Tröster, Ryan, & Koller, 1997). However, sole use of the Barona Index has presented with systematic under- and overestimate of WAIS-R FSIQ across the intelligence continuum (Griffin, Mindt, Rankin, Ritchie, & Scott, 2002). Equally problematic, the standard error associated with the Barona FSIQ estimate is quite high, leading to suggestions that it be used to provide only a general estimate of the range of premorbid functioning. Thus, utilization of either of the standardized assessment measures in conjunction with demographic information can assist in the estimation of premorbid function with individuals presenting with memory complaints.

The Wechsler group has recently published their newest version, the Test of Premorbid Functioning (TOPF), which has been revised and re-normed with the WAIS-IV and WMS-IV. Unfortunately, developers have not provided normed data for the WASI-II; requiring practitioners to administer certain portions of the WAIS-IV in for computer software to generate profile interpretation.

There is some evidence that reading measures to assess premorbid functioning may be compromised with AD patients (McFarlane, Welch, & Rodgers, 2006). Additionally, each of the assessments has its limitations and therefore should be used with caution. For example, the WRAT reading subtest and the NART-Revised estimations have been found to underestimate average and higher FSIQ scores (Wiens, Bryan, & Crossen, 1993). The WTAR has varying results in traumatic brain injury populations (e.g., Green & Bavelier, 2008; Mathias, Bowden, Bigler, & Rosenfeld, 2007), while Barona Index score has presented with the lowest correlation with FSIQ in a brain impaired population (Scott, Krull, Williamson, Adams, & Iverson, 1997). The AMNART may underestimate premorbid functioning in those with lower education and higher cognitive dysfunction (Lowe & Rogers, 2011). Interestingly, the Barona Index was found to be as good as a predictor when compared to methods combining performance on cognitive measures and demographics (Axelrod, Vanderploeg, & Schinka, 1999). It is generally recommended that all information presented to the clinician should be utilized in conjunction with one of the above reading measures in order to develop an estimate of premorbid functioning.

Extended Cognitive Measures:

As discussed, a screening battery developed to assess for dementia is the Repeatable Battery for the Assessment of Neuropsychological Status (RBANS) (Randolph, Tierney, Mohr, & Chase, 1998). This measure takes approximately 25 minutes to administer and has normative data for patients' ages 20 to 89 years. Unlike other memory assessments, the RBANS was developed specifically for elderly adults and therefore may allow for finer distinctions of memory compared to others (Lezak et al., 2012). The measure assesses five different indices: Immediate Memory, Visuospatial/Constructional, Language, Attention, and Delayed Memory. Four different versions are available to allow for repeated assessment. A large advantage of the RBANS, which most screening measures lack, is the assessment of non-verbal memory and story memory. Sensitivity and specificity in the detection of cognitive impairment associated with AD are excellent; 0.96 and 0.98, respectively (Duff et al., 2008).

The Dementia Rating Scale-2 (DRS-2) (Jurica et al., 2001) is a commonly used screening assessment in the elderly population. The measure is intended for individuals 56 to 105 years of age. Administration time is typically 10 to 15 minutes for healthy adults, and up to 45 minutes for demented patients. The DRS-2 has 24 subtests that are combined into 5 subscales: Attention, Initiation/Perseveration, Construction, Conceptualization, and Memory. Given that performances vary by age, education, and IQ, it is recommended for individuals to use age and education normative data (Strauss, Sherman, & Spreen, 2006). The original total cut-off score of 137 out of 144 from the developers has been considered to have limited value since sample sizes of the score is based on an extremely small sample of 31 (Strauss et al., 2006).

Norms are always an issue. It is recommended to utilize the Mayo's Older Americans Normative Studies (MOANS) norms available in Strauss et al. (2006), which also provides cut-off scores for subscales (Lucas et al., 1998). The MOANS developers have since reassessed their previous subjects in order to remove patients from the data set that later developed MCI or dementia (Pedraza et al., 2010). The DRS-2 was designed with the intention of discriminating between healthy adults with dementia. The measure has shown to be sensitive in detecting early stages of dementia and has been used as a tool to track the progression of cognitive decline (see Strauss et al., 2006). Individual subscale performance can also assist with differing neuropathological conditions (Lezak et al., 2011). Individuals with higher intelligence suffering from early stages of dementia may not be identified. The DRS-2 was developed to avoid floor effects and not ceiling effects (Strauss et al., 2006). Scores are also

negatively correlated with age and have shown to be variable with cultural factors (Lezak et al., 2011). Thus, the measure may not detect MCI dependent on cut-off scores, especially with those of higher education. Future investigation and revisions of the norms may improve the sensitivity of the assessment to capture MCI presentation.

Finally, the Clinical Dementia Rating scale (CDR) is a freely assessable assessment tool that assists in the formulation of the client's presentation and diagnosis. The semi-structure interview assesses several areas of functioning including: Memory, Orientation, Judgment and Problem Solving, Community Affairs, Home and Hobbies, and Personal Care. Completion of the assessment is based on information gathered from the patient and the collateral. After each separate domain is scored ranging from no impairment to severe impairment, a total global score is calculated. The scoring system is such that the patient does not need to score on the same level of severity on each of the domains. It is recommended to not solely rely on the global scale for identification of potential presentation of cognitive impairment, including MCI (Chang et al., 2011).

Different global and sum of boxes (SOB) cutoff scores have been suggested. Typically, a global score of 0.5 is suggestive of MCI and very early AD. However, subsequent research has demonstrated that utilization of this method produced a 3-fold increased risk of being diagnosed as having AD for every 1-point increment in CDR-SOB scores (Bryant et al., 2008). Authors recommend using the CDR-SOB scores to distinguish levels of AD. The measure was found to be generally stable in the assessment of dementia over the course of three decades. The CDR is an extremely informative measure that can assist in the understanding of functioning in areas that may be missed by a clinical interview.

Recommended Brief Assessments of Cognition

Assessment	Areas of Functioning	Administration Time	Features
Montreal Cognitive Assessment	Visuospatial/ Executive, Naming, Memory, Attention, Language, Abstraction, Delayed Recall, and Orientation	10 minutes	Three versions; assessments and manual free online. Shortened TMT-B, Clock Drawing, serial 7's, phonemic fluency, similarities, construction of a cube
<ul style="list-style-type: none"> • Numerous studies favor the MoCA over the MMSE (e.g., Damian et al., 2011; Freitas, Simões, Alves, & Santana, 2013; Nazem et al., 2009; Smith, Gildeh, & Holmes, 2007). • Compared to the MMSE, the MoCA is superior in the detection of MCI and dementia in Parkinson's disease (Hoops et al., 2009; Zadikoff et al., 2008), transient ischemic attack and stroke (Godefroy et al., 2011; Pendlebury, Cuthbertson, Welch, Mehta, & Rothwell, 2010), vascular cognitive impairment (Dong et al., 2010; Schweizer, Al-Khindi, & Macdonald, 2012) and behavioral variant frontotemporal dementia (Freitas et al., 2013). • Less of a ceiling effect than the MMSE and has a stronger association with functional status post-discharge of inpatient stroke patients (Toglia et al., 2011). 			
Repeatable Battery for the Assessment of Neuropsychological Status (RBANS)	Attention, Immediate Memory, Language, Delayed Memory, and Visuospatial/ Constructional	25 minutes	Four versions; shortened Judgment Line Orientation, Coding, Figure Copy and Recall, Story Memory
<ul style="list-style-type: none"> • Developed specifically for elderly adults; may allow for finer distinction of memory difficulties (Lezak et al., 2012). • Better sensitivity than MMSE and DRS (Gold, Queem, Iannone, & Buchanan, 1999). • Sensitivity and specificity for AD are excellent; 0.96 and 0.98 (Duff et al., 2008). 			

<ul style="list-style-type: none"> • Test-retest reliability ranges between .53 and .83 on indices with minimum practice effects (Duff et al., 2005). • Education can account for a proportion of all indices, ranging from 1.9-7.6% (Gontkovsky, Mold, & Beatty, 2002). • Updated version has norms based on education and other demographics. 			
Clock Drawing	Visuospatial, Attention, Receptive Language, Numerical Knowledge, Working Memory, Executive Functioning	2 minutes	● One of the top 40 assessments used by neuropsychologists (Rabin, Paolillo, & Barr, 2016); high intercorrelation of scoring techniques
<ul style="list-style-type: none"> • High sensitivity and specificity; each 85%, high correlations with other cognitive measures including the MMSE (Shulman, 2000). • Level of education impacts performance of non-demented elderly adults (Ainslie & Murden, 1993) 			
TMT-B	Attention, Executive Functioning, Working Memory	2 to 5 minutes	Second most utilized executive function measure (Rabin, Paolillo, & Barr, 2016)
<ul style="list-style-type: none"> • Reliability of TMT vary markedly; however, less so on TMT-B, $r = .65$ (Strauss et al., 2006). • The combination of TMT-B and delayed verbal recall was found to be the best discriminator for individuals who developed AD 1.5 years later and those who remained cognitively intact (Chen et al., 2000). • Found to be the single best neuropsychological predictor of the conversion from MCI to AD with a 64.6% prediction accuracy (Ewers et al., 2012). • Significant moderate association with ADLsg and driving ability (Martyr & Clare, 2012). 			

Wechsler Test of Adult Reading (WTAR)	Premorbid Functioning	2 minutes	Co-normed with the Wechsler Adult Intelligence Scale-III
<ul style="list-style-type: none"> • WTAR and Wide Range Achievement Test-4 (WRAT) Reading subtest are highly correlated (.75-.78) (Mullen & Fouty, 2014), National Adult Reading Test (NART) is a good predictor of WAIS Full-Scale IQ (Strauss et al., 2006), therefore, these measures can produce similar outcomes. • These reading tests have been found to be effective in the estimation of premorbid function in elderly adults (Ferraro & Sturgill, 1998; Law & Carroll, 1998; Maddrey et al., 1996; Ryan & Paolo, 1992). • The NART in combination with the Barona Index of Intelligence accurately predicted IQ for individuals with mild to moderate AD (Paolo et al., 1997). 			
Clinical Dementia Rating scale (CDR)	Memory, Orientation, Judgment and Problem Solving, Community Affairs, Home and Hobbies, and Personal Care	5 to 10 minutes	Semi-structured interview with patient and the collateral
<ul style="list-style-type: none"> • Generally stable in the assessment of dementia over the course of three decades (Williams, Roe, and Morris, 2009). • Assists in the differentiation of frontotemporal dementia patients and Alzheimer's patients (Rosness, Haugen, & Engedal, 2011). 			

Standard Neuropsychological Measures

More thorough testing may be required in order to differentiate the presentation of a Mild or Major NCD, as well as the identification of subtype. This is outside of the scope of the intention of the book; therefore, a brief overview is provided. The reader is directed to Smith and Farias (2018) and Tuokko and Smart (2016) for further review.

One of the most utilized intelligence measures is the Wechsler Adult Intelligence Scale-IV (WAIS-IV). Administration time significantly varies based on patient presentation. A briefer assessment can be used to gain insight into level of intelligence. The Wechsler Abbreviated Scale of Intelligence-II can generate a full scale IQ score based on either two (Vocabulary and Block Design) or four subtests (Similarities and Matrix

Reasoning added). Assessment of these domains is important to grasp patients' level of functioning. Specifically, crystallized intelligence has higher stability compared to fluid intelligence; however, crystallized knowledge itself decreases around age 70 (Sattler & Ryan, 2009). The WASI (Wechsler Abbreviated Scale of Intelligence (WASI Test) is also applied and delivers an estimation of general intellectual ability by measuring the verbal, nonverbal, and general cognition of individuals from 6 to 89 years of age (see pearson.com).

In regards to cognitive domains, there are many components of assessments that need to be considered. There are several advantages to longer assessments, such as assessing learning curves and delayed memory. However, these types of assessments may not be appropriate for fast paced settings or for patients that present with moderate to severe cognitive impairment and may not withstand hours of testing. A review of highly used measures and their descriptions is provided below.

Neuropsychological Assessments of Cognition

Assessment	Administration Time	Description
<i>Attention</i>		
WAIS-IV Digit Span	5 to 10 minutes	<i>Directions (D):</i> Patient repeats numbers to facilitator (forwards, backwards, sequential order) <i>Areas of Functioning (AoF):</i> Auditory attention (digits forwards), and working memory (digits backwards and sequencing). Embedded validity measure of effort; reliable span (number of digits in last series of two correct) of forwards and backwards ≥ 7
WAIS-IV Letter Number Sequencing	2 to 5 minutes	<i>D:</i> Patient repeats numbers and letters and numbers; numbers first in numerical order followed by letters in alphabetical order <i>AoF:</i> Auditory working memory
Wechsler Memory Scale (WMS)-III Spatial Span	2 to 5 minutes	<i>D:</i> Patient repeats pattern of blocks touched by the administrator forwards and backwards <i>AoF:</i> Visual attention (forward) and visual working memory (backward)

<i>Processing Speed</i>		
WAIS-IV Symbol Search	2 minutes	<i>D:</i> Patient scans a row of symbols and identifies if one of the symbols on the right is the same as one of the target symbols on the left <i>AoF:</i> Visual scanning and discrimination, processing speed
WAIS-IV Coding	2 minutes	<i>D:</i> Patient draws as many symbols corresponding to their paired number as indicated in the key as quickly as possible <i>AoF:</i> Visual-motor processing speed, working memory
Symbol Digit Modality Test	90 seconds	<i>D:</i> Oral and written modalities: patient either writes or says numbers that corresponds to their paired symbols as indicated in the key as quickly as possible <i>AoF:</i> Visual-motor processing speed, working memory
<i>Psychomotor Function</i>		
Grooved Pegboard	Up to 5 minutes	<i>D:</i> Patient places pegs with a ridge along one side into slotted holes angled in different directions one at a time, one hand at a time, as quickly as possible <i>AoF:</i> Fine motor coordination and dexterity, and visual-spatial monitoring
Hand Dynamometer	Under 1 minute	<i>D:</i> Patient squeezes and holds lever as hard as he/she can for two to three seconds at least twice per hand, that is averaged <i>AoF:</i> Hand strength
<i>Language</i>		
Boston Naming Test	10 to 20 minutes	<i>D:</i> Patient is to say the name of the drawing <i>AoF:</i> Word finding
Token Test	~5 minutes	<i>D:</i> Patient is to complete basic to more challenging commands with stimuli of different shapes and colors <i>AoF:</i> Auditory comprehension, praxis

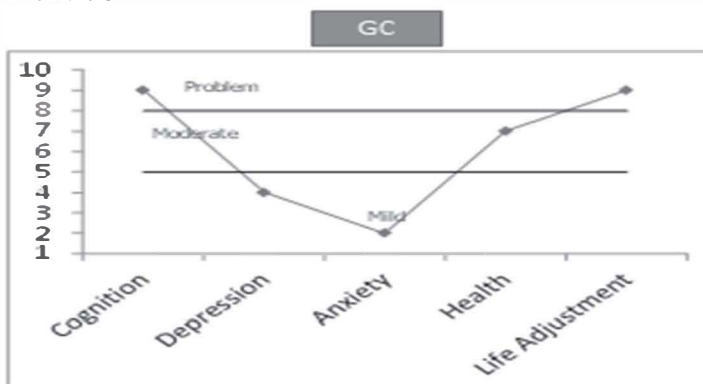
<i>Memory</i>		
WMS-IV Logical Memory	~40 minutes	<i>D:</i> Patient repeats two short stories read by the administrator after an immediate delay and after a longer delay; discriminate if information was part of the story or not <i>AoF:</i> Verbal attention, memory, and recognition
California Verbal Learning Test-3	~40 minutes	<i>D:</i> Patient repeats a list of words to the administrator over 5 trials; repeats a different list of words from a distractor list; recalls the original list immediately after the distractor list (with and without semantic cueing), and after a longer delay (with and without semantic cueing); discriminates between words either from the first list or not; forced recognition between two items <i>AoF:</i> Verbal attention, learning, immediate and delayed memory, recognition, and effort
Brief Visual Memory Test-Revised	~40 minutes	<i>D:</i> Patient is to draw as many of the simple geometric shapes as they can recall immediately after exposure across three trials, and again after a longer delay; discriminates between figures from previously presented shapes and non-presented shapes; copy the figures <i>AoF:</i> Visual attention, learning, delayed memory, recognition, basic visual-constructional abilities
WMS-IV Visual Reproduction	~40 minutes	<i>D:</i> Patient is to draw progressively challenging geometric shapes immediately after exposure, and again after a longer delay; discriminates between figures from previously presented shapes and non-presented shapes; copy the figures <i>AoF:</i> Visual attention, memory,

		recognition, and basic visual-constructional abilities
Tombaugh-Taylor Complex Figure	~30 minutes	<i>D:</i> Patient is to draw a complex figure immediately after exposure across four trials, and again after a longer delay; copy the figure <i>AoF:</i> Visual-spatial perception; visual attention, learning, memory, and complex visual-constructional abilities, approach to task (e.g., gestalt figure vs. details)
Rey-Osterrieth Complex Figure	~40 minutes	<i>D:</i> Patient is to copy a complex figure, reproduce the figure after a short and longer delay; discriminates between parts of the complex figure and distractor items <i>AoF:</i> Visual-spatial perception, attention, immediate and delayed memory, complex visual-constructional abilities, approach to visual task (e.g., gestalt figure vs. details)
<i>Executive Functioning</i>		
Short Category Test	~25 minutes	<i>D:</i> Patient is to test, develop, and implement different underlying principles from visual stimuli based on administrator feedback <i>AoF:</i> Deductive reasoning, problem solving abilities, ability to shift set
Wisconsin Card Sorting Test	~25 minutes	<i>D:</i> Patient is to test, develop, and implement different strategies from cards based on administrator feedback <i>AoF:</i> Deductive reasoning, problem solving abilities, ability to shift set, impulsivity
Controlled Oral Word Association (COWA)/	6 to 10 minutes	<i>D:</i> Patient provides as many words as possible based on a phonemic or semantic target within a specific time frame; patient is to alternate between semantic categories (DKEFS only) <i>AoF:</i> Mental generativity, processing speed, inhibition

Case 2

GC reported a rapid progression of cognitive symptoms and functional difficulties. Importantly, patients may not recognize cognitive changes until being informed by others or a significant event (e.g., couldn't find the car for an hour); thus, they may not be accurate historians in regard to time considerations. This is especially true within an elderly population given that cognitive demands and social interactions may be limited due to retirement, smaller social networks, and less social involvement.

Patient: GC



Cognition

DOB: 1943

Cognition:

MMSE= 18/30 raw

MoCA = 15/30 raw

TMT-A: <10%ile

TMT-B: Unable to complete

Life Adjustment

FAQ =15, abnormal

IADLs: Needs supervision for all IADLs

SES/Education: No Problem

Environment: Reasonably safe

Relationship: Dependent on spouse

Meaning in life: Unable to now process

Caregiving: Wife available

Health

8 medications

Cardiac problems

Inactive

Possible ETOH abuse

Pain = 3/10

Sleep = ESS = 11

Depression

Mild problems for BDI-2 = 12, GDS-SF = 4, MBMD normal

Anxiety

Normal scores for STAI, GAD-7, MBMD

CHIEF COMPLAINT: Evaluation for cognitive impairment; referred from family medicine

HISTORY OF PRESENT ILLNESS: The patient is a 74 year-old Caucasian male. His wife reported difficulties with word finding, memory, and problem solving. Reported onset was within one to two years and has significantly progressed. Mr. GC was driven by his wife to the interview. Both he and his wife were interviewed. He retired 10 years ago and remains in his home most of the time with his wife. He had limited insight to his difficulties but becomes frustrated with his expressive difficulties. He is inactive and at times confused.

RELEVANT DEVELOPMENTAL HISTORY: The patient is a Georgia native, and youngest of two children. He met his milestones on time and had no reported learning disorders or attention deficit disorder. Mr. GC graduated from a state college with his bachelor's degree. Subsequently, he served in the Navy for 6 years. After time served, he owned a telephone company until his retirement in 2003. He was married in 1961 and has two daughters and four grandchildren. He lives locally with his wife. He is very active in retirement and depends on his wife.

RELEVANT MEDICAL HISTORY: Mr. GC has had four CABG operations, the last in 2008. He consumes one to two glasses of wine per day and does not utilize tobacco products. He denies pain. He sleeps approximately 6 hours per night that is often disrupted. He exercises mildly and is social. There is no psychiatric history. He is currently on several medications: Levemir, metoprolol, simvastatin, lisinopril, pantoprazole, Niaspan, donepezil, and Foltanx.

MENTAL STATUS: The patient is a 74-year-old Caucasian male who has difficulty expressing himself. He stammered and presented with circumlocutious when providing his history. He was unable to provide much information as he reported significant memory issues. Processing of

information was slow. He denied depression and anxiety, but reported frustration surrounding his current situation. There was no evidence of perceptual anomalies or delusory thinking. He has minimal insight to his situation, but demonstrates some difficulty with judgment and denied memory difficulties.

TEST RESULTS: His estimated premorbid functioning was in the average range (WRAT-4 SS = 94). Mr. GC's intelligence score fell into the mildly to moderately impaired range (WAIS-IV FSIQ SS = 76, 5%), while subtests scores varied (ss = 3-9). His cognitive screening performances were all below normal. MoCA and MMSE were impaired, especially memory. He made several errors on TMT-A (60"), and was unable to complete TMT-B due to numerous errors and becoming confused. He had no issue with clock drawing, but was unable to copy a cube. Total RBANS Index was in the moderately impaired range (SS = 70, 2%). Performance on RBANS Visuospatial/Construction was intact (Index = 131; 98%), while all other indices were impaired (Attention Index = 68, 1%; Language Index = 60, 1%; Immediate Memory Index = 57, .2%; Delayed Memory = 44, .1%).

LIFE ADJUSTMENT: Total score on the FAQ was 15, demonstrating deficits in executive functioning. Mr. GC barely leaves the home and does not perform the majority of his IADL's. He is then inactive.

EMOTIONAL: Emotional self-report scales indicated mildly problematic depression (BDI-II = 12, GDS-SF = 4) and no anxiety (GAD-7 = 1, SAST=15). PAI and MBMD were within normal limits except for concerns about his thinking problems. His personality pattern also was nondescript, as he shows no penchant for any style. Sleep may be an issue.

GC

Psychiatric Indicators	AA	2	40			ANXIETY-TENSION
	BB	2	20			DEPRESSION
	CC	18	85			COGNITIVE DYSFUNCTION
	DD	3	40			EMOTIONAL LABILITY
	EE	14	68			GUARDEDNESS
Coping Styles	1	6	55			INTROVERSIVE
	2A	0	0			INHIBITED
	2B	0	0			DEJECTED
	3	10	65			COOPERATIVE
	4	14	62			SOCIABLE
	5	12	55			CONFIDENT
	6A	10	51			NONCONFORMING
	6B	18	70			FORCEFUL
	7	21	58			RESPECTFUL
	8A	11	64			OPPOSITIONAL
	8B	5	61			DENIGRATED

Watch and Wait

Summary: This is a 74 year-old Caucasian male who was referred for an evaluation for his cognitive status. He reportedly presented with deficits in memory, verbal expression, and executive function over the past one to two years. Performance in all assessed cognitive domains was within the impaired range, with the exception of visuospatial abilities. These changes are dramatic given his history, and he is rapidly declining. Mr. GC shows mild evidence of depression, which he attributed to frustration with speech difficulties. His cognitive scores coupled with impaired IADL functioning indicate he is suffering from a form of dementia.

Interventions:

Checklist

Discernment: Caregiving only

Core markers

Validate Problem

Psychoeducation of Model X (caregiver only)

Assessment X

Alliance

Monitoring X

Case formulation X

The Watch and Wait markers were in place with the exception of validation and alliance as these were problematic for a dementia. Caregiving supplanted these. Based on presentation of significant cognitive difficulties and reported onset of one to two years with rapid decline, it is recommended for Mr. GC undergo further neuropsychological testing to determine likely etiology and course of progression, as well as intervention strategies. It is likely Mr. GC experienced more subtle cognitive difficulties prior to reported onset that he was able effectively compensate. An MRI may also be warranted to better delineate potential dementia presentation. Advanced directives should also be completed given his current cognitive status. He should become as socially involved as possible and this will be arranged with his wife and senior center. Given his multiple cardiac risk factors, Mr. GC is strongly recommended to follow a heart healthy diet, his medication regimen, and get appropriate exercise. Contact will involve the primary care physician and repeat cognitive screening will be conducted to track changes over time. The patient is now supported by his wife. She will require assistance from other family members and community members (e.g., church members) and may require respite care.

Problem List

1. Cognition: Referral for neuropsychological testing, monitor behaviors, simple behavioral activation, exercise, HABIT (Healthy Action to Benefit Independence and Thinking, Mayo Clinic) or Memory protocol for his level, activate stimulation and leisure activities, socialization, possible Senior Center
2. Life Adjustment: Caregiver protocol, monitor behavior for possible long term care placement or extended caregiver application, caregiver support

Self or other monitoring: Moni-targets of behavior over time (with caregiver)

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks:

Managing situational contingencies: X

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation:

Concreteness training:

Imagery exercise for counter-experience:

Compassion training:

Communication and Assertiveness training: X

Behavioral experiments:

Emotional exposure:

Homework: X

Longer Therapy processes:

None applied

Lifestyles:

- Practice Spirituality/Religion
- Socialize: Less decline and live longer
- Exercise: Even housework
- Diet
- Values and Leisure

Follow-up: Re-evaluate every 3 to 6 months

Re-Test 6 months later

Cognition:

RBANS: Not repeated

MoCA/MMSE: Recommended

TMT-A: Not repeated

TMT-B: Not repeated

Life Adjustment

FAQ =14

IADLs: same as before but more active

Environment: Safe and supportive

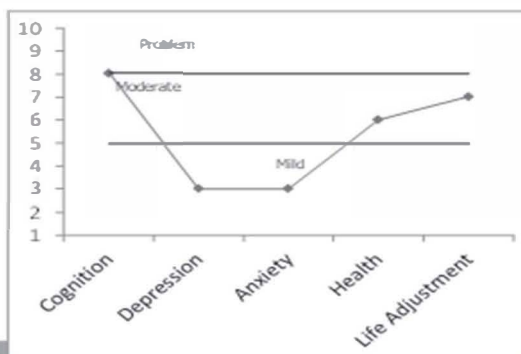
Relationship: Good, dependent on spouse

Caregiving: Wife given Zarit Caregiving Scale; burden lower and relationship improved

Relapse Issues: none noted

He was largely absent symptoms of depression and anxiety. These are common problems within this population. Based on the Cochrane review (Orgeta, Qazi, Spector, & Orrell, 2014), few were initiated. More effective psychosocial interventions (e.g., counseling, cognitive behavioral therapy, interpersonal psychodynamic therapy) are actually less effective compared to those without severe cognitive impairment.

Here is the follow-up profile. Cognition remains the key issue.



Cognition

This is a typical presentation of someone who suffers from major NCD. There are several areas of severely impaired functioning, along with some level of affective involvement. Therefore, life adjustment and cognition were significantly impacted. At this level of impairment, interventions are required in order to appropriately assist with the management of the patient and his care provider, in this case, his wife. Given his level of impairment, he was provided cognitive stimulation. Selected cognitive and social targets were also targeted, including HABIT (Healthy Action to Benefit Independence and Thinking; Mayo Clinic) and the Cognitive Rehabilitation Manual (Haskins, 2012-2014). Social engagement and behavioral activation were especially addressed.

Treatment

Cognitive Training:

Previously we noted that two consensus statements about brain training offered conflicting views on the validity. Regardless, cognitive training (CT) has become a popular intervention for this demographic to assist in the development of compensatory strategies and to strengthen and reinforce neural networks by repetitive challenging activities of increasing difficulty. The National Institute of Health (National Institute of Health, 2011) Conference for Alzheimer's Disease and Cognitive Decline assessed 6,713 studies on risks and protective factors related to cognitive functioning. Results demonstrated that CT and behavioral factors were the most influential components associated with risk reduction for cognitive decline.

CT has traditionally been offered in two different formats; pencil and paper and computer-based. Given the ease of access, computer based CT programs are on the rise (e.g., Lumosity, Happy Neuron, Cogmed); thus, more recent studies have focused on this format of intervention. There is increasing evidence suggesting that older adults who are impaired can benefit from CT. A review of the literature by Hyer, Mullen, and McKenzie (2015), provides information of recent major studies of CT in adults with MCI or mild AD. General results reveal that interventions show positive effects on cognition, affect, and overall functioning, regardless of treatment modality. However, studies vary in type and duration of intervention, as well as population samples. Effect sizes are generally small but significant, and comparison non-intervention groups even demonstrated some non-significant improvements.

Recent meta-analysis demonstrated that computerized CT is efficacious in the improvement on global cognition as well as varying

domains of cognitive functioning within mild NCD populations (Hill, Mowszowski, Naismith, Chadwick, Valenzuela, & Lampit, 2016). In that meta-analysis the authors examined the effects of CT on at least one cognitive or behavioral outcome in older adults with MCI or dementia. CT had to include a minimum of four hours with a clear rational and virtual reality or video game component. Results indicated limited benefit for dementia patients. Moderate effects on cognition were observed in MCI populations; specifically, attention, working memory, learning, memory, and global cognition. Importantly, there was evidence for improved psychosocial functioning and depression. Thus, if administered properly, CT can have meaningful effects in those with MCI. As suggested in the review, future research needs to examine longer-term intervention to develop an understanding the potential attenuation of dementia.

In previous chapters we also noted that careful and scientific reviews suggest caution in strong recommendations. In general, overall results indicated that it is important for elderly adults to continue to be cognitively challenged throughout their life in order to stave off cognitive decline. However, research pertaining to generalization of gains to daily functioning and the means of CT mechanisms is still in its infancy. Jak, Seelye, & Jurick (2013) indicated that such computer based CT programs provide the most significant improvements in trained tasks, especially for processing speed. Importantly, external techniques (e.g., calendars, reminders, memory books) can be coupled with such programs that focus on internal mechanisms in order to maximize potential gains. All together, data supports utilization of CT programs; however, effect sizes are variable. Therefore, it is recommended that patients presenting with MCI or early stages of dementia engage in some form of CT intervention, given that programs are reasonably affordable and there are minimal negative side-effects (Hyer et al., 2015). Again, commercial claims of these interventions pertaining to their efficacy of cognitive enhancement is suspect and may be premature (Jak et al., 2013). Interestingly, the Alzheimer's Association has concluded cognitive training may reduce the risk of cognitive decline, based on population-based analyses (Baumgart, Snyder, Carrillo, Faxio, Kim, & Johns, 2015).

Importantly, CT has shown to improve psychosocial functioning (e.g., Hill et al., 2016), which is a factor that can be easily overlooked when considering recommendations for patients. In considering overall presentation of a patient, this form of intervention – their life, resulting in overall improvement. Caregivers also seem to benefit.

In 2011 the Centers for Disease Control and Prevention's Healthy Brain Initiative developed a Public Health Road Map for the promotion of cognitive health. The recommendations included several areas of intervention including cognitively stimulating activities, physical activity, and healthy vascular function. There are several interventions that can be recommended to patients at varying levels of cognitive functioning. Importantly, these suggestions pointed to numerous lifestyle changes that can be implemented such as diet and exercise that are outlined in detail in the Watch and Wait Model (Chapter 4) and Health (Chapter 6) chapters.

This has also led to an initiative put forth in the National Alzheimer Plan Act to develop a disease-modifying treatment (DMT) by 2025 (Cummings et al., 2016). To complement efforts to develop a DMT for individuals with symptomatic AD, a concerted effort is underway to initiate preventive measures in asymptomatic individuals. For example, several medical conditions are being found to increase the risk of the development of cognitive decline and subsequently dementia. Pharmaceutical studies have been unsuccessful in the prevention and ultimate progression of the disease process. Comparatively, prevention studies focusing on risk reduction and lifestyle modification do appear to offer additional benefits. Getting people motivated, getting them to practice, and having some challenge to the effort seem optimal for whatever positive change will occur.

Efforts to prevent cognitive decline and development of dementia may be more successful when they are multimodal and directed to at-risk individuals based on their personal health profile, rather than using "one-size-fits-all" approaches. AD especially offers a large array of potentially modifiable risk factors (e.g. lifestyle, exposure, environment, comorbid disease) that are excellent targets to personalize the approach to medical care. Precision medicine approaches specifically target the heterogeneity of AD by identifying person-specific risk factors and applying a customized intervention directed against this risk profile. Even if these precision approaches do not cure or prevent AD, removing other pathways to neurodegeneration may greatly improve the likelihood that amyloid- or tau-specific therapies reach their endpoints. Perhaps it is time to abandon generalized approaches to AD and consider neurodegenerative disorders as diseases of a lifetime and that there may be individualized ways to build a better brain as we age.

Medication:

In regards to pharmaceutical intervention, several medications are routinely prescribed within this population. Specifically, the medications

of choice to assist with memory performance are cholinesterase inhibitors (ChEIs) (e.g., donepezil, galantamine, rivastigmine) and low-affinity *N*-methyl-D-aspartate (NMDA) (i.e., antagonist memantine). These medications have not received approval for treatment of mild NCD. Several investigations have assessed whether ChEIs can improve cognitive functioning or delay onset of dementia. Research has failed to show any significant difference in cognitive functioning. These medications, however, did demonstrate significant adverse reactions: gastrointestinal side effects, cardiac syncope, and headaches (Buckley & Salpeter, 2015).

ChEIs are marketing approved for the treatment of mild to moderate dementia. Unfortunately, almost all published trials on ChEIs in the dementia population were industry sponsored. Systematic evidence reviews concluded that ChEI treatment has a small statistically significant improvement in cognitive functioning in dementia patients, yet is not considered to be clinically important (Qaseem et al., 2008). The majority of research has investigated effects in mild to moderate AD. There is less information surrounding Lewy body and vascular dementia. Limited trials with Lewy body dementia reveal better results than in AD, yet there is little improvement with vascular dementia (Buckley & Salpeter, 2015). However, these studies only examined short-term effects; studies that were extended to a year demonstrated reduced benefit overtime (Buckley & Salpeter, 2015). Additionally, there was no benefit for those with advanced disease or for those who were older than 85 years old. Adverse effects were dose-dependent; there was a two to five fold increase in cardiovascular neurological, and gastrointestinal side effects. Those who are 85 and older have twice the associated risk. Also, it appears that almost 25% of dementia patients do not metabolize ChEIs well. Interestingly, drugs typically applied to attention deficit problems may have some benefit.

Comparatively, as noted with ChEIs studies pertaining to the efficacy of memantine were all short-term and industry sponsored. Meta-analysis demonstrated small cognitive benefits in patients with moderate to severe AD and vascular dementia at three months of treatment, which was not observed at six months. When memantine was added to ChEI treatment, there was no statistical improvement in cognitive, functional, or behavioral outcomes (Buckley & Salpeter, 2015). There were no significant benefits observed in mild dementia or Lewy body dementia. Side effects were significantly less compared to ChEIs. Nonetheless, the combination of ChEIs and memantine is used frequently for AD.

The American College of Physicians and American Academy of Family Physicians developed guidelines pertaining to these medications

within the elderly population (Qaseem et al., 2008). Specifically, they recommend that potential initiation of a trial of either ChEIs or memantine should be based on individualized assessment. Decision of medication selection should be based on the patient's tolerability, medication side-effect profile, ease of use, and cost. Additionally, authors stated there is an "urgent need for further research on clinical effectiveness of pharmacological management of dementia" (Qaseem et al., 2008, p. 1).

In general, polypharmacy is not recommended for the elderly population. A systematic review demonstrates that improvement in appropriate polypharmacy is beneficial in reducing inappropriate prescribing and medication-related problems (Patterson, Hughes, Kerse, Cardwell, & Bradley, 2012). AS noted previously, data analyzed from the National Alzheimer's Coordinating Center of 1994 of individuals' age 65 and older with dementia revealed those taking at least five medications were more likely to have functional decline (Lau, Mercaldo, Shega, Rademaker, & Weintraub, 2011). However, this pattern was only significant in adults who were not prescribed with inappropriate prescription medications. Specific to cognitive decline, medications with anticholinergic properties; such as some antidepressants, antipsychotics, antihistamines, and bronchodilators, can cause cognitive worsening or delirium in patients with dementia (Buckley & Salpeter, 2015). Research surrounding discontinuation of anticholinergic medication in the elderly is poorly studied and understood. A systematic review only found four studies that met criteria; one of which found demonstrated improvement of cognitive performance after discontinuation (Salahudeen, Duffull, & Nishtala, 2014). Thus, it is critical for practitioners to be mindful of prescriptions that have anticholinergic properties in adults 65 years and older.

Exercise:

This topic is especially apt in the cognition chapter. We address it in virtually every chapter. Several reviews demonstrated that exercise in older individuals with and without cognitive decline have beneficial effects (e.g., Bherer, 2015; Lee et al., 2010; van Uffelen, Chin A Paw, Hopman-Rock, & van Mechelen, 2008). Lee and colleagues (2010) showed a dose-response gradient with exercise and risk factors of associated with cognitive decline, AD, and cardiovascular disease: diabetes, hypertension, obesity, depression, sedentary lifestyle, and current smoking. The authors recommended that prevention for both cardiovascular disease and cognitive decline should be coupled. Analysis of seminal and recent studies support the concept of cognitive plasticity is

preserved in late adulthood, specific to attentional control as promoted through cognitive training and exercise (Bherer, 2015). Physical exercise demonstrated not only improvement in physical capacity, but also in some cognitive domains and quality of life. Results impacted participants who were both frail and non-frail.

Even more revealing, meta-analysis conducted by Sofi et al., (2011) evaluated the impact of exercise on cognitive decline in a non-demented sample. Results of 33,816 patients suggest a significant protection against cognitive decline for physical activity ranging from high to low-to-moderate levels. Similarly, a meta-analysis examining 2020 participants presenting with dementia or related cognitive impairments, exercise also improved cognitive and physical functioning (Heyn, Abreu, & Ottenbacher, 2004).

Exercise assists all levels of older people with and without problems. Hall and colleagues (2016) assessed the physical performance across the adult lifespan and noted correlates with age and physical inactivity. As to be expected, physical performance decreased across age. Men outperformed their counterparts, yet decrement was similar within age group. Importantly, increased physical activity was associated with better physical performance (World Health Organization, 2018).

Programs that increase physical exercise among people with MCI have also revealed cognitive benefits. A review of the literature revealed older adults who were at risk of AD and engaged in aerobic exercise had a moderate effect on cognitive function (Panza et al., 2018). A similar trend presented in healthy older adults. Specifically, Launtenschlager et al., (2008) randomized individuals with and without MCI were either in treatment as usual or encouraged to complete at least 150 minutes of moderate intensity physical exercise per week for 24 weeks. Those in the intervention group demonstrated modestly better short-term memory, language, processing speed and depression compared with the usual care.

However, some research does not support these findings. For example, Gates, Singh, Sachdev, and Valenzuela (2013) conducted a meta-analysis on the improvements of exercise on patients with MCI. These participants included adults' ages above 65 years diagnosed with mild NCD/MCI or have a MMSE mean score of 24-28. Results indicated there is very limited evidence that exercise improves cognitive functioning within this population. However, given the issues surrounding diagnosing mild NCD/MCI with a single screening measure, these results are suspect since the analysis may be examining individuals not truly representative of the diagnosis.

Another important consideration regarding exercise/physical activity is the patient's ability to engage in these activities. Falls are the leading cause of injury in older adults, which could result in head injury and/or death. Therefore, physical activity can be recommended based on the determination of the patient's physician. It is critical to have open communication with the patient's primary care physician in order to ensure appropriate intervention.

Diet:

As with exercise, we again address diet. Research supports lifestyle and dietary factors for AD that are congruent with recommendations for cardiovascular diseases and diabetes (Barnard et al., 2014). Currently, the Alzheimer's Association recommends two different types of diets: Diet Approaches to Stop Hypertension (DASH) and Mediterranean Diet ("Adopt a Healthy Diet", 2017). Similar to the recommendations of Lee et al. (2010), these diet interventions target both high blood pressure and cognition.

DASH consists of eating foods low in saturated fat, total fat, cholesterol, and decrease intake of fats, red meat, salt, and sugar. The diet focuses on consuming more fruits, vegetables, low-fat dairy, whole grains, poultry, fish and nuts. The Mediterranean diet is quite similar, with slight differences – replace butter with healthy fats, use herbs to flavor food instead of salt, eat fish and poultry at least twice a week. The Mediterranean diet has become very popular within the health field. Multiple studies have been conducted across several populations. Specific to mild NCD and AD, meta-analysis demonstrates those with highest adherence to the diet had 33% less risk of cognitive impairment and reduce risk of mild NCD (Singh et al., 2014).

Preliminary guidelines were developed regarding diet and lifestyle for the prevention of AD from the International Conference on Nutrition and the Brain (Barnard, 2014). In addition to the dietary recommendations outlined above, several other factors were included: (1) vitamin E should come from food, (2) consistent intake of vitamin B12, and (3) only consume iron supplements when directed by physician. Authors also suggest that aluminum contribution to the development of AD is still suspect.

Lifestyle factors such as smoking and heavy alcohol consumption should be eliminated. Specific to smoking, research is confounding. It is hypothesized that smokers develop dementia younger, but were more likely to die before onset due to other health related factors (Cooper, Sommerlad, Lyketsos, Livingston, 2015). Regarding alcohol consumption,

the evidence is also mixed, but this is attributed to low power of studies examining heavy alcohol use. ●f the one clinical study with appropriate power, those who had a lifetime history of heavy drinking were more likely to covert to AD, while moderate drinkers were less likely than abstainers to convert from mild NCD to a dementia (Xu, Liu, Yin, Zhu, Zhang, & Fan, 2009).

As previously discussed, it is critical for these potential interventions be reviewed with patients' medical care providers. There are many potential issues that could present with changing diet and lifestyle, including contraindications of medications. Thus, it is recommended for clinicians to develop strong relationships with these health care providers in order to optimize patient outcomes. See the chapter on Health.

Social engagement:

Establishing and maintaining social connections and engagements is also an important component for quality of life, but this has also shown to have benefits for cognitive functioning. According to the Alzheimer's Association, social engagement has been associated with reduced rates of disability and mortality ("Stay Mentally and Socially Active", 2017). They also suggest social involvement may support brain health and potentially delay onset of dementia.

These claims are well supported through several studies. Hughes and colleagues (2013) examined the effects of social engagement with 816 older adults (mean age = 78 years) who were diagnosed with mild NCD, over the course of three years. Results showed significant lower risk of progression to severe cognitive impairment was associated with greater variety and frequency of involvement in social activities. Similar findings were also found when examining late-life social activity. James, Wilson, Barnes and Bennett (2011) conducted a 12-year longitudinal study (mean follow up = 5.2 years) of 1,138 older community-dwelling adults without dementia at baseline (mean age = 79.6 years). Investigators accounted for many factors including social network size, depression, chronic conditions, disability, cognitive activity, physical activity, neuroticism, and extraversion. With all of these adjusted parameters, results demonstrated an association of a one-point increase in social activity to a 47% decrease in rate of global cognitive decline. Further, global cognitive decline was reduced in those who more frequently engaged in social activity, by an average of 70%. Individuals who were more socially active were also found to have fewer depressive symptoms and higher levels of global cognition at baseline. Perhaps most importantly, these findings remain robust even when the more severe cognitively impaired

participants and those with mild NCD were removed from the analysis; meaning, everyone benefited cognitively from social engagement.

The Alzheimer's Association noted that there have been few systematic reviews within this area (Baumgart et al., 2015). Four longitudinal studies found no effects on cognitive functioning (Brown et al., 2012). Comparatively, a recent meta-analysis conducted over 19 longitudinal cohort studies contradicts these findings. The investigation showed low social participation, less frequent social contact, and heightened loneliness were all significantly associated with incident of dementia (Kuiper et al., 2015). Authors conclude the strength of these findings is comparable with other factors that have been found to contribute to the incident of dementia: low education, physical inactivity, and late-life depression. Naturally, socialization is a base factor for behavioral activation and personal meaning, both highly related to better quality of life.

Case 3

Patient: LV

DOB: 1940

Domains

Cognition:

MoCA: 14/30 raw; memory 0/5

MMSE: 19/30 raw; memory 2/3

TMT-A: WNL

TMT-B: Unable to complete

Health

Medications: Greater than 8 medications including ChEI.

Rated health 8/10

BMI is normal

Sleep: nocturia

Pain: mild (4/10)

Exercise: None

Life Adjustment

Function problems

Well cared for

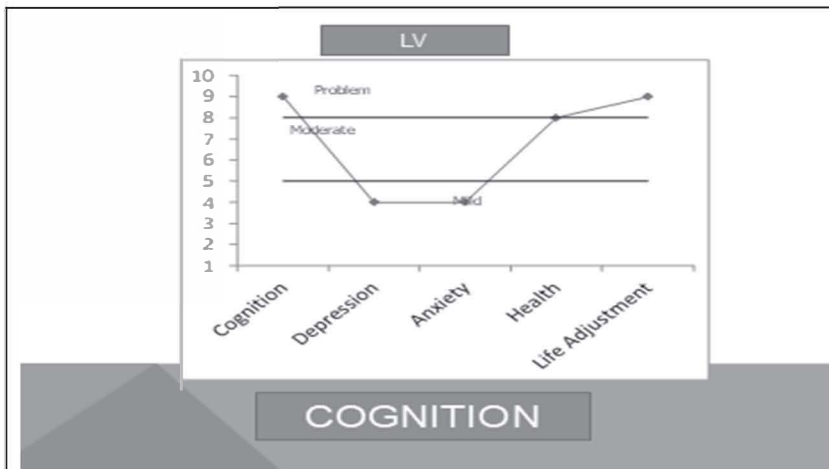
Good SES and education

Depression

Normal scores (see below), MBMD refused

Anxiety

Normal scores (see below), MBMD refused



CHIEF COMPLAINT: Evaluation for cognitive impairment; referred from family medicine.

HISTORY OF PRESENT ILLNESS: The patient is a 77-year-old Caucasian female who reported mild memory problems occurring over the last year. She is independent with ADLs but has problems with some IADLs. She is currently active around the house, but is isolated. Mrs. LV has become increasingly fearful of driving; her husband has assumed this responsibility. Her husband is a medical doctor and completes most of the functions required outside of the home. She was a nurse.

RELEVANT DEVELOPMENTAL HISTORY: The patient is one of nine children. She met milestone markers on time and describes her life as positive. She received her RN degree, but did not practice. She became a homemaker and had four children. She married her husband in 1956, with whom she currently resides. She has been a “homebody.” Her husband has retired and cares for her more and more.

RELEVANT MEDICAL HISTORY: Medication utilization is unclear – the patient and husband did not bring a list to sessions, nor were able to accurately recall the medications. Loosely, she is taking HTN, thyroid and cholesterol medications as well as an RA medication. She has been taking memory enhancement medication for approximately one year. Mrs. LV does not utilize alcohol or tobacco products or drugs. She denies pain, depression, and anxiety. Sleep is interrupted by nocturia. She also suffers from rheumatoid arthritis (RA), which has caused her to fall several times.

MENTAL STATUS: The patient is a 77-year-old Caucasian female who is oriented and able to provide some insight to her situation during

interview. She is oriented and conversed well even if defensive. She endorsed some memory issues but endorsed good functioning, including her ability to recall information she reads. She is reportedly a voracious reader. She seems to resent being placed in the position of being evaluated. Her husband, who was present during the interview, substantiated this information. She denied any perceptual anomalies.

TEST RESULTS: Mrs. LV's predicted premorbid intelligence was within the above average range (WRAT-4 Reading). She had problems on the screen. Her MoCA and MMSE were impaired; she was not able to draw the clock or cube from the MoCA, and was unable to perform Serial 7's, nor able to grasp the concept of TMT-B. Comparatively, performance of TMT Part A was within the average range. Her RBANS performance was impaired across most domains (Total Index SS = 64, 1%; Attention Index = 75, 4%; Visuospatial/Constructional Index = 69, 2%; Immediate Memory Index = 73, 4%; Delayed Memory Index = 44, .1%). Her language was low normal (Language Index = 90, 25%). Subsequent assessment one month later indicated similar results (Total RBANS Index SS = 60).

LIFE ADJUSTMENT: She is reasonably healthy and well managed but does take many medications. Mrs. LV presented with more problems than she would relate to. She was not able to perform tasks around the house; FAQ was 13, demonstrating deficits in executive functioning and ability to complete IADL's. She has problems with sleep, nocturia. Again she has good support.

EMOTIONAL: Emotional self-report scales indicated no depression (BDI-II = 3; GDS-SF = 1) and no anxiety (GAD-7 = 1).

Watch and Wait

Summary: This is a 77-year-old Caucasian female who was referred for an evaluation for her cognitive status. She lives with her husband and receives significant support from both him and their daughter. She is able to perform only some IADLs and does not engage well. Any task that is complicated is a concern for her. Upon feedback, the husband endorsed a slow decline of functioning over the course of years. Mrs. LV meets criteria for major NCD, likely Alzheimer's disease. She is not depressed or anxious and pain and sleep (despite nocturia) are in the normal range. She seems content and resistant to any interventions but has some appreciation for her situation.

Some Watch and Wait checklist items were lacking due to her cognition and commitment. She was provided considerable education on

her situation. Her husband was involved in all sessions.

Domains

1. **Cognition:** She will continue to be monitored, as she has much support from her husband and daughter, who have good command of the situation. We will work with the husband as the primary care giver. He was asked to join a support group and we will see him periodically. Cognitive stimulation for Ms. LV may potentially assist with compensatory skill development. She has scores in the dementia range.
2. **Life Adjustment:** As noted, caregiver protocols were also initiated for the husband. She will be given tasks to become more social, behaviorally activated, and cognitively stimulated.
3. **Health:** Mrs. LV needs to be seen by a geriatrician to assess and monitor her RA symptoms that have resulted in falls, as well as medication management and exercise recommendations. Her nightly incontinence will be addressed. Mrs. LV will be asked to become as socially active as possible (senior center and increased behavioral activation) and to maintain responsibilities in the home, as appropriate date.

Checklist

Discernment: (not fully motivated but compliant: good caregiving)

Core markers

Validate Problem (caregiver) X

Psychoeducation of Model (caregiver) X

Assessment X

Alliance

Monitoring X

Case formulation X

Self or other monitoring: Mini-targets of behavior over time

Activity scheduling: collaborate scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:
 Compassion training:
 Communication and Assertiveness training:
 Behavioral experiments: X
 Emotional exposure:
 Homework: X

Longer Therapy processes:

Caregiving protocol

Lifestyles:

- Practice Spirituality/Religion
- Socialize:
- Practice sleep hygiene
- Exercise:
- Increase cognition:

Follow-up

Cognition:

MoCA and MMSE no change

TMT-A: still WNL

TMT-B: Unable to complete

Health

7 Medications: none added but oxybutynin. Her ChEI was ceased.

Rated health 8/10

Sleep and Pain: normal

PCP follow-up successful

Life Adjustment

Good caregiver support

Function problems

Depression

Normal scores

Anxiety

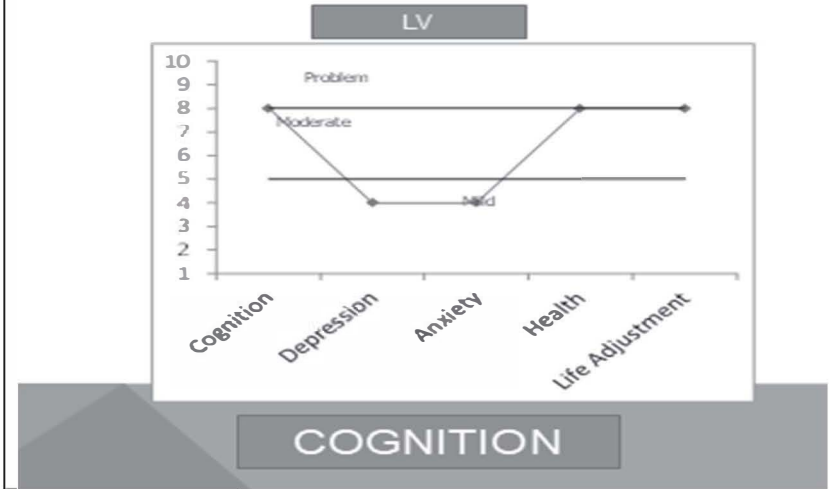
Normal scores

Relapse: N/A

Mrs. LV presented with worsening cognitive functioning, not yet diagnosed as a dementia. For Mrs. LV, cognitive stimulation intervention will not only potentially assist in daily functioning with compensatory strategy development, but will also potentially improve quality of life. Within our clinic, we offered both individual and group cognitive rehabilitation services; individual typically recommended for those with

more severe presentation or who do not benefit from a group setting. Yet, as intervention proves to be too challenging, accommodations can be made. The ACRM manual is a good place to see where alterations can apply (Haskins, 2012-2014).

Post domain profile is similar to the original – cognition and life adjustment issues are paramount.



Conclusion

It is rare to grow old with a completely healthy brain. Until we can titrate the brain and body with function and behavior as well as cognition, we fall short. The biomarkers in the room are helpful but only approximations; poor brain changes in fractional anisotropy, hippocampal atrophy, quantitative EEG alterations, resonance imaging changes, regional blood glucose alterations, increased AB burden, and neuropsychological changes. Importantly, the condition of someone's brain postmortem only partially tells how well functioning was in the years leading up to the person's death, while neuropsychological testing provides good insight to current functional abilities; thus, playing a critical role in health care.

Hopefully this chapter conveys at least that psychological assessment in the context of the person's life is relevant and adds value. The aptitude-treatment interaction may be a better model where the individuation of the person (N of 1) matters and evaluations at this level are important alone. The application of care-based ethics is as important as our tests and

treatment.

From our view though, cognition is an important phenotype, equal to age and biomarkers. Importantly, cognitive performance cannot be captured within a vacuum. Many elements can impact cognitive presentation on assessment. Therefore, it is critical for clinicians to conduct thoughtful and thorough clinical interviews, as well as assess other areas of functioning, as outlined in subsequent chapters: depression, anxiety, health, and life adjustment. Each of the five factors also impact decision-making pertaining to treatment. As always, if other domains outside of cognition present as problematic – these should be addressed and cognition re-assessed once the areas have been treated effectively. However, some of the potential interventions for cognitive functioning can benefit other areas, such as the impact of cognitive training and social engagement on affect.

In sum, each patient presents with different nuances of strengths and weaknesses within all aspects of their lives. This should be considered when examining potential interventions, as discussed in each of the five areas of the model. Dementia cases are all different. Collaborative treatment planning with patients is key to improving outcomes, as it provides a sense of ownership for patients. Outcomes will be further enhanced when integrating patients' health care providers with treatment, when available. This in turn provides patients with more comprehensive treatment and gives the clinician an inside track to health (medical) functioning and potentially acquiring information on a more consistent basis; therefore, being able to more effectively, "Watch and Wait."

References

- 2016 Alzheimer's Disease Facts and Figures. (2016). Retrieved from https://www.alz.org/documents_custom/2016-facts-and-figures.pdf
- Adopt a Healthy Diet. (2017, January). Retrieved from http://www.alz.org/brain-health/adopt_healthy_diet.asp
- Ainslie, N. K., & Murden, R. A. (1993). Effect of education on the clock-drawing dementia screen in non-demented elderly persons. *Journal of the American Geriatrics Society*, 41(3), 249–252.
- Aksu, Y., Miller, D. J., Kesidis, G., Bigler, D. C., & Yang, Q. X. (2011). An MRI-derived definition of MCI-to-AD conversion for long-term, automatic prognosis of MCI patients. *PLoS One*, 6(10), e25074.
- Albert, M. S., DeKosky, S. T., Dickson, D., Dubois, B., Feldman, H. H., Fox, N. C., ... Phelps, C. H. (2011). The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the

- National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia*, 7(3), 270–279. <http://doi.org/10.1016/j.jalz.2011.03.008>
- Alzheimer's disease facts and figures. (2014). Retrieved from http://www.alz.org/alzheimers_disease_facts_and_figures.asp.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Arevalo Rodriguez, I., Smailagic, N., Roqué i Figuls, M., Ciapponi, A., Sanchez-Perez, E., Giannakou, A., ... & Cullum, S. (2015). Mini-Mental State Examination (MMSE) for the detection of Alzheimer's disease and other dementias in people with mild cognitive impairment (MCI). *The Cochrane Library*.
- Attems, J., & Jellinger, K. A. (2014). The overlap between vascular disease and Alzheimer's disease—lessons from pathology. *BMC medicine*, 12(1), 206.
- Axelrod, B. N., Vanderploeg, R. D., & Schinka, J. A. (1999). Comparing methods for estimating premorbid intellectual functioning. *Archives of Clinical Neuropsychology: The Official Journal of the National Academy of Neuropsychologists*, 14(4), 341–346.
- Barnard, N. D., Bush, A. I., Ceccarelli, A., Cooper, J., de Jager, C. A., Erickson, K. I., ... & Morris, M. C. (2014). Dietary and lifestyle guidelines for the prevention of Alzheimer's disease. *Neurobiology of Aging*, 35, S74-S78.
- Barnes, D. E., & Yaffe, K. (2011). The projected effect of risk factor reduction on Alzheimer's disease prevalence. *The Lancet Neurology*, 10(9), 819–828. [http://doi.org/10.1016/S1474-4422\(11\)70072-2](http://doi.org/10.1016/S1474-4422(11)70072-2)
- Baumgart, M., Snyder, H. M., Carrillo, M. C., Fazio, S., Kim, H., & Johns, H. (2015). Summary of the evidence on modifiable risk factors for cognitive decline and dementia: A population-based perspective. *Alzheimer's & Dementia*, 11(6), 718-726.
- Bherer, L. (2015). Cognitive plasticity in older adults: effects of cognitive training and physical exercise. *Annals of the New York Academy of Sciences*, 1337, 1–6. <http://doi.org/10.1111/nyas.12682>
- Bird, T. D. (2015). Alzheimer Disease Overview. In R. A. Pagon, M. P. Adam, H. H. Ardinger, S. E. Wallace, A. Amemiya, L. J. Bean, ... K. Stephens (Eds.), *GeneReviews*(®). Seattle (WA): University of Washington, Seattle.
- Bondi, M. W., & Smith, G. E. (2014). Mild cognitive impairment: A concept and diagnostic entity in need of input from neuropsychology. *Journal of The International Neuropsychological Society*, 20(2), 129-

134. doi:10.1017/S1355617714000010
- Braak, H., & Braak, E. (1991). Neuropathological staging of Alzheimer-related changes. *Acta neuropathologica*, 82(4), 239-259.
- Braak, H., & Del Tredici, K. (2011). Alzheimer's pathogenesis: is there neuron-to-neuron propagation?. *Acta neuropathologica*, 121(5), 589-595.
- Brown, C. L., Gibbons, L. E., Kennison, R. F., Robitaille, A., Lindwall, M., Mitchell, M. B., ... & MacDonald, S. W. (2012). Social activity and cognitive functioning over time: a coordinated analysis of four longitudinal studies. *Journal of Aging Research*, 2012.
- Bruscoli, M., & Lovestone, S. (2004). Is MCI really just early dementia? A systematic review of conversion studies. *International Psychogeriatrics*, 16(2), 129-140.
<http://doi.org/10.1017/S1041610204000092>
- Buckley, J. S., & Salpeter, S. R. (2015). A Risk-Benefit Assessment of Dementia Medications: Systematic Review of the Evidence. *Drugs & Aging*, 32(6), 453-467. <http://doi.org/10.1007/s40266-015-0266-9>
- Centers for Disease Control and Prevention. (2011). *Healthy brain initiative*. Retrieved from:
<http://www.alz.org/publichealth/downloads/2013-RoadMap.pdf>
- Centers for Disease Control and Prevention (CDC). (2013). Self-reported increased confusion or memory loss and associated functional difficulties among adults aged ≥ 60 years - 21 States, 2011. *MMWR. Morbidity and Mortality Weekly Report*, 62(18), 347-350.
- Chang, Y.-L., Bondi, M. W., McEvoy, L. K., Fennema-Notestine, C., Salmon, D. P., Galasko, D., ... For the Alzheimer's Disease Neuroimaging Initiative. (2011). Global clinical dementia rating of 0.5 in MCI masks variability related to level of function. *Neurology*, 76(7), 652-659. <http://doi.org/10.1212/WNL.0b013e31820ce6a5>
- Chen, P., Ratcliff, G., Belle, S. H., Cauley, J. A., DeKosky, S. T., & Ganguli, M. (2000). Cognitive tests that best discriminate between presymptomatic AD and those who remain nondemented. *Neurology*, 55(12), 1847-1853. <http://doi.org/10.1212/WNL.55.12.1847>
- Cooper, C., Sommerlad, A., Lyketsos, C. G., & Livingston, G. (2015). Modifiable predictors of dementia in mild cognitive impairment: a systematic review and meta-analysis. *American Journal of Psychiatry*, 172(4), 323-334.
- Corsentino, E. A., Sawyer, K., Sachs-Ericsson, N., & Blazer, D. G. (2009). Depressive Symptoms Moderate the Influence of the ApolipoproteinE polipoproteinEtoMs Moderate the Influence of the D. G. (2009). Depressive Sym *The American Journal of Geriatric Psychiatry*, 17(2),

155-165.

- Crum, R. M., Anthony, J. C., Bassett, S. S., & Folstein, M. F. (1993). Population-based norms for the Mini-Mental State Examination by age and educational level. *JAMA*, 269(18), 2386–2391.
- Cummings, J., Aisen, P. S., DuBois, B., Frölich, L., Jack, C. R., Jones, R. W., ... & Scheltens, P. (2016). Drug development in Alzheimer's disease: the path to 2025. *Alzheimer's research & therapy*, 8(1), 39.
- Damian, A. M., Jacobson, S. A., Hentz, J. G., Belden, C. M., Shill, H. A., Sabbagh, M. N., ... Adler, C. H. (2011). The Montreal Cognitive Assessment and the mini-mental state examination as screening instruments for cognitive impairment: item analyses and threshold scores. *Dementia and Geriatric Cognitive Disorders*, 31(2), 126–131. <http://doi.org/10.1159/000323867>
- (Donohue, M., Spurling, R., Peterson, R., Sun, C., Weiner, M., and Aisen, P. as part of the ADNI, Alzheimer's Disease Neuroimaging Initiative).
- Donovan, N. J., Amariglio, R. E., Zoller, A. S., Rudel, R. K., Gomez-Isla, T., Blacker, D., ... & Marshall, G. A. (2014). Subjective cognitive concerns and neuropsychiatric predictors of progression to the early clinical stages of Alzheimer disease. *The American Journal of Geriatric Psychiatry*, 22(12), 1642-1651.
- Dong, Y., Sharma, V. K., Chan, B. P.-L., Venketasubramanian, N., Teoh, H. L., Seet, R. C. S., ... Chen, C. (2010). The Montreal Cognitive Assessment (MoCA) is superior to the Mini-Mental State Examination (MMSE) for the detection of vascular cognitive impairment after acute stroke. *Journal of the Neurological Sciences*, 299(1-2), 15–18. <http://doi.org/10.1016/j.jns.2010.08.051>
- Duff, K., Beglinger, L. J., Schoenberg, M. R., Patton, D. E., Mold, J., Scott, J. G., & Adams, R. L. (2005). Test-retest stability and practice effects of the RBANS in a community dwelling elderly sample. *Journal of clinical and experimental neuropsychology*, 27(5), 565-575.
- Duff, K., Humphreysclark, J., Bryant, S., Mold, J., Schiffer, R., & Sutker, P. (2008). Utility of the RBANS in detecting cognitive impairment associated with Alzheimer's disease: Sensitivity, specificity, and positive and negative predictive powers. *Archives of Clinical Neuropsychology*, 23(5), 603–612. <http://doi.org/10.1016/j.acn.2008.06.004>
- Duyckaerts, C., & Boller, F. (2003). Greenfield's Neuropathology. *European Journal of Neurology*, 10(4), 467-468.
- Edmonds et al. (2015).
- Ewers, M., Walsh, C., Trojanowski, J. Q., Shaw, L. M., Petersen, R. C., Jack, C. R., ... Hampel, H. (2012). Prediction of conversion from mild

- cognitive impairment to Alzheimer's disease dementia based upon biomarkers and neuropsychological test performance. *Neurobiology of Aging*, 33(7), 1203–1214.e2.
<http://doi.org/10.1016/j.neurobiolaging.2010.10.019>
- Ferraro, F. R., & Sturgill, D. (1998). Lexical effects and lexical properties associated with National Adult Reading Test (NART) stimuli in healthy young adults and healthy elderly adults. *Journal of Clinical Psychology*, 54(5), 577–584.
- Ferreira, D., Perestelo-PÃ©rez, L., Westman, E., Wahlund, L.-O., SarrÃ-a, A., & Serrano-Aguilar, P. (2014). Meta-Review of CSF Core Biomarkers in Alzheimer's Disease: The State-of-the-Art after the New Revised Diagnostic Criteria. *Frontiers in Aging Neuroscience*, 6.
<http://doi.org/10.3389/fnagi.2014.00047>
- Fleisher, A. S., Sun, S., Taylor, C., Ward, C. P., Gamst, A. C., Petersen, R. C., ... & Thal, L. J. (2008). Volumetric MRI vs clinical predictors of Alzheimer disease in mild cognitive impairment. *Neurology*, 70(3), 191–199.
- Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). "Mini-mental state": A practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, 12(3), 189–198.
- Freitas, S., Simões, M. R., Alves, L., & Santana, I. (2013). Montreal Cognitive Assessment: Validation Study for Mild Cognitive Impairment and Alzheimer Disease. *Alzheimer Disease & Associated Disorders*, 27(1), 37–43.
<http://doi.org/10.1097/WAD.0b013e3182420bfe>
- Gates, N., Singh, M. A. F., Sachdev, P. S., & Valenzuela, M. (2013). The effect of exercise training on cognitive function in older adults with mild cognitive impairment: a meta-analysis of randomized controlled trials. *The American Journal of Geriatric Psychiatry*, 21(11), 1086–1097.
- Godefroy, O., Fickl, A., Roussel, M., Auribault, C., Bugnicourt, J. M., Lamy, C., ... Petitnicolas, G. (2011). Is the Montreal Cognitive Assessment Superior to the Mini-Mental State Examination to Detect Poststroke Cognitive Impairment?: A Study With Neuropsychological Evaluation. *Stroke*, 42(6), 1712–1716.
<http://doi.org/10.1161/STROKEAHA.110.606277>
- Gold, J. M., Queern, C., Iannone, V. N., & Buchanan, R. W. (1999). Repeatable battery for the assessment of neuropsychological status as a screening test in schizophrenia, I: sensitivity, reliability, and validity. *American Journal of Psychiatry*, 156(12), 1944–1950.
- Green, C. S., & Bavelier, D. (2008). Exercising your brain: a review of

- human brain plasticity and training-induced learning. *Psychology and Aging*, 23(4), 692–701. <http://doi.org/10.1037/a0014345>
- Griffin, S. L., Mindt, M. R., Rankin, E. J., Ritchie, A. J., & Scott, J. G. (2002). Estimating premorbid intelligence: Comparison of traditional and contemporary methods across the intelligence continuum. *Archives of Clinical Neuropsychology*, 17(5), 497–507.
- Hall, K. S., Cohen, H. J., Pieper, C. F., Fillenbaum, G. G., Kraus, W. E., Huffman, K. M., ... & Newby, L. K. (2016). Physical performance across the adult life span: correlates with age and physical activity. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, 72(4), 572–578.
- Haskins, E. (2012-2014). *Cognitive Rehabilitation Manual: Translating evidence-based recommendations into practice*. ACRM, Reston, VA.
- Henry, M. S., Passmore, A. P., Todd, S., McGuinness, B., Craig, D., & Johnston, J. A. (2013). The development of effective biomarkers for Alzheimer's disease: a review: AD biomarker review. *International Journal of Geriatric Psychiatry*, 28(4), 331–340. <http://doi.org/10.1002/gps.3829>
- Heyn, P., Abreu, B. C., & Ottenbacher, K. J. (2004). The effects of exercise training on elderly persons with cognitive impairment and dementia: a meta-analysis. *Archives of physical medicine and rehabilitation*, 85(10), 1694–1704.
- Hill, N. T., Mowszowski, L., Naismith, S. L., Chadwick, V. L., Valenzuela, M., & Lampit, A. (2016). Computerized Cognitive Training in Older Adults With Mild Cognitive Impairment or Dementia: A Systematic Review and Meta-Analysis. *American Journal of Psychiatry*, appiajp201616030360.
- Hoops, S., Nazem, S., Siderowf, A. D., Duda, J. E., Xie, S. X., Stem, M. B., & Weintraub, D. (2009). Validity of the MoCA and MMSE in the detection of MCI and dementia in Parkinson disease. *Neurology*, 73(21), 1738–1745. <http://doi.org/10.1212/WNL.0b013e3181c34b47>
- Hughes, T. F., Flatt, J. D., Fu, B., Chang, C. C. H., & Ganguli, M. (2013). Engagement in social activities and progression from mild to severe cognitive impairment: the MYHAT study. *International psychogeriatrics*, 25(04), 587–595.
- Hyer, L. (2014). *Psychological treatment of older adults: a holistic model*. New York, NY: Springer Publishing Company.
- Hyer, L., Mullen, C., & McKenzie, L. (2015). Cognitive training for mildly impaired older adults. In P. A. Lichtenberg, B. T. Mast, B. D. Carpenter, & J. Loebach Wetherell (Eds.), *APA handbook of clinical geropsychology, Vol. 2: Assessment, treatment, and issues of later life*.

- (pp. 341–368). Washington: American Psychological Association. Retrieved from <http://content.apa.org/books/14459-013>
- Jack CR, Bennett DA, Blennow K, Carrillo MC, Dunn B, et al. (2017). 2017 NIA-AA research framework to investigate the Alzheimer's Disease continuum. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 13(7):P890 - P891.
- Jak, A. J., Seelye, A. M., & Jurick, S. M. (2013). Crosswords to Computers: A Critical Review of Popular Approaches to Cognitive Enhancement. *Neuropsychology Review*, 23(1), 13–26. <http://doi.org/10.1007/s11065-013-9226-5>
- James, B. D., Wilson, R. S., Barnes, L. L., & Bennett, D. A. (2011). Late-life social activity and cognitive decline in old age. *Journal of the International Neuropsychological Society*, 17(06), 998-1005.
- Jedynak, B. M., Lang, A., Liu, B., Katz, E., Zhang, Y., Wyman, B. T., ... & Prince, J. L. (2012). A computational neurodegenerative disease progression score: method and results with the Alzheimer's Disease Neuroimaging Initiative cohort. *Neuroimage*, 63(3), 1478-1486.
- Jessen, F., Wiese, B., Bachmann, C., Eifflaender-Gorfer, S., Haller, F., Kölsch, H., ... & Wollny, A. (2010). Prediction of dementia by subjective memory impairment: effects of severity and temporal association with cognitive impairment. *Archives of general psychiatry*, 67(4), 414-422.
- Julayanont, P., Brousseau, M., Chertkow, H., Phillips, N., & Nasreddine, Z. S. (2014). Montreal Cognitive Assessment Memory Index Score (MoCA-MIS) as a Predictor of Conversion from Mild Cognitive Impairment to Alzheimer's Disease. *Journal of the American Geriatrics Society*, 62(4), 679-684.
- Juricia, P. J., Leitten, C. L., & Mattis, S. (2001). *Dementia Rating Scale-2 (DRS-2)*. Professional Manual. Odessa, FL: Psychological Assessment Resources.
- Kivipelto, M. (2001). Midlife vascular risk factors and Alzheimer's disease in later life: longitudinal, population based study. *BMJ*, 322(7300), 1447–1451. <http://doi.org/10.1136/bmj.322.7300.1447>
- Kuiper, J. S., Zuidersma, M., Voshaar, R. C. ●., Zuidema, S. U., van den Heuvel, E. R., Stolk, R. P., & Smidt, N. (2015). Social relationships and risk of dementia: A systematic review and meta-analysis of longitudinal cohort studies. *Ageing research reviews*, 22, 39-57.
- Lau, D. T., Mercaldo, N. D., Shega, J. W., Rademaker, A., & Weintraub, S. (2011). Functional Decline Associated With Polypharmacy and Potentially Inappropriate Medications in Community-Dwelling Older Adults With Dementia. *American Journal of Alzheimer's Disease and*

- Other Dementias, 26(8), 606–615.
<http://doi.org/10.1177/1533317511432734>
- Law, R., & Carroll, R. E. (1998). A comparison of three measures of estimating premorbid intellectual level in dementia of the Alzheimer type. *International Journal of Geriatric Psychiatry*, 13(10), 727–730.
- Le Couteur, D. G., & Sinclair, D. A. (2010). A Blueprint for Developing Therapeutic Approaches That Increase Healthspan and Delay Death. *The Journals of Gerontology Series A: Biological Sciences and Medical Sciences*, 65A(7), 693–694.
<http://doi.org/10.1093/gerona/glq048>
- Lee, Y., Back, J. H., Kim, J., Kim, S.-H., Na, D. L., Cheong, H.-K., ... Kim, Y. G. (2010). Systematic review of health behavioral risks and cognitive health in older adults. *International Psychogeriatrics / IPA*, 22(2), 174–187. <http://doi.org/10.1017/S1041610209991189>
- Levy, B. R., Slade, M. D., Murphy, T. E., & Gill, T. M. (2012). Association Between Positive Age Stereotypes and Recovery From Disability in Older Persons. *JAMA*, 308(19), 1972.
<http://doi.org/10.1001/jama.2012.14541>
- Levy, B. R., Zonderman, A. B., Slade, M. D., & Ferrucci, L. (2009). Age Stereotypes Held Earlier in Life Predict Cardiovascular Events in Later Life. *Psychological Science*, 20(3), 296–298.
<http://doi.org/10.1111/j.1467-9280.2009.02298.x>
- Levy, B. R., Zonderman, A. B., Slade, M. D., & Ferrucci, L. (2012). Memory Shaped by Age Stereotypes over Time. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 67(4), 432–436. <http://doi.org/10.1093/geronb/gbr120>
- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (Eds.). (2012). *Neuropsychological assessment* (5th ed). Oxford; New York: Oxford University Press.
- Lezak, M. D., Howieson, D. B., Bigler, E. D., & Tranel, D. (Eds.). (2012). *Neuropsychological assessment* (5th ed). Oxford; New York: Oxford University Press.
- Lezak, M. D., Howieson, D. B., & Loring, D. W. (Eds.). (2004). *Neuropsychological assessment* (4th ed). Oxford; New York: Oxford University Press.
- Lichtenberg, P. A. (2010). *Handbook of assessment in clinical gerontology*. Amsterdam; Boston: Academic. Retrieved from <https://www.clinicalkey.com/dura/browse/bookChapter/3-s2.0-C20090019114>
- Lowe, D. A., & Rogers, S. A. (2011). Estimating Premorbid Intelligence among Older Adults: The Utility of the AMNART. *Journal of Aging*

- Research*, 2011, 1–7. <http://doi.org/10.4061/2011/428132>
- Lucas, J. A., Smith, G. E., Bohac, D. L., Tangalos, E. G., Kokmen, E., Graff-Radford, N. R., ... Ivnik, R. J. (1998). Normative Data for the Mattis Dementia Rating Scale. *Journal of Clinical and Experimental Neuropsychology (Neuropsychology, Development and Cognition: Section A)*, 20(4), 536–547. <http://doi.org/10.1076/jcen.20.4.536.1469>
- Mackin, R., Nelson, J., Delucchi, K., Raue, P., Satre, D., Kiesses, D., Alexopoulos, G., & Arean, P. (2014). Association of age and depression onset with cognitive functioning in individuals with late life depression and executive dysfunction. *American Journal of Geriatric Psychiatry*, 22, 12, 1633-1642.
- Maddrey, A. M., Cullum, C. M., Weiner, M. F., & Filley, C. M. (1996). Premorbid intelligence estimation and level of dementia in Alzheimer's disease. *Journal of the International Neuropsychological Society: JINS*, 2(6), 551–555.
- Martyr, A., & Clare, L. (2012). Executive function and activities of daily living in Alzheimer's disease: a correlational meta-analysis. *Dementia and Geriatric Cognitive Disorders*, 33(2-3), 189–203. <http://doi.org/10.1159/000338233>
- Mathias, J. L., Bowden, S. C., Bigler, E. D., & Rosenfeld, J. V. (2007). Is performance on the Wechsler test of adult reading affected by traumatic brain injury? *British Journal of Clinical Psychology*, 46(4), 457-466. <http://doi.org/10.1348/014466507X190197>
- Matias-Guiu, J., Cbrera-Martin, M., Valles-Salgado, M, et al. Neural basis of cognitive assessment in Alzheimer disease, amnesic mild cognitive impairment, and subjective memory complaints. *American Journal of Geriatric Psychiatry*, 25, 730-740.
- McFarlane, J., Welch, J., & Rodgers, J. (2006). Severity of Alzheimer's disease and effect on premorbid measures of intelligence. *The British Journal of Clinical Psychology / the British Psychological Society*, 45(Pt 4), 453–463. <http://doi.org/10.1348/014466505X71245>
- McNair, D. M., & Kahn, R. J. (1983). Self-assessment of cognitive deficits. *Assessment in geriatric psychopharmacology*, 137-143.
- Merrill, D. A., Siddarth, P., Raji, C. A., Emerson, N. D., Rueda, F., Ercoli, L. M., ... & Bookheimer, S. Y. (2016). Modifiable risk factors and brain positron emission tomography measures of amyloid and tau in nondemented adults with memory complaints. *The American Journal of Geriatric Psychiatry*, 24(9), 729-737.
- Mendez, M. F. (2017). Early-onset Alzheimer disease. *Neurologic clinics*, 35(2), 263-281.
- Meyer, G. J., Finn, S. E., Eyde, L. D., Kay, G. G., Moreland, K. L., Dies,

- R. R., ... & Reed, G. M. (2001). Psychological testing and psychological assessment: A review of evidence and issues. *American psychologist*, 56(2), 128.
- Miebach, L., Wolfsgruber, S., Polcher, A., Peters, ●., Menne, F., Luther, K., ... & Buerger, K. (2019). Which features of subjective cognitive decline are related to amyloid pathology? Findings from the DELC●DE study. *Alzheimer's research & therapy*, 11(1), 66.
- Mitchell, A. J. (2009). A meta-analysis of the accuracy of the mini-mental state examination in the detection of dementia and mild cognitive impairment. *Journal of psychiatric research*, 43(4), 411-431.
- Molinari, V. (Ed.). (2011). *Specialty competencies in geropsychology*. ●xford ; New York: ●xford University Press.
- Morris, J. C., Storandt, M., Miller, J. P., McKeel, D. W., Price, J. L., Rubin, E. H., & Berg, L. (2001). Mild Cognitive Impairment Represents Early-Stage Alzheimer Disease. *Archives of Neurology*, 58(3). <http://doi.org/10.1001/archneur.58.3.397>
- Mullen, C. M., & Fouty, H. E. (2014). Comparison of the WRAT4 Reading Subtest and the WTAR for Estimating Premorbid Ability Level. *Applied Neuropsychology: Adult*, 21(1), 69–72. <http://doi.org/10.1080/09084282.2012.727111>
- Murry, M. & DeTure, M. (2018) The neuropathology of dementia (pp 41-66). In G. Smith's APA handbook of dementia. APA, Washington, DC.
- Chertkow, H. (2005). The Montreal Cognitive Assessment, MoCA: a brief screening tool for mild cognitive impairment. *Journal of the American Geriatrics Society*, 53(4), 695–699. <http://doi.org/10.1111/j.1532-5415.2005.53221.x>
- Nasreddine, Z. (2019). 90% of untrained raters make these MoCA test mistakes. info@mocatetest.org
- National Institutes of Health. (2011). NHI state of the science conference: Preventing Alzheimer's disease and cognitive decline. Bethesda, MD: U.S. Department of Health and Human Services.
- Nazem, S., Siderowf, A. D., Duda, J. E., Have, T. T., Colcher, A., Hom, S. S., ... Weintraub, D. (2009). Montreal cognitive assessment performance in patients with Parkinson's disease with "normal" global cognition according to mini-mental state examination score. *Journal of the American Geriatrics Society*, 57(2), 304–308. <http://doi.org/10.1111/j.1532-5415.2008.02096.x>
- brien, J. T. (2014). Role of imaging techniques in the diagnosis of dementia. *The British journal of radiology*.
- Bryant, S. E., Waring, S. C., Cullum, M., Hall, J., Lacritz, L., Massman, P. J., ... Doody, R. (2008). Staging Dementia Using Clinical Dementia

- Rating Scale Sum of Boxes Scores: A Texas Alzheimer's Research Consortium Study. *Archives of Neurology*, 65(8), 1091.
<http://doi.org/10.1001/archneur.65.8.1091>
- Orgeta, V., Qazi, A., Spector, A. E., & Orrell, M. (2014). Psychological treatments for depression and anxiety in dementia and mild cognitive impairment. *The Cochrane Library*.
- Paolo, A. M., Tröster, A. I., Ryan, J. J., & Koller, W. C. (1997). Comparison of NART and Barona demographic equation premorbid IQ estimates in Alzheimer's disease. *Journal of Clinical Psychology*, 53(7), 713–722.
- Panza, G. A., Taylor, B. A., MacDonald, H. V., Johnson, B. T., Zaleski, A. L., Livingston, J., ... & Pescatello, L. S. (2018). Can Exercise Improve Cognitive Symptoms of Alzheimer's Disease?. *Journal of the American Geriatrics Society*, 66(3), 487-495.
- Parsons, M. W., & Hammeke, T. E. (2014). *Clinical neuropsychology: a pocket handbook for assessment / Michael W. Parsons and Thomas A. Hammeke, editors ; Peter J. Snyder, founding editor.*
- Patterson, S. M., Hughes, C., Kerse, N., Cardwell, C. R., & Bradley, M. C. (2012). Interventions to improve the appropriate use of polypharmacy for older people. In The Cochrane Collaboration (Ed.), *Cochrane Database of Systematic Reviews*. Chichester, UK: John Wiley & Sons, Ltd. Retrieved from
<http://doi.wiley.com/10.1002/14651858.CD008165.pub2>
- Pedraza, O., Lucas, J. A., Smith, G. E., Petersen, R. C., Graff-Radford, N. R., & Ivnik, R. J. (2010). Robust and Expanded Norms for the Dementia Rating Scale. *Archives of Clinical Neuropsychology*, 25(5), 347–358. <http://doi.org/10.1093/arclin/acq030>
- Pendlebury, S. T., Cuthbertson, F. C., Welch, S. J. V., Mehta, Z., & Rothwell, P. M. (2010). Underestimation of Cognitive Impairment by Mini-Mental State Examination Versus the Montreal Cognitive Assessment in Patients With Transient Ischemic Attack and Stroke: A Population-Based Study. *Stroke*, 41(6), 1290–1293.
<http://doi.org/10.1161/STROKEAHA.110.579888>
- Petersen, R. C. (2004). Mild cognitive impairment as a diagnostic entity. *Journal of internal medicine*, 256(3), 183-194.
- Petersen, R. C., Doody, R., Kurz, A., Mohs, R. C., Morris, J. C., Rabins, P. V., ... & Winblad, B. (2001). Current concepts in mild cognitive impairment. *Archives of neurology*, 58(12), 1985-1992.
- Poey, J. L., Burr, J. A., & Roberts, J. S. (2017). Social connectedness, perceived isolation, and dementia: does the social environment moderate the relationship between genetic risk and cognitive well-

- being?. *The Gerontologist*, 57(6), 1031-1040.
- Qaseem, A., Snow, V., Cross, T., Forcica, M. A., Hopkins, R., Shekelle, P., ... Owens, D. K. (2008). Current Pharmacologic Treatment of Dementia: A Clinical Practice Guideline from the American College of Physicians and the American Academy of Family Physicians. *Annals of Internal Medicine*, 148(5), 370. <http://doi.org/10.7326/0003-4819-148-5-200803040-00008>
- Rabin, L. A., Barr, W. B., & Burton, L. A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: a survey of INS, NAN, and APA Division 40 members. *Archives of Clinical Neuropsychology: The Official Journal of the National Academy of Neuropsychologists*, 20(1), 33-65. <http://doi.org/10.1016/j.acn.2004.02.005>
- Rami, L., Mollica, M. A., García-Sánchez, C., Saldaña, J., Sanchez, B., Sala, I., ... & Molinuevo, J. L. (2014). The subjective cognitive decline questionnaire (SCD-Q): a validation study. *Journal of Alzheimer's Disease*, 41(2), 453-466.
- Randolph, C., Tierney, M. C., Mohr, E., & Chase, T. N. (1998). The Repeatable Battery for the Assessment of Neuropsychological Status (RBANS): preliminary clinical validity. *Journal of Clinical and Experimental Neuropsychology*, 20(3), 310-319. <http://doi.org/10.1076/jcen.20.3.310.823>
- Richardson, E. D., Nadler, J. D., & Malloy, P. F. (1995). Neuropsychologic prediction of performance measures of daily living skills in geriatric patients. *Neuropsychology*, 9(4), 565-572. <http://doi.org/10.1037/0894-4105.9.4.565>
- Roberts, R., & Knopman, D. S. (2013). Classification and Epidemiology of MCI. *Clinics in Geriatric Medicine*, 29(4), 753-772. <http://doi.org/10.1016/j.cger.2013.07.003>
- Rosness, T. A., Haugen, P. K., & Engedal, K. (2011). The clinical dementia rating scale could be helpful in differentiating frontotemporal dementia from Alzheimer's disease. *International Journal of Geriatric Psychiatry*, 26(8), 879-880. <http://doi.org/10.1002/gps.2555>
- Ryan, J. J., & Paolo, A. M. (1992). A screening procedure for estimating premorbid intelligence in the elderly. *Clinical Neuropsychologist*, 6(1), 53-62. <http://doi.org/10.1080/13854049208404117>
- Salahudeen, M. S., Duffull, S. B., & Nishtala, P. S. (2014). Impact of Anticholinergic Discontinuation on Cognitive Outcomes in Older People: A Systematic Review. *Drugs & Aging*, 31(3), 185-192. <http://doi.org/10.1007/s40266-014-0158-4>
- Sattler, J. M., & Ryan, J. J. (2009). *Assessment with the WAIS-IV*. San

- Diego, Calif.: Jerome M. Sattler.
- Savica, R., Wennberg, A., Hagen, C., Edwards, K., Roberts, R. ●., Hollman, J. H., ... & Mielke, M. M. (2017). Comparison of gait parameters for predicting cognitive decline: the mayo clinic study of aging. *Journal of Alzheimer's Disease*, 55(2), 559-567.
- Scheltens, P., & Korf, E. S. (2000). Contribution of neuroimaging in the diagnosis of Alzheimer's disease and other dementias. *Current opinion in neurology*, 13(4), 391-396.
- Schmidtke, K., Pohlmann, S., & Mettemich, B. (2008). The syndrome of functional memory disorder: definition, etiology, and natural course. *The American Journal of Geriatric Psychiatry*, 16(12), 981-988.
- Schretlen, D., Munro, C., Anthony, J., & Pearlson, G. (2003). Examining the range of normal intraindividual variability in neuropsychological test performance. *Journal of International Neuropsychological Society*, 9, 864-870.
- Schweizer, T. A., Al-Khindi, T., & Macdonald, R. L. (2012). Mini-Mental State Examination versus Montreal Cognitive Assessment: Rapid assessment tools for cognitive and functional outcome after aneurysmal subarachnoid hemorrhage. *Journal of the Neurological Sciences*, 316(1-2), 137-140. <http://doi.org/10.1016/j.jns.2012.01.003>
- Scott, J. G., Krull, K. R., Williamson, D. J. G., Adams, R. L., & Iverson, G. L. (1997). ●klahoma premorbid intelligence estimation (opie): Utilization in clinical samples. *The Clinical Neuropsychologist*, 11(2), 146-154. <http://doi.org/10.1080/13854049708407043>
- Shulman, K. I. (2000). Clock-drawing: is it the ideal cognitive screening test? *International Journal of Geriatric Psychiatry*, 15(6), 548-561.
- Silbert, L. C., Howieson, D. B., Dodge, H., & Kaye, J. A. (2009). Cognitive impairment risk White matter hyperintensity progression matters. *Neurology*, 73(2), 120-125.
- Singh, B., Parsaik, A. K., Mielke, M. M., Erwin, P. J., Knopman, D. S., Petersen, R. C., & Roberts, R. ●. (2014). Association of mediterranean diet with mild cognitive impairment and Alzheimer's disease: a systematic review and meta-analysis. *Journal of Alzheimer's Disease*, 39(2), 271-282.
- Smith, G. E., & Farias, S. T. (Eds.). (2018). *APA handbooks in psychology series. APA handbook of dementia*. Washington, DC, US: American Psychological Association.
- Smith, T., Gildeh, N., & Holmes, C. (2007). The Montreal Cognitive Assessment: validity and utility in a memory clinic setting. *Canadian Journal of Psychiatry. Revue Canadienne De Psychiatrie*, 52(5), 329-332.

- Sofi, F., Valecchi, D., Bacci, D., Abbate, R., Gensini, G. F., Casini, A., & Macchi, C. (2011). Physical activity and risk of cognitive decline: a meta-analysis of prospective studies. *Journal of internal medicine*, *269*(1), 107-117.
- Sperling, R. A., Aisen, P. S., Beckett, L. A., Bennett, D. A., Craft, S., Fagan, A. M., ... Phelps, C. H. (2011). Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia*, *7*(3), 280-292. <http://doi.org/10.1016/j.jalz.2011.03.003>
- Stay Active. (2017, January). Retrieved from http://www.alz.org/brain-health/stay_physically_active.asp
- Stay Mentally and Socially Active. (2017, January). Retrieved from http://www.alz.org/brain-health/stay_mentally_socially_active.asp#social
- Strauss, E., Sherman, E. M. S., & Spreen, O. (2006). *A compendium of neuropsychological tests: administration, norms, and commentary* (3rd ed). Oxford; New York: Oxford University Press.
- Sundermann, E. E., Maki, P. M., Rubin, L. H., Lipton, R. B., Landau, S., & Biegon, A. (2016). Female advantage in verbal memory: Evidence of sex-specific cognitive reserve. *Neurology*, *87*(18), 1916-1924. doi:<http://dx.doi.org.ezproxy3.lhl.uab.edu/10.1212/WNL.0000000000003288>
- Susman, E. (2019). *Combating memory loss: Common problems and treatments*. Mind, Mood and Memory and Massachusetts General Hospital. Boston, MA.
- Toglia, J., Fitzgerald, K. A., O'Dell, M. W., Mastrogiovanni, A. R., & Lin, C. D. (2011). The Mini-Mental State Examination and Montreal Cognitive Assessment in Persons With Mild Subacute Stroke: Relationship to Functional Outcome. *Archives of Physical Medicine and Rehabilitation*, *92*(5), 792-798. <http://doi.org/10.1016/j.apmr.2010.12.034>
- Tombaugh, T. N., McDowell, I., Kristjansson, B., & Hubble, A. M. (1996). Mini-Mental State Examination (MMSE) and the Modified MMSE (3MS): A psychometric comparison and normative data. *Psychological Assessment*, *8*(1), 48-59. <http://doi.org/10.1037/1040-3590.8.1.48>
- Tuokko, H. A., & Smart, C. M. (2018). *Neuropsychology of Cognitive Decline: A Developmental Approach to Assessment and Intervention*. Guilford Publications.
- Van Uffelen, J. G. Z., Chin A Paw, M. J. M., Hopman-Rock, M., & van

- Mechelen, W. (2008). The effects of exercise on cognition in older adults with and without cognitive decline: a systematic review. *Clinical Journal of Sport Medicine: Official Journal of the Canadian Academy of Sport Medicine*, 18(6), 486–500. <http://doi.org/10.1097/JSM.0b013e3181845f0b>
- Wiens, A. N., Bryan, J. E., & Crossen, J. R. (1993). Estimating WAIS-R FSIQ from the national adult reading test-revised in normal subjects. *Clinical Neuropsychologist*, 7(1), 70–84. <http://doi.org/10.1080/13854049308401889>
- World Health Organization (2018). Global strategy on diets, physical activity and health: Physical activity and older adults. Retrieved from: http://www.who.int/dietphysicalactivity/factsheet_olderadults/en/
- Williams, M. M., Roe, C. M., & Morris, J. C. (2009). Stability of the Clinical Dementia Rating, 1979–2007. *Archives of Neurology*, 66(6), 773–777. <http://doi.org/10.1001/archneurol.2009.69>
- Xu, G., Liu, X., Yin, Q., Zhu, W., Zhang, R., & Fan, X. (2009). Alcohol consumption and transition of mild cognitive impairment to dementia. *Psychiatry and clinical neurosciences*, 63(1), 43–49.
- Zadikoff, C., Fox, S. H., Tang-Wai, D. F., Thomsen, T., de Bie, R. M. A., Wadia, P., ... Marras, C. (2008). A comparison of the mini mental state exam to the montreal cognitive assessment in identifying cognitive deficits in Parkinson's disease: MoCA Versus MMSE in Assessing Cognition in PD. *Movement Disorders*, 23(2), 297–299. <http://doi.org/10.1002/mds.21837>
- Zhu, X. C., Tan, L., Wang, H. F., Jiang, T., Cao, L., Wang, C., ... & Yu, J. T. (2015). Rate of early onset Alzheimer's disease: a systematic review and meta-analysis. *Annals of translational medicine*, 3(3).

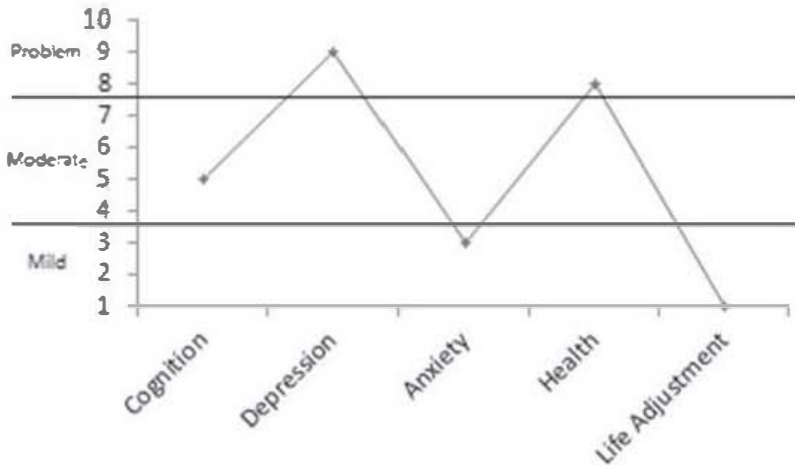
CHAPTER 8

DEPRESSION

LEE HYER, CATHERINE A. YEAGER
AND IAN A. YEAGER

As in the other chapters we pick up on the case of SB from Chapter 5.

PATIENT: SB
DOB: 1942



SB Case

Domains

Depression

MINI: Positive for Depression

BDI = 23

PHQ-9 = 13

GDS-SF = 7

MMBD: Depression

Health

Chronic Diseases: 4 (CHF)

Health Rating: 2/10

ESS = 10

Pain: 20

Cigarettes: No

Alcohol: None

BMI: 27

Exercise: None, excessive sedentary behavior

Anxiety

GAD-7 and STAI are normal

Cognition

MoCA was normal and normal range for other scales (see below)

Life Adjustment

Retired professional

Good support

Interactive

Adequate SES and good education

Does all IADLs

HISTORY OF PRESENT ILLNESS: Mrs. B indicated that for some time now she has had problems with her memory. She moved from New Orleans, Louisiana roughly four years ago to be near her daughter in Georgia. She has health issues that involve a recent cardiac bypass and other medical issues related to the vasculature system.

RELEVANT DEVELOPMENTAL HISTORY: Mrs. B is a native of Louisiana. She is the older of two children. Her father worked for the city government and her mother was a librarian. She was graduated from high school and went on to become an RN. She practiced at that profession in different ways; ER, home health, public health, and for 25 years as a school nurse. She was married at age 21. That marriage lasted 42 years when her husband died in 2005. They had two girls. She mentions 2005 and 2006 as being a momentous time in her life because her husband died

and because she developed cardiac problems and also retired. She now lives alone near one of her daughters.

RELEVANT MEDICAL HISTORY: Mrs. B indicated that she is concerned about her memory and has been so for a number of years. She also admitted to being unhappy. She noted that she had a psychological evaluation in Louisiana in roughly 2012, which indicated that she has been diagnosed as one with dementia. She did not give good information regarding this assessment. She does not drink alcohol because, she reports, she is allergic to it. She quit smoking tobacco in 1965. She indicates that she has very mild pain. She has an esophageal spasm that causes trouble and she takes belladonna alkaloid for it. She has sleep apnea and uses a CPAP for the past 10 years. She also has poor sleep habits, sleeping until roughly 10:00 in the morning. She takes naps often and late into the evening. She does not have a formal exercise program, but does have a Fitbit to count her steps. She takes the belladonna alkaloid, Imdur, Coreg, a statin, carvedilol, isosorbide, HCTZ, Zoloft, Plavix, ranitidine, 0.81 aspirin, cranberry supplement, a woman's vitamin, and CoQ10. She has a psychiatric history that dates back to roughly 2005 when she was placed on Zoloft because of distress stemming from her retirement, her husband's death, and some health issues. She indicates that she does not have a lot of stress these days, but is very inactive and stays at home a good deal of the time.

MENTAL STATUS: The patient is a 74-year-old Caucasian female who drove to this appointment. She was well dressed and interacted in a very matter-of-fact way. Over time she smiled more. She related that she now leads her life in a very structured way. She answered questions appropriately and in-depth. She said she is not happy, but not depressed either. There was little indication that she was anxious. There were no perceptual anomalies or delusional thinking. She had a need to be quite precise and complete in her description of her life. Her judgment appears to be good and her insight appears to be adequate.

PREMORBID FUNCTIONING and OVERALL COGNITIVE FUNCTIONING: Mrs. B has a premorbid intelligence score placing her in the high average area intellectually based on the Barona Index of Intelligence. Her WASI Full Scale is 109; Verbal = 114 and Performance = 103. This is a score reflective of high average skills. Her MoCA was 26/30, low average. Her RBANS Index was 99, average. She showed little variability on the domains, including memory, attention, and visuospatial areas. She has excellent language. Her executive functioning is average (Trails B and WCS). Based on these scores, she is responding now about average commensurate to her per-morbid abilities.

EMOTIONAL ASSESSMENT: On the MINI, Mrs. B scored in the range for depression. Emotional self-report scales indicated depression (BDI = 23, PHQ-9 = 13 and Mood Scale = 7), but not anxiety (GAD-7= 4). STAI is normal also. The PAI and MBMD indicated that she is depressed and has somatic/health concerns. She has a respectful personality profile (MBMD). This suggests that she is treatment compliant and in need of structure. She wants to be on top of her life.

PAIN and SLEEP: Mrs. B has no pain sensitivity problems on the MBMD. She experiences pain at 3 out of 10 and this does not interfere with her IADLs. Sleep is a problem. She awakens later in the day, uses a CPAP, and naps frequently.

WATCH and WAIT: This is a 74 y/o female who was referred for an evaluation of her cognitive status. She lost her husband 12 years ago. She is a retired RN and has raised a family. She can do all ADLs and all IADLs. She lives alone but has support. Cognitively she has a normal intelligence. She is better verbally than non-verbally. She has reasonable skills in all domains, especially memory and new learning. She is NOT in a dementia.

Mrs. B is depressed and has health issues. She is a (mild) loner and has been by herself for a few years. Her self-concept is somewhat rigid in process but she has a rather positive self-evaluation. She can be self-critical, however, and has been focusing upon past failures and lost opportunities. She is troubled inwardly by self-doubt and misgivings about her adequacy that is apparent to others. She is a very reserved person who represses conflict and, to some extent, feelings. She is mildly active, sleeps somewhat poorly, and has been treated for depression for years. She does not have an exercise program. She is, however, one who wants to comply and please.

Psychiatric Indications	AA	7	60		ANXIETY-TENSION
	BB	12	77		DEPRESSION
	CC	14	72		COGNITIVE DYSFUNCTION
	DD	5	40		EMOTIONAL LABILITY
	EE	7	35		GUARDEDNESS
Coping Styles	1	4	45		INTROVERSIVE
	2A	3	60		INHIBITED
	2B	3	45		DEJECTED
	3	8	60		COOPERATIVE
	4	6	35		SOCIABLE
	5	7	40		CONFIDENT
	6A	4	25		NONCONFORMING
	6B	7	35		FORCEFUL
	7	24	80		RESPECTFUL
	8A	5	50		OPPOSITIONAL
	8B	6	71		DENIGRATED

Checklist

Discernment: X (very motivated)

Core markers

Validate Problem X

Psychoeducation of Model X

Assessment X

Alliance X

Monitoring X

Case formulation X

Problem List

1. **DEPRESSION:** Monitor mood, behavioral activation, CBT group, somatic softening techniques, ACT modules, psychoeducation, mild grief intervention, social interaction
2. **HEALTH:** Titrate care with involvement of Cardiology, help monitor HTN and heart markers, check with daughter on medications, encourage wellness center and make a nutritional consult.

Mini-Modules

Self or other monitoring: targets of behavior over time X

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Contingency plans (If-then): X

Apply progressive relaxation: X

Concreteness training:

Imagery exercise for counter-experience: X

Compassion training: X

Communication and Assertiveness training: X

Behavioral experiments: X

Emotional exposure:

Homework: X

Longer Therapy processes:

CBT: Group CBT and use of 3-6 column evidence record

ACT: use of acceptance interventions

Lifestyle:
 Practice happiness
 Practice Spirituality/Religion
 Socialize: Less decline and live longer
 Develop resilience
 Exercise: Attend Wellness Center
 Somatic softening: Meditate and Mindfulness
 Values and Leisure
 Follow-up domain profile (3 months) does not differ from the original.

She will be given feedback on this report. We will attempt to engage her and optimize response to the problems noted. She has solid life values and respectful personality traits, but the need for psychological treatment is evident. She will be encouraged to appreciate her health, to receive some counseling, and to alter her life mildly. We will monitor her depression and health.

Depression Model

Measure	Healthy	Moderate Problem	Problematic or Syndromic
Description	The patient is not depressed, mood is expressed in a healthy range and not consistently low.	The patient may have a subclinical depression or dysthymia. Mood is slightly depressed without significant distress or impairment.	The patient is likely experiencing depressed mood, anhedonia, and/or suicidal ideation. Many DSM-5 symptoms will be endorsed, some with great severity. Functioning is impaired
MINI	No positive Hits	Borderline MDD or Dysthymia	MDD or Dysthymia
BDI-II	≤10	11-22	≥23
PHQ-9	≤6	7-11	≥12
GDS-SF	≤5	6-10	≥11

ASSESSMENT

Screen:

A useful measure of depression is the Mini International Neuropsychiatric Interview (MINI) which is a brief structured interview meant to aid in the assessment of 16 DSM-III disorders and was later updated to be current with the DSM-IV (Lecrubier, et al., 1997; Sheehan et al., 1998). The MINI shows good agreement with the Composite International Diagnostic Interview (CIDI; kappa = .73); the MINI accomplished this in less than

one fourth of the time on average. The average time to administer the MINI is 21 minutes (SD = 7.7 minutes); for the purpose of assessing depression, many modules could conceivably be excluded from the interview. This relatively quick interview has high interrater reliability ($\kappa = .88-1.0$) and a test retest κ of .83 (Lecrubier et al., 1997). The brevity of the MINI, along with good convergence with more time consuming structured interviews, makes it an appealing choice for a guided interview. That said, an analysis of the accuracy or utility of the MINI when used with older adults has not yet been published.

The Beck Depression Inventory-II (BDI or BDI-II: Beck et al., 1996) is probably the most commonly used self-report screener for depression. It consists of 21 items and is recommended for use in people 13 years and older. It has been validated on older adults; it shows good internal consistency with coefficient alphas of .84 - .90 with the lowest correlating item related to sexual interest ($r = .05$; Jefferson, et al., 2001). When BDI-II items are compared across age groups, older adults endorse the following items less often: sadness, guilty feelings, punishment feelings, self-dislike, self-criticalness, crying, agitation, and irritability. Loss of interest in sex and loss of energy are endorsed more often by elders than by younger adults (Segal et al., 2008). Steer and colleagues (1999; 2000) compared the BDI-II factor structure by age and found that it is different in older adults when compared to a younger sample on several items. For example, crying was found to be a cognitive factor in older adults, whereas pessimism was a non-cognitive factor. Yet, other similar studies have shown pessimism to be a cognitive factor in older adults (Segal et al., 2008). It is notable that the BDI-II was unable to differentiate between older adults with MDD and older adults with adjustment disorder with depressive symptoms. This is one area in which clinical discretion and additional assessments are probably necessary to distinguish major depression from adjustment problems.

The 9 item Patient Health questionnaire (PHQ-9) is a self-report questionnaire derived from the full Patient Health Questionnaire (PHQ: Kroenke, et al., 2001). The PHQ and PHQ-9 were developed to be useful in a variety of medical settings and are easy to understand. Scores on the PHQ-9 range from 0-27; each of the items is scored 0-3, where each number reflects the frequency of each symptom (0-not at all; 1-several days; 2-more than half the days; 3-nearly every day) in the previous two weeks. There is an additional question that asks the person to rate his/her level of functioning over this period. The PHQ-9 has high internal reliability of .86 to .89 (Löwe et al., 2004). Manea, et al. (2012) conducted a meta-analysis of the psychometric properties of the PHQ-9 and determined that a cutoff score

between 8-11 strongly suggests a diagnosis for depression.

The Geriatric Depression Scale (GDS: Yesavage et al. 1983) is a 30-item measure of depression designed for older adults. The GDS uses YES/NO responses. The 15 item Geriatric Depression Scale-Short Form (GDS-SF: Sheikh & Yesavage, 1986) is face valid, reliable and very easy to use. It is more popular than the long version. It factors into one domain, unsatisfactory quality of life (Hyer, 2014). The GDS-SF is internally consistent and features items that reflect unhappiness and less than optimal enjoyment in life. It is not indicative of clinical depression unless the score is quite high (>10). A score of 5 reflects mild dysphoria and below 5 is within normal limits. The GDS-SF is an often used screener in Primary Care settings because it is quick to administer, easy to score, and shows good sensitivity for the detection of depression. Of note, the GDS excludes somatic symptoms because depressed older adults often misinterpret somatic symptoms as a medical comorbidity. As such, the clinician will want to elucidate somatic complaints in addition to giving the GDS. Finally, it is worth emphasizing that there are several other scales that have adequate psychometrics that can be applied to older adults (Balsamo, Cataldi, Carlucci, Padulo, & Fairfield, 2018).

All depression scales have problems where older adults are at issue. Virtually all scales used on older adults were developed on younger adults. Unfortunately there is no careful understanding or assessment of other related psychiatric disorders, such as bipolar disorder, dysthymia and adjustment reactions. Also caregivers over-report problems, especially depression. Below is a listing of other problems with self-report measures.

PROBLEMS WITH DEPRESSION SELF-REPORT SCALES

- overlap between depression and dementia
- overlap with somatic symptoms
- Little accounting for level of sensory or cognitive impairment
- Little accounting for end-of-life phenomenology in depression issues
- overlap with anxiety
- Under-recognition of depressive symptoms
 - Less likely to verbalize moods
 - Less likely to know low mood symptoms (e.g., anhedonia)
 - Attribute problems to normal aging
 - Less likely to report cognitive problems as symptoms of depression
 - Less likely to see inactivity as depression
 - Less likely to connect sexual problems with depression
 - Time is problematic as skills/behaviors/moods vary by day

Assessment of Suicide and Grief:

One special area of concern for older adults is suicide because they have the highest rate among age groups (>18/100,000). Older adults with depression are likely to express hopelessness and sometimes suicidal ideation. However, suicide risk assessment is usually not addressed. Of elders who have died by suicide, 71%-90% had a diagnosable psychiatric disorder (Conwell, 2011), with depression being the most common condition. Hopelessness and perceived burden to others are cognitions that are associated with increased suicidal ideation and risk of suicidal behavior (Conwell, 2011). Serious medical conditions such as cancer, severe cardiovascular disease, and neurological disease are associated with between 35% and 70% of suicides in older adults (Fiske et al., 2009; Fiske, Riley, and Widoe, 2008). Functional disability and poor quality of social support also increase the risk for depression and suicide. There is evidence, too, that older adults who commit suicide have more vascular brain involvement (WMH, microbleeds). Finally, access to firearms is significantly associated with suicide risk in this group, mostly among men.

There are few suicide risk assessments specifically standardized for older adults. Item 9 on the PHQ-9 addresses it directly and should be quickly monitored. The Geriatric Suicide Ideation Scale (GSIS: Heisel & Flett, 2006) is a 31-item measure developed to measure suicidal ideation, death ideation, loss of personal and social meaning, and perceived meaning in life. This scale converges well with other measures of suicidal ideation and has strong internal inter-item consistency. The GSIS has been shown to differentiate older adults with low levels of suicidal ideation from older adults with high levels of suicidal ideation. Heisel & Flett (2016) recommend using a cutoff of 12 on the GSIS and a cutoff score of 6 on the GDS-SF to achieve the best sensitivity and specificity in predicting suicidal ideation.

Two other scales are worth noting. The Terminally Ill Grief and Depression Scale (TIGDS: Periyakoil et al., 2005) is a 42-item measure developed to assess preparatory grief and depression in adults with terminal illness. The depression subscale of the TIGDS is convergent with other measures of depression, whereas the Grief Scale does not correlate with other depression measures. Also, Prigerson and colleagues (Prigerson et al., 1995) have addressed complicated grief for many years. The Inventory of Complicated Grief (ICG) that measures maladaptive symptoms (scores > 25 are clinically significant). This scale assesses a constellation of symptoms including a preoccupation with the deceased, longing and yearning, disbelief and inability to accept the death, and avoidance of reminders of the loss.

To more fully understand suicide risk, one should also assess for protective factors that promote resiliency and decrease the risk of suicide (Heisel & Flett, 2008). One way to do this is to apply the Reasons for Living Scale-Older Adult version (RFL-OA; Edelstein et al., 2009) in the suicide risk assessment. The RFL-OA is a 69-item Likert scale that measures factors that can influence one's decision-making about suicide. For example, the respondent is quizzed on reasons for living, such as, "my religious beliefs forbid it;" "I want to see my grandchildren grow up;" "Tomorrow I may feel better."

Overview of Depression

Depression is one of most common ailments. About 7%-9% of adults are afflicted with depression annually, and there is somewhere around a 17% lifetime risk, with some studies showing prevalences as high as 1 in every 5 people (Patten, 2010). Almost 14% of individuals older than 55 years have depression (Beekman et al., 1999). When viewed as a medical problem, depression is #8 on the list of the 10 most common chronic diseases in adults (Gerteis et al., 2014). It would therefore be accurate to say that most people have either dealt with depression themselves or have family members or friends who've struggled with depression. While there is not total agreement, depression may well be the leading cause of disability worldwide. Depressive syndromes are the leading contributor of disability-adjusted life years (Ferrari et al., 2013). Women are more likely to have depression than men. The condition also is more prevalent in people living in poverty and in stressful environments. Of note, depression in older adults increases mortality rates regardless of SES and other medical conditions (Schulz et al., 2000). Of older adults who have depression, less than half are experiencing it for the first time. Such late onset depression may warrant a different conceptualization than recurrent depression. Older adults who first experienced depression at a younger age more frequently have elevated personality scales on neuroticism/anxiousness and/or a family history of depression.

As we have noted, depression encompasses a number of variants, including: major depression, minor or subsyndromal depression, bipolar depression, 'late life' depression, and dysthymia. Signs and symptoms vary in number, intensity, and chronicity. MDD is defined by the presence of at least 5 of 9 criteria in the DSM-5 (APA, 2013) that must be present for most days for at least 2 weeks. These symptoms must cause distress or impairment in some realm of functioning. MDD peaks in prevalence between 30-44 years with an overall 12 month prevalence of 7%; the rate

of MDD among adults >60 years is significantly lower than that of any other age group (Kessler et al., 2005). Prevalence of MDD increases substantially, however, among elders older than 85 years and among nursing home residents (Luppa et al., 2012; Seitz et al., 2010). Subsyndromal or minor depression, in which at least two DSM 5 symptoms are present for at least 2 weeks - one of which is depressed affect - impairs 15%-25% of older adults (Jeste, et al., 1999). Similarly, dysthymia, that is, a low grade, chronic depressive style from which episodes of MDD can arise, affects about 3%-6% of the population, but, like MDD, is thought to be less prevalent in older adults. In older individuals, dysthymia may have its onset in middle or late life, and appears to have less associated psychiatric comorbidity but closer ties to severe life stressors and medical conditions (Kocsis, 1998). In general, epidemiologic data on these diagnoses in older patients should be cautiously considered because factors such as cognitive deterioration, chronic medical diseases, and frequent adverse life events (e.g. bereavement) can complicate the clinical picture, making an accurate diagnosis more difficult to make.

The idea that subsyndromal depression (SSD) is less problematic or influential on adjustment, especially cognition, is a misconception. SSD is defined as depressive symptomatology below the frequency and severity for the diagnosis of MDD. SSD has been associated with more rapid conversion from MCI to AD and dementia, implying that SSD may be an etiological risk factor or prodromal manifestation of dementia neuropathology. Individuals with MCI and chronic SSD have been found to display more rapid deterioration in global cognitive functioning, as well as accelerated progression to dementia.

Neuroimaging assessments of cortical atrophy may hold particular promise to elucidate the neurobiological mechanisms of SSD-related accelerated cognitive decline. Cortical atrophy in the hippocampus and entorhinal cortex has been consistently reported in MCI. Similarly, late-life depression has been associated with cortical atrophy, particularly within the anterior cingulate, prefrontal cortex, and orbital frontal cortex. In a study we have previously cited by Gonzales, et al. (2017), individuals with chronic SSD seemed to represent a sub-group that is highly vulnerable to accelerated cognitive decline, an effect that may be governed by frontal lobe and anterior cingulate atrophy. It remains to be seen, however, whether these additional anatomical changes in SSD place individuals at greater risk for the development of atypical Alzheimer's disease with greater frontal systems involvement.

Because of the fuzzy nature of depression in older adults, 'depression' from here on will refer to a broader depressed state rather than one specific diagnosis. The cause of depression is unknown. As noted above, it is clearly a heterogeneous condition, and there are a number of likely etiologies. Because of this, consideration of depression as a nonspecific response to brain injury or systemic inflammation, rather than as a specific entity, is also necessary (see Chapter 5 for more on depression as a reflection of systemic inflammation).

Again, the signs and symptoms of MDD are fairly defined in the DSM. They include low mood and anhedonia. Anhedonia is most likely the more specific diagnostic symptom of the two. Many of these signs and symptoms—for example, low mood or anhedonia—are common in the general population, perhaps as high as 30% (for low mood), and aren't always associated with a depressive syndrome. Thus, accurate diagnosis can be rather elusive in older adults because of the reciprocal relationships among depression symptoms, social functioning, cognition, higher disease burden, and medication side effects. Identification of depression can also be complicated by age-related factors and tacit social expectations of older adults to display behaviors characteristic of depression. For example, a noted slowness in movement in older adults may be cautiousness, deliberativeness, drug induced slowing, or depression. Also, the importance of executive functioning (EF) in the presence of depression is often underestimated (i.e., Depressive Executive Dysfunction; Alexopoulos, et al., 2002). In short, teasing out when an apparent symptom of depression is actually depression is more challenging in older adults.

Depression, then, is broad in scope. Lu and Ahmed (2010) note: "There is increasing evidence that symptoms of elderly depression may be etiologically distinct (e.g., more psychomotor retardation and anhedonia in vascular depression) and that focusing on subclusters of depressive symptoms, rather than relying on general depression assessment tools may help enhance construct validity..." (p.379). After several cluster analyses on depression at late life, Hybels et al. (2009) noted: "The heterogeneity in symptom presentation among older adults diagnosed with MDD can potentially inform the development of the DSM-5" (p.387).

The heterogeneity of depression is further complicated, in many cases, by anxiety. Representative estimates of mood, anxiety, and combined mood and anxiety symptoms using a sample of 2,575 survey participants age 55 and older found that 5% developed a mood disorder, 12% developed anxiety disorders, and 3% developed mood and anxiety disorders within the previous year. Prevalence of all conditions declined with age. Women were, again, more likely than men to have combined

depression/anxiety (Byers et al., 2010).

There are a number of pitfalls that clinicians are susceptible to when it comes to making a depression diagnosis. Many clinicians have their own conceptualizations of depressive disorders that may lack sufficient flexibility to widen these to a new population of patients. Other clinicians take a less personalized but no less rigid approach to the diagnosis of depression, wherein meeting the ICD-10/DSM criteria, or being one point above the cutoff on an assessment, is the necessary and sufficient template for depression. That said, neither the conceptual approach nor criteria-based approach takes into account the totality of a patient's experience. That is to say, a symptom such as anhedonia may be indicative of depression in 100 patients, but in patient 101, it may just be a normative variant. That hundred and first patient may also report an apparently benign thought or behavior or even a world view that, for that person, is indicative of a problem. It is because of that "one-off" patient that clinicians must exercise diagnostic flexibility informed by clinical experience and research to properly identify and treat the depressive syndromes. In fact, the specific symptoms of depression can be reliably diagnosed reasonably well. In the DSM-5 Field Trial, the kappa measure of reliability tended to be about 0.6. So, researchers were able to recognize specific symptoms in a person when two separate raters conducted the evaluation on two separate occasions. However, the reliability of the *syndrome* of depression itself—when you accumulate the signs, symptoms, and behaviors whereby the patient meet at least five DSM criteria and at least one of these is depressed mood or anhedonia—was poor. This suggests that the clinician will need to sometimes think outside the box to correctly diagnose depression in an older adult.

We note too, that, the DSM is not helpful for more serious variants of later life depression. For example, it does not help with bipolar disorder at later life. About 5% of new bipolar cases occur in older age groups. This in fact may be a secondary mania caused by a medical condition such as multiple sclerosis. Bipolar disorder also can be secondary to various types of brain injury such as closed head injury or TBI, stroke, subarachnoid bleeds or other hemorrhages. There is no specific treatment for this and no specific diagnostic technique for its designation. For new symptom presentations, medical assessment becomes paramount.

Myths about Aging and Depression

Haigh, Bogucki, Sigmon, and Blazer (2018), in a review article, revisited common myths and misconceptions about depression in late life first raised by Blazer in 2003. They identified 5 myths and sought to set the record straight. Myth 1 is that depression at later life is symptomatically different from depression at younger ages, with more somatic and cognitive complaints and less frequent emotional complaints. There is of course some evidence, as described above, that the phenomenology of depression differs in older adults compared to younger adults. Most notably, somatic symptoms are more prevalent in older adults and especially in elders with late-onset depression. Nevertheless, there is not a preponderance of high quality evidence to suggest that such symptomatic differences are either consistent across different samples or clinically meaningful. The severity of depressive symptoms may be overestimated in older adults because of comorbid physical problems and underestimated in the oldest old because of their tendency to under-report depressive symptoms altogether. Again, this has not been shown to be consistent, especially in community-dwelling elders and those whose symptoms do not meet full diagnostic criteria for MDD.

Myth 2 states that depression is more common later in life than at younger ages. Again, as described above, MDD is less common in older adults than in younger or middle aged adults. Twelve-month prevalence rates for MDD in older adults range from 0.3% to 10.2% compared with most recent estimates of 10.4% in younger adults and 7.7 to 9.4% in middle-age adults. Higher incidence rates (i.e., new cases in a given period) of other depressive disorders, such as dysthymia or minor depression or MDD-NO have been reported in older adults and have important clinical implications. It has been argued that the presence of SSD, which is more prevalent in the elderly (10-50% prevalence in older adults compared with 14-15% prevalence in younger and middle-age adults) have a significant impact on functioning and likely warrants clinical intervention. Older adults with clinically significant depressive symptoms as well as sub-threshold symptoms still suffer impairment in physical, social, and role functioning similar to patients with MDD.

Myth 3 presumes that depression is more chronic in later life than at younger ages. As it now appears, prevalence and incidence rates do not differ based on age. That said, older adults who had MDD when younger do have a greater likelihood of reoccurrence later in life and less likelihood of full remission. The authors note that more recent longitudinal studies indicate that depression in older adulthood is associated with a worse tra-

jectory, probably moderated by symptom severity, number of previous episodes/age of first onset, medical comorbidities, and an external locus of control. Frequent relapse and recurrence are associated with increased mortality. Unfortunately there are data that suggest that older adults with depression persist in that state. In one study older adults with MDD had a substantially worse prognosis than their younger counterparts. A 2-year observational study showed that the course of MDD worsened linearly with age, such that individuals aged 70 years and older had the worst outcomes. These findings persisted after the researchers adjusted for clinical, social, and health factors. It might then be worthwhile to treat depression more intensively (Schaakxs, et al., 2018).

Myth 4 holds that depression in late life is more difficult to treat than depression at younger ages. Not true. Several sub-group analyses confirm that the effectiveness of CBT, life review therapy, and problem-solving therapy (PST), but not for non-directive or supportive therapies. For example, a large number of older and younger highly depressed veterans received 12-16 sessions of CBT, using a treatment protocol developed for veterans and military service members with depression (Karlin, Trockel, Brown, et al., 2015). Both younger and older adults experienced significant equivalent reductions in depressive symptoms, and the magnitude of this effect was identical for both groups. Using a similar analysis, Karlin and colleagues (2013) demonstrated equivalent effectiveness of the acceptance and commitment therapy (ACT) for depression in a sample of older and younger adults.

Myth 5 poses that depression in late life is more often caused by psychosocial factors. Although older adults, like younger adults, can become depressed as a result of an interaction between stressful life events and psychological vulnerability, in general older adults tend to shift focus toward the positive. This shift in subjective well-being likely results in spared or enhanced emotional regulation. Newer thinking points toward the increased importance of biological factors in the development of depressive symptoms over the role of psychosocial variables. Three major etiological hypotheses have been proposed. They are: 1) the vascular hypothesis (which implicates the involvement of WMH); 2) the inflammatory hypothesis (which suggests that depression is associated with age-related increases in inflammation); and 3) the degenerative hypothesis (which suggests that depression is a prodromal marker of cognitive impairment and dementia). Additionally, links between depression in several age-related pathological processes, such as cardiovascular, neuroanatomical, endocrine, and immune systems strongly refute the belief that depression in older adulthood is primarily due to psychosocial stressors.

Perhaps the message here is that older adults actively generate their world. They enact reality through the actions and lenses they form. Of course, all events are perceived through the developmental lens of age, cohort and most centrally the person's lived experience. The older adult makes a commitment to living that, when perturbed, is measured, albeit crudely, by core variables of the person (measured by the domains). With depression the person is pulled down. Having a network of social support, an optimistic attitude, better self-control, and a sense of purpose in life, matters. But it is often absent in the person with depression. When asked to "un-depress" their situation, older adults note that depression is hidden or at least contaminated with the lament of living and physical maladies. Content analyses (on depression at late life) suggest that older adults often reluctantly want to converse. They address four themes; revealing hidden depression, reducing "blind spots," having an opportunity to talk, and moving on from sadness or confusion (Overend, 2015). We might surmise that older adults want to connect, to be relevant, to unburden themselves in the changes of life. They seek to un-trap themselves and grab onto life in ways at which research can only guess.

Stress, Depression, and Timing

Pre-clinical models of acute stress, neuroimaging, and postmortem investigations have provided invaluable information on the neurobiology of stress on the brain. This relates directly to depression. Stress results in neuronal atrophy and volume loss within the prefrontal cortex, decreased neuronal and glial proliferation, and decreased expression of brain-derived neurotrophic factor (BDNF). Stress and depression-related disrupted connectivity between cortical and limbic regions is associated with aberrant feedback loops, endocrine abnormalities, and increased inflammatory markers. Other brain regions, such as the amygdala and the cingulate cortex become overactive.

These neurobiological changes are associated with the duration and severity of depression. Age-related normative changes in neuronal loss and synaptic inefficiency may further increase the susceptibility to depression at later life. Finally, white matter lesions, often markers of stress, are associated with depressive symptoms in older adults. However, it is not clear whether different symptom dimensions of depression have distinct associations with white matter lesions. Recall the Kirton et al. (2014) study where higher CES-D total scores were associated with greater white matter lesion volume and with faster rate of volume increases over time in women, especially at older ages. Higher depressed mood and somatic

symptoms sub-scores were associated with the greatest increases in white matter lesions.

The older age range appears to have a high incidence of depression risk factors. Findings are mixed, but provide support for the “on-time, off-time” hypothesis of variable exposures to depression risk factors across the lifespan (Schaakxs et al., 2017). Risk factors such as chronic disease and increased BMI (body mass index), which occur most frequently at older ages, had the strongest association with depression in younger ages. In contrast, other depression risk factors, such as reduced social functioning and recent negative life events, remain constant throughout the lifespan. Pertinent to late-life depression, other depression-specific variables (for example number of depressive episodes) also influence the relationship between psychosocial stressors and depression. Recurrent depressive episodes throughout the lifespan may be associated with a “kindling” model, which argues that the effects of psychosocial precipitants or stressors will diminish with progressive depressive episodes. In other words, stressful life events are strongly associated with initial depressive episodes, but this relationship attenuates with repeat episodes. At a certain threshold, depression recurrences take on a life of its own.

That said, social isolation may still be a factor in recurrent depression. This is one of many stressors that seems to have a special impact on people at later life. Social interactions are an important human experience that can decline with age. A decrease in social activation and connectedness can be a product of depression and a result of depression (Cornwell & Waite, 2009). Any improvement of expectations (real or otherwise) in regard to social engagement is a healthy endeavor for older adults. We will see more on this in other chapters.

Comorbidities

As implied, Depression is perhaps the most common comorbid condition in all of medicine. It is the most common comorbidity in psychiatry. With such high comorbidity rates, depression is more common in the presence of another psychiatric condition than it is in the general population. Again, this suggests that the underlying pathology causing the other psychiatric condition may be putting people at risk for depression, so depression likely is a nonspecific response to another (brain) pathology.

The Most Common Comorbidity in Psychiatry: Major Depression*

Bipolar I disorder	90%
Generalized anxiety disorder	67%
Panic disorder	50%
Schizophrenia	50%
Alcohol use disorder	40%+
Drug use disorder	40%+
Post-traumatic stress disorder	37%
Borderline personality disorder	30%+
●bsessive-compulsive disorder	30%
ADHD	25%

* Strakowski, et al 2013.

Similarly, depression is common in other types of medical conditions. It appears that any disease that affects the brain increases the likelihood of depression. Furthermore, any medication that crosses the blood-brain barrier appears to increase the risk for major depression. That is a risk that often is not considered by prescribers. If blood pressure medication is causing depression, that risk to the patient's health may be higher than, say, mild hypertension. In AD, multiple sclerosis, and migraine, the rates of depression tend to be as high as 50%. In stroke, it ranges as low as 30% depending upon where the stroke occurred. But the rates of depression are very high following a prefrontal stroke. Even cardiovascular disease, renal disease, cancer and diabetes, which aren't thought to directly affect the brain, all have increased rates of depression. The mechanism here may be through inflammatory processes or other nonspecific general responses to illness. But again, this supports the notion that depression can often be a nonspecific brain response to another underlying medical pathology.

Depression Comorbidity in Medicine*

Alzheimer's disease	50%
Multiple sclerosis	50%
Migraine	47%
Parkinson's disease	40%
●CPD	40%
Epilepsy	35%
Cardiovascular disease	35%
Diabetes	33%
Stroke	30%+
Chronic renal disease	30%
Cancer	30%

* Strakowski, et al 2013.

In one example of this intertwining of depression and disease, Choi and colleagues (2014) shed light on the relationship between depression and cardiovascular disease by examining data from the National Health and Age Trend Study of over 5,000 patients over 65 years at two waves. They found both cross-sectional associations and longitudinal reciprocal relationships between late-life depression and cardiovascular symptoms. They concluded that the care of older adults with cardiovascular disease and/or depression must include interventions focusing on lifestyle and psychological needs that can reduce risks to both of these disorders. As such, depression prevention and treatment also must be an integral part of cardiovascular disease management.

There is likely a genetic depressive condition that is recurrent and treatment resistant that has not been well defined yet. As genetic typing, biomarker development, and other technologies progress, this is likely to change. So it is something of a surprise that MDD is one of our least heritable psychiatric conditions. It has a heritability risk of somewhere between 28% and 44% (Fernandez-Pujals, et al. 2015). Most of the major psychiatric illnesses we diagnose are very heritable. For example, bipolar spectrum disorders have a heritability estimate of between 60%-80% (Kerner, 2014). Schizophrenia has a similar heritability estimate of about 79% (Hilker et al, 2017). This risk of heritability for MDD increases in people who have very well-characterized recurrent MDD, suggesting that depression is more susceptible to environmental factors than other major psychiatric disorders. Finally, in families with primary depressive syndromes, PTSD, and personality disorders, there are higher rates of depression - often higher than rates of the primary genetic condition.

Pain clearly has a powerful negative impact on mood. First, at least 25-50% of older adults complain of pain, and the pain is often chronic in nature. The factors most significantly linked to increased risk of chronic wide-spread pain are non-restorative sleep, anxiety, memory impairment, and poor quality of life (McBeth, et al., 2014). This study suggested that changes in the body chemistry, anatomy, and function associated with initial injury or disease process have re-designed the brain's circuits so that pain signals can no longer be trusted. The person no longer is, therefore, acting normally. Clinical studies have revealed that up to 85% of patients with chronic pain are affected by severe depression (Bair, Robinson, Katon & Kroenke, 2003). In addition, patients with pain are found to have higher levels of stress-generated cortisol, smaller hippocampal volumes, and greater activity in brain regions associated anticipatory anxiety and associative learning. Additional research has found evidence of impaired neuroplasticity in chronic pain patients. Serotonin also is decreased,

impacting both pain perception and mood. Further, chronic pain and depression, together, further aggravate the severity of both disorders. Problems expand to cause trouble with exercise, sleep, socialization, and ability to engage in pleasurable activities, resulting in a poorer prognosis. Pain, then, may be considered a key marker of depression.

A medication evaluation is vital when assessing depressive symptoms in patients with medical comorbidities. A number of commonly prescribed medications are linked to depression. Indeed, a recently published cross-sectional US population study in JAMA (Qato, Zenberger & Olsson, 2018) calculated an overall prevalence of 37.2% of US adults using medications are prescribed a medication with depression as a potential adverse effect. Furthermore, the adjusted percentage of adults with concurrent depression was higher among those using more of these depressogenic medications at the same time (e.g, estimated 15% for ≥ 3 medications). Below is a list of commonly prescribed medications that can cause depressive symptoms.

Medication class	Use	Common Drugs	Alternatives
Beta Blockers	Hypertension, angina, migraines, tremors, irregular heart-beat, and, in eye-drop form, certain kinds of glaucoma.	Atenolol Carvedilol Metoprolol Propranolol Sotalol Timolol	Calcium channel blockers Benzothiazepine
Corticosteroids	Inflammation, rheumatoid arthritis, lupus, Sjögren's syndrome and gout.	Cortisone Methylprednisolone Prednisone Triamcinolone.	Acetaminophen Aspirin Tramadol
Benzodiazepines	Anxiety, insomnia, and to relax muscles.	Alprazolam Diazepam Estazolam Flurazepam Lorazepam Temazepam Triazolam	Psychological treatments such as ACT, CBT, CBT-Insomnia and HRV bio-feedback

Parkinson's drugs	Parkinson's disease, Restless Legs Syndrome	Levodopa/ Carbidopa Pramipexole Ropinirole	If using levodopa, dose can be reduced with addition a COMT-inhibitor
Hormone replacement drugs	Post-menopausal symptoms, especially hot flashes	Premarin Estrogen patch Progesterone	SSRI for hot flashes
Stimulants	Excessive daytime sleepiness 2 nd to hypersomnia, narcolepsy, or sleep apnea. Also, ADHD, chronic fatigue	Methylphenidate Dexedrine Modafinil	Confirm cause of sleepiness and treat (e.g., CPAP for sleep apnea) Eliminate drugs with sedating effects
Anticonvulsants	Seizures, neuropathic pain, bipolar disorder, migraines	Carbamazepine Gabapentin Lamotrigine Pregabalin Topiramate	Phenytoin (seizures) Venlafaxine (neuropathic pain)
Proton Pump Inhibitors/H2 drugs	GERD/acid reflux	●omeprazole Lansoprazole Pantoprazole Nizatidine Famotidine Cimetidine Ranitidine	Non-calcium-carbonate-based antacid, avoid offending foods, sleep with head above belly
Statins and other lipid-lowering drugs	Cholesterol reduction	Atorvastatin Lovastatin Pravastatin Simvastatin Fibrates Ezetimibe Colesevelam	A combination of vitamin B12, vitamin B6, folic acid and fish oil to lower homocysteine in the body (homocysteine increases blood lipids), exercise
Anticholinergic drugs	Irritable bowel syndrome	Dicyclomine	A calcium-free antacid

Vascular Interference

Vascular risk factors are very much a part of depression at later life. Vascular Depression (VaD) is characterized by feelings of sadness, hopelessness, irritability, and other symptoms that last two or more weeks. These patients also experience cognitive deficits and have a poor response to antidepressant drugs. VaD is caused by decreased cerebral blood flow that results in lower oxygen supply and damage to the brain's white matter connections in the frontal cortex and basal ganglia. EF is especially poor in these cases. Risk factors include HTN, mini-strokes or TIAs, smoking, obesity, family history of cardiac problems, high LDL cholesterol, atherosclerosis, chronic stress, and diabetes. MRI studies show extensive WMH, subcortical microvascular lesions, lacunes, and microinfarcts. In short, vascular risk factors are pervasive in later life depression. Post-mortem studies of elderly persons who have died from suicide reveal lacunes as well as small vessel and Alzheimer-related pathologies (Alexopoulos, 2017).

Reasonable speculation is that recurrent and persistent depression in early or mid-life may lead to cerebrovascular compromise. In turn, cerebrovascular compromise may lead to more frequent depressive episodes with poor treatment response and outcomes. Interrupting the vicious cycle of depression and vascular disease is a critical clinical and public health task. We note again that it remains unclear whether treatment of depression can reduce the risk of cerebrovascular pathology and its complications.

Hybels, Pieper, Payne, and Steffens (2016) found that vascular lesions, as seen through brain imaging, are associated with both depression and functional impairment in older adults. They studied 381 patients, ages 60 years and older, and followed them up 16 years later. They measured WMH volume through brain MRI data. Participants who were both depressed and had higher volume of WMH at baseline were the most at risk for functional decline across all measures of function. Conclusions were that older adults with both cerebrovascular risk factors and depression are at an increased risk of functional decline, and may benefit from the treatment for both conditions.

It is not clear whether different symptom dimensions of depression have distinct associations with WMH. Higher depression scores (CES-D) were associated with greater white matter lesion volume and with faster rate of volume increases over time in women, especially at older ages. Higher depressed mood and somatic symptoms sub-scores were associated with the greatest increases in WMH and somatic symptoms sub-scale

scores were associated with the greater increases in WMH volume over time in older ages. In men, depressed mood and somatic symptoms were associated with WMH volumes at baseline.

Depressed individuals have a 45% higher risk for stroke than non-depressed individuals and 25% higher risk for stroke-related mortality. Depressive symptoms and stress lead to the greatest elevation of the risk for vascular disease mainly in low income populations. Physiologic changes occurring during depressive episodes may increase both risk for strokes and directly compromise cerebrovasculature.

A word about the pharmacogenomic age. It is upon us. It is improving, but at present may not be ready for prime time. Genomind's Genecept Assay, GenSight, RightMed, CNSDose and others are seeking over-the-counter privileges. Now it requires a physician's order. More than 750,000 people have availed themselves of GeneSight. Roughly 50% of patients with depression respond to their first antidepressant and maybe ~30% will not respond to any drug. In fact, the efficacy of psychiatric medication treatment is mostly suppressive. Psychiatric medications only seem to stabilize the person in the short run. Antidepressants may actually help by blunting the rise of cortisol or helping with inflammation or the HPA system. Now genotyping is on board for predicting adverse effects, helping the patient take more ownership of the treatment process, and improving treatment after a few failures.

Best evidence seems to center on medications that address the CPY450 gene family (Abbasi, 2018). At present marketing is ahead of the data. Most of the extant support is from smaller samples, and poorer validation. In fact, the FDA has highlighted the need for caution by physicians and consumers because most genetic tests that make claims about the effects of a specific medicine are not supported by enough scientific information or clinical evidence (FDA Safety Communication, Nov 2018, updated April 2019).

Late Life Depression, Cognition, SMI, MCI, and Dementia

In some ways this is an extension of the previous section. Cognitive changes, neuropsychological changes, and depression are a messy mix. Cognitive aging is characterized by gradual changes in cognitive functioning associated with normal aging. These changes can be variable between patients, are not secondary to a neurodegenerative illness or typically accompanied by functional decline, and generally accelerate in later life. Given the relatively high prevalence of depression in older adults and the growing focus on modifiable risk factors of dementia, there is

strong interest in understanding better the complex relationship between depression and cognitive impairment. In their review, Greene and Wang (2018), highlighted neurological and psychological changes that occur in depression at later life, and emphasized the importance of gathering the full complement of interview, medical, and neuropsychological data on these patients, not only to understand the wide variety and complexity of symptom presentations in this condition, but also because there are reversible causes of cognitive impairment, such as drug-drug interactions, drug side effects, substance misuse, and metabolic derangement. Longitudinal monitoring of the patient to track symptom progression is equally important to understanding the full clinical picture: Does the patient exhibit primarily a cognitive disorder, a mood disorder, or both? Which symptoms remit, stabilize, or progress over time in the context of aggressive treatment of mood symptoms?

As suggested above, imaging studies have found that late-life depression is associated with higher ischemic burden on structural MRI, as well as impaired executive and memory functions (Makin, Nelson, Delucchi, et al., 2014). Furthermore, baseline impairments in encoding and EF in patients with depression seem to be risk factors for the progression to dementia. However, this progression seems to be highly variable. For example, Steffens and colleagues (2009) found that, in a two-year follow-up of cognition in a large group of non-demented depressed older adults, approximately 25% of the group reverted back to normal cognitive function, 15% progressed to dementia, and the remainder continued to display cognitive impairments without functional decline.

A range of patient characteristics is associated with worse neuropsychological performance in late-life depression, including older age, later age of onset of depression, greater severity of depression, more medical comorbidities, and presence of pre-existing cognitive problems (Reynolds, et al., 2001). Also, there can be neuropsychological roots for late-life depression, characterized by structural and functional brain abnormalities in middle age. These include reduced frontal and hippocampal volumes, WMH, vascular changes, and problems with blood oxygen uptake as seen on fMRI and SPECT. PET studies also have documented functional differences in metabolic activity between individuals with late-life depression and comparison subjects. Finally, the role of neurotransmitter deficits, including serotonin, dopamine, norepinephrine, and the cholinergic system have been implicated in late life depression (Tadayonnejad et al., 2014). Interestingly, the value of neurotransmitters may be more effective in patients in a higher SES as it seems to precipitate a positive interaction between the environment and

the struggling depressed older person.

A word should be said about SMI or Subjective Cognitive Decline (SCD), a condition in which the patient self-reports memory problems but shows unimpaired performances on neuropsychological testing. SCD was once brushed off by clinicians as part of normal aging, anxiety (“the worried-well”), or depression. Indeed, SCD is often difficult to distinguish clinically from normal cognitive aging, which is characterized by gradual changes in cognitive functions associated with the aging brain. Normal cognitive aging features can vary in older adults, but they are not secondary to a neurodegenerative disease, nor are they accompanied by troublesome functional decline. Typical changes include difficulty with free recall (e.g., names), some executive dysfunction (e.g., multi-tasking), and slowed processing speed.

SCD, however, is increasingly viewed as representing the first symptomatic manifestations of dementia – that is, pre-MCI (Jessen, et al., 2014). In longitudinal studies, SCD has been associated with higher rates of incident cognitive impairment and dementia (Waldorff, et al., 2012). But here, too, depression complicates the picture. For example, multiple studies have shown that SCD is common in individuals with late-life depression, ranging from 50-60% of individuals (Bartley et al. 2012). In a recent study by Chu and colleagues (2017), 55% of older adult participants with histories of major depression but no current depressive symptoms complained of SCD. It is noteworthy that this same group was significantly more likely to be medically compromised by 4 or more diseases (34%) as compared to 12% of subjects with depression histories but no SCD, and 16% of healthy controls. It is also worth noting that the subjects with depression histories and SCD were significantly more likely to be taking cardiovascular medications.

Archer et al. (2015) describe how SCD can result from mood disorders/personality factors or systemic illness. These individuals will differ, however, in terms of complaints, personal characteristics, and help seeking behavior. Findings by Yates and colleagues (2015) also reveal that subjective memory complaints are more likely to be related to mood problems than objective cognitive impairment. Qualitative research by Burke et al. (2015) developed 8 complaint themes espoused by individuals in the earliest stages of AD and individuals experiencing normal cognitive aging. They included issues of changing self-identity, the causal attribution of cognitive decline, anxiety related to perceived decline, the negative perceptions attached to a diagnosis of dementia, changing levels of insight, and perception of well-being in aging. Miebach et al. (2018)

also assessed cognitive complaint profiles in memory clinic patients and depressed patients. Healthy controls were also involved. Twelve complaint themes were applied with a qualitative expert rating. Seven complaint themes were endorsed more often by the cognitive complaint group; complaint themes in the depression group aligned with depression symptoms and separated them from the other two groups.

Complaint Themes

Absence of Contextualization: "I do not know"*

Increasing frequency: "Happens all the time now"^^

Situational: "Especially if I am in the supermarket"

Attentional fluctuation: "I am not concentrating on where I put my glasses"

Affective influence of memory: "I was stressed"

Progression: "It is happening more and more"* ^^

Impact on affect: "I get frustrated with myself"*^^

Predomination: "I waste time on looking for stuff"* ^^

Burdensome coping: "I just have to leave things alone" ^^

Dependency: "I ask my wife"^^

●Over-endorsed complaints: "It's like all the time"

Dismissive attitude: It's age"^^

Distractible speech: Not answering questions well ^^

* MDD patients endorse this theme more than controls.

^^ Cognitive complaint patients endorse this them more than controls.

MCI problems are of course a step beyond SCD. ●One prospective study showed that tau-mediated degeneration, but not A β -deposition was significantly higher in patients with MCI compared to SCD (Wolfsgruber, et al., 2017). MCI suggests that cerebrovascular risk factors affect white matter tracts in the frontal striatal pathways resulting in executive dysfunction and potentially the affective symptoms of depression. Another epidemiological study highlighted that baseline depressive symptoms independently predicted a subsequent diagnosis of MCI 6 years later (Barnes et al., 2006). ●Of interest, SSRI treatment has been associated with slower progression from MCI to AD in individuals with a history of depression, according to results from the prospective Alzheimer's Disease Neuroimaging Initiative (ADNI). SSRIs appear to reduce amyloid plaque burden and cognitive impairment in animal models, but evidence from clinical studies is less conclusive. Bartels, Schneider and colleagues (2017) used data from 755 currently nondepressed ADNI participants to evaluate the impact of SSRI treatment on cerebrospinal fluid (CSF) biomarkers and

progression from MCI to AD. A baseline diagnosis of MCI was associated with a 2.6-fold higher likelihood of a history of depression, and a baseline diagnosis of AD was associated with a 3.77-fold higher likelihood, compared with the cognitively normal group. However, a history of depression was not associated with an elevated rate of conversion from MCI to AD.

Dementia, of course, occurs with noxious frequency. Epidemiological findings indicate that late-life depression is a risk factor for dementia (Steenland et al, 2012). Longitudinal data from the Women's Health and Aging Studies similarly show that baseline depressive symptoms predicted subsequent cognitive decline (Rosenberg, et al., 2010). Other studies have reported either that remote history or number of past depressive episodes appears to increase the likelihood of later-developing dementia (Green, et al. 2003). By contrast, some epidemiological studies have not found an association between depression in late life and subsequent development of dementia (Becker, et al. 2009). Data from recent epidemiologic studies suggest that late-life depression is associated with approximately 50% increased likelihood of developing dementia in general, including both AD and vascular dementia. These new findings clearly suggest, then, that late-life depression is a risk factor for dementia (Mirza et al., 2016).

As a whole, cognitive deficits in depressed older adults appear to have significant clinical consequences and have been associated with increased rates of relapse, disability, and a poorer response to antidepressant medication. Recent studies suggest therapeutic approaches to late-life depression may partially ameliorate the cognitive deficits associated with this disorder, but much of the impairment persists after treatment and that late-life depression is likely a risk factor for dementia itself. The persistent cognitive deficits in older depressed patients have led some researchers to suggest that late-life depression is a syndrome that is distinct from depression in younger patients, especially in those with depression onset after 60yo. Others have argued that cognitive impairments observed in many depressed patients are simply exacerbated by increasing age.

Biomarkers

To date, there are no biological markers for depression. That said, there are of course many brain changes in late life depression: WMH, inflammation, GABA, Glutamate, BDNF, IL-6, cardiovascular disease, platelet activation, T-cell response, and elevated homocysteine levels. Currently, APOE-4, beta-amyloid, and tau are dementia biomarkers that have received the most interest. For example, there seems to be a significant relationship between depressive symptoms and APOE-4 in

regard to the progression from MCI to dementia. Specifically, longitudinal studies show that APOE-4 carriers with depression were 4.4 times more likely to progress to AD compared to the non-APOE-4 carriers with depression (Irie, et al., 2008). In addition, recent research indicates that individuals with late life depression display a reduction in neurotrophins, including nerve growth factor, glial-derived neurotrophic factor, and brain-derived neurotrophic factor. As discussed, there also is clear evidence suggesting that the HPA system and inflammation play a role in late-life depression and comorbid cognitive decline, perhaps by way of chronic stress (Köhler et al., 2010).

Neuroimaging is supportive of the above findings. Recent fMRI studies suggest that structural lesions associated with late-life depression also can lead to a disruption of neural networks associated with the clinical features of late-life depression (Tadayonnejad et al., 2014). Research with amyloid and neurofibrillary tangle binding agent (FDDNP) in individuals with MCI and depression found variable temporal and parietal lobe binding. This finding was further confirmed by research coming from the Alzheimer's Disease Neuroimaging Initiative (ADNI) showing that life-long depressive symptoms reliably predict A- β accumulation in patients with MCI (Chung, et al., 2015). Even the EEG seems to be a marker, revealing that individuals with late-life depression demonstrate more slow wave activity and prolonged P300 latencies than their nondepressed peers, indicating hypoactive cerebral arousal and slower information processing (Kindermann et al., 1998).

In time, depression will eventually be tethered to brain networks. Several teams across the US found that people whose depression improved the most had greater brain network connectivity between the anterior insular cortex (a region involved in identifying the importance of events) and the middle temporal gyrus (which plays a role in the subjective experience of emotion). Observing how people's brains perform when they are doing complex tasks also may shed some light on how they will respond to therapy. The proposed mechanisms underlying depression (noted above) may be the core biological components of this heterogeneous, nonspecific response to a variety of underlying brain pathologies.

Information on these and depression are just unfolding. Dunlop, et al. (2012) found that specific patterns of activity on brain scans may help clinicians identify whether psychotherapy or antidepressant medication is more likely to help individual patients recover from depression. The study, called PRedICT, randomly assigned patients to 12 weeks of treatment with one of two antidepressant medications or with cognitive behavioral therapy (CBT). At the start of the study, patients underwent a fMRI, which

was then analyzed to see whether the outcome from CBT or from medication depends on the state of the brain prior to starting treatment. The scan showed that the degree of functional connectivity among the subcallosal cingulate cortex (an important emotion processing center) and three other areas of the brain was associated with treatment outcome. Specifically, patients with positive connectivity between the brain regions were significantly more likely to achieve remission with CBT, whereas patients with negative or absent connectivity were more likely to improve with antidepressant medication. These findings strongly suggest that all depressive syndromes are not equal and, like different types of cancer, different types of depression require individually tailored treatments.

In sum, diagnosing (and treating) depression in people with MCI or dementia presents special challenges as we have noted. Researchers are still trying to tease out the relationship between depression and dementia. Depression does not appear to cause dementia; it is a likely risk factor, just as dementia is a risk factor for depression. Often depression comes first. In fact, depression in early life is a risk factor for dementia while depression in late life can be a prodrome of dementia itself.

What we know about the brain and depression is that depression is probably an expression of some underlying neurodegenerative process, probably related to biological changes such as inflammation, vascular disease, and high stress hormone levels. Life-long depressive symptoms reliably predict A- β accumulation in patients with MCI. As noted SSRI treatment has been associated with slower progression from MCI to AD in individuals with a history of depression (Weiner et al., 2015). Little is known about whether drugs or other interventions developed to treat AD have any effect on depression. Therefore, depression in the context of an older person with some cognitive decline might be considered a sentinel event. Increasing awareness of this should be a concern.

Apathy and Depression in Dementia

Apathy and depression vie for type and level of problems with older adults, especially older adults with dementia. Apathy is far from a new construct. It is the most common psychiatric symptom in dementia. Growing evidence of the distinct pathophysiology and differences in appropriate pharmacologic and psychosocial interventions has led to the conclusion that apathy and depression have divergent natural histories and that apathy is a separate and distinct syndrome from depression (Tagarello, Giradi, & Amore, 2009). In fact, apathy is generally associated with more impairment in ADLs than cognitive status would otherwise dictate. It is also associated with more cognitive and functional decline, a longer illness duration, and an increased dementia severity (van Dalen et al., 2018).

Zhu, Grossman, & Sano (2019) assessed the relationship among the constructs of apathy and depression, along with function. This was a cross-sectional study of AD patients with Clinical Dementia Ratings between .5 and 2. Over 7000 subjects were evaluated, with an average age of mid 70's. Findings showed that apathy, and not depression, was significantly associated with worse function, with the strongest connection in mild dementia. While apathy was found to occur early in the disease, it also was common across the AD spectrum. This was determined from both the clinician's and informant's perspectives. The prevalence of apathy was greater in those with more severe dementia. Independent of apathy, results showed that presence of depression was not associated with function in any dementia severity group.

This makes an assessment of depression with some consideration of apathy a problem. Clearly, the need to assess apathy and depression separately is important in patients with dementia. The independent effects on function will help identify patients who may benefit from more targeted management strategies.

Complete Assessment

The measures suggested here to assess depression in older adults are generally applicable to younger adults, too, but these have less of the noise than other relevant measures in regard to physical/somatic symptoms and just normal aging. It is difficult to say whether any one scale will be the sure way to identify depression; no one scale is perfect and no one of the scales discussed here can tell clinicians that depression is leading the charge or caused by the wake of another problem. Clinicians can choose from many depression measures and they must exercise discretion when interpreting results due to the very issues discussed in this chapter.

Accordingly, we have provided a grouping of measures. The omnibus symptom inventories are especially relevant because they place the patient in the context of many psychopathologies and coping strategies. The MBMD is particularly useful as it provides information on psychopathology as well as personality, stress moderators, and treatment prognostics. The MBMD also includes a subjective validity test (SVT). In addition, clinician rating scales allow for a structured review diagnostic criteria for depression. The self-report scales allow for subjective ratings or "numbers." The clinician can then monitor severity of the numbers and use classification ratios for probability markers. Note that the complete assessment is a simple extension of the screen.

Of note, depression in the context of a dementia is a special problem. Three scales are suggested below. Finally, because depression impacts all

areas of the person, a quality of life measure is important.

Assessment for Depression

- **Omnibus Measure:** MBMD¹, PAI², MMPI-2-RF³, MINI⁴
- **Clinician Ratings:**
 - HAM-D⁵ (17 or 24, use 10)
 - MADRS⁶
- **Self Report scales:**
 - PHQ-9⁷ (10)
 - BDI-II⁸ (10)
 - GDS⁹ (11) or GDS-SF (5)
 - CESD¹⁰ (20 item, cut-off 6)
 - Zung Depression Self Rating Scale¹¹ (50)
- **Dementia and Depression:**
 - Cornell Scale for Depression in Dementia¹² (19 items use to get severity after depression is established)
 - Dementia Mood Assessment Scale¹³ (17 items)
 - Provisional Depression in Dementia¹⁴
- **Overall distress:**
 - GQ-45¹⁵
 - BASIS-32¹⁶
- **Apathy:**
 - Starkstein Apathy Scale (SAS)¹⁷

¹ Millon Behavioral Medicine Diagnostic (Millon et al., 2001)

² Personality Assessment Inventory (Morey, 1997)

³ Minnesota Multiphasic Personality Inventory-2-Restructured Form (Ben-Porath & Tellegen, 2008)

⁴ Mini-International Neuropsychiatric Interview (Sheehan et al. 1998)

⁵ Hamilton Depression Rating Scale (Hamilton, 1967)

⁶ Montgomery-Asberg Depression Rating Scale (Williams & Kobak, 2008)

⁷ Patient Health Questionnaire-9 item (Kroenke et al., 2001)

⁸ Beck Depression Inventory-II (Beck et al., 1998)

⁹ Geriatric Depression Scale (Yesavage et al. 1983)

¹⁰ Center for Epidemiological Studies-Depression (Radloff, 1977)

¹¹ Zung Depression Self Rating Scale (Zung, 1965)

¹² Cornell Scale for Depression in Dementia (Alexopoulos et al, 1988)

¹³ Dementia Mood Assessment Scale (Sunderland & Minichiello, 1996).

¹⁴ Provisional diagnostic criteria for depression of Alzheimer disease (Celin et al., 2002)

¹⁵ Outcome Questionnaire-45 (Lambert & Finch, 1999)

¹⁶ Behavior and symptom identification scale-32 (Jerrell, 2005)

- Assess medical and psychiatric comorbidities: Charlson Index¹⁸
- Assess social support, cognition, sensory, sleep, pain, meds

Treatment

Overall, we can say that MDD and other depressive syndromes respond to a number of treatments. One in six American adults is taking a psychiatric drug. The majority of these are antidepressants. Indications are that antidepressant use increases with age, with 25% of adults over 65 years taking these drugs. The SSRIs are relatively specific, but in any given patient, we don't know whether the patient will respond to an SSRI, SNRI, bupropion, the older tricyclics, or another treatment option, such as psychotherapy. The SSRIs and SNRIs have been effective at levels comparable to the psychotherapies. Surprisingly, the most effective antidepressant treatment we have, electroconvulsive therapy (ECT), is among the least specific treatments we have. It suggests, again, that different types of depression are, perhaps, responding to different types of interventions. Unfortunately, at this point, we are not good at identifying which intervention is the most likely to be effective for a given depression presentation. This offers yet more evidence to suggest that depression is a heterogeneous and often nonspecific construct within the setting of other neuropathology. It is noteworthy that SSRIs and SNRIs have been found to improve episodic memory and EF on occasion (Rosenblatt, et al. 2016). Furthermore, Donepezil, an anticholinergic drug prescribed for memory impairment, also has shown some positive results regarding depression in patients with cognitive decline (Pelton, et al., 2008).

Traditionally, anti-depression treatment is viewed as altering low levels of the neurochemicals, GABA, serotonin, epinephrine and dopamine. Presumably this leads to an increase in BDNF (brain derived neurotrophic factor) and an improved HPA system. Presumably, too, the brain structures (hippocampus, amygdala, and frontal lobes especially) alter as does the function of the interconnected neural networks. Antidepressants are beneficial for older adults with depression but the effects are modest. In general, remission is seen in about one third of patients; relapse is, however, robust. Cohen's d values are often less than .3 (.5 at least is considered to be a moderate effect size). In fact, it seems that antidepressants

¹⁷ Starkstein Apathy Scale (Pedersen et al., 2012)

¹⁸ Charlson Comorbidity Index (Charlson et al., 1987)

have the best effect on patients with more severe symptoms (see Cassels, 2010).

All that said, antidepressant medications are a first line intervention. Research over the past 20 years has provided robust evidence for the efficacy of antidepressant medication in the acute phase of late-life major depression and, once the person is euthymic, for their use in the maintenance phase. Unfortunately, they are to behavioral medicine what antibiotics are to physical medicine when it comes to overuse. More is not better. Additionally, SSRIs and SNRIs are provided first, but up to 50% – 66% of patients, depending upon its definition, are treatment resistant. Often “oppositional tolerance” and “antidepressant-induced tardive dysphoria” can develop: In other words, the medication can result in further problems. In a general way, depression is far too complex for a pill to fix, regardless how novel any future drugs might be. Much of the improvement with antidepressants may be attributed to a placebo effect. In a meta-analysis of acute depression phase, parallel group, double blinded, placebo controlled studies with random assignment, for 2nd generation antidepressants not associated with a medical disorder, and being 60 or older, ten unique trials with 13 contrasts (N=2377 active drug and 1788 placebo) were included (Nelson & Papakostas, 2009). Results showed: response rates for Drug = 44.4% vs. response rate for Placebo=34.7%. In most placebo-control studies, placebo rates vary between 19% - 47%.

In general, roughly 40% of elders will either not respond or respond poorly to medication. Among those who do respond, roughly 50% will show a relapse within 2 years. Additionally, roughly 25% experience spontaneous remission. For the remainder, several characteristics predict difficulty in achieving remission with therapy. These include: a greater number of concurrent physical conditions; more severe depressive symptomatology; higher anxiety; inadequate prior response to antidepressant treatment; dissatisfaction with social support; suicidal ideation; a history of recurrent episodes; and the use of concomitant psychotropics, such as sleep aids or anxiety agents. Sundermann, Katz, and Lipton (2016) even highlighted sex differences. Following 572 women and 345 men with a mean age just above age 77yo for 4.2 years, on average 90 women and 65 men developed amnesic MCI during follow-up. Results indicated that mild depressive symptoms in men and moderate-severe symptoms in women represented a marker for treatment resistance or future cognitive impairment.

We do note this: Pizzagalli, et al (2018) showed that a patient's response to an antidepressant medication can be assessed by looking at the activation level of the rostral anterior cingulate cortex (ACC) region of the

brain by an electroencephalogram (EEG). This study seems to demonstrate the 'incremental predictive validity' of a neural marker, that is, the fact that activity in this brain region predicts the likelihood of treatment response above and beyond the contribution of a range of low-cost and easily administered clinical and demographic characteristics previously shown to predict treatment outcome. More than 300 patients were tested at four sites in the United States -- using sertraline for the treatment group. They showed that the rostral ACC marker predicted clinical response eight weeks later, even when statistically controlling for demographics and clinical variables previously linked to treatment response. For those with the marker of good response, a clinician could tell patients that they have a high chance of benefitting from the intervention, and they should stay engaged in treatment. Or, for patients with the marker of low response, clinicians could decide to start with more aggressive treatment at the outset, such as a combination of pharmacotherapy and psychotherapy, and monitor these patients more closely.

Nonsurgical brain stimulation, also known as somatic psychiatric therapy, is a viable alternative or add-on treatment for MDD in adults. Investigators reviewed 113 clinical trials that randomized over 6700 patients (average age 48 years, approximately half women) with MDD or bipolar disorder in an array of nonsurgical stimulations or sham therapy, focusing on response (efficacy) and all-cause discontinuation (acceptability). Stimulations included ECT, several types of transcranial magnetic stimulation (TMS), theta burst stimulation, magnetic seizure therapy, and transcranial direct current stimulation (tDCS). Researchers found that several types of ECT as well as high-frequency left repetitive TMS (rTMS) and tDCS were more effective than sham therapy in improving depressive symptoms (Mutz et al., 2019). ECT and transcranial magnetic stimulation also have especially been shown to have positive effects on depression (Greene and Wang, 2018). ECT, the oldest and most effective treatment for treatment resistant late life depression has remission rates of approximately 60% (Lisanby, 2007). Repeated Transcranial Magnetic Stimulation (rTMS), a fairly recent addition to the somatic psychiatric treatments, involves the delivery of magnetic pulses to the cortex. There is good evidence to support the use of rTMS for depression in working age adults, but efficacy research for older adults is lacking. What studies have been done on older adults suggest that rTMS can be helpful, but older adults are likely to require longer treatments and higher frequency of pulses for a positive effect (Sabesan et al. 2015). Nonsurgical brain stimulation treatments then should then be considered as alternative or add-on for severe depression in patients who have not

responded to drug treatment (Mutz et al., 2019).

Psychotherapies of course are effective. We have already reviewed the treatment efficacy studies on depression (Hyer, 2014). In brief, the efficacy for psychotherapies and medications are roughly equal. Twenty-seven randomized trials of psychotherapy for late-life depression show that late-life depression can be treated with this intervention (Mackin & Areal, 2005). Effect sizes range from .35 to .75 in these studies (Cuijpers et al., 2017). The psychotherapies favorably reviewed include CBT, PST, brief psychodynamic therapy, ACT, and supportive therapy. Bibliotherapy also has been reviewed as helpful. In addition, there are several preventative programs that we have identified in other chapters that are effective. It should be noted that Leichsenring and Steinert (2017) indicated that CBT should not be considered the gold standard as the CBT studies are flawed, effect sizes are lower than desired, and/or the comparison sample is wait list or placebo (the so-called “intent to fail”) as opposed to using another psychotherapy intervention as the comparator.

Smart phone “apps” as therapy adjunct for depression and anxiety

There are seemingly countless “apps” for various behavioral health maladies, including anxiety, depression, PTSD, OCD, insomnia, and various addictions such as tobacco cessation. Many are not evidence-based, some are scams. Deciding which ones to recommend to your patients can be difficult without trying them out yourself to determine which are credible.

The nonprofit website, *PsyberGuide.org*, reviews behavioral health apps and digital tools. The purpose of each app is described and rated for credibility, user experience and how transparent the app is about the ways in which it stores and uses patient data. The search function enables users to narrow down apps based on platform, cost, target audience, condition, or treatment type.

PsyberGuide is supported by Northwestern University and the University of California at Irvine. It works in partnership with mental-health organizations such as the National Alliance on Mental Illness and the International OCD Foundation.

Research findings suggest that older and younger patients do have some differences when it comes to treatment effectiveness, but also many similarities. Despite the ideas of Knight and Pachana (2015), treatment with older adults is somewhat different from that provided younger people

in each domain. We believe that at least in two areas differences are considerable; extended health needs and life adjustment problems. These problems are broad and intertwined. Basic medical and environmental needs must be addressed or the person will falter. Secondary problems actually become less of an issue when the key targets are addressed. Success depends on the conviction by the patient that the therapist cares and is competent. Quality of the patient's and family's participation is a significant determinate of outcome. If the patient buys into the treatment plan, the psychotherapist is competent, some change occurs, the patient feels an empathic reaction, and s/he is monitored, improvement stands a good chance of occurring. As we have suggested, treatment fails because an intervention is made too precipitously and quickly – this is the core tenet of the Watch and Wait Model.

It is reasonable to speculate further that the combined use of CBT, IPT, and PST, along with the judicious application of medication, may provide the best chance of change. Typical CBT protocols seem to do this as they include education about anxiety, self-monitoring, relaxation training, exposure to anxiety-provoking thoughts and situations using systematic desensitization, and cognitive restructuring. Some protocols also included problem solving skills training, behavioral activation, sleep hygiene, reflective listening, life review, and memory aids.

In general however, the effect is more modest than that for younger adults. Modifications such as shortening sessions, presenting material at a slower rate, providing extra psycho-education, and including caregivers and family members in the treatment plan may be necessary with older adults. Group CBT can improve outcome for those who have a limited social support network. Although some studies suggest there are long-term effects of pharmacological treatments, an incomplete response is common. For patients who do not respond completely to SSRIs, augmenting treatment with CBT may improve treatment response and reduce worry symptoms. Furthermore, those who wish to taper off medications, providing CBT with medication management may improve outcomes over medication taper alone.

In 2019, The American Psychological Association (APA Policy Feb. 16, 2019 (APA, 2019)) provided empirical suggestions on older adults on depression. They saw MDD at later life as prevalent but to a lesser extent than subthreshold depression which was seen as 2–3 times more common than MDD in older adults (Meeks, Vahia, Lavretsky, Kulkarni, & Jeste, 2011). As we noted too, cognitive decline, age-associated neurobiological changes, stressful events, and sleep disturbance are clear risk factors for late-life depression. Additionally, the review board noted that the clinical

presentation of depression in older adults is distinctive and differs from early periods in the life cycle by following a relapsing and recurrent course. Depression in older adults was seen as often treatment resistant (Ng & Schweitzer, 2002). Partial response, marked by the presence of residual symptoms, is associated both with continuing disability, caregiver burden, and elevated risk for early relapse and recurrence.

As we have reviewed, the panel noted further that depression in older adults typically co-occurs with often chronic medical disorders, amplifying disability (Lin et al., 2003). Pain was especially highlighted. Lin and colleagues (2003) found that enhancing care for depression was related to lower levels of pain as well as higher quality of life and functional status among a diverse sample of older adults with comorbid depression and arthritis. No surprise, depression either coexists with or foreshadows the development of cognitive impairment and dementia. And as we have reviewed, this group cited a meta-analysis by Diniz and colleagues (2013) finding a link between depression in older adults and risk of dementia.

The table below provides a summary of treatment recommendations. The panel's recommendations and suggestions for CBT, problem-solving therapy (PST), and life-review therapy rather than no treatment or treatment as usual (TAU) is consistent with other reviews of older adult depression treatment research (Cuijpers, Karyotaki, Pot, et al., 2014). The panel endorses similar therapies to the ones we endorse. There is a place for pharmacotherapy (especially combined pharmacotherapy). The panel also reviews subthreshold/minor depression, conditionally suggesting CBT, life-review treatment, and problem-solving therapy. Note that for persistent depressive disorder, the panel conditionally suggests PST (individual). For Relapse Prevention, the panel recommends combination IPT and pharmacotherapy or combination supportive care and pharmacotherapy.

Initial Treatment: MDD

1. Group life review over TAU
2. Group CBT over TAU
3. Combined Pharmacotherapy plus IPT over IPT alone
(nortriptyline used and now recommended against)

Mildly Valid

4. CBT over No Rx, TAU, desipramine
5. CBT + Non-specific support with Pharmacotherapy over Pharmacotherapy
6. IPT + Pharmacotherapy over supportive care, IPT + supportive care

<p>7. PST over reminiscence therapy Insufficient Evidence PST and Phone Support</p>
<p>Initial Treatment: Subthreshold or Minor Depression</p> <ol style="list-style-type: none"> 1. CBT-Internet 2. CBT-Individual 3. CBT + TAU 4. Life Review over education control 5. PST 6. Paroxetine (This SSRI has anticholinergic side effects) Insufficient Evidence <ol style="list-style-type: none"> 1. Behavioral Bibliotherapy vs TAU 2. Life Review vs TAU
<p>Initial Treatment: MDD or Minor Depression + Cognitive impairment/Dementia</p> <ol style="list-style-type: none"> 1. PST 2. Pleasant Events Behavioral Therapy Insufficient Evidence Behavioral Activation Therapy TAU
<p>Initial Treatment: Persistent Depressive Disorder</p> <ol style="list-style-type: none"> 1. PST 2. Paroxetine
<p>Initial Treatment: MDD with Medical or Other Complications</p> <ol style="list-style-type: none"> 1. CBT + Support for T2D or COPD 2. Multicomponent intervention 3. Coping group
<p>Prevention of Recurrence – MDD</p> <ol style="list-style-type: none"> 1. IPT + Pharmacotherapy 2. Supportive Therapy + Pharmacotherapy

We now address a few of these problem conditions and make several suggestions.

Treating Complexity

Conditions more common in older adults can complicate the diagnosis and treatment of depression in older adults. Chronic conditions, cognitive impairments, and medications warrant considerations when conceptualizing and treating patients. As discussed above, the impact of comorbid medical conditions on depression is striking; rates of depression (MDD) in the

community tend to range from 3%-5%. This is in sharp contrast to the 10%-14% found in inpatient medical settings (Feldman et al., 1987; Katon, 2003). Interestingly enough, the type of medical disorder has relatively little impact on rates of depression; social problems and previous history of depression were more impactful. Patten (2001; 2018) noted that long term medical conditions were warning signs of future depression and that more chronic conditions related to greater risk of developing depression in the future. It is worthwhile for clinicians to probe more for depression in patients who have chronic medical conditions or who use medical services more than the average patient. Prophylaxis treatment for depression in patients who have one or more of these risk factors is recommended. Mindfulness Based Cognitive Therapy (MBCT) with a focus on health enhancing activities such as proper diet, yoga, and medication adherence has demonstrated effectiveness at preventing depression relapse (Segal, et al., 2012). At the least a careful watch for the complexity of the older adult should be in effect when treating with psychotherapy.

Above we suggested that heritability for depression is projected to be ~30%. That leaves considerable room for individual input. As implied, depression is the gift that keeps on giving. It is the leading influence on obesity, diabetes, HTN, and vascular diseases in general, WMH, and inflammatory problems. It is very much a vascular disease and influences its behavioral sequelae, inactivity, cognitive decline (especially executive dysfunction), and poor health/morbidity. An integrated care package incorporating first line psychological and pharmacological treatments, along with evidence-based lifestyle changes, have a more robust effect on depression. In fact, newer data on diabetes programs and other health problems pave the way for therapeutic interventions that address common behavioral health disorders simultaneously recognizing common risk determinants and shared end points.

Treating Emotional Processing Deficits

Treatment of emotional processing is a special issue. The ability to effectively deploy emotion regulation strategies relies on modulation of bottom-up affective processes by top-down (emotional- focused therapy) or cognitive control regulatory networks. Gross (2007) defines emotion regulation as an attempt one makes to influence which types of emotions they experience, when they experience them, and how emotions are expressed. Successful emotion regulation relies on a well-functioning neural network that includes the cognitive control network (CCN) and default mode network (DMN), which include brain structures that tend to

deteriorate in the aging process. Deterioration in relevant neural networks and structures, resulting in MCI or mild dementia, reduces the ability to regulate emotions in the face of stressors. A real-time fMRI neurofeedback study demonstrated that positive induction-based emotion regulation training increased connectivity between the amygdala and other brain regions, particularly the frontal gyrus, as well as frontal regions involved in affect regulation (Zotev et al., 2018). Psychotherapies that promote emotion identification and regulation, such as ACT (Petkus & Wetherell, 2013), and mindfulness-based interventions (Segal et al., 2012), IPT (Miller & Reynolds 2007), Problem Adaptive Therapy (PATH: Kiosses et al., 2011) and even heart rate variability biofeedback to address sympathetic nervous system over-arousal (Jester, Rozek & McKelley, 2018) can be effective here. Again, emotions are registered differently in the older cohort and need watching.

This is more reason for a transdiagnostic approach to treatment. We have discussed the value of the transdiagnostic model where core strategies are applied to foster adaptive emotion, especially mindful emotional awareness, cognitive flexibility, countering emotion-driven behaviors, tolerance of physical sensations, and imaginal emotional exposures. In several newer programs, like LEAF, WELLCUS, and PATH, the emphasis is on on-the-ground coaching (see Sirey et al., 2016). While focusing on more impaired populations, individually tailored skills are employed to counter the negative reality of life and build on strengths. This is the building of skills for increasing positive effect. Typically these include the usual suspects; positive events, gratitude, behavioral activation, mindfulness, positive reappraisal, attainable goals, altruistic behaviors, and a value-based emphasis of life change. Skills are taught to lay counselors and made handy for community application. Importantly, in one of these models (Open Door), the counselor collaborates with the patient to identify and address both attitudinal and structural barriers to seeking treatment as these are prepotent in the perceived need of the patient. Not addressing these issues results in non-commitment or non-compliance.

Accommodating Cognition

Empirically supported, evidence-based psychotherapy, such as CBTs that teach emotion regulation skills (e.g., cognitive reappraisal), may be beneficial to amnesic MCI patients. Goodkind et al. (2015) found that CBT for older adults with depression was especially effective for reducing depressive symptoms among those with poor cognitive flexibility, one aspect of EF, at baseline. These findings suggest that CBT may help older

adults with cognitive decrements to better engage the neural resources necessary for effective emotional regulation. In that same study, participants also performed a cognitive flexibility task using fMRI before the CBT trial. Higher patterns of activation were noted and lower activation in the left frontal inferior triangle and right superior frontal gyrus prior to CBT were associated with better response. Since fMRI was performed prior to CBT only, it is unknown whether the CBT changed activation patterns in these brain regions and the response to cognitive flexibility; this would be an interesting investigation. It seems clear that the prefrontal regions are crucial in modulating emotional responses to stressors and that it is possible to modify decrements in these skills with cognitive-behavioral interventions.

The treatment of patients with frank EF problems is a special problem. We have addressed this before because of its importance in depression. Pimontel et al. (2016) conducted a meta-analysis of executive dysfunction and antidepressant treatment response in late-life depression. A Medline search was conducted to identify regimented treatment trials contrasting EF between the older adult responders and non-responders. Measures of EF were extracted from eight studies and six domains were identified. These included cognitive flexibility, planning and organization, response inhibition, selective attention, verbal fluency, and the Dementia Rating Scale Initiation/Perseveration composite score. Results showed that the effect of planning and organization was significantly different from zero, whereas cognitive flexibility, response inhibition, and the others were not. Therefore, the domain of planning and organization is meaningfully associated with poor antidepressant treatment response in late-life depression. These findings suggest that therapies that focus on planning and organization may provide effective augmentation strategies for antidepressant non-responders with late-life depression.

Stroke is of course a special case. Increased mortality after post-stroke depression in both short-term and long-term follow-up studies has been reported in the stroke literature since at least the 1990s. Robinson, Long, and Jorge (2017) reexamined the patients from a one-year randomized control study in a double-blind of escitalopram, problem-solving therapy, or placebo to prevent depression in patients less than 3 months post-stroke. The objective of that study was to determine if preventive treatment would predict time to death 8-10 years in follow-up. Earlier research showed that stopping escitalopram by 18 months follow-up, but not problem-solving therapy, led to a significant increase in depression. At a mean of 8 years after stroke, that study found that problem-solving therapy had statistically significant delay in mortality after stroke compared with escitalopram and

placebo groups. This is the first time that evidence has been found to suggest that psychotherapy for depression is effective in delaying mortality after stroke.

Whatever the organic insult, the clinician needs to be wary of overt and occult effects and modify treatment accordingly. Watch and Wait is especially relevant here. In Chapter 7 we discuss the value of CT (cognitive training). Even here, the devil is in the details: Computer-based CT results improved cognition with MCI patients, but more so with those with no or less WMH (Djabelkhir-Jemmi et al., 2018). What this requires is to be thoughtful and careful when assessing depression, to look for potential associations with other conditions. As implied, this obviously reaches well into the medical (health domain) arena: Do we really need to add a statin or antihypertensive or other drug that crosses the blood-brain barrier and increases the risk for depression? Clearly, the need to orchestrate care when a disorder, such as depression, has so many connectors, is important.

Treatment through Exercise

As we argue in every chapter: Exercise Rules! There is no better intervention in the thoughtful attempt at change than exercise. Hallgren, et al. (2016) designed a study to quantify and compare the long-term effectiveness of prescribed exercise and clinician-supported CBT with usual care for patients with mild to moderate depression. Participants included 945 Swedish adults (mean age, 43 years; 73% women) recruited from primary care clinics located throughout Sweden. They were randomly allocated to 12 weeks of group exercise (three 60-min sessions per week), clinician-supported CBT, or usual care. CBT involved having the patients work through online modules while being monitored by a supervising psychologist. The usual-care group received standard treatment for depression at the discretion of their primary care provider. Most patients in the usual-care group received regular face-to-face counseling. Of the 945 patents, 740 (78%) returned the 3-month questionnaire, and 797 (84%) returned the 12-month questionnaire. Patients in all three groups experienced a significant reduction in depression severity from baseline to 3 months and from baseline to 12 months. The researchers noted that exercise and CBT (internet) are at least equally effective long-term treatments for mild to moderate depression compared with usual care.

It is important to highlight that holistic or “non-traditional” approaches to depression treatment may be more palatable to older adults than psychopharmacological or psychotherapeutic approaches. Non-traditional

treatments, such as exercise, art therapy, meditation, and are used by many for non-behavioral health reasons. Older adults may be more open to their use, especially if they have never had psychiatric treatment. This especially applies to physical activity. A systematic review by Mura and Carta (2013) showed that most studies found a reduction in depressive symptoms with no other interventions than increasing activity. Multiple studies have shown that exercise alone is equally effective, if not a more powerful addition to antidepressant medications (Blumenthal, Babyak, & Moore, 1999). We will see this again in other chapters.

Treatment through Diet

Nutritional psychiatry is on the rise. Randomized controlled trials are now showing significant efficacy for the treatment of clinical depression with dietary and nutritional interventions. Even people with severe depression can learn and benefit from dietary interventions, dispelling the notion that only people with mild depression can benefit (Jacka et al., 2017). Parletta and colleagues (2017) examined the relationship between more severe depression and diet in a randomized controlled trial that included 3 arms; one group received a traditional Mediterranean diet (n = 75), one group received a traditional Mediterranean diet supplemented with fish oil, and the third group received a control intervention. The researchers recruited 152 adults aged 18 to 65 years (105 women and 47 men), 38% of whom reported a diagnosis of depression, and 36% reported taking an antidepressant. The Mediterranean diet intervention included an initial nutritional consultation, biweekly group cooking workshops, easy, inexpensive recipes, and take-home food hampers for making the recipes. The control intervention involved biweekly meetings that included social activities such as board games, book clubs, and photo sharing. Participants were provided with snacks, such as biscuits, dips, cheese, crackers, coffee, tea, juice, or water. Participants were assessed at baseline, 3 months, and 6 months. Primary behavioral health outcomes were assessed with the Depression, Anxiety and Stress Scale and the Assessment of Quality of Life.

The investigators found that the Mediterranean diet was significantly associated with lower anxiety, negative affect, better coping and overall quality of life. They also found associations between food groups and behavioral health outcomes. For example, eating more vegetables was associated with less stress and more positive emotions, and eating more fruit was associated with less anxiety and more positive emotions and relationships. Eating a wide range of vegetables and fruits also improved

behavioral health outcomes. Importantly, eating a traditional Mediterranean diet decreased the amount of take-out or “fast” food and unhealthy snacks the participants ate. Indeed, a 2019 meta-analysis of 16 RCTs that targeted depression and anxiety symptoms with dietary interventions (Firth et al., 2019), which found a positive impact of a healthful diet on depressive and anxiety symptoms (especially among women), also found that, though specific dietary interventions varied among studies, a common denominator was the reduction/elimination of processed or “junk” foods and replacing them with high-fiber, nutrient dense alternatives such as vegetables.

There are many ways to introduce healthful eating to patients using the Mediterranean diet as a model, including learning and teaching basic nutrition, using simple food assessments, prescribing healthful foods (i.e., shopping the perimeter of the grocery store where all fresh foods are displayed) and avoiding processed or “manufactured” foods. Another option is to partner with registered dietitians, nutritional health coaches, or local chefs. The challenge for medicine and psychiatry/psychology is to get clinicians to start thinking about the value of nutritional interventions. The psychologist/psychiatrist must not be hesitant to develop competence in nutrition as it relates to behavioral health, chronic disease, and obesity. This knowledge needs to be in the clinician’s wheelhouse as most patients do not have access to a nutritionist and their PCPs are often ill informed about nutrition (see Chapter 5 for more on this topic).

Nutritional psychiatry has banked on the Mediterranean diet or DASH diet. These can even result in longer telomeres over time (Leung, Fung, McEvoy, Lin, & Epel, 2018). There appear to be clear advantages to a good diet. In the past decade the advantages of “superfoods,” like berries, fish, eggs, fortified whole grain cereal, and even chocolate, are noted (see *Mind Mood & Memory*, 2008). Increasingly, “brain-boosting” herbs and spices, like garlic, rosemary, clove, sage, turmeric, and cinnamon, are touted (See *Mind, Mood & Memory*, 2010). For some practitioners the application of health foods is more important than medications or psychotherapies.

In sum, the standard fare for depression involves interventions for reduced levels of serotonin, increased norepinephrine, fatty acid imbalance, inflammation, dysregulation of the HPA system, and a focus on neurodegeneration. Now nutrition can have a role in treating these deficits. Below is a listing of usual input for nutrition for depression (Ruiz, 2019).

Nutrition for Depression

- Anti-inflammation diet
- Dietary source of folate and B vitamins
- Mild stimulants (caffeine, green tea)
- Serotonergic input (tryptophan from eggs, meat, dairy)
- Curcumin
- mega 3 oils
- Avoid alcohol, other substances
- ... and Sunlight and Exercise

Treatment of Bereavement

Bereavement is a special situation. Unfortunately its designation in the DSM-5 has made it a psychiatric disorder, probably beyond its merit. Theorists and researchers currently embrace a range of empirically validated frameworks to account for the variegated trajectories of bereavement, but two theories have emerged as the most demonstrable in empirical promise. First, the *dual-process* model of bereavement posits that grieving a loved one entails oscillating between orientation to the loss (i.e., continuing bonds with the deceased by expressing emotion related to the death and reconnecting with meaningful memories) and restoration of contact with a changed world (i.e., reengaging relationships and experimenting with new life roles). Second, the *meaning reconstruction* model of grief views grieving as a process of reaffirming or reforming a world of meaning that has been challenged by loss. These two predominant bereavement theories are complementary. Both the dual-process model and the meaning reconstruction model view grief as a life-long process of renegotiating continuing bonds with the deceased and formulating meaning into one's life after the loss. Most empirically informed grief therapies aim to provide avenues for patients to explore continued bonds with the deceased, the personalized meaning behind their loss, and a reconstruction of purpose in life without their loved one. In short, bereavement theory has evolved away from the ubiquitous stage models (e.g., Kübler-Ross's 5 stages of grief) toward a more contextualized processing of varied responses to loss.

Additionally, the constructs of grief and bereavement have vied with one another for having depression in the background. This includes prolonged grief response and persistent complex bereavement disorder (DSM-5 appendix), for example. These are of course beyond the normal grief response. Regardless, the constructs seem to agree that grief or bereavement is different than depression. It is a yearning, like an addiction.

Depression does not show activity in the brain's reward centers; grief does. There is a reward that people feel that is related to wanting to remain in contact with someone that you love. It does generally not improve with an antidepressant. Reasonably agreed-to treatment constructs include psychoeducation (understanding grief), managing painful emotions, thinking about the future, strengthening relationships, telling the story of death, learning to live with reminders and remembering the person who died.

The treatment of bereavement is beyond the bounds of this book. It has many tentacles. Fortunately, it is grounded in loss and inner pain familiar to many treatment models. It is modal at late life, the season of loss. Considering treatment of anxiety or depression with CBT, intervention modules should consider combating demoralization, teaching coping skills, and problem-solving techniques, shifting to the soft view of an active, resourceful, and competent person, encouraging behavioral activation. Similarly they should focus on learning to alter associations between thoughts, feelings, and behaviors that do not promote avoidance, learning relaxation skills and facilitating maintenance and generalization of skills. Will to Live (WTL) also is apt and typically declines with age. It is also a construct that touches on grief both from a phenomenological and a construct level. It is inner turmoil that is universal and ineluctable. Carmel, Tovel, Raveis, & O'Rourke (2018) showed that older adults with a strong WTL continue to maintain their satisfaction with life. Weakening of the WTL is a marker of depression or depressive symptoms, as well as an indicator of poor response to bereavement. Health seems to be the key mediator; healthy people have a stronger WTL.

One very helpful model that incorporates the above discussed problems of grief is ACT (Acceptance and Commitment Therapy), as proposed by Hayes (2018). It is a straightforward therapy that requires an acceptance of the loss, the feeling and changes in life. It also advocates for a larger vision for the psychological experience of loss and a connection/commitment to one's new or altered life.

Grief Psychotherapy

Acknowledge the loss: There is a wound that needs healing.

Remember positive events

Embrace feelings of the loss: Do not push away the loss or avoid.

Embrace the feelings of loss.

Expand scope of vision: Expect positive emotions.

Good feelings do belong in this process.

Watch out for unhelpful thoughts:

Accept feelings as part of the reaction to loss. Be open to feelings.

Connect with what matters: Pain suggests that you are very human and still alive. Feelings are power!

Take committed action: Act on your values.

Hayes (2018)

Applying the Watch and Wait Model to Depression

In general, treatment for older adults is always a challenge. When patients present with both significant emotional and cognitive complaints, clinicians should aggressively treat the depressive symptoms first and then reassess cognitive symptoms after some resolution of the severe emotional distress. Also, clinicians should always approach complex cases by considering potential reversible causes of the patient's mood and cognitive symptoms. One of the most overlooked, but easily reversible causes of mood and cognitive impairment in older adults is medication side effects. Substance abuse/misuse also is another one. Cerebrovascular risk factors clearly are often involved, too. As we have highlighted in this chapter, the underlying cause of depression is often unknown, so the clinician must use due diligence to figure out likely effective targets for treatment. If the patient has a major stress component, for example, simply providing antidepressant medication without relieving the stress is likely to be ineffective. If there is an underlying primary psychiatric condition, such as bipolar disorder, OCD, or alcohol abuse, failing to recognize and treat the primary condition is going to make it much more difficult to manage the depression. In fact, managing the underlying condition may be what's necessary and may be *all* that's necessary to manage depression. Regardless, there are many roads to Rome and likely more than one is necessary for remission.

Below is an outline of what we have been discussing. Assessment leads the parade. The Watch and Wait process then unfolds with validation/alliance, psychoeducation, module-interventions, lifestyles, and, if needed, more formal empirically supported interventions (e.g., CBT). The outline below is familiar but is targeted for depression.

Treatment Approach

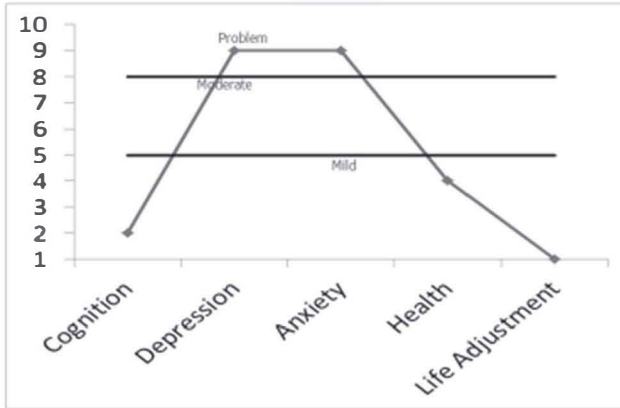
- Assess with screeners and cognitive measures
- Watch and Wait Initial Phase
- Psychoeducation
- Simple Modules
 - Behavioral activation/relaxation
 - Assess dietary quality and refer to Nutrition if available

- Introduce Meds or Psychotherapy (CBT, IPT, ACT, PST)
- Spouse/Family/Kids/Friends/Enemies
- If problems with compliance: Motivational Interviewing
- Lifestyles
- Formal Watch and Wait Treatment
- Use Mini-modules
- CBT, PST, ACT, IPT
- Use case manager for home help/phone/email/text
- Monitor
- Follow-up: Assume slow recovery and keep family involved
-
- **Response WILL be slower than in younger patients**

We should add here that clinicians must always think in terms of prevention. This is a marker of importance. Karp and colleagues (Karp, Dew, Wahed, Fitzgerald, Bolon, et al., 2018) argue for an individualized approach. The prevention of depression and anxiety disorders involve adapting to the patient's chronic and changing course, delivering appropriate treatment when needed most, reacting to non-compliance or side effects, reducing treatment burden, delivering only what is necessary, delivering early treatments with a positive and durable downstream prevention effect and sifting through available treatment options. This leads to a more personalized preventative care approach. Multi-disciplinary teams with the kind expertise noted in this chapter are usually needed to effectively treat. Watch and Wait always endorses this. Assessment always considers this.

Case

MR



Depression

Depression			
Measure	Healthy	Moderate Problem	Problematic or Syndromic
Description	The patient is not depressed, mood is expressed in a healthy range and not consistently low.	The patient may have a subclinical depression or dysthymia. Mood is slightly depressed without significant distress or impairment.	The patient is likely experiencing depressed mood, anhedonia, and/or suicidal ideation. Many DSM-5 symptoms will be endorsed, some with great severity. Functioning is impaired
MINI			MDD or Dysthymia
BDI-II			X
PHQ-9			X
GDS-SF		X	

Page * 41

Domains

Depression

MINI: Positive for Depression

BDI = 29

PHQ-9 = 16

GDS-SF = 6

MBMD: Depression

Anxiety

MINI: Positive for anxiety

GAD-7 = 7

BAI = 24

STAI = 43

Cognition

Normal (see below)

Health

Chronic Diseases: 4 (thyroid disease, hyperlipidemia, HTN, A-fib)

Health Rating: 5/10

ESS = 10

Pain: 2/10

Cigarettes: No
 Alcohol: None
 BMI: 27
 Exercise: None, excessive Stationary Behavior
 Life Adjustment
 Lives alone with a significant other
 High SES and good support
 Functions well

HISTORY OF PRESENT ILLNESS: Patient indicated that for the past two years she has been complaining of memory problems, forgetting things that she does during the day, and on occasion losing her keys. She also is unhappy and has been treated for some depressive symptoms with medication. She also indicated that she forgets issues like cooking rituals and recipes.

RELEVANT DEVELOPMENTAL ISSUES: She relates normal developmental milestones. She was graduated from high school and was married shortly thereafter. She was divorced in 2009 from her husband. This was after 50 years of marriage. He has a schizophrenia diagnosis. She has two children, a boy and a girl, and they are very supportive. She has worked over the years, first at a school, then at the Post Office. She was let go on disability in 1991 and receives Workers' Compensation. She lives alone.

RELEVANT MEDICAL PROBLEMS: She is fairly healthy. She does not use tobacco or alcohol. Indications are that she sleeps roughly 6 hours at night, but it is disrupted across the night. She does not have an exercise program, but plans on doing one. She is an active member of a church, attending twice on Sundays and once on Wednesdays. She takes several medications, including Synthroid, Cardizem HCT, a statin, Coumadin, as well as Tylenol. She also takes psychiatric medications including Wellbutrin, Zoloft, and an anxiolytic. She has had 1 psychiatric hospitalization for depression, roughly in 1991. She experiences mild pain.

PREMORBID FUNCTIONING and OVERALL COGNITIVE FUNCTIONING: Her WASI Full Scale Score is 107 (68%): Verbal = 109 (73%) and Performance = 103 (58%). This suggests a normal intelligence and good skills now. Currently Mrs. R had a MoCA of 27/30, normal. Her RBANS was 103 (58%), also normal.

FUNCTION: She scored in the normal range on the FAQ, indicating few problems on this measure of executive function in activities of daily living. She was rated as needing no help with transfers, cooking, shopping, managing meds, housework, doing laundry, driving, or managing finances.

AFFECT: Her MINI indicated that she is currently depressed and has mild anxiety. She receives some help from her current significant other. Her PHQ-9 was 16; her BDI-II was 29, both depressed. Her GDS-SF was 6, mild depression. She has an anxiety rating on the GAD-7, BAI and STAI. She has a complicated personality style: detachment, dejection, denigration and independence. This suggests that therapy will have to be attuned to these long-standing patterns. She has been treated by psychiatrists for over 20 years and has one psychiatric hospitalization. In sum, she has had psychiatric problems off and on over the years.

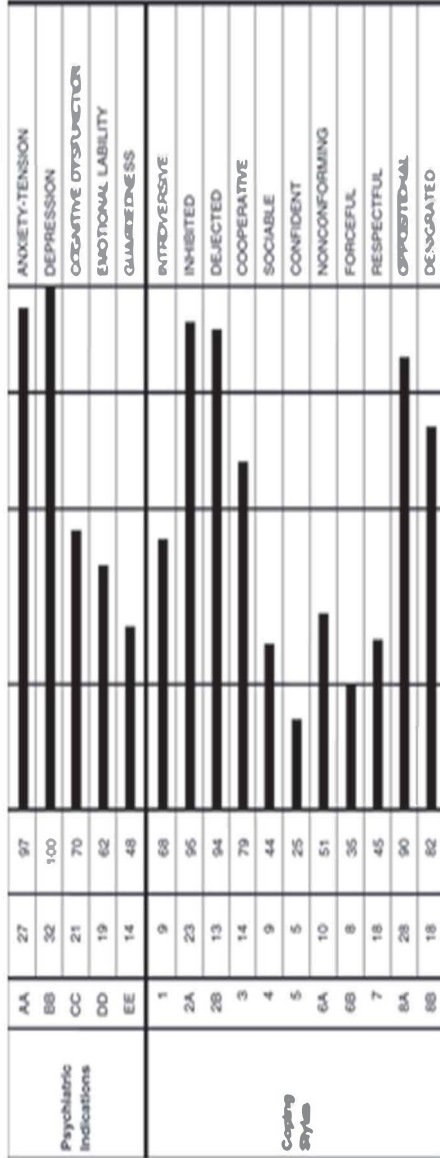
PAIN and SLEEP: She rated her pain at present as 4/10; average 4/10. She has back problems.

WATCH and WAIT: She has depression and anxiety. She is living well and seems to be enjoying her life. Her health is also stable. She is benefiting from on-going psychiatric care (medications). She is reasonably healthy. She does not use tobacco or alcohol. Indications are that she sleeps roughly 6 hours at night, but it is disrupted across the night. She does not have an exercise program. She is an active member of a church. She is also social with a significant other. She takes several medications, including psychiatric medications of Wellbutrin, Zoloft, and an anxiolytic. Her GAF is ~60.

Cognitively she has many strengths. This is in distinction to her complaints. This needs monitoring, however. At base, she is depressed. She has a long psychiatric history. The quality of her depression seems to be influenced by her complex personality where she has strong patterns of depression thinking, detachment, denigration and independence. She does not appear to be reporting a significant degree of thoughts of worthlessness and hopelessness. Her interpersonal style is best characterized as self-effacing and lacking confidence in social interactions. She is likely to have difficulty in having her needs met in personal relationships and instead will subordinate her own interests to those of others in a manner that may seem self-punitive. Her failure to assert herself may result in mistreatment or exploitation by others, although, at this point, it appears that she has been recently effective in maintaining her important relationships. She is likely to be compliant in therapy, however. Sleep and pain are not in play at present.

MBMD Profile

MR



ANXIETY-TENSION

DEPRESSION

COGNITIVE DYSFUNCTION

EMOTIONAL LABILITY

GUARDIANCESS

INTRUDERSIVE

INHIBITED

DEJECTED

COOPERATIVE

SOCIAL

CONFIDENT

NONCONFORMING

FORCEFUL

RESPECTFUL

OPPOSITIONAL

DENIGATED

Psychiatric Indications

Coping Styles

Interventions:

All Watch and Wait markers are in place. She needs help in a few areas but mostly depression, a probable reason for her less adaptive functioning reports. Her medications are managed by a local psychiatrist. This involves only medication, no psychotherapy. We therefore provided psychoeducation, validated her, and suggested other interventions. She will join the CBT group and will be given several behavioral activation strategies. Her social support connectors will be supported as well. She will be requested to increase her exercise. She may benefit from some individual counseling as she seems unsure of herself and has self-doubt. The CBT group will help determine her need for counseling.

Checklist

Discernment: Committed and compliant: X

Core markers

Validate Problem	X
Psychoeducation of Model	X
Assessment	X
Alliance	X
Monitoring	X
Case formulation	X

Problem List

3. **DEPRESSION:** Monitor mood, behavioral activation, counseling for interpersonal issues, CBT group (negative thinking protocol), exercise, ACT modules, interface with psychiatrist

4. **ANXIETY:** Monitor anxiety daily with SUDS, CBT group weekly, teach relaxation, apply worry control, counseling focusing on self-image, coping and assertiveness needs, HRV bio-feedback

Mini-modules: targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks:

Managing situational contingencies:

Role playing and modeling: X

Environmental control:

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:
 Imagery exercise for counter-experience: X
 Compassion training: X
 Communication and Assertiveness training: X
 Behavioral experiments: X
 Emotional exposure:
 Homework: X

Longer Therapy processes:

CBT: Use of group and 3-6 column evidence record
 ACT: use of acceptance interventions
 IPT: Interpersonal role play

Lifestyles:

- Practice happiness
- Practice Spirituality/Religion
- Socialize: Less decline and live longer
- Develop resilience
- Exercise: Even housework
- Diet
- Somatic softening: Meditate and Mindfulness
- Values and Leisure

Follow-up: Re-evaluate every 6 months

Depression

MINI = Mild Depression

BDI = 17

PHQ-9 = 7

GDS-SF = 4

Anxiety

MINI = No Anxiety

GAD-7 = 7

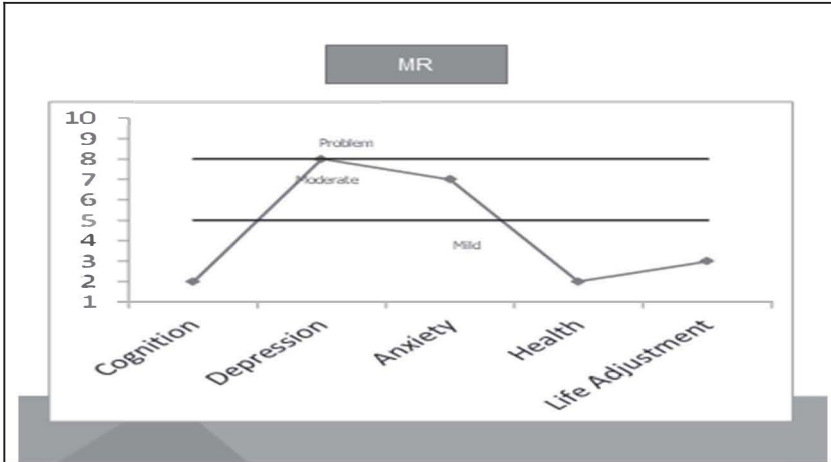
BAI = 4

STAI = 23

MBMD: Depression/Tension

Relapse Issues: In process

Follow-up domain profile suggests that there is little change. She continues to need monitoring in depression and anxiety. She has good support, is medically compliant and has plans for her days.



Conclusion

Depression and its friends are everywhere at later life. It has attachments in the brain, in the body, in relationships, for self-views, and in one's purpose in life. It is the final common pathway of difficult living or poorly adjusted coping. One is never whole with depression. Unfortunately, it meanders in the soul of the lived life and the life now-experienced. It can, however, be recognized and treated.

Depression needs measuring and prioritizing. Late-life depression is clearly a disabling illness associated with significant economic and societal cost. Cognitive dysfunction additionally represents a concurrent and debilitating aspect of this disorder. It brings in other problems. Mild cognitive impairments, especially, have consistently been documented in over half of individuals with late-life depression and these cognitive impairments contribute to processing speed deficits. These are often considered to be hallmark cognitive features in late-life depression. Anxiety and health, too, are usually not far behind the presentation of dysphoria. Given the heterogeneity of impairments exhibited by individuals with depression, differentiating the direct impact of late-life depression from the effects of other concurrent conditions or diseases represents a significant challenge. Watch and wait attempts to provide order and clarity.

References

- Abbasi, J. (2018). Companies tout psychiatric pharmacogenomic testing, but it is ready for a store near you? *JAMA*, October, 320(16), 1627-1629.
- Alexopoulos, G. (2017). Depression and cerebral vascular disease: What is to be done? *American Journal of Geriatric Psychiatry*, 25(2), 129-131.
- Alexopoulos GS, Abrams RC, Young RC, and Shamoian CA (1988). Cornell Scale for Depression in Dementia. *Biol Psychiatry*, 23: 271-284.
- Alexopoulos GS, Kiosses DN, Klimstra S, Kalayam B, Bruce ML (2002). Clinical Presentation of the "Depression-Executive Dysfunction Syndrome" of Late Life. *Am J Geriatric Psychiatry*, 10(1):98-106.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders (5th ed.)*. Arlington, VA: American Psychiatric Publishing.
- American Psychological Association (2019). *Clinical Practice Guideline for the Treatment of Depression Across Three Age Cohorts*. Guideline Development Panel for the Treatment of Depressive Disorders, American Psychological Association, Washington, D.C.
- Archer HA, Newson MA, Coulthard EJ (2015). Subjective memory complaints: Symptoms and outcome in different research settings. *Jl Alzheimers Disease*, 48:S109-S114.
- Balsamo, M, Cataldi, F., Carlucci, L., Padulo, C., & Fairfield, B. (2018). Assessment of late-life depression via self-report measures: A review. *Clinical Interventions in Aging*, 13, 2021-2044.
- Barnes DE, Alexopoulos GS, Lopez OL, Williamson JD, Yaffe K (2006). Depressive symptoms, vascular disease, and mild cognitive impairment: Findings from the cardiovascular health study. *Archives General Psychiatry*, 63(3):273-279.
- Bartels C, Wagner M, Wolfgruber S, Ehrenreich H, & Schneider A. (2017). Impact of SSRI therapy on risk of conversion from mild cognitive impairment to Alzheimer's Dementia in individuals with previous depression. *American Journal of Psychiatry*, 175(3):232-241 <https://doi.org/10.1176/appi.ajp.2017.17040404> .
- Bartley M, Bokde AL, Ewers M, Faluyi YO, Tobin WO, Snow, A, et al. (2012). Subjective memory complaints in community dwelling healthy older people: the influence of brain and psychopathology. *International Jl Geriatric Psychiatry*, 27: 836-43.
- Beck AT, Steer RA, & Brown GK. (1996). *BDI-II*, Beck Depression Inventory Manual. Minneapolis: Pearson Clinical.
- Becker JT, Yue-Fang C, Lopez OL, Dew MA, Sweer RA, et al. (2009).

- Depressed Mood is Not a Risk Factor for Incident Dementia in a Community-Based Cohort. *American Journal Geriatric Psychiatry*, 17(8): 653-663.
- Beekman A, Copeland J, Prince M (1999). Review of community prevalence of depression in later life. *British J Psychiatry*, 174:307-311.
- Ben-Porath YS & Tellegen A. (2008). Minnesota Multiphasic Personality Inventory-2-Restructured Form. (Professional Manual). Minneapolis: University of Minnesota Press.
- Blazer, D (2003). Depression in late life: Review and commentary. *The Journals of Gerontology*, 58(3): 249-265.
- Blumenthal JA, Babyak MA, Moore KA, Craighead WE, Herman S, et al (1999). Effects of Exercise Training on Older Patients with Major Depression. *Archives of Internal Medicine*, 159(19):2349-2356.
- British Medical Journal* (2019). Published online March 27, 2019.
- Buckley, RF, Saling, MM, Frommarm, I, Wolfsgruber, S, Wagner, M (2015). Subjective cognitive decline from a phenomenological perspective: A review of the qualitative literature. *J Alzheimers Disease*, 48(suppl 1):S125-S140.
- Byers AL, Yaffe K, Covinsky KE, Friedman MB, Bruce ML (2010). High occurrence of mood and anxiety disorders among older adults: The National Comorbidity Survey Replication. *Arch General Psychiatry*, 67(5):489-496.
- Byers AL, Nelson C, Yaffe K, & Lai AX (2017). Predictors of Mental Health Services Use Across the Life Course among Racially-Ethnically Diverse Adults. *Am J Geriatric Psychiatry*, 25(11): 1213-1222.
- Cassels, C. (2010). [Medscape.com/viewarticle/714847](https://www.medscape.com/viewarticle/714847).
- Charlson ME, Pompei P, Ales KL, MacKenzie CR (1987). A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Diseases*, 40(5):373-83.
- Choi NG, Kim J, Marti CN, Chen GJ (2014). Late-life depression and cardiovascular disease burden: Examination of reciprocal relationship. *Am J Geriatric Psychiatry*, 22(12):1522-1529.
- Chu CS, Sun IW, Begum A (2017). The association between subjective memory complaint and objective cognitive function in older people with previous major depression. *PLoS One*, 12:e0173027
- Chung JK, Plitman E, Nakajima S, et al (2015). Lifetime history of depression predicts increased amyloid-B accumulation in patients with mild cognitive impairment. *J Alzheimers Disease*, 45:907-919.
- Conwell Y, Van Orden K, Caine ED (2011). Suicide in older adults. *Psychiatric Clinics North America*, 34:451-468.
- Cornwell EY & Waite LJ (2009). Social disconnectedness, perceived

- isolation, and health among older adults. *J Health Social Behavior*, 50(1):31-48.
- Cuijpers P, Cristea IA, Karyotaki E, Reijnders M, Hollon SD (2017). Component studies of psychological treatments of adult depression: A systematic review and meta-analysis, *Psychotherapy Research*, DOI: 10.1080/10503307.2017.1395922
- Cuijpers, P., Karyotaki, E., Pot, A. M., Park, M., & Reynolds, C. F., III. (2014). Managing depression in older age: Psychological interventions. *Maturitas*, 79(2), 160–169.
- Diniz, B. S., Butters, M. A., Albert, S. M., Dew, M. A., & Reynolds, C. F., III. (2013). Late-life depression and risk of vascular dementia and Alzheimer's disease: Systematic review and meta-analysis of community-based cohort studies. *British Journal of Psychiatry*, 202(5), 329–335. doi: 10.1192/bjp.bp.112.118307
- Djabekhir-Jemmi, L, Wu, Y, Boubaya, M, Marlats, F, Lewis, M, et al. (2018). Differential effects of a computerized cognitive stimulation program on older adults with mild cognitive impairment according to the severity of white matter hyperintensities. *Clinical Interventions in Aging*, 13, 1542-1554.
- Dunlop BW, Binder EB, Cubells JF, Goodman MM, Kelley ME, et al. (2012). Predictors of remission in depression to individual and combined treatments (PREdict): study protocol for a randomized controlled trial. *Trials*, 13, 106. <http://doi.org/10.1186/1745-6215-13-106>
- Edelstein B, Heisel MJ, McKee DR, Martin RR, Koven LP, et al. (2009). Development and Psychometric Evaluation of the Reasons for Living—Older Adults Scale: A Suicide Risk Assessment Inventory, *The Gerontologist*, 49(6): 736–745.
- FDA Safety Communications (2018). The FDA Warns Against the use of Many Genetic Tests with Unapproved Claims to Predict Patient Response to Specific Medications. Federal Drug Administration: November 1, 2018, updated April 4, 2019. Accessed April 21, 2019: <https://www.fda.gov/MedicalDevices/Safety/AlertsandNotices/ucm624725.htm#actions>
- Feldman E, Mayou R, Hawton K, Ardem M, Smith EB (1987). Psychiatric disorders in medical in-patients. *QJM*, 63(2): 405-412.
- Fernandez-Pujals A, Adams MJ, Thomson P, McKeachie AG, Blackwood, DH, et al. (2015). Epidemiology and Heritability of Major Depressive Disorder, Stratified by Age of Onset, Sex, and Illness Course in Generation Scotland: Scottish Family Health Study. *PLoS ONE*, 10(11) e0142197

- <http://doi.org/10.1371/journal.pone.0142197>
- Ferrari AJ, Charlson FJ, Norman RE, Patten SB, Freedman G, et al. (2013). Burden of depressive disorder by country, sex, age, and year: findings from the global burden of disease study *PLoS Med*, 10(11), e1001547.
- Firth J, Marx W, Dash S, Carney R, Teasdale SB, et al. (2019). The effects of dietary improvement on symptoms of depression and anxiety: A meta-analysis of randomized controlled trials. *Psychosomatic Medicine*, 81:265-280.
- Fiske A, Riley AA, Widoe RK (2008). Physical Health and Suicide in Late Life: An Evaluative Review, *Clinical Gerontologist*, 31:4, 31-50.
- Fiske A, Wetherell JL, Gatz M (2009). Depression in older adults. *Annual Review Clinical Psychology*, 5:363-389.
- Gerteis J, Izrael D, Deitz D, LeRoy L, Ricciardi R, Miller T, Basu J (2014). Multiple Chronic Conditions Chartbook. AHRQ Publications No. Q14-0038. Rockville, MD: Agency for Healthcare Research and Quality.
- Goodkind M, Gallagher-Thompson D, Thompson L, Kesler S, Anker L, et al. (2015). The impact of executive function on response to cognitive behavioral therapy in late-life depression. *International J Geriatric Psychiatry*, 31(4):334-339.
- Gonzales MM, Insel PS, Nelson C, Tosun D, Mattsson N, et al. (2017). Cortical atrophy is associated with accelerated cognitive decline in mild cognitive impairment with subsyndromal depression. *Am J Geriatric Psychiatry* 25, 980-991.
- Green RC, Cupples LA, Kurz A, Auerbach S, Go R, et al. (2003). Depression as a risk factor for Alzheimer disease: The MDRAGE study. *Archives of Neurology*, 60(5), 753-759.
- Greene RD, & Wang S (2018). Neurologic Changes and Depression. *Psychiatric Clinics of North America*, 41(1):111-126.
- Gross JJ (2007). *Handbook of emotion regulation*. NY: Guilford Press; 2007.
- Hallgren M, Helgadóttir B, Herring MP, Zeebari Z, Lindefors N, et al. (2016). Exercise and internet-based cognitive-behavioural therapy for depression: Multicentre randomised controlled trial with 12-month follow-up. *The British Journal of Psychiatry*, 209(5): 414-420.
- Hamilton M (1967). Development of a rating scale for primary depressive illness. *British Journal of Social and Clinical Psychology*, 6: 278-96
- Hayes, S. (2018). Reaction to grief. *Psychology Today*, July/August, 72-78.
- Heisel MJ & Flett GL (2006). The development and initial validation of the geriatric suicide ideation scale (GSIS). *American Journal of Geriatric Psychiatry*, 14:742-751.
- Heisel MJ, & Flett GL (2008). Psychological resilience to suicide ideation

- among older adults. *Clinical Gerontologist*, 31:51–70.
- Heisel MJ, & Flett GL (2016). Investigating the psychometric properties of the geriatric suicide ideation scale (GSIS) among community-residing older adults. *Aging and Mental Health*, 20:208–221.
- Hilker R, Helenius D, Fagerlund B, Skytthe A, Christensen K, et al. (2017). Heritability of schizophrenia and schizophrenia spectrum based on the nationwide Danish Twin Register. *Biological Psychiatry*; DOI: 10.1016/j.biopsych.2017.08.017
- Hybels CF, Blazer DG, Pieper CF, Landerman LR, Steffens DC (2009). Profiles of depressive symptoms in older adults diagnosed with major depression: A latent cluster analysis. *Am J Geriatric Psychiatry*, 17(5):387–396.
- Hybels CF, Pieper CF, Payne ME, Steffens, DC (2016). Late-life depression modifies the association between cerebral white matter hyperintensities and functional decline among older adults. *The American Journal of Geriatric Psychiatry*, 24(1):42–49.
- Hyer L (2014). *Psychological Treatment of Older Adults*, NY: Springer Publishers.
- Irie F, Masaki KH, Petrovitch H, Abbott RD, Ross GW, et al. (2008). Apolipoprotein E ε4 allele genotype and the effect of depressive symptoms on the risk of dementia in men: The Honolulu-Asia Aging Study. *Archives General Psychiatry*, 65(8): 906–912.
- Jacka FN, Neil A, Pie R, Itsiopoulos C, Cotton S, et al. (2017). A randomised controlled trial of dietary improvement for adults with major depression (the ‘SMILES’ trial). *BMC Medicine*, 15:23 1-13 <https://doi.org/10.1186/s12916-017-0791-y>
- Jefferson AL, Powers DV, Pope M. (2001). Beck depression inventory-II (BDI-II) and the geriatric depression scale (GDS) in older women. *Clinical Gerontologist*, 22(3-4):3-12.
- Jerrell, JM (2005). Behavior and symptom identification scale 32. *The Journal of Behavioral Health Services & Research*, 32: 341.
- Jessen F, Amariglio RE, van Boxtel M, Breteler M, Ceccaldi M, et al. (2014). A conceptual framework for research on subjective cognitive decline in preclinical Alzheimer's disease. *Alzheimers Dementia*, 10(6):844–852.
- Jeste DV, Alexopoulos GS, Bartels SJ, Cummings JL, Gallo JJ, et al. (1999). Consensus statement on the upcoming crisis in geriatric mental health: research agenda for the next 2 decades. *Archives General Psychiatry*, 56(9):848–853.
- Jester DJ, Rozek EK, McKelley RA (2018). Heart rate variability biofeedback: implications for cognitive and psychiatric effects in older

- adults. *Aging and Mental Health*,
<https://doi.org/10.1080/13607863.2018.1432031>
- Katon WJ (2003). Clinical and health services relationships between major depression, depressive symptoms, and general medical illness. *Biological Psychiatry*, 54(3):216-226.
- Kerner B (2014). Genetics of bipolar disorder. *The Application of Clinical Genetics*, 7:33-42. <http://doi.org/10.2147/TACG.S39297>
- Kessler RC, Berglund P, Demler M, Jin R, Merikangas KR, Walters EE (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62(6):593-602.
- Kindermann SS, Kalayam B, Brown GG, Burdick KE, Alexopoulos GS (1998). Executive Functions and P300 Latency in Elderly Depressed Patients and Control Subjects. *American J Geriatric Psychiatry*, 8(1):57-65.
- Kiosses DN, Teri L, Velligan DI, Alexopoulos GS (2011). A home-delivered intervention for depressed, cognitively impaired, disabled elders. *Int J Geriatr Psychiatry*, 26(3):256-62.
- Knight BG, Pachana NA (2015). *Psychological Assessment and Treatment with Older Adults*. Oxford, UK: Oxford University Press.
- Kocsis JH (1998). Geriatric Dysthymia. *J Clin Psychiatry*, 59 Suppl 10:13-5.
- Köhler S, Thomas AJ, Lloyd A et al. (2010). White matter hyperintensities, cortisol levels, brain atrophy and continuing cognitive deficits in late-life depression. *Br J Psychiatry*, 196:143-149.
- Kroenke K, Spitzer RL, & Williams JB (2001). The PHQ-9. *Journal of General Internal Medicine*, 16(9): 606-613.
- Lambert MJ, & Finch AE (1999). The Outcome Questionnaire. In M. E. Maruish (Ed.), *The use of psychological testing for treatment planning and outcomes assessment* (2nd ed.), pp. 831-869. Mahwah, NJ: Erlbaum.
- Lecrubier Y, Sheehan DV, Weiller E, Amorim P, Bonora I (1997). The Mini International Neuropsychiatric Interview (MINI). A short diagnostic structured interview: reliability and validity according to the CIDI. *European Psychiatry*, 12(5):224-231.
- Leichsenring F & Steinert C (2017). Is Cognitive Behavioral Therapy the Gold Standard for Psychotherapy? The Need for Plurality in Treatment and Research. *JAMA*, 318(14): doi:10.1001/jama.2017.13737.
- Leung, C., Fung, T., McEvoy, C., Lin, J., & Epel, E. (2018). Diet quality indices and leukocyte telomere length among health US adults: Data from NHANES 1992-2002. *American Journal of Epidemiology*, D0:

- 10.1093/aje/kwyl24)
- Lin, E. H. B., Katon, W., Von Korff, M., Tang, L. Q., Williams, J. W., Kroenke, K., . . . Unützer, J., for the IMPACT Investigators. (2003). Effect of improving depression care on pain and functional outcomes among older adults with arthritis: A randomized controlled trial. *JAMA*, 290(18), 2428–2434. doi: 10.1001/jama.290.18.2428
- Lisanby SH (2007). Electroconvulsive therapy for depression. *N Engl J Med*. 357(19):1939-45.
- Löwe B, Spitzer R, Gräfe K, Kronke K, Quenter A, Zipfel S, et al. (2004). Comparative validity of three screening questionnaires for DSM-IV depressive disorders and physicians' diagnoses. *Journal of Affective Disorders*, 78(2):131–140.
- Lu B & Ahmed I (2010). The Mind-Body Conundrum: The Somatopsychic Perspective in Geriatric Depression. *Am J Geriatric Psychiatry*, 18(5):378–381.
- Luppa M, Luck T, Riedel-Heller SG, Koenig H, Angermeyer MC (2012). Natural course of depressive symptoms in late life. An 8-year population-based prospective study. *J Affective Disorders*, 142(1-3): 166-171.
- Mackin RS, Areán P (2005). Evidence-based psychotherapeutic interventions for geriatric depression. *Psychiatric Clinics of North America*, 28(4):805-820.
- Manea L, Gilbody S, McMillan D (2012). Optimal cut-off score for diagnosing depression with the Patient Health Questionnaire (PHQ-9): a meta-analysis. *Canadian Medical Association Journal*, 184(3): E191-E196.
- McBeth J, Lacey RJ & Ross W (2014). Predictors of new-onset widespread pain in older adults: results from a population-based prospective cohort study in the UK. *Arthritis and Rheumatology*, 66(3):757-767. <https://doi.org/10.1002/art.38284>
- Meeks, T. W., Vahia, I. V., Lavretsky, H., Kulkarni, G., & Jeste, D. V. (2011). A tune in “a minor” can “b major”: A review of epidemiology, illness course, and public health implications of subthreshold depression in older adults. *Journal of Affective Disorders*, 129(1-3), 126–142.
- Miebach L, Wolfsgruber S, Frommann I, Buckley R, Wagner M (2018). Different Cognitive Complaint Profiles in Memory Clinic and Depressive Patients. *Am J Geriatric Psychiatry*, 26(4):463-475.
- Miller MD, Reynolds CF (2007). Expanding the usefulness of Interpersonal Psychotherapy (IPT) for depressed elders with co-morbid cognitive impairment. *Int J Geriatr Psychiatry*, 22(2): 101-5.

- Mind, Mood & Memory (2010). Massachusetts General Hospital, July, 10.
- Mind, Mood & Memory (2008). Massachusetts General Hospital, August, 3.
- Mirza SS, Wolters FJ, Swanson SA, Koudstaal PJ, Hofman A, et al. (2016). 10-year trajectories of depressive symptoms and risk of dementia: a population-based study. *The Lancet Psychiatry*, DOI: 10.1016/S2215-0366(16)00097-3
- Morey LC (1997). *The Personality Assessment Screener (professional manual)*. Lutz, FL: Psychological Assessment Resources.
- Mura G & Carta MG (2013). Physical activity in depressed elderly. A systematic review. *Clinical Practice and Epidemiology in Mental Health*, 9:125–135.
- Mutz J, Vipulanathan V, Carter B, Hurlmann R, Fu CHY, et al. (2019). Comparative efficacy and acceptability of non-surgical brain stimulation for the acute treatment of major depressive episodes in adults: systematic review and network meta-analysis. *British Medical Journal*, 364 :11079.
- Nelson J & Papakostas G (2009). Atypical antipsychotic augmentation in Major Depressive Disorder: A meta-analysis of placebo-controlled randomized trials. *Am J Psychiatry*, 166(9):980-9.
- Ng, C.-H., & Schweitzer, I. (2002). An overview of the management of treatment resistant depression in late life. *Psychogeriatrics*, 2, 156–164.
- lin JT, Katz IR, Meyers BS, Schneider LS, Lebowitz BD (2002). Provisional diagnostic criteria for depression of Alzheimer disease: rationale and background. *Am J Geriatric Psychiatry*, 10(2):129-41.
- verend, K, Bosanquet, K, Bailey, D, Foster, D, Gascoyne, S, Lewis, H, Nutbrown, S, Woodhouse, R, Gilbody, S, & Chew-Graham, C. Revealing hidden depression in older adults: A qualitative study within a randomized trial. *MBC Family Practice* 16, 142, 1-6.
- Parletta N, Zamowiecki D, Cho J, Wilson A, Bogomolova S, et al. (2017). A Mediterranean-style dietary intervention supplemented with fish oil improves diet quality and mental health in people with depression: A randomized controlled trial (HELFINMED). *Nutritional Neuroscience*, 7:1-14.
- Patten SB (2001). Long-term medical conditions and major depression in a Canadian population study at waves 1 and 2. *J Affective Disorders*, 63(1-3):35-41.
- Patten SB (2010). Accumulation of major depressive episodes over time in a prospective study indicates that retrospectively assessed lifetime prevalence estimates are too low. *BMC Psychiatry*, 9: 19.
<http://www.biomedcentral.com/1471-244X/9/19>

- Patten SB, Williams JVA, Lavorato DH, Wang JL, Jetté N, et al. (2018). Patterns of association of chronic medical conditions and major depression. *Epidemiology and Psychiatric Science*, 27(1):42-50. doi: 10.1017/S204579601600072X.
- Pedersen KF, Alves G, Larsen JP, Tysnes OB, Møller SG, Brønnick K (2012). Psychometric properties of the Starkstein Apathy Scale in patients with early untreated Parkinson disease. *American J Geriatric Psychiatry*, 20(2):142-8. doi: 10.1097/JGP.0b013e31823038f2.
- Pelton GH, Harper OL, Tabert MH, Sackeim HA, Scarmeas N, et al. (2008). Randomized double-blind placebo-controlled donepezil augmentation in antidepressant-treated elderly patients with depression and cognitive impairment: a pilot study. *International J Geriatric Psychiatry*, 23(7): 670-676.
- Periyakoil VS, Kraemer HC, Noda A, Moos R, Hallenbeck J, et al. (2005) The development and initial validation of the Terminally Ill Grief or Depression Scale (TIGDS). *International J Methods Psychiatric Research*, 14:203-212.
- Petkus AJ, Wetherell JL (2013). Acceptance and Commitment Therapy with Older Adults: Rationale and Considerations. *Cognitive and Behavioral Practice*, 20(1), 47-56.
- Pimontel MA, Rindskopf D, Rutherford BR, Brown PJ, Roose SP & Sneed JR (2016). A Meta-Analysis of Executive Dysfunction and Antidepressant Treatment Response in Late-Life Depression. *Am J Geriatric Psychiatry*, 24(1): 31- 41.
<http://doi.org/10.1016/j.jagp.2015.05.010>
- Pizzagalli DA, Webb CA, Dillon DG, Tenke CE, Kayser J, et al. (2018). Pretreatment Rostral Anterior Cingulate Cortex Theta Activity in Relation to Symptom Improvement in Depression: A Randomized Clinical Trial. *JAMA Psychiatry*, 75(6):547-554.
- Qato DM, Ozenberger K, Olfson M (2018). Prevalence of Prescription Medications with Depression as a Potential Adverse Effect Among Adults in the United States. *JAMA*, 319(22):2289-2298.
- Radloff LS (June 1977). The CES-D Scale: A Self-Report Depression Scale for Research in the General Population. *Applied Psychological Measurement*, 1(3): 385-401.
- Reynolds C, Alexopoulos GS, Katz R, Lebowitz BD (2001). Chronic depression in the elderly: approaches for prevention. *Drugs & Aging*, 18(7): 507-514.
- Robinson RG, Long J, Jorge RE (2017). Prevention of post-stroke mortality using problem-solving therapy or escitalopram. *Am J Geriatric Psychiatry*, 25(5): 512-519.

- Rosenberg PB, Mielke MM, Qian-Li X, Carlson MC (2010). Depressive symptoms predict incident cognitive impairment in cognitive healthy older women. *Am J Geriatric Psychiatry*, 18(3): 204-211.
- Rosenblat JD, Kakar R, & McIntyre RS (2016). The Cognitive Effects of Antidepressants in Major Depressive Disorder: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. *International Journal of Neuropsychopharmacology*, 19(2), pyv082. <http://doi.org/10.1093/ijnp/pyv082>
- Ruiz, V. (2019). Nutritional and integrative interventions for mental health disorders: Non-pharmaceutical interventions for depression, anxiety, bipolar, & ADHD. PESI Presentation, Savannah GA, February, 2019.
- Schaakxs R, Comijs HC, van der Mast RC, Schoevers RA, Beekman ATF, et al. (2017). Risk Factors for Depression: Differential Across Age? *Am J Geriatr Psychiatry*, 25(9):966-977.
- Schaakxs R, Comijs HC, Lamers F, Kok RM, Beekman AT (2018). Associations between age and the course of major depressive disorder: a 2-year longitudinal cohort study. *Lancet Psychiatry*, 5(7):581-590.
- Sabesan P, Lankappa S, Khalifa N, Krishnan V, Gandhi R, Palaniyappan L (2015). Transcranial magnetic stimulation for geriatric depression: Promises and pitfalls. *World J Psychiatry*, 5(2):170-181.
- Segal DL, Coolidge FL, Cahill BS, Riley AA (2008). Psychometric properties of the Beck Depression Inventory—II (BDI-II) among community-dwelling older adults. *Behavior Modification*, 32(1):3-20.
- Segal ZV, Williams JMG, & Teasdale JD (2012). *Mindfulness-based cognitive therapy for depression* NY: Guilford Press.
- Seitz D, Purandare N, Conn D (2010). Prevalence of psychiatric disorders among older adults in long-term care homes: A systematic review. *International Psychogeriatrics*, 22(7):1025-39.
- Schulz R, Beach SR, Ives DG, Martire LM, Ariyo AA, Kop WJ (2000). Association between depression and mortality in older adults: the Cardiovascular Health Study. *Archives of Internal Medicine*, 160(12): 1761-1768.
- Sheehan DV, Lecrubier Y, Sheehan KH, Amorim P, Janavs J, Weiller E, Hergueta T, Baker R, Dunbar GC. (1998). The Mini-International Neuropsychiatric Interview (M.I.N.I.): the development and validation of a structured diagnostic psychiatric interview for DSM-IV and ICD-10. *J Clinical Psychiatry*, 59 Suppl 20:22-33.
- Sheikh JI, & Yesavage JA (1986). Geriatric Depression Scale (GDS): Recent evidence and development of a shorter version. *Clinical Gerontologist: The Journal of Aging and Mental Health*, 5(1-2): 165-173.

- Sirey, J, Banerjee, S, Marino, P, Halkett, A, Raefifar, E, Paggi, M, & Bruce, M. (2016). Improving mental health treatment initiation among depressed community dwelling older adults. *American Journal of Geriatric Psychiatry*, 24, 4, 310—320.
- Sperling RA, Aisen PS, Beckett LA, Bennett DA, Craft S, et al. (2011). Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimers Association workgroups on diagnostic guidelines for Alzheimers disease. *Alzheimers and Dementia*, 7(3):280-292.
- Steenland K, Karnes C, Seals R, et al (2012). Late-life depression as a risk factor for mild cognitive impairment or Alzheimer's disease in 30 US Alzheimer's disease centers. *J Alzheimers Disease*, 31:265-275.
- Steer RA, Ball R, Ranieri F, Beck AT (1999). Dimensions of the Beck Depression Inventory-II in clinically depressed outpatients. *J Clinical Psychology*, 55:117-128.
- Steer, RA, Rissmiller DJ, Beck AT (2000). Use of the Beck Depression Inventory-II with depressed geriatric inpatients. *Behaviour Research and Therapy*, 38(3): 311-318.
- Steffens D, Fisher G, Langa K, Potter G, Plassman B (2009). Prevalence of depression among older Americans: The Aging, Demographics and Memory Study. *International Psychogeriatrics*, 21(5):879-888.
- Strakowski SM, Adler CM, DelBello MP (2013). Is depression simply a nonspecific response to brain injury? *Current Psychiatry Reports*, 15:386-402.
- Sunderland T & Minichiello M (1996). Dementia Mood Assessment Scale. *Int Psychogeriatr*. 8 Suppl 3:329-31.
- Sundermann EE, Katz MJ, Lipton RB, Lichtenstein AH, Derby CA (2016). A Brief Dietary Assessment Predicts Executive Dysfunction in an Elderly Cohort: Results from the Einstein Aging Study (EAS). *Journal of the American Geriatrics Society*, 64(11): e131–e136.
- Tadayonnejad R & Ajilore ● (2014). Brain network dysfunction in late-life depression: A literature review. *J Geriatric Psychiatry And Neurology*, 27(1), 5-12.
- Tagarello, F., Giradi, P., & Amore, M. (2009). Depression and apathy in dementia: Same syndrome or different construct. *The American Journal of Geriatric Psychiatry*, 49, 246-249.
- Tampubolon G & Maharani A (2017). Depression Trajectories of Older Americans and Britons 2002-2012: A Rejoinder and a Hope. *Am J Geriatric Psychiatry*, 25(11): 1198.
- Van Dalen, J., Van Wanrooij, I., Moli van Charante, E. et al. (2018). Association of apathy with risk of incident dementia: A systematic

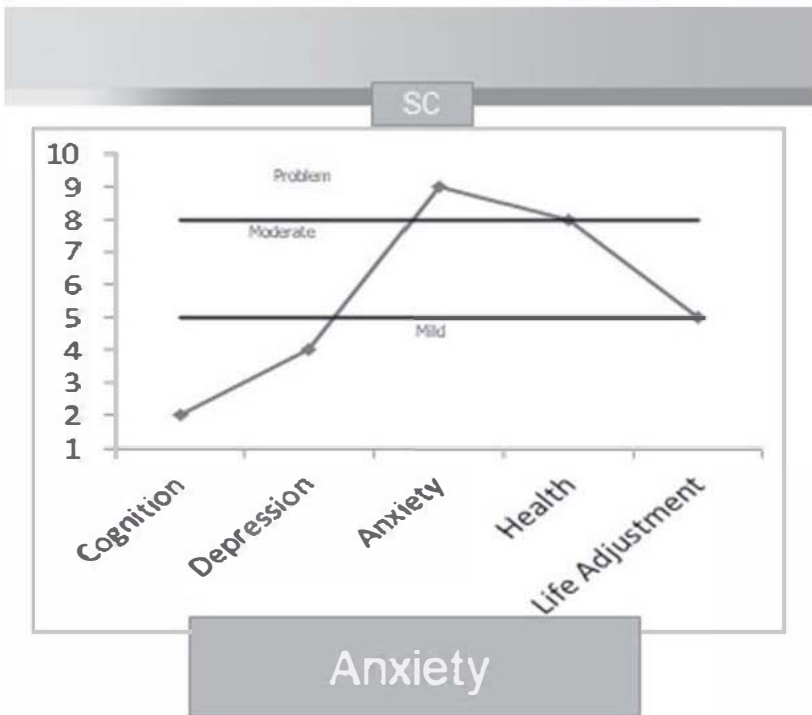
- review and meta-analysis. *JAMA Psychiatry*, 75, 1012-1021.
- Waldorff FB, Siersma V, Vogel A, Waldemar G (2012). Subjective memory complaints in general practice predicts future dementia: A 4-year follow-up study. *International J Geriatric Psychiatry*, 27:1180-1188.
- Weiner MW, Veitch DP, Aisen PS, Beckett LA, Cairns NJ, et al. (2015). 2014 Update of the Alzheimer's Disease Neuroimaging Initiative: A review of papers published since its inception. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 11(6), e1-120. <http://doi.org/10.1016/j.jalz.2014.11.001>
- Williams JBW, Kobak KA (2008). Development and reliability of a structured interview guide for the Montgomery-Asberg Depression Rating Scale. *The British Journal of Psychiatry*, 192(1):52-58.
- Wolfsgruber S, Polcher A, Koppa A, Kleineidam L, Frölich L, et al. (2017). Cerebrospinal fluid biomarkers and clinical progression in patients with subjective cognitive decline and mild cognitive impairment. *J Alzheimers Disease*, 58(3):939-950. doi: 10.3233/JAD-161252.
- Yates JA, Clare L, Woods RT, Matthews FE (2015). The Cognitive Function and Ageing Study Wales Subjective memory complaints are involved in the relationship between mood and mild cognitive impairment. *J Alzheimers Disease*, 48:S115-S123.
- Yesavage JA, Brink TL, Rose T, Lum ●, Huang ●, Adey V, Leier V. (1983). Development and validation of a geriatric depression screening scale: A preliminary report. *Journal of Psychiatric Research*, 17, 37-49.
- Zhu, C., Grossman, H., & Sano, M. (2019). Why do they just sit: Apathy as a score symptom of Alzheimer's Disease. *The American Journal of Geriatric Psychiatry*, 27, 4, 395-405.
- Zotev V, Phillips R, Misaki M, Wong CK, Wurfel BE, et al. (2018). Real-time fMRI neurofeedback training of the amygdala activity with simultaneous EEG in veterans with combat-related PTSD. *NeuroImage: Clinical*, 19:106-121. <https://doi.org/10.1016/j.nicl.2018.04.010>
- Zung, WW (1965). A self-rating depression scale. *Archives of General Psychiatry*, 12: 63-70.

CHAPTER 9

ANXIETY

LEE HYER AND KRYSTAL JACKSON

We pick up from Chapter 5



SC Case

Measures

Anxiety

MINI: Generalized Anxiety Disorder

GAD-7: 16

STAI =41

MBMD: Anxiety

PAI: Anxiety

Depression

Normal BDI-II, GDS-SF, and PHQ-9

Life Situation

Lives in ALF, married

Low education and SES

Health

Number of medications and medical comorbidities excessive

Chronic Diseases: 5

Health Rating: 3/10

Epworth Sleep Scale = 5

Pain: 8/10

Cigarettes: No

Alcohol: None

BMI: 33

Exercise: None

Cognition

Low normal (see below)

HISTORY: Patient has been a resident of an ALF for 7 years. This senior living facility provides some assistance. She is married, and she attends a CBT group. She is having trouble with anxiety and several medical issues that involve pain and considerable physical problems. She is on several medications and is attempting to deal better with her life in these areas. She also lost her son 2 years ago, and she has had a major grief reaction. She has resolved this to a reasonable degree. She has 3 daughters in the area who are supportive. She complains of worry and confusion. She is married and helps her husband.

RELEVANT DEVELOPMENTAL INFORMATION: Patient is the oldest child of 5, all the rest being boys. She has indicated that her life growing up was difficult. Her mother was a schizophrenic and this was a troubling experience for her as her mother was often sick. She graduated from high school and undertook some college at different points in her life. She initially joined the Air Force, but was only there for a year as her

mother became sick. She returned home to assist her dad raising her brothers. She worked on and off at different places and last worked about 10 to 15 years ago in an ALF. She had to leave because of pain in her back. She was married twice, the first time at age 19, and had 3 girls from this union. She later married at age 29 to her current husband, and they had 1 son. This son was eventually killed at age 31 in a car accident. Her husband is mildly impaired.

MEDICAL HISTORY: Patient has several physical illnesses. She does not drink alcohol or use tobacco. She is in constant pain with her back and also has fibromyalgia and MS. She noted further that she has asthma. She believes further that she has CHF. Her sleep at the present time is adequate, and she takes Xanax. She also takes Wellbutrin, Zoloft, Synthroid, omeprazole, and ibuprofen, as well as Lortab. She also experiences esophageal spasms, and she takes Nitro. She has had several surgeries including lung surgeries, hysterectomy, colon surgery (for cancer), gallbladder surgery, and bariatric surgery roughly 10 years ago. She had lost 100 pounds, but then has put that weight back on. There is a psychiatric history that largely involves her receiving medications.

OVERALL TEST RESULTS: MoCA score was 26/30, low normal. She scored in the average range for memory (4/5). She was below average on a test of phonemic fluency (7). She can do abstractions well. She was not able to do Serial 7s well. She had no problems either with recognition, a good sign. Her RBANS total was low normal. She had mild problems in language, abstraction, fluency, and visuospatial areas. On brief testing for executive functioning she was low average (Trails B and WCS). She does her ADLs and most IADLs. She does not have command of her medications, or her finances. She drives. Again she is in an ALF with minimal support.

EMOTIONAL ASSESSMENT: On the MINI, she was anxious (GAD). She scored in the normal depression range on the BDI-II, 10, as well as on the GDS-SF, 4. Her PHQ-9 was normal (3). She scored as highly anxious on the GAD-7 (16) and the STAI (41). In sum, she scored as anxious. Her interview fully corroborated these findings.

On the MBMD she registers as having anxiety for Axis I problems. She scores high on pain sensitivity and functional deficits. Her personality profile is one of confident, social, and cooperative traits.

PAIN and SLEEP: She has high pain sensitivity on the MBMD. She rated her pain at present as 5/10; average 6/10. She has problems (with pain) with activity (4/10), work (3/10), walking 8/10, work 7/10, relations with people (5/10), sleep (5/10), and enjoyment in life (4/10). Sleep is currently less of a problem as she is an ESS (5). She relates that she does sleep well.

WATCH and WAIT: This is a 65 y/o woman who is evaluated for psychological problems. She is currently living with her husband in a senior living environment. She has many health problems, including CHF, back pain, fibromyalgia, lung issues, and IBS. She has also had several surgeries including one for colon CA. She has an HS grade education. She had a difficult childhood and some abuse through the years. She can do all her ADLs and some IADLs. She does receive support. ● On the basis of test results today, she has a low average intelligence and shows some cognitive problems that do not seem to impact her IADLs. She seems to be able to do what is necessary in her life. She also scores as one with anxiety. She is somatically focused. Pain is an issue. Her pain and sleep are less salient issues these days. They are well managed in the ALF.

Checklist

Discernment: X

Core markers

Validate Problem X

Psychoeducation of Model X

Assessment X

Alliance X

Monitoring X

Case formulation X

Problem List

1. **ANXIETY:** High anxiety scores and expressed worry. Diagnosis of GAD. Monitor anxiety daily with SUDS, CBT group weekly, teach relaxation, coordinate exercise 4x/week with ALF, coping and assertive needs. Introduce HRV biofeedback, mindfulness exercises, time management, caregiver training (for husband)
2. **HEALTH:** Address pain and sleep with monitoring, coordinate with PCP and ALF on pain and med management, exercise and behavioral activation

Anxiety and health are major issues. She was admitted to a CBT group and targets were her anxious concerns about her health, her family, and her inability to assert herself and be clear about her goals. Mindfulness and relaxation as well as components of ACT therapy are to be enacted. She will also be educated in exercise and plans for upcoming medical appointments and decisions. She will also be supported and efforts will be made to have her family in therapy over time. Her husband is now sick but is cared for. Her daughter is bipolar. She lives in an ALF.

Self or other monitoring: Mni-targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks:

Managing situational contingencies:

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation: X

Concrete PS training: X

Imagery exercise for counter-experience: X

Compassion training:

Communication and Assertiveness training: X

Behavioral experiments: X

Emotional exposure:

Homework: X

Longer therapy processes:

CBT: Anxiety management. CBT group targets were her anxious concerns about her health, her family, and her inability to assert herself and be clear about her goals. Mindfulness and relaxation as well as components of ACT therapy are to be enacted.

PST: Plan for caregiving problems.

ACT: use of acceptance interventions in the context of her caregiving stress

Lifestyles:

- Practice Spirituality/Religion
- Socialize: Less decline and live longer
- Develop mild resilience with coping methods
- Exercise: Daily walking
- Diet: ●rganize through ALF
- Somatic softening: Meditate and Mindfulness
- Values and Leisure: Initiate in ALF with social worker

Follow-up: Re-evaluate every 6 months

Anxiety

GAD-7: 9

STAI=32

MBMD: Anxiety < PR 45

<p>Health</p> <p>Same number of meds</p> <p>Chronic Diseases: 5</p> <p>Health Rating: 5</p> <p>ESS = 3</p> <p>Pain: 70</p> <p>BMI: 33</p> <p>Exercise: Improved</p> <p>Depression: Normal</p> <p>Cognition: Normal MoCA (26/30)</p> <p>Life Adjustment: Same</p> <p>Relapse Issues: Follow over time</p> <p>Post Profile improved but still Health and Anxiety are Problems</p>

Model:**Anxiety**

Measure	Healthy	Moderate Problem	Problematic or Syndromic
Description	Daily functioning is not impaired by worry or fear.	Some activities are impaired by worry or fear, but the person is still functional at work and interpersonally.	Some activities are impaired by worry or fear, but the person is still functional at work and interpersonally.
MINI	No positive hits	Subsyndromal hits	Syndromal hits
GAD-7	≤6	7-14	≥15
BAI	≤9	10-16	≥17
STAI	≤29	30-44	≥45

Brief Measures

Anxiety and depression are intimately related and highly comorbid. The number of symptom combinations with these two disorders is staggering. We suggest the use of the GAD-7, BAI, and STAI. We note here that occult anxiety (subsyndromal) is especially insidious in an older population. Anxiety is generally factored into four areas: worry, somatic issues, interpersonal, and physiological problems. Remember that worry is present across numerous diagnoses and can impact life in ways that isn't always captured on typical measures. Additionally, we use PTSD scales (Clinician Administered PTSD Scale, CAPS), if necessary, as well as the

MINI and the Hamilton Anxiety Rating Scale on occasion. There are of course other scales as anxiety is reasonably generic and assesses perturbation in general. Distinct forms of anxiety or worry identify the type of disorder.

Generalized Anxiety Disorder-7: The GAD-7 is a brief assessment using the seven most correlated items of 13 original diagnostic criteria items for GAD itself. The GAD-7 has excellent internal consistency, good test-retest reliability, good convergent validity, and good criterion validity. The tool includes a measure of severity, and it is able to distinguish between depressive and anxious symptoms (Spitzer, Kroenke, Williams, & Lowe, 2006).

State-Trait Anxiety Inventory: The STAI is a self-report questionnaire with 20 questions about how the person feels “right now, at this moment” (p. 6) and 20 questions about how the person “generally” (p. 6) feels. The STAI is designed to measure apprehension, tension, nervousness, and worry. The STAI was normed for high school students, college students, working adults, and military recruits through age 69 (Spielberger, 1983). Kvaal, Ulstein, Nordhus, and Engedal (2005) have been able to provide the specificity and sensitivity of the STAI for adults of 60 years old and older when compared to the DSM-IV-TR criteria.

Beck Anxiety Inventory: The BAI is designed to discriminate between anxiety symptoms and depression symptoms when examining their presence in the previous seven days. Although the BAI has been shown to have adequate reliability and validity for older adults, some concerns have been noted about the 14 somatic-focused questions. This is a concern because these symptoms may not be present in patients with GAD, and older adults may not experience the same type of physical symptoms as younger adults. Although the BAI was found to have good internal consistency, there was no discriminant validity found between the BAI and measures of depression (Wetherell & Gatz, 2005). It has been suggested that the BAI could distinguish between older patients with anxiety and a control group (Lauderdale & Sheikh, 2003). A factor analysis of the BAI identified four components: GI symptoms, CNS hyperarousal, excessive worry, and difficulty in making decisions.

Added Clinician Rated Assessments

Hamilton Anxiety Rating Scale: The HARS is commonly used to detect anxiety. It is a 14-item scale in which an observer rates each item on a scale from 0 (none) to 4 (severe). A score of 18 or higher would indicate the presence of clinical anxiety. It has been suggested that the HARS can detect GAD in the elderly compared to control (Lauderdale & Sheikh,

2003).

Clinician Administered PTSD Scale: The CAPS is a widely used interview designed to diagnose PTSD. Although originally designed to be used with veterans, it is currently used with a variety of traumas. It is based on the DSM-IV criteria for PTSD. For each symptom, a severity rating is determined using a five point rating scale (0-4) for frequency and duration. A literature review indicated that the CAPS has very positive psychometric properties (Weathers, Keane, & Davidson, 2001).

Complete Assessment

This is perhaps a misnomer. The above measures/ratings are generally sufficient for an assessment of anxiety. Therrien and Hunsley (2012) identified 91 different measures across 213 articles examining the assessment of anxiety in older adults. These included overall ratings of various anxiety problems, behavioral assessments, and anxiety-related measures, and the use of a diary. They have adequate reliability and validation for older adults. They can also be the most helpful in cases of PTSD and with panic.

- Ratings
ADIS-IV, SCID, MINI, HRSRSD (17 items) or HARS (13 items)
- Self-Report Measures
PSWQ, STAI, MAQ, SAST, Worry Questionnaire, Worry Scale for Older Adults, DASS, ASI, POMS, BAI, PANAS, ATQ (Automatic Thoughts Quest, 21 items)
Diary (interpersonal avoidance issues)
- Omnibus Measures: MBMD, PAI
- Unique Measures: Anxiety and Aging Scale (AAS), Death Anxiety Scale (DAS).

ADIS-IV is the Anxiety Disorders Interview Schedule for DSM-IV; SCID is the Structured Clinical Interview for DSM; MINI is the Mini International Neuropsychiatric Interview; HSRSD is the Hamilton Rating Scale for Depression; HARS is the Hamilton Anxiety Rating Scale; PSWQ is the Penn State Worry Questionnaire; STAI is the State Trait Anxiety Test; DASS is the Depression Anxiety and Stress Scale; ASI is the Anxiety Sensitivity Index; POMS is the Profile of Mood States; BAI is the Beck Anxiety Index; PANAS is the Positive and Negative Affect Scale; MBMD is the Millon Behavioral Medical Diagnostic, PAI is the Personality Assessment Index

Background

Overall, anxiety is complex, and it has many parts and problems. For starters, it is a syndrome, a sign and a symptom. It is also comorbid with depression, somatization, and dementia (American Psychiatric Association, 2013). Medical problems can confound its expression and causality (Cassidy & Rector, 2008). Subthreshold worry is also a concern. Anxiety appears to have a relationship but also exists independently of worry or tension. About half of older adults incur worry later in life. Anxiety and executive dysfunction also have a distinct relationship. Treatment is best considered with the use of modules rather than treatment manuals.

Facts of Anxiety at Later Life

- Syndrome, sign and a symptom.
- Need functional attachment with symptoms and real world issues (medical, social, financial, family).
- Problem “true anxiety” and “secondary anxiety.”
- Subthreshold is serious.
- Anxiety is a precursor to cognitive problems (perhaps dementia).
- Anxiety often progresses to depression (and vice versa).
- Modules better than inflexible manuals.

As with younger adults, each anxiety disorder has a unique component: GAD has chronic uncontrolled worry; panic shows a somatic overconcern/avoidance; phobias involve fearful avoidance; social anxiety has a cognitive self-evaluation and avoidance expression; OCD has requisite obsessions and compulsions; and PTSD involves numbing, re-experiencing, avoidance, and arousal (American Psychiatric Association, 2013). Fear itself is complex and is more than one emotion. In general, PTSD and phobia involve an under-active prefrontal cortex and disinhibited amygdala, and GAD and OCD involve an overactive prefrontal cortex. Also in general (as we shall see), cross sectional investigations mainly support the hypothesis that the presence or severity of anxiety is associated with lower cognitive performance in older adults. Those who develop GAD later in life report more functional impairment and poor health than those with earlier onset GAD. This may actually involve negative affectivity and a disposition state (neuroticism), and be correlated with mood disorders, anxiety disorders, somatoform disorders, eating disorders and alcohol problems, as well as some Axis II disorders.

For older adults, anxiety itself is the most common psychiatric diagnosis (Beaudreau & O'Hara, 2008). Generalized anxiety or worry is

the best marker of the problems experienced by older adults. For older adults too, anxiety is often seen at subthreshold levels and is more prominent than depression (Bryant, Jackson, & Ames, 2008). Even at subthreshold levels, anxiety can be problematic, especially over time. As many as 20% of older adults may experience clinical anxiety in some form (Byers, Yaffe, Covinsky, Friedman, & Bruce, 2010). This is twice as many as occurrences of dementias and is significantly more prevalent than instances of MDD (Cassidy & Rector, 2008). Unfortunately, these prevalence rates may be underestimated because older adults tend to minimize anxiety symptoms or fail to recognize them (Gurian & Minor, 1991). Additionally, physicians may not recognize the symptoms when they are presented (Vink, Aartsen, & Schoevers, 2008). As mentioned previously, a diagnosis of depression typically includes a comorbid anxiety diagnosis in about half of all cases. When anxiety is diagnosed first, this number is less (Wolitzky-Taylor, Castriotta, Lenze, Stanley, & Craske, 2010). For older adults, anxiety and depression are seen frequently in primary care clinics where visits and medication use have increased (Porensky et al., 2009).

The underlying structural and functional neural anatomy of late-life GAD is relatively understudied and has received little attention. This gap in our knowledge is problematic since the treatment response to late-life GAD is relatively poor, especially when compared to midlife GAD treatment with CBT. To date, there are no published studies addressing the neural changes following pharmacotherapy in late-life GAD, but the identification of these changes would be very beneficial. Andrescu et al. (2015) showed that functional connectivity during worry and reappraisal indicates the presence of deficits in both the generation and reappraisal of worry. With pharmacologic treatment, they found greater connectivity in the prefrontal nodes of the executive control network during reappraisal of memory. Compared with non-anxious participants, elderly GAD participants showed multiple differences in the neural networks involved in emotional generation and regulation. Pharmacology treatment of late-life GAD also produced significant changes in the same networks.

In many cases, older adults may report their problems as concern or worry, and they may also misattribute their somatic anxiety symptoms to a medical problem (Kawachi, Sparrow, Vokonas, & Weiss, 1994). The concern for the clinician is often what is primary and what is secondary anxiety. Issues that may arise with anxiety include physical complications, decrease in overall well-being, increased mortality, and an extensive over use of services. Anxiety disorders in older adults occur frequently with other conditions, both psychiatric and medical, with somatic symptoms

being a hallmark component. Cardiovascular disease, pain disorders, lung disease, and gastrointestinal problems are all found to be significantly associated with anxiety disorders. As a result, older adults are likely taking multiple medications, some of which can worsen the anxiety symptoms (Cassidy & Rector, 2008). As one might expect, anxiety complicates these issues, and the physical issues may exacerbate the anxiety.

For the most part, psychiatric illnesses are seen early in life and become exacerbated later in life (Wolitzky-Taylor, Castriotta, Lenze, Stanley, & Craske, 2010). However, of those with current or lifetime GAD, half experience their first onset after age 50. In late onset GAD, older adults report more functional impairment and poorer health than those who have an earlier anxiety onset (Cassidy & Rector, 2008). We should note too, that the common problems are avoidance and excessiveness (that is out of proportion to an actual danger).

When looking for a differential diagnosis, overlapping symptoms between anxiety, depression, and dementia complicate the picture. When GAD is diagnosed in patients with AD, the anxiety symptoms are primarily hoarding, agitation, and/or aggression. Furthermore, research has suggested that anxiety may be a predictor of cognitive decline (Wolitzky-Taylor, Castriotta, Lenze, Stanley, & Craske, 2010). Research to differentiate GAD from dementia has resulted in a proposed symptomology for anxiety in AD that includes irritability, muscle tension, fears, restlessness, and respiratory problems (Starkstein, Jorge, Petracca, & Robinson, 2007). This has also been considered a form of depression in dementia.

Anxiety and Cognitive Decline

Anxiety has been shown to result in poor cognitive performance in older adults (Beaudreau & O'Hara, 2008). As adults age, reduced working memory and processing speed occur. This may mean that older adults in particular may be more vulnerable than their younger counterparts to the effects of anxiety on cognitive functioning (Hyer, 2014). Anxiety and depression together also predict more rapid decline in memory later in life than depression by itself (O'Hara, 2012).

We should note that anxiety adds problems everywhere. Below we see the effect of how anxiety influences subjective memory impairment (SMI) and MCI by its mere presence (Jessen et al., 2010). In this article, subjects were asked if they had memory problems (no objective data) and whether they worry. Some were diagnosed as having MCI. The MCI group resulted in the highest conversion rates to dementia. Those with SMI and worry

also had a substantial likelihood of developing a dementia compared to those without worry. Worry matters!

Worry and Memory

Summary: SMI Ss were more likely than people without SMI to show changes in brain activity resembling those seen in the early stages of AD (based on MRI). This was measurably enhanced by worry.

Method: Adults 75 and older (N=2415): Asked if memory were a problem and, if so, did they worry.

Results: Ss with SMI and No Worry: 2x more likely to develop AD as no SMI.

Ss with SMI and Worry: 6x more likely to develop AD as no SMI.

Ss with MCI: 10x more likely to develop dementia and 20x for AD.

Jensen, et al., 2010 Archives of General Psychiatry

Related to the study cited above, Andreescu et al. (2015) assessed the complex associations between depression, anxiety, and MCI. The authors assessed just under 2,000 individuals aged 65 and over. The results suggested that over time chronic anxiety was associated with MCI on all conditions. Overall, the results indicated that subgroups with different anxiety and depression profiles had different relationships with cognitive and functional definitions of MCI. In another study by Mah, Binns, & Steffens (2015), anxiety symptoms in amnesic MCI patients predicted conversion to AD independent of depression and memory loss. These findings suggested that anxiety is not a prodromal non-cognitive feature of AD. It may actually accelerate the decline into AD through direct and indirect effects. The risk for AD was increased by 33%, 78%, and 135% for mild, moderate, and severe anxiety, respectively. When baseline hippocampal volume, amygdala markers, executive function, and atrophy were included in the model, the association between anxiety and AD remained significant. If anxiety in amnesic MCI increases the rate of decline toward AD, anxiety may be a prodromal symptom of AD. Perhaps anxiety is a subjective reaction to worsening memory instead. Anxiety, in this study, was related to an increased rate of atrophy in the entorhinal cortex volume, suggesting that anxiety may accelerate decline to AD through multiple different pathways.

Patients with MCI and dementia have similar rates of anxiety (77% and 80% respectively), and anxiety may also be affected by the progression of the impairment (Diefenbach, Bragdon, & Blank, 2014). Anxiety symptoms can most often correspond to deficits in executive

functioning with MCI, vascular dementia, and behavioral symptoms. The identification of the role of anxiety in dementia is crucial because symptoms dictate the treatment and overall prognosis. For those with anxiety, a lower quality of life, decreased functioning, increased caregiver burden, and increased risk of placement outside the home are likely. Furthermore, the presence of anxiety increases the likelihood of a depressive disorder in this population, and, as noted above, the combination of anxiety and depression is associated with more severe impairment and more rapid decline in cognitive functioning compared to those with only depression. In healthy older adults, worry was a predictor of the onset of MCI over the course of two years.

Gulpers, et al. (2016) found that anxiety predicts the onset of both cognitive impairment and dementia. The authors noted that the relationship could also be the reverse with the decline resulting in anxiety. Antidepressants such as selective serotonin reuptake inhibitors do not reverse cognitive problems. Furthermore, they are effective in only severe forms of anxiety, such as generalized anxiety disorder or major depression with co-occurring anxiety. Evidence-based psychotherapy, such as CBT, may have limited benefits in this age as well, particularly when comorbid cognitive impairment is present. In light of these limitations, the best chance to manage cognitive decline in the context of anxiety is to use a multimodal integrative approach. This includes physical exercise (aerobic and strength training), mindfulness, and cognitive training.

Data from our clinic show that among patients who presented for memory problems in the past year the average anxiety score was high depending on the level of cognition problems; dementia was worst. In effect, either anxiety served as a problem state before the cognitive issue or became worse after cognitive problems.

Anxiety and Cognition			
Measure	AAMI (n=35)	MCI (n=34)	Early Dementia (n=42)
GAD-7	4	6	10
SAST	17	22	23
PSWQ	38	51	56
MBMD-TEN	68	71	72

One more problem to consider is falls. Decreases in functional motor skills are often seen in conjunction with anxiety and depression (Vetter & Ford, 1989). For many older adults, the fear of falling leads to a drastic decrease in the quality of life (Iglesias, Manca, & Torgerson, 2008),

possibly because they restrict their lives as a result of fear. Agoraphobia also occurs frequently in this population and can happen after a traumatic event like falling. Fear of falling can result in increased isolation, increased depression, and interference with rehabilitation (Cassidy & Rector, 2003).

PTSD

PTSD is a special problem. It ranges from 1.5% to 4% in older adults in the United States (Acierno et al., 2007). The prevalence of subclinical PTSD is significantly higher in younger adults compared to younger adults (van Zelst, de Beurs, Beekman, Deeg, & van Dyck, 2003). Authors of the DSM-5 made important changes to the diagnostic criteria for PTSD, and it's not clear yet how this may change how PTSD is identified in older adults. Results of the National Health and Resilience in Veterans study (Mota et al., 2016) showed that in US veterans, the probabilities of PTSD and psychiatric comorbidity were similar to in studies using DSM-IV based measures. In older adults with PTSD, the symptoms of avoidance and arousal are more prevalent with early life traumatization (Böttche, Kuwert & Knaevelsrud, 2012) and dissociation is decreased (Labinsky, Blair & Yehuda, 2006).

PTSD may be a moving target. It continues to be conceptualized by an ever increasing number of factors. Recall that most trauma for older adults percolates from emergency rooms after a medical event. This is important as this form of trauma is less studied. In the past few years the DSM-5 and the ICD-11 have altered the components of the trauma response. The DSM-5 criteria are now richer by three criteria against a backdrop of four symptom clusters with differences in the wording in items. The ICD-11 settled on three clusters of questions (re-experiencing, avoidance, and a persistent sense of current threat manifested by exaggerated startle and hypervigilance). In fact, the phenomenology of PTSD at later life is elusive but is featured by the anxious trauma as well as dysphoria. The addition of other life and medical problems makes the care of this disorder complex.

The trauma response in later life may be due in part to unique cohort features. Understanding early life trauma is important but difficult (Ladson & Bienenfeld, 2007). Those adults in the older old category (>75) may be faced with feelings of shame and fear about self-disclosure which could prevent them from talking about psychological complaints (Franco, 2007). Older adults may be more likely to report physical concerns or pain instead of psychological symptoms making them more difficult to detect.

Furthermore, negative life events can trigger a past grief that has been unprocessed. As one gets older, avoidance-based coping strategies become less effective (Khouzam, 2008). PTSD is comorbid with many other diagnoses, and this also complicates recognition and treatment. Furthermore, illnesses often present in old age such as dementia, cerebrovascular accident or Parkinson's disease may result in cognitive impairment, which in turn may result in difficulty with communication.

Cognitive impairment is more common in adults with PTSD compared to those exposed to trauma but without PTSD (Qureshi et al., 2011). Several studies have shown that pre-existing brain abnormalities that result in cognitive problems may be a risk factor for PTSD (Kremen, Koenen, Afari, & Lyons, 2012). The National Health and Resilience in Veterans Study suggested that late life exacerbated PTSD may be associated with executive dysfunction in one out of ten veterans (Mota et al., 2016). Cognitive deficits such as learning disabilities, memory dysfunction, and attention deficits can also result from neurobiological changes of PTSD (Samuelson, 2011).

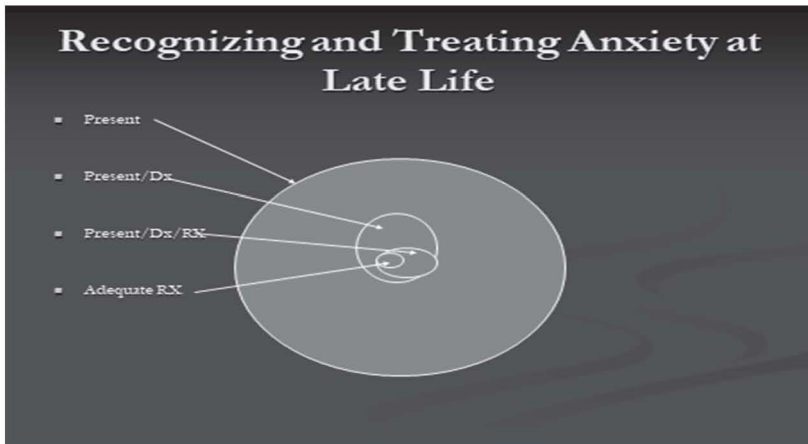
Co-occurring medical problems can impede the assessment and treatment of PTSD because of symptom overlap and/or exacerbation of symptoms: PTSD can both cause and exacerbate somatic problems. PTSD is associated with conditions such as cardiovascular disease, type 2 diabetes mellitus, and dementia (Lohr et al., 2015). Approximately 80%-90% of people with PTSD have at least one, and approximately two thirds have two or more co-occurring psychiatric disorders (Van Minnen, Zoellner, Hamed, & Mills, 2015). Pietrzak, Southwick, Galea, and Norris (2012) found that PTSD and partial PTSD in a sample of older adults (60+) in the US were associated with an increased likelihood of lifetime mood and anxiety symptoms, drug use, and borderline and narcissistic personality disorders.

Even though older adults have a high stress rate and multiple stressors, they do not seem to be more vulnerable or sensitive to trauma than young adults (Hyer, Kramer, & Sohnle, 2004). This population is still susceptible to Acute Stress Disorder (ASD) though. The most common acute trauma in late adulthood is often considered motor vehicle accidents (MVAs; Norris, 1992). MVAs are the second leading cause of injury in this population and older adults show the highest crash fatality rate. In most cases, these accidents could be attributed to driver error (Mandavia & Newton, 1998). Many other kinds of trauma common in the elderly are often overlooked, however, in primary care and mental health settings. Anxiety is also often found in conjunction with medical disorders in older adults (Lauderdale & Sheikh, 2003). Medical issues, such as cancer and

cancer treatment and serious health problems such as cardiac care using CABG and ventricular assist devices (VADs) may also result in PTSD. Additional traumas to consider for possible PTSD may include the loss of a spouse, victimization by crime, and abuse (Newton, 2010; McCartney & Severson, 1997).

Treatment

The treatment of anxiety at later life is challenging. This is so for many reasons but perhaps mainly because it is difficult to formally diagnose and then come up with an empirically valid treatment. Anxiety at later life also differs depending on when the person incurs the disorder: Late-life GAD, for example, is associated with less severe worry than early onset GAD. It is also true that, when patients see problems as physical, then no mental health diagnosis is provided; if attributions are anxiety-based, then one is given. Primary care physicians, the usual gatekeepers for older adults and anxiety problems, are often at a loss as to the best attribution (cause) of the problem. In fact, most older adults who meet criteria for an anxiety disorder do not receive treatment. It is not uncommon for one to have some depression, some physical problems, and some recent stresses and to have considerable anxiety. The other components will be addressed first and often solely.



Given a diagnosis, the most effective models of treatment include the implementation of several different components (Lauderdale & Sheikh, 2003). Ayers, Sorrell, Thorp, and Wetherell (2007) reviewed the literature

for geriatric anxiety treatment and identified 17 studies with evidence-based treatments (EBTs) for older adults with GAD or mixed anxiety disorders: relaxation training, cognitive-behavioral therapy (CBT), supportive therapy, and cognitive therapy. All showed some value. CBT for late-life GAD seems to have the most consistent support (Bartels et al., 2002), and relaxation training can be utilized as an effective but low-cost intervention (Thorp et al., 2009). Studies have also shown promise for the use of CBT and executive function (EF) training in older adults with anxiety and cognitive decline (e.g., Mohlman & Gorman, 2005). More recently, Hall, Kellett, Berríos, Bains, and Scott (2016) conducted a meta-analysis of several studies on GAD. They found that at six-months post-treatment, CBT had significant treatment effects compared to wait-list or treatment as usual controls. When compared to active controls, no CBT treatment had showed a significant advantage at the end of treatment or at follow-up. In sum, CBT is likely more effective than no treatment at all, but more research is needed.

Similarly, mindfulness (Segal, Williams, & Teasdale, 2012), well-being therapy (e.g., Frish, 1998), and positive psychotherapy (Seligman, Rashid, & Parks, 2006) are treatments that have been shown to be effective for anxiety, but not specifically researched with older adults. Acceptance and Commitment Therapy (ACT) has been applied to older clients with GAD with positive results (Wetherell et al., 2011). A specific, stand-alone effective treatment is exercise (Herring, O'Connor, & Dishman, 2010). It can be used to treat depression, cognitive decline, medical problems, and anxiety, as well as to increase quality of life (Hyer, 2014).

Research has also indicated that anxiety medications are effective for providing quick relief for older adults (Pinquart, Duberstein, & Lyness, 2006), and have increased effectiveness compared to psychotherapy (Pinquart, Duberstein, & Lyness, 2007). Benzodiazepines and SSRIs are often recommended for the treatment of GAD, Panic Disorder, and PTSD in the elderly. Although benzodiazepines may provide effective relief for anxiety symptoms, in the older population they may also present some risks such as cognitive decline (Patemiti, Dufouil, & Alperovitch, 2002). Because of the risks with benzodiazepines, especially with long term use, SSRI medications may prove to be a safer alternative, but studies on the long-term effects for the elderly are still limited (Katz, Reynolds, Alexopoulos, & Hackett, 2002). Antidepressant medication is usually used to treat most patients with anxiety disorders. Increasing numbers of patients also use antidepressants long term. Unfortunately response to antidepressants in patients is often variable due to a lack of continuous

treatment with an antidepressant or a reduction of antidepressant potency due to repeated exposure. A response failure could also be the result of progression of the disease.

Regardless, the Pinquart et al. (2006, 2007) study does indicate that medication seems to have an impact. The results indicated that both pharmacotherapy and behavioral interventions are reasonably effective, though more robust effects were found for pharmacotherapy. Meds work and can be used short term, especially with psychotherapy modules (e.g., CBT), to good effect. We caution that the number of studies examining the effectiveness of medications combined with psychotherapy at later life are limited. Medication and psychotherapy are, however, likely to be recommended when anxiety and depression are comorbid (Sheikh & Cassidy, 2000).

There are other treatments or formats. Brenes, Danhauer, Lyles, Anderson, and Miller (2017) examined the long-term effects of the telephone delivery of CBT-T compared with non-directive supportive therapy in rural older adults with GAD. They divided 141 older adults with GAD into these two groups. At 15 months follow-up, there was a significantly greater decline in general anxiety symptoms and worry difference among participants who were in the CBT group compared to those in the non-directive supportive therapy group. There were no significant differences between the conditions with depressive and GAD symptoms. The conclusion was that CBT-T is superior to non-directive support therapy in reducing anxiety and depression after one instance of completing treatment. Additionally, Goncalves and Byrne (2012) found that for older adults, nonspecific, non-pharmacological, and group treatment for GAD may be key factors for an effective intervention.

Common Anxiety Treatments

- Meds, both anxiolytic and SSRI (e.g., Pinquart, Duberstein, & Lyness 2007)
- CBT (Bartels et al., 2002)
- Problem Solving Therapy (Alexopolous, Raue, Kanellopoulos, Mackin, & Arean, 2008)
- ACT (Wetherell et al., 2011)
- Attention Training (Papageorgiou & Wells, 1998)
- Intolerance of uncertainty (Ladouceur & Gosselin, 2000)
- Emotional dysregulation (Memmin, Heimberg, Turk & Fresco, 2005)
- Metacognition (Wells & King, 2006)

Having the option of an effective behavioral intervention as an alternative to medication is important for many, but this is especially true for older adults. This population presents with unique issues that make the use of medication much more complicated. When determining if medication is appropriate, the previous treatment and response to that treatment, current symptoms, current medications, and side effects should all be taken into account (Lauderdale & Sheikh, 2003). Again, overall medication can be effective by itself, and it can enhance the effectiveness of behavioral interventions. On the other hand, the behavioral interventions can also enhance the effectiveness of the medications (Hyer, 2014).

Regardless, think of the following for better care. These general ideas especially apply to primary care.

Overall Anxiety Treatment Ideas

1. Do not wait for the patient to report anxiety
2. Assess carefully the type of anxiety (GAD and Social Anxiety are prevalent)
3. Manage direction and time
4. Discern primary from secondary anxiety
5. Be empathic and use MI
6. Culture matters
7. Collaborate and refer

In no other disorder are treatment modules more relevant than anxiety. This is most often in combination with CBT/ACT/PST. Relaxation or somatic softening is applied at the gate along with psychoeducation. Monitoring is frequently applied, and motivational interviewing is an early intervention. Again, follow-up and booster sessions are important.

ANXIETY MODULAR INTERVENTIONS

RELAXATION	PAIN MANAGEMENT
SLEEP HYGIENE	PLEASANT ACTIVITIES
PROBLEM SOLVING	ASSERTIVENESS TRAINING
WORRY CONTROL	TIME MANAGEMENT

ACCEPTANCE	COGNITIVE THERAPY
MINDFULNESS	EXPOSURE
BEHAVIORAL ACTIVATION	FAMILY INVOLVEMENT
TOLERANCE OF UNCERTAINTY	BIOFEEDBACK

The format applied by the Watch and Wait model is applied as always. WE suggest subtle differences below. One difference involves the need to be aggressive with anxiety as it is omnipresent but elusive and often camouflaged. Medication also is a first option. As noted, modules are introduced early and often. This runs the gamut from standard psychological interventions to sleep and exercise.

<p>Treatment Format</p> <ul style="list-style-type: none"> • Assess in usual way plus cognition • Watch and Wait model applied • Monitor and assess <p>Treatment Phase</p> <ul style="list-style-type: none"> • Set stage for treatment is SSRI/SNRI • Modules: Worry Control, Assertiveness, Sleep interventions, CBT/PST/ACT <p>Consider relaxation training/ACT</p> <p>If problems: Motivational interviewing to encourage a medication trial</p> <p>Always: Supportive contact and communication with prescriber/case mgr.</p> <p>Monitor other domains and watch for relapse</p> <ul style="list-style-type: none"> • Use case manager for frequency and follow up • Follow up and use boosters • Overall response WILL generally be slower than in patients with depression
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Worry may require a more targeted approach. It is prevalent and different from fear. It is rather cognitive. Worry habits develop through a process of automatic association between a behavioral response and any context that occurs repeatedly with the performance of that behavior. The behavior, thinking, and emotion resulting in anxiety cohabit. An issue

of importance is the contexts (cues) that become automatic triggers for worry. This occurs without the mediation of an implicit or explicit goal. Thus, any response repeated frequently that is contingent on a context results in a habitual response. Worry becomes rumination quickly and can occur without direct intention or effort. Habits like this do not alter easily. They do respond to alterations in external cues or an acceptance of the internal experience. This is why we see modules, motivational interviewing, acceptance, and altered external environments as key parts to the usual interventions. A focus on thought content alone is generally insufficient to stop habits. The environment needs to be tweaked or some counterconditioning involving an extinction of the habit connection is in order. This usually means a spouse, caregiver, or focus on the context of the behaviors.

In essence, the patient needs to appreciate the process – expect the cue, label it (or notice it distinctly), tolerate it, and continue on with the normal activity (absent the anxiety). Worry is then a habit plus. It is a type of learning, a flawed coping strategy to deal with habitual situations that is ingrained and repetitive. Worry is the avoiding of risk by thinking (rumination), problem solving without making plans of action, avoiding negative aspects of the situation, controlling unwanted feelings, using understanding for avoidance, or feeling certain about beliefs. For older adults worry is less about helping or solving a problem and more about easing tension in a negative reinforcement way: “I worry about this to avoid something else that I see as worse.” The targets are predictable: health, pain, family, self-concerns, or aging concerns. Again, interventions involve altered settings, alternate behaviors, and acceptance.

The intervention, worry control, is especially helpful as it corrals the problem and provides some degree of control. The goal is not to stop anxiety but to shift repetitive thinking about real problems so that it becomes adaptive and not intrusive. The core principles of CBT (contextual-functional-analytic perspective), reducing habits, normalizing the experience, encouraging active, concrete, experiential, and specific behavior taking a functional approach, and linking triggers as changed habits, as well as forming a focus on the non-specific factors of therapy (warmth, empathy, optimism, validation, and persistence), are helpful. In a sense worry is initially treated by psychoeducation (this is what you are doing), providing a plan (coping, acceptance, worry control, etc.) and practice. Installation of the coping strategy is the easy part; activation and practice involve work. Motivational interviewing and monitoring are at the ready for help.

PTSD treatment requires special care. Overall, models related to exposure, cognitive processing therapy (CPT), eye movement and desensitization and reprocessing (EMDR), and CBT have been effective. There is also some evidence that memory reappraisal techniques may have benefit. Older adult therapy has a long history of reminiscence therapies. Of interest, Cook, McCarthy, and Thorp (2017) outlined the importance of older adults with PTSD getting treatment with the usage of exposure therapy. In their review, evidence-based practices for PTSD indicated that prolonged exposure is safe, acceptable, and efficacious with cognitively intact older adults. Given the rapid growth of the aging population and the potential impact on mental health practices, it is important to provide older adults with this form of treatment.

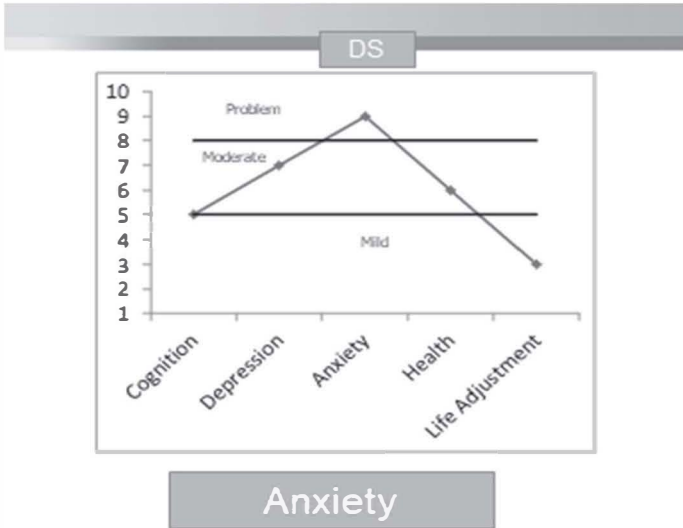
Post Script

Perhaps a unique factor for stress/anxiety in older adults is how it transfers to the phenomenology of worry; this pervades life and health issues. Worry and anxiety, then, almost become a substrate of human pathology and concern. One might say there are two tracks for a therapy for anxiety at later life. One is that therapy demands of the person a change in being – to accept and surrender, not fight and challenge. As noted above, change requires a rewiring of the habit system and a reduction in the anxious brain.

Or, therapy may require a battle. Some time ago, Lazarus and Folkman (1984) described the process of coping with stress as constantly changing cognitive and behavioral efforts to manage specific demands that are appraised as taxing or exceeding the resources of the person. Stress is of course anxiety in a violent form. It is stress that matters, as well as the process used by the person to handle it. This occurs simply when the demands of the situation outweigh the resources of the person. This can be the cognitive, emotional, and behavioral response to the stressor. It is then a transactional process that is applied, including the appraisal of the problem, the physiological arousal, and the coping style applied. It is not surrender.

The therapy needs a director who is aware of the person, the problem, and what is entailed in change. A series of therapies that involve coping and stress reduction have been effective. These include stress inoculation therapy, stress reduction therapy, and a grouping of coping strategies using a sequence and process format. These ideas largely involve a process for stress confrontation migrating from the stimulus to the response and post response (see Hyer, 2014).

Case 2: DS



Patient: DS

DOB: 1949

CHIEF COMPLAINT: Evaluation for memory problems.

Screen

Anxiety

GAD-9 = 18

SAST = 22

BDI-II = 15

PHQ-9=6

MBMD-anxious and depressed

PAI- anxiety

Health

Set stage for treatment is SSRI/SNRI S/P brain tumor

Migraines (monthly)

Chronic Diseases: 5 and > 10 medications

Health Rating: 5/10

ESS = 3 (normal)

Pain: 3/10

Cigarettes: Quit 10 yrs ago

Alcohol: No
 Exercise: none

Depression:

BDI-II = 11
 GDS-SF = 5
 PHQ-9 = 6
 MBMD and PAI are normal

Life Situation:

Supportive marriage
 Lower SES and education
 Inactive and adequate functioning
 Adherent to medical regimen

HISTORY OF PRESENT ILLNESS: The patient indicated that she had been having memory problems since the removal of a brain tumor along with some depression since the death of her father two years ago. She was referred by her neurologist. She complains also of worry or stress in her job. She notes that she has always been a worrier.

RELEVANT DEVELOPMENTAL HISTORY: The patient was born in the South and moved around with her family due to her father's job. She is the youngest of three children with two older brothers, one of whom has a mental illness. She graduated from high school and has some college. She has been married twice, about 10 years to her current husband who is supportive. She has two children by her first husband. She reported that she has a considerable amount of stress (worry) in her life including from the recent death of her father and work. She is currently on medical leave from her job. She lives with her husband. No indication of IADL problems was noted.

RELEVANT MEDICAL HISTORY: The patient had a brain tumor removed in 2002 and has been seeing a neurologist since. She has migraine headaches about once a month. Her medications include Neurontin, Cymbalta, pravastatin, Synthroid, Klonopin (sleep), and something for migraines. She does not drink alcohol and quit smoking her pack-a-day cigarette habit 10 years ago. She is able to sleep well with medication. She does not exercise and reported a recent weight gain in the last few months. She reported memory problems with lapses around the time of her early 20s, and she described herself as probably depressed.

MENTAL STATUS: The patient is a 68-year-old Caucasian female who drove herself to the appointment. She was animated but also cried during the interview. She indicates that she is not as happy as she used to be. She was emotionally labile and is very anxious with worry. Her

anxiety seems to have gotten worse in the last year and she notes that she worries like never before. Although she reported memory problems for earlier memories, she did not report any memory problems in her current functioning. There was no evidence of word finding issues. She functions well and is savvy about current events. There was no evidence of perceptual anomalies or delusional thinking. Her judgement and insight appear to be adequate.

PREMORBID FUNCTIONING and OVERALL COGNITIVE FUNCTIONING: Her MoCA was normal (28/30; 4/5 on memory). The patient had a premorbid intelligence score placing her in the low average area intellectually based on the Barona Index of Intelligence. Her WAIS-IV showed a low average IQ. She had a low and minimal scatter on the WAIS-IV subscales. She had a 9 on the Vocabulary subscale and 9 on the Information subscale. She scored an overall RBANS index of 86 (18%). She was lower on language and visuospatial areas. Attention was low average. Her responses were variable, and overall her current functioning was reasonably commensurate with her intelligence. She can do all ADLs and IADLs. Memory is average.

EMOTIONAL ASSESSMENT: Emotional self-report scales indicated anxiety and mild depression. She scored 11 on the BDI-II, 5 on the GDS-SF, and 6 on the PHQ-9, all mild depression. She scored as having a syndromal pattern on the MBMD for anxiety. Her PAI also showed excessive worry. Both anxiety measures were high: SAST (22) and GAD-7 (18). She also has a respectful personality indicating compliance and suppressed feelings of irritability. This results in worry.

Watch and Wait: Patient presents with primary problems of worry and secondary ones of managing her health. She also has mild depression but her life situation seems stable and she has sufficient cognitive skills for day-to-day adjustment. The patient is taking no psychiatric medications and has minimal socialization in spite of a supportive environment. She has also been inactive. Sleep is not an issue and she is compliant with her health regimen.

Her Watch and Wait markers were set. She was provided considerable education on her anxiety and brain processes. She was also taught HRV biofeedback targeting anxiety. Her coherence rates are in the positive direction. She monitors her fear-based cognitions (regarding health and being alone) and uses worry control techniques. She is also in a CBT group of older adults for reinforcement and socialization. A Memory Group was also recommended. She has attended.

Brain tumor is no longer a problem. Coordinate with neurologist. Monitor migraine headaches. Initiate an anxiolytic med. She will be encouraged to exercise. Weight is stable.

Checklist

Discernment: X (motivated and compliant)

Core markers

Validation X

Psychoeducation of Model X

Assessment X

Alliance X

Monitoring X

Case formulation X

Problem List

1. Anxiety: Contact PCP for SSRI with anxiolytic effects, monitor anxiety daily with SUDS, CBT group weekly, teach relaxation, initiate exercise in ALF, counseling focusing on problem solving, coping and assertive needs, HRV biofeedback. Suggest Memory Group. This should handle depression.
2. Health: Interface with PCP, help monitor HTN and A1c and migraines, check with family on medications, assure that medical regimen is followed.
3. Life Adjustment: Caregiving education and training, behavioral activation

Self or other monitoring: Mini targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:

Imagery exercise for counter-experience: X

Compassion training:

Communication and Assertiveness training: X

Behavioral experiments: X

Emotional exposure:

Homework: X

Longer Therapy processes:

CBT: Use of 3-6 column evidence record

ACT: use of acceptance interventions

Lifestyles:

- Practice happiness
- Practice Spirituality/Religion
- Socialize: Less decline and live longer
- Develop resilience
- Exercise: Even housework
- Diet
- Somatic softening: Meditate and Mindfulness
- Values and Leisure

Follow-up: Re-evaluate every 6 months

Anxiety

GAD-7 = 5

SAST = 23

BDI-II = 4

PHQ-9 = 6

MBMD-non-anxious

PAI- no anxiety

Health

Chronic Diseases: 5 and > 7 medications

Health Rating: 5/10

ESS = 3

Pain: 3/10

Alcohol: No

Exercise: 5 days/week

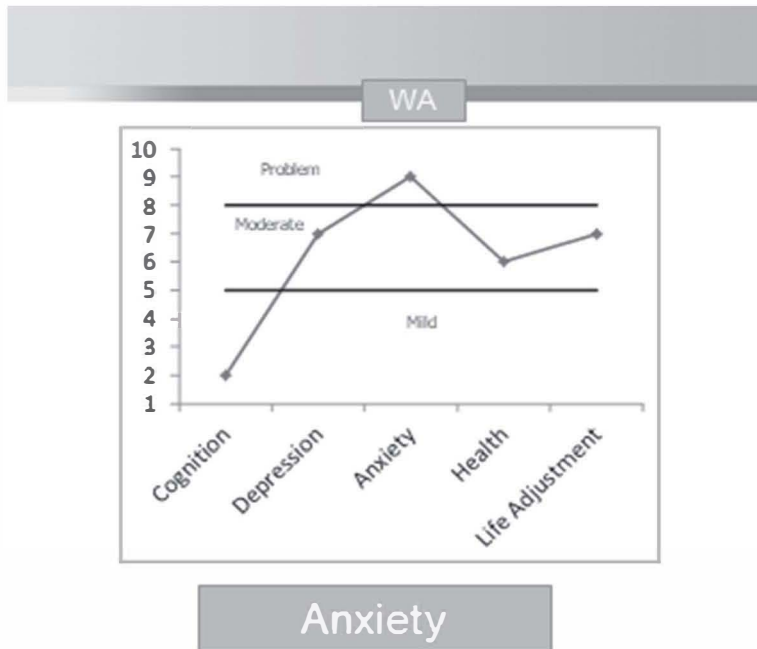
Depression: Improved (BDI-II = 8)

Cognition: No change (MoCA = 26/30)

Life Adjustment: No change

Follow up domain profile is similar to the original one. Key target are anxiety and to some extent depression and health.

Case 3: WA



PATIENT: WA

DOB: 1945

CHIEF COMPLAINT: Evaluation for anxiety and depression.

Screen

Anxiety

GAD-9= 13

SAST = 45

MINI-depressed and anxious

MBMD-anxious and depressed

Depression

BDI-2 = 19

PHQ-9 =17

Mood Scale =4

PAI and MBMD depressed

Health

Cancer in remission

Chronic Diseases: 3 and > 10 medications

Health Rating: 3/10

ESS = 17

Pain: 5/10

Cigarettes: No

Alcohol: 3-4 drinks a week

Exercise: occasional walking

Cognition

Normal (see below)

Life Adjustment

Lives alone and has support

HISTORY OF PRESENT ILLNESS: The patient indicated that she was having difficulty with attention and fearfulness and was referred by her primary care physician for an evaluation. This has been ongoing since childhood.

RELEVANT DEVELOPMENTAL HISTORY: The patient was born and raised in Georgia. She had problems with self-esteem most of her childhood. Her mother died when she was 10 years old, and she began to experience fears about being abandoned. She was able to graduate from high school and received a BA. She worked right after college as a travel agent for over 30 years. She has been married to her current husband for 40 years, and they have no children. She is currently retired but works part time at a florist's. The patient reported that she feels stress in her marriage and experienced depression when her father died ten years ago. She says that she worries all the time about her life and marriage.

RELEVANT MEDICAL HISTORY: Patient indicated that she is on several medications including Celebrex, Imitrex (for migraines), naproxen for pain, Zoloft, and Ativan (for sleep). She has arthritis and occasional migraines. She is also a cancer survivor from age 40, and this is in remission. She indicated that stressors in her life are her marriage, her health, and her self-esteem. She drinks about 3 to 4 drinks a week (different days) but does not use tobacco. She indicated that sleep is a mild problem for her, but she does sleep 7 hours a night. She is tired during the day and does nap at times. She walks occasionally when she is not in pain. She has been in therapy previously and has only been on Zoloft for about two years.

MENTAL STATUS: Patient is a 72-year-old African American female who was quite easy to talk with. She was upbeat, positive, laughed, and provided specific information to all questions asked. She was not overweight. There was no evidence of cognitive problems or word finding issues. She indicated that she is a worrier and on occasion can get depressed. That said, she was very upbeat and smiled. She would occasionally become very frustrated at herself when questions were missed on mental status testing. She demonstrates no perceptual anomalies or delusional thinking. She appears to show rather good insight into her situation and to assert good judgment.

PREMORBID FUNCTIONING and OVERALL COGNITIVE FUNCTIONING: The patient has a premorbid intelligence score placing her in the average range based on the Barona Index of Intelligence. She has a college education and had been a travel agent for > 30 years. Her WAIS-IV Vocabulary was 13. Her MoCA was 29/30, normal. Her RBANS was 114, 84%. All domains were in the normal or better ranges. As such, she has above average premorbid intelligence.

EMOTIONAL ASSESSMENT: Emotional self-report scales indicated considerable psychiatric problems. On the MINI she scored as having problems on depression and anxiety. She scored 17 on the PHQ-9 and 19 on the BDI-II, both depressed. Her Mood Score was 4, normal. She did score as largely anxious on the MBMD and PAI also. She scored as anxious on the anxiety scale of the SAST (25). She also had a high anxiety score on the GAD-7 (13). She is concerned about her cognition (MBMD). Her personality was cooperative and inhibited but shows no prototype patterns. She is then one who is likely to comply and has good self-resources for her therapy, and to be to herself. Sleep and pain are also issues.

Psychiatric Indications	AA	24	103		ANXIETY-TENSION
	BB	9	71		DEPRESSION
	CC	21	83		COGNITIVE DYSFUNCTION
	DD	9	60		EMOTIONAL LABILITY
	EE	2	5		GUARDEDNESS
Coping Styles	1	2	20		INTROVERSIVE
	2A	3	65		INHIBITED
	2B	2	20		DEJECTED
	3	8	65		COOPERATIVE
	4	9	55		SOCIABLE
	5	4	30		CONFIDENT
	6A	2	20		NONCONFORMING
	6B	2	20		FORCEFUL
	7	17	50		RESPECTFUL
	8A	1	20		OPPOSITIONAL
	8B	2	20		DENIGRATED

Watch and Wait: This woman has primary problems of anxiety and some depression. Her health is stable and her life adjustment, despite a shaky marriage, is worth focusing on but stable. Her cognition is stable and normal. She is a worrier and has reasons (marriage) to be sad. She takes Zoloft and Ativan (for sleep).

Checklist

Discernment: X (motivated and compliant)

Core Markers:

Validate Problem X

Psychoeducation of Model X

Assessment X

Alliance X

Monitoring X

Case formulation X

Domains

1. **Anxiety:** Targets for her include what to do to reduce her anxiety and stabilize her life. She is now in counseling. She was asked to cut out alcohol for a few months; to walk a half hour a day for five day/week; and to maximize her out-of-the-house socialization at church and with a friend. She was introduced to worry control successfully. She was trained on slow breathing and mindfulness. She is to be educated on and to practice sleep hygiene. Her medication needs changing and interfacing with her PCP is underway. She was taught how to be assertive.
2. **Depression:** Medication and behavioral activation. She was also asked to monitor her marriage – what she enjoys and what the problems are. This would allow her to develop a perspective on her marriage and provide data for future interventions. Grief counseling (father) was also suggested.

Self or other monitoring: Mini-Targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks:

Managing situational contingencies: X

Role playing and modeling: X

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:
 Absorption training:
 Compassion training: X
 Communication and Assertiveness training: X
 Behavioral experiments: X
 Emotional exposure:
 Homework: X

Longer Therapy processes:

CBT: Use of 3-6 column evidence record

ACT: use of acceptance interventions

PST: improve on EF skills

Worry Control

Lifestyles:

- Practice happiness
- Practice Spirituality/Religion
- Socialize
- Altruism (increases dopamine and endorphins) and volunteer
- Practice sleep hygiene
- Exercise
- Somatic softening: Meditate and Mindfulness
- Values and Leisure
- Increase cognition

Follow-up

Anxiety

GAD-7 = 5

SAST = 25

MINI-no depressed nor anxious states

MBMD-no anxious and depressed PR range

Depression

BDI-2 = 6

PHQ-9 = 8

Mood Scale = 2

Health

Chronic Diseases: 3 and > 10 medications

Health Rating: 5/10

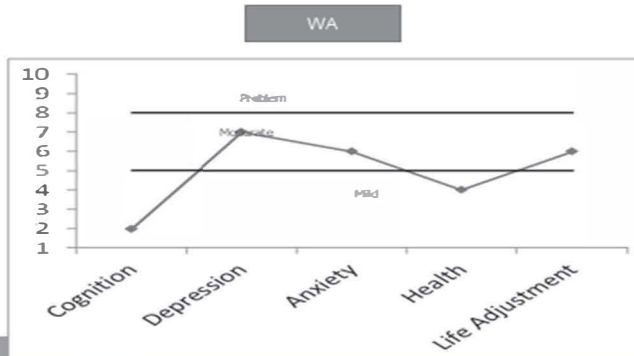
ESS = 7

Pain: 3/10

Exercise: improved walking

Relapse: N/A

This is an updated domain profile. She still has some problems with depression and anxiety. Her life adjustment is stable as is her health. Her cognition is quite good.



Conclusion

Anxiety is the gift that keeps on giving. It is sneaky and is often the causative agent for medical and cognitive problems. It reduces quality of life. It is insidious and almost certainly a predictor for dementia. What GAD is at later life is difficult to absolutely know and define (like so many psychiatric entities) but is apparent when present.

The Watch and Wait model is an effort to calm this angst. It is a slow unfolding of semi-precision care for worried unwell older adults. The National Institute of Mental Health argues for specific mechanisms of psychological disorders in a highly focused fashion that can be disseminated at a population level as part of a stepped care approach that can be administered in a clinical setting. They are referring to panic and avoidance. Regardless, this is Watch and Wait.

References

- Acierno, R., Lawyer, S. R., Rheingold, A., Kilpatrick, D. G., Resnick, H. S., & Saunders, B. E. (2007). Current psychopathology in previously assaulted older adults. *Journal of Interpersonal Violence, 22*(2), 250-258.
- Alexopoulos, G. S., Raue, P. J., Kanellopoulos, D., Mackin, S., & Arean, P. A. (2008). Problem solving therapy for the depression-executive dysfunction syndrome of late life. *International Journal of Geriatric Psychiatry, 23*(8), 782-788. doi:10.1002/gps.1988
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- Andreescu, C., Sheu, L. K., Tudorascu, D., Gross, J. J., Walker, S., Banihashemi, L., & Aizenstein, H. (2015). Emotion reactivity and regulation in late-life generalized anxiety disorder: Functional connectivity at baseline and post-treatment. *The American Journal of Geriatric Psychiatry, 23*(2), 200-214. doi: <http://doi.org/10.1016/j.jagp.2014.05.003>
- Ayers, C. R., Sorrell, J. T., Thorp, S. R., & Wetherell, J. L. (2007). Evidence-based psychological treatments for late-life anxiety. *Psychology and Aging, 22*(1), 8-17. doi: <http://dx.doi.org/10.1037/0882-7974.22.1.8>
- Bartels, S. J., Dums, A. R., Oxman, T. E., Schneide, L. S., Arean, P. A., Alexopoulos, G. S., & Jeste, D. V. (2002). Evidence-based practices in geriatric mental health care: An overview of systematic reviews and meta-analyses. *Psychiatric Services, 53*(11), 1419-1431. Retrieved from <http://dx.doi.org.medlib-proxy.mercer.edu/10.1176/appi.ps.53.11.1419>
- Beaudreau, S.A. & O'Hara, R. (2008). Late-life anxiety and cognitive impairment: A review. *American Journal of Geriatric Psychiatry, 16*(10), 790-803. Retrieved from <http://dx.doi.org.medlib-proxy.mercer.edu/10.1097/JGP.0b013e31817945c3>
- Böttche, M., Kuwert, P., & Knaevelsrud, C. (2012). Posttraumatic stress disorder in older adults: An overview of characteristics and treatment approaches. *International Journal of Geriatric Psychiatry, 27*(3), 230-239. doi:10.1002/gps.2725
- Brenes, G. A., Danhauer, S. C., Lyles, M. F., Anderson, A., & Miller, M. E. (2017). Long-Term Effects of Telephone-Delivered Psychotherapy for Late-Life GAD. *The American Journal of Geriatric Psychiatry, 25*, 1249-1257. doi:10.1016/j.jagp.2017.05.013

- Bryant, C., Jackson, H., & Ames, D. (2008). The prevalence of anxiety in older adults: Methodological issues and a review of literature. *Journal of Affective Disorders, 109*, 233-250. doi: 10.1016/j.jad.2007.11.008
- Byers, A. L., Yaffe, K., Covinsky, K. E., Friedman, M. B., & Bruce, M. L. (2010). High occurrence of mood and anxiety disorders among older adults. *Archives of General Psychiatry, 67*(5), 489-496. doi:10.1001/archgenpsychiatry.2010.35
- Cassidy, K. & Rector, N. A. (2008). The silent geriatric giant: Anxiety disorders in late life. *Geriatrics & Aging, 11*(3), 150-156. Retrieved from <https://www.healthplexus.net/files/content/2008/April/1103anxiety.pdf>
- Cook, J. M., McCarthy, E., & Thorp, S. R. (2017). Older Adults with PTSD: Brief State of Research and Evidence-Based Psychotherapy Case Illustration. *The American Journal of Geriatric Psychiatry, 25*, 522-530. doi:10.1016/j.jagp.2016.12.016
- Diefenbach, G. J., Bragdon, L. B., & Blank, K. (2014). Geriatric anxiety inventory: Factor structure and associations with cognitive status. *American Journal of Geriatric Psychiatry, 22*(12), 1418-1426. Retrieved from <https://www-clinicalkey-com.medlib-proxy.mercer.edu/#!/content/playContent/1-s2.0-S1064748113002327?returnurl=null&referrer=null>
- Franco, M. (2007). Posttraumatic Stress Disorder and Older Women. *Journal of Women & Aging, 19*(1/2), 103-117. doi:10.1300/J074v19n01_07
- Frisch, M. B. (1998). Quality of life therapy and assessment of in health care. *Clinical Psychology: Science and Practice, 5*(1), 19-40. doi: 10.1111/j.1468-2850.1998.tb00132.x
- Goncalves, D. C. & Byrne, G. J. (2012). Interventions for generalized anxiety disorder in older adults: Systematic review and meta-analysis. *Journal of Anxiety Disorders, 26*, 1-11. doi: 10.1016/j.janxdis.2001.08.010
- Gulpers, B., Ramakers, I., Hamel, R., Kohler, S., Oude Voshaar, R., & Verhey, F. (2016). Anxiety as a Predictor for Cognitive Decline and Dementia: A Systematic Review and Meta-Analysis. *American Journal of Geriatric Psychiatry, 24*(10), 823-842. Doi: <https://doi.org/10.1016/j.jagp.2016.05.015>
- Gurian, B. S., & Minor, J. H. (1991). *Anxiety in the elderly: treatment and research. Clinical presentation of anxiety in the elderly*. New York: Springer.
- Hall, J., Kellett, S., Berrios, R., Bains, M. K., & Scott, S. (2016). Efficacy of Cognitive Behavioral Therapy for Generalized Anxiety Disorder in

- Older Adults: Systematic Review, Meta-Analysis, and Meta-Regression. *The American Journal of Geriatric Psychiatry*, 24(11), 1063-1073. doi: 10.1016/j.jagp.2016.06.006
- Herring, M. P., O'Connor, P. J., Dishman, R. K. (2010). The effect of exercise training on anxiety symptoms among patients: A systematic review. *Archives of Internal Medicine*, 170(4), 321-331. doi: 10.1001/archinternmed.2009.530
- Hyer, L. (2014). *Psychological treatment of older adults: A holistic model*. New York: Springer.
- Hyer, L., Kramer, D. & Sohnle, S. (2004). CBT with older people: Alterations and the value of the therapeutic alliance. *Psychotherapy: Theory, Research, Practice, Training*, 41(3), 276-291. Retrieved from <http://dx.doi.org.medlib-proxy.mercer.edu/10.1037/0033-3204.41.3.276>
- Iglesias, C. P., Manca, A., & Torgerson, D. J. (2008). The health-related quality of life and cost implications of falls in elderly women. *Osteoporosis International*, 20(6), 869-878. doi: 10.1007/s00198-008-0753-5
- Jessen, F., Wiese, B., Bachmann, C., Eifflaender-Gorfer, S., Haller, F., Kölsch, H., . . . & Bickel, H. (2010). Prediction of dementia by subjective memory impairment. *Archives of General Psychiatry*, 67(4), 414-422. doi: <http://dx.doi.org/10.1001/archgenpsychiatry.2010.30>
- Katz, I. R., Reynolds, C. F., Alexopoulos, G. S., & Hackett, D. (2002). Venlafaxine ER as a treatment for generalized anxiety disorder in older adults: Pooled analysis of five randomized placebo-controlled trials. *Journal of the American Geriatrics Society*, 50(1), 18-25. doi: 10.1046/j.1532-5415.2002.50003.x
- Kawachi, I., Sparrow, D., Vokonas, P. S., & Weiss, S. T. (1994). Symptoms of anxiety and risk of coronary heart disease: The normative aging study. *Circulation*, 90(5), 2225-2229. doi: <https://doi.org/10.1161/01.CIR.90.5.2225>
- Khouzam, H. R. (2008). Posttraumatic stress disorder and aging. *Postgraduate Medicine*, 120(3), 122-129. doi: 10.3810/pgm.2008.09.1913
- Kremen, W. S., Koenen, K. C., Afari, N., & Lyons, M. J. (2012). Twin studies of posttraumatic stress disorder: Differentiating vulnerability factors from sequelae. *Neuropharmacology*, 62(2), 647-653. doi:10.1016/j.neuropharm.2011.03.012
- Kvaal, K., Ulstein, I., Nordhus, I. H., & Engedal, K. (2005). The Spielberger State-Trait Anxiety Inventory (STAI): the state scale in

- detecting mental disorders in geriatric patients. *International Journal of Geriatric Psychiatry*, 20(7), 629–634. doi:10.1002/gps.1330
- Labinsky, E., Blair, W., & Yehuda, R. (2006). Longitudinal Assessment of Dissociation in Holocaust Survivors with and without PTSD and Nonexposed Aged Jewish Adults. *Annals of The New York Academy of Sciences*, 1071(1), 459-462. doi:10.1196/annals.1364.042
- Ladson, D., & Bienenfeld, D. (2007). Delayed reaction to trauma in an aging woman. *Psychiatry*, 4(6), 46-50.
- Ladouceur, R., & Gosselin, P. (2000). Experimental manipulation of intolerance of uncertainty: a study of a theoretical model of worry. *Behaviour Research & Therapy*, 38(9), 933-941. doi: 10.1016/S0005-7967(99)00133-3
- Lauderdale, S. A., & Sheikh, J. I. (2003). Anxiety disorders in older adults. *Clinics in Geriatric Medicine*, 19, 721-741. doi: 10.1016/S0749-0690(03)00047-8
- Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. New York: Springer Pub. Co.
- Lohr, J. B., Palmer, B. W., Eidt, C. A., Aailaboyina, S., Mausbach, B. T., Wolkowitz, O. M., & ... Jeste, D. V. (2015). Regular Research Article: Is Post-Traumatic Stress Disorder Associated with Premature Senescence? A Review of the Literature. *The American Journal of Geriatric Psychiatry*, 23, 709-725. doi:10.1016/j.jagp.2015.04.001
- Mah, L., Binns, M. A., & Steffens, D. C. (2015). Anxiety symptoms in amnesic mild cognitive impairment are associated with medial temporal atrophy and predict conversion to Alzheimer disease. *The American Journal of Geriatric Psychiatry*, 23(5), 466-476. doi:10.1016/j.jagp.2014.10.005
- Mandavia, D. & Newton, K. (1998). Geriatric trauma. *Emergency Medicine Clinics of North America*, 16(1), 257-274. Retrieved from [http://dx.doi.org.medlib-proxy.mercer.edu/10.1016/S0733-8627\(05\)70358-8](http://dx.doi.org.medlib-proxy.mercer.edu/10.1016/S0733-8627(05)70358-8)
- McCartney, J. R. & Severson, K. (1997). Sexual violence, post-traumatic stress disorder and dementia. *Journal of the American Geriatrics Society*, 45(1), 76-78. doi: 10.1111/j.1532-5415.1997.tb00982.x
- Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2005). Preliminary evidence for an emotion dysregulation model of generalized anxiety disorder. *Behaviour Research and Therapy*, 43, 1281-1310. doi:10.1016/j.brat.2004.08.008
- Mohlman, J. & Gorman, J. M. (2005). The role of executive functioning in CBT: A pilot study with anxious older adults. *Behavior Research and*

- Therapy*, 43(4), 447-465. Retrieved from <http://dx.doi.org.medlib-proxy.mercer.edu/10.1016/j.brat.2004.03.007>
- Mota, N. P., Tsai, J., Sareen, J., Marx, B. P., Wisco, B. E., Harpaz-Rotem, L., & ... Pietrzak, R. H. (2016). High burden of subthreshold DSM-5 post-traumatic stress disorder in U.S. military veterans. *World Psychiatry: Official Journal of The World Psychiatric Association (WPA)*, 15(2), 185-186. doi:10.1002/wps.20313
- Newton, J. P. (2010). Elder abuse: An issue not to be ignored. *Gerontology*, 27(2), 83-84. doi: 10.1111/j.1741-2358.2010.00387.x
- Norris, F. H. (1992). Epidemiology of trauma: Frequency and impact of different potentially traumatic events on different demographic groups. *Journal of Consulting and Clinical Psychology*, 30(3), 409-418. doi: <http://dx.doi.org/10.1037/0022-006X.60.3.409>
- Hara, R. ●. (2012). The reciprocal relationship of neurocognitive and neuropsychiatric function in late life. *The American Journal of Geriatric Psychiatry*, 20(12), 1001-1005. Retrieved from <https://www-clinicalkey-com.medlib-proxy.mercer.edu/#!/content/playContent/1-s2.0-S1064748112620119?returnurl=null&referrer=null>
- Papageorgiou, C., & Wells, A. (1998). Effects of attention training on hypochondriasis: A brief case series. *Psychological Medicine*, 28(1), 193-200. doi:10.1017/S0033291797005825
- Patemiti, S., Dufouil, C., & Alperovitch, A. (2002). Long-term benzodiazepine use and cognitive decline in the elderly: The epidemiology of vascular aging study. *Journal of Clinical Psychopharmacology*, 22(3), 285-293. Retrieved from http://journals.lww.com.medlib-proxy.mercer.edu/psychopharmacology/Abstract/2002/06000/Long_Term_Benzodiazepine_Use_and_Cognitive_Decline.9.aspx
- Pietrzak, R. H., Southwick, S. M., Tracy, M., Galea, S., & Norris, F. H. (2012). Posttraumatic stress disorder, depression, and perceived needs for psychological care in older persons affected by Hurricane Ike. *Journal of Affective Disorders*, 138(1-2), 96-103. doi:10.1016/j.jad.2011.12.018
- Pinquart, M., Duberstein, P., & Lyness, J. (2006). Treatment for later-life depressive conditions: A meta-analytic comparison of pharmacotherapy and psychotherapy. *American Journal of Psychiatry*, 163(9), 1493-1501. Retrieved from <http://proxygsu-mer3.galileo.usg.edu/login?url=http://search.proquest.com/docview/220503490?accountid=12383>
- Pinquart, M., Duberstein, P., & Lyness, J. (2007). Effects of psychotherapy and other behavioral interventions on clinically depressed older adults:

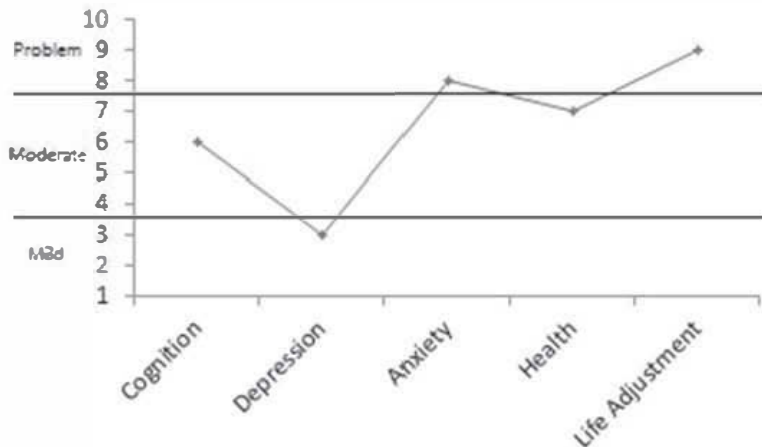
- A meta-analysis. *Aging and Mental Health*, 11(6), 645-657. doi: <http://dx.doi.org/10.1080/13607860701529635>
- Porensky, E. K., Dew, M. A., Karp, J. F., Skidmore, E., Rollman, B. L., Shear, M. K., & Lenze, E. J. (2009). The burden of late-life generalized anxiety disorder: Effects on disability, health-related quality of life, and healthcare utilization. *The American Journal of Geriatric Psychiatry*, 17(6), 473-482. Retrieved from <http://dx.doi.org.medlib-proxy.mercer.edu/10.1097/JGP.0b013e31819b87b2>
- Qureshi, S. U., Long, M. E., Bradshaw, M. R., Pyne, J. M., Magruder, K. M., Kimbrell, T., & ... Kunik, M. E. (2011). Does PTSD Impair Cognition Beyond the Effect of Trauma?. *The Journal of Neuropsychiatry And Clinical Neurosciences: Official Journal of The American Neuropsychiatric Association*, 23(1), 16-28. doi:10.1176/jnp.23.1.jnp16
- Samuelson, K. W. (2011). Post-traumatic stress disorder and declarative memory functioning: a review. *Dialogues in Clinical Neuroscience*, 13(3), 346-351.
- Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2012). *Mindfulness-based cognitive therapy for depression*. New York: The Guilford Press.
- Seligman, M., Rashid, T., & Parks, A. C. (2006). Positive psychotherapy. *American Psychologist*, 61(8), 774-788. doi: <http://dx.doi.org/10.1037/0003-066X.61.8.774>
- Sheikh, J. I. & Cassidy, E. L. (2000). Treatment of anxiety disorders in the elderly: Issues and strategies. *Journal of Anxiety Disorders*, 14(2), 173-190. doi: 10.1016/S0887-6185(99)00047-X
- Spielberger (1983). *Manual for the state-trait anxiety inventory STAI (form Y)* ("self-evaluation questionnaire"). Palo Alto, CA: Consulting Psychologists Press.
- Spitzer, R. L., Kroenke, K., Williams, J. B., & Lowe, B. (2006). A brief measure for assessing generalized anxiety disorder: The GAD-7. *Archives of Internal Medicine*, 166(10), 1092-1097. doi:10.1001/archinte.166.10.1092
- Starkstein, S. E., Jorge, R., Petracca, G., & Robinson, R. G. (2007). The construct of generalized anxiety disorder in Alzheimer disease. *The American Journal of Geriatric Psychiatry*, 15(1), 42-49. Retrieved from <http://dx.doi.org.medlib-proxy.mercer.edu/10.1097/01.JGP.0000229664.11306.b9>

- Therrien, Z. & Hunsley, J. (2012). Assessment of anxiety in older adults: A systematic review of commonly used measures. *Aging and Mental Health, 16*(1), 1-16. doi: 10.1080/13607863.2011.602960
- Thorp, S. R., Ayers, C. R., Nuevo, R., Stoddard, J. A., Sorrell, J. T., & Wetherell, J. L. (2009). Meta-analysis comparing different behavioral treatments for late-life anxiety. *The American Journal of Geriatric Psychiatry, 17*(2), 105-115. doi:10.1097/JGP.0b013e31818b3f7e
- Van Minnen, A., Zoellner, L. A., Hamed, M. S., & Mills, K. (2015). Changes in comorbid conditions after prolonged exposure for PTSD: A literature review. *Current Psychiatry Reports, 17*(3), 1-17. doi: 10.1007/s11920-015-0549-1
- van Zelst, W. H., de Beurs, E., Beekman, A. F., Deeg, D. H., & van Dyck, R. (2003). Prevalence and risk factors of posttraumatic stress disorder in older adults. *Psychotherapy and Psychosomatics, 72*(6), 333-342. doi:10.1159/000073030
- Vetter, N. J. & Ford, D. (1989). Anxiety and depression scores in elderly fallers. *Geriatric Psychiatry, 4*(3), 159-163. doi: 10.1002/gps.930040307
- Vink, D., Aartsen, M. J., & Schoevers, R. A. (2008). Risk factors for anxiety and depression in the elderly: A review. *Journal of Affective Disorders, 106*(1-2), 29-44. doi: 10.1016/j.jad.2007.06.005
- Weathers, F. W., Keane, T. M., & Davidson, J. R. T., (2001). Clinician-administered PTSD scale: A review of the first ten years of research. *Depression and Anxiety, 13*(3), 132-156. doi: 10.1002/da.1029
- Wells, A., & King, P. (2006). Metacognitive therapy for generalized anxiety disorder: An open trial. *Journal of Behavior Therapy and Experimental Psychiatry, 37*, 206-212. doi:10.1016/j.jbtep.2005.07.002
- Wetherell, J. L. & Gatz, M. (2005). The beck anxiety inventory in older adults with generalized anxiety disorder. *Journal of Psychopathology and Behavioral Assessment, 27*(1), 17-24. doi: 10.1007/s10862-005-3261-3
- Wetherell, J. L., Liu, L., Patterson, T. L., Afari, N., Ayers, C. R., Thorp, S. R., ... Petkus, A. J. (2011). Acceptance and commitment therapy for generalized anxiety disorder in older adults: A preliminary report. *Behavior Therapy, 42*(1), 127-134. Retrieved from <http://dx.doi.org.medlib-proxy.mercer.edu/10.1016/j.beth.2010.07.002>
- Wolitzky-Taylor, K. B., Castriotta, N., Lenze, E. J., Stanley, M. A., & Craske, M. G. (2010). Anxiety disorders in older adults: A comprehensive review. *Depression and Anxiety, 27*(2), 190-211. doi: 10.1002/da.20653

CHAPTER 10

LIFE ADJUSTMENT

Again, we pick up from chapter 5 on the case.



Case MS

DOB: 02/27/1930

CHIEF COMPLAINT: Evaluation for memory problems.

Screen Problems

Anxiety

MMI: Positive for GAD

GAD-7 = 6

BAI = 13

MMQ: Anxiety/Tension

Life Adjustment

SES/Education: Problem

Function: IADLs deficient

Environment: Reasonably safe

Relationship: Loner but has her daughter

Meaning in life: Few activities and no leisure activities

Caregiving: Daughter available

HISTORY OF PRESENT ILLNESS: The patient and daughter were interviewed. The patient was driven to this appointment by her daughter. The indications are that roughly 3 years ago after a heart procedure, and then other procedures including stent and pacemaker, the patient started having difficulty with her memory and some behavioral issues. Despite this, the patient does not see herself as having a problem with cognition. She lives alone locally and is on Social Security. She has been divorced for a number of years. She has some problems with IADLs.

RELEVANT DEVELOPMENTAL HISTORY: The patient is a native of Oklahoma. She indicated that she was the oldest of 3 children. Her father was a geologist. She indicated that growing up was difficult in that her father was quite strict. She graduated from high school and went to a local university for approximately a year. After high school she was quickly married to a person who was then in the Air Force. She was married for 12 years and had 3 children. She was divorced and had another child out of wedlock, and then married again at age 38 for 3 years, and divorced that person. She has been divorced, therefore, for a substantial number of years. She herself migrated at some point to Georgia, apparently after her second divorce, and worked at the Air Force Base as a secretary for 23 years. She was fired from this position because of a threatening letter to command. She has not worked there for over 20 years, although she relates that she has done some minimal temp work. She is currently getting Social Security. She has problems with making ends meet.

MEDICAL HISTORY: The patient's medical records indicate that she has a pacemaker and stent. She also has hypertension and thyroid problems. Her surgical history includes these as well as a thyroid operation, tonsillectomy, and cataract removal. She is currently on Eliquis, aspirin, Zolof, HTN and other medications. She does not drink alcohol or use tobacco. She indicated that roughly a month ago she had an accident in the house injuring her leg, causing her difficulties to the present. She indicates that she sleeps okay. She is also a member of a local church. She had one psychiatric hospitalization in 1958, and apparently has not

received any treatments since that time. She does all of her ADLs but has problems with IADLs. She does not drive. She does not do her finances. She also has someone take her shopping and someone to administer her medications.

MENTAL STATUS: The patient is an 84-year-old Caucasian female who was driven to this appointment. She entered in a wheelchair but was able to walk to the office. She clearly has difficulty with some pain in one of her legs. She smiled and answered questions with some specificity, but did have difficulty hearing on occasion. Questions had to be repeated. She indicated that she is not depressed but is increasingly anxious. There was no evidence of perceptual anomalies or delusionary thinking. Her memory for dates was suspect, and she had to check out timelines and questions regarding her life history. She was however able to provide some information with specificity. She is aware that she needs help in her home.

Watch and Wait Summary: This is an 84 y/o Caucasian female who was referred for an evaluation for her cognitive status. She lives alone and has had problems with attention and memory for about two years. She has a caregiver daughter who drives and takes care of her finances, medications, and shopping. She can do all ADLs but not all IADLs. She has been alone for about 20 years after being fired in the 1990s. She receives SS disability. Money and home adjustment are issues.

Cognitively she is average premorbidly and has problems now. Her MoCA was 22/30 with 1/5 on memory and poor attention. Very brief testing revealed some problems: RBANS was low normal (90), and new learning and memory were below normal. Her executive functioning was also lower (Trails B and WCS). She is average in language and on visuospatial tasks. She can read adequately. She makes criteria for MCI but is able to function in virtually all areas with some minor help from her daughter.

Emotionally she scored as very defensive. She relates to anxiety only. She has a personality pattern of a confident and social as well as respectful pattern. She marches to her own drummer. She is mildly concerned about her thinking. She scores high on poor compliance. In general, she appears to be moody and irritable but admits to none of this. Sleep is not an issue. Pain is not an issue.

Psychiatric Indications	AA	3	55				ANXIETY-TENSION
	BB	0	15				DEPRESSION
	CC	9	70				COGNITIVE DYSFUNCTION
	DD	1	20				EMOTIONAL LABILITY
	EE	3	35				GUARDEDNESS
Coping Styles	1	0	15				INTROVERSIVE
	2A	1	50				INHIBITED
	2B	0	15				DEJECTED
	3	1	20				COOPERATIVE
	4	11	70				SOCIABLE
	5	10	64				CONFIDENT
	6A	0	15				NONCONFORMING
	6B	2	20				FORCEFUL
	7	20	67				RESPECTFUL
	8A	0	15				OPPOSITIONAL
	8B	0	15				DENIGRATED

She is, then, responding as one with many problems. She has life adjustment problems as well as anxiety and some health issues. Cognitively also she has some deficits, as she cannot do her IADLs. She is cared for by her daughter who sees after her needs. Her daughter is frustrated as her mother is very independent. Regardless, she requires some assistance and the areas of most concern are memory and handling new tasks. She qualifies for MCI (Mild Neurocognitive Disorder). She is at some risk for a dementia over time.

Treatment

She agreed to be seen bi-weekly with her daughter. She is not willing to attend treatment for biofeedback. A careful monitoring of her IADL habits was instituted along with a plan for her to become more involved in her church. Her daughter was also to monitor behaviors related to her day-to-day actions, especially those related to anxiety (statements and behaviors) and basic behaviors. This will serve as an entre for discussion of a possible move. She will also be retested in 6 months.

Checklist

Discernment: Not in place
Core markers

Validate Problem	
Psychoeducation of Model	X
Assessment	X
Alliance	
Monitoring	X
Case formulation	X

Problem List

1. Life Adjustment: She requires assistance in her living situation. This involves caregiving and independence management. Activate commitment to therapy, continue validation and establish an alliance, instruct daughter on caregiving, have caregiver monitor anxiety and assess problem behaviors, teach IADL skills, organize possible Senior Center membership as well as church involvement, set up CT system with caregiver
2. Anxiety: She worries excessively. Interact with PCP on anxiety med, monitor anxiety daily with SUDS, teach relaxation, initiate exercise 4x/week with PT.

3. Health: Titrate care with PCP, help monitor HTN and A1c, check with family on medications,
4. Depression: behavioral activation

Self or other monitoring: Targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:

Imagery exercise for counter-experience:

Compassion training:

Communication and Assertiveness training:

Behavioral experiments: X

Emotional exposure:

Homework: X

Longer Therapy processes:

CBT: Behavioral activation, monitoring

ACT: use of acceptance interventions

Lifestyles:

- Practice Spirituality/Religion
- Socialize: Less decline and live longer
- Develop resilience
- Exercise: Even housework
- Somatic softening: Meditate and Mindfulness
- Values and Leisure

Follow-up: Re-evaluate every 6 months

Life Adjustment: Caregiving and independence management improved. Daughter improved caregiving, monitored anxiety and assessed problem behaviors. Senior Center membership and church involvement was activated.

Anxiety

MINI: GAD in remission

GAD-7 = 4

BAI = 8

SAST =22

Health

- Chronic Diseases: 5
- Health Rating: 4
- ESS = 5
- Pain: 40
- BMI: 37
- Exercise: Less Stationary Behavior

Relapse Issues: Profile remains the same but with improvement. Patient shows slow progress in life adjustment and worry. Cognitive deficits prohibit further interventions. Caregiving is positive at present.

Life Adjustment

Measure	Healthy Intact	Mildly Problematic	Problematic or Syndromic
Description	Overall lifestyle is positive, patient engages in the core components of lifestyle without major problems	Lifestyle choices are likely to contribute to disorder or have begun to do so.	Lifestyle is compromising patient's mental and/or physical health. Self-care is lacking or not present.
Education and SES	College or professional certificate and continuing interests	HS education or extensive interests in reading/leisure	No HS education and little intellectual interests
FAQ (or AD-8)	≤10	11-18	≥19
Safe/supportive environment	Safe	Limited safety	Unsafe
Relationship Status	Married/in a Relationship	Single/divorced	Widowed in the last five years
Meaning in Life	Purpose in life/happy	Limited purpose or happiness	No purpose/unhappy
Stress (1-10)	0-3	4-7	>7

Background

First we set the stage. Douma, Steverink, Hutter, and Meijering (2017) used a participant-generated word method to obtain a lay view understanding of social wellbeing from 66 older adults of varied gender and age. They had different housing arrangements in the Netherlands. In a detailed empirically grounded knowledge of aspects categorized under domains of the participant views of social wellbeing, results found that 15 main domains were important to the participants: social life, activities, health, space and place, independence, mobility, financial situation,

societal criticism, political situation, personal characteristics, way of life, religion, healthcare and support, personal development, and others. The large variety of domains identified indicates that social wellbeing is a multi-dimensional concept. Social wellbeing is also contextual in nature because the participants related their social wellbeing not only to individual characteristics, for example health, but also to the characteristics of their social, physical, and political environments – that is their space and place and political situation.

Similarly, Kok, Aartsen, Deeg, and Huisman (2017) studied the prevalence of successful aging from a holistic definition. The implications of this study are that many older adults age successfully, but the character of successful functioning over time varies between indicators and the combination of successful indicators varied between individuals. Subjective assessments and respect for individuality are important in the quality of life for older adults.

Match these facts with the new older life in this century. We have changed in the last few decades. The magnitude of changes in the healthcare system is testament to the potential of culture to alter physical functioning. Compared to our ancestors at the turn of the 20th century, we are taller, stronger, and healthier. Our brains process information faster and we live a lot longer. For the last 50 years each cohort has arrived at older age and has been healthier than the one before. Interestingly, healthy life expectancy has dropped since 1990. Again, these are generally calculated in the form of DALYs or disability adjusted life-years. One DALY equals one lost year of healthy life and is measured by the sum of years of a life lost to early death and years lived with disability. The leading global causes of health loss as measured by DALYs in 2016 were ischemic heart disease, lower respiratory infections, and stroke, but have been added to with depression, low back and neck pain, and road injuries. As we have suggested many times in this book, the world has made great progress in health and now the challenge is to invest in finding more and effective ways at preventing or treating the major causes of illness and disability. This, of course, concerns chronic disease.

In this context function eventually falters: we have noted that as people age, ADLs decline dramatically; looking more closely at functional decline, adults older than 75 years account for most of the ER visits and falls; and a sedentary lifestyle leaves older adults at risk for just about everything. Both obesity and malnutrition increase with age, and, although smoking and alcohol use are lower, it is still a concern. We pay a price for our living patterns but we are living longer. We have already highlighted the Nash study (Nash et al., 2017) where the younger one was to cease

smoking, the longer one lived; fortunately former smokers were at substantially reduced risk of mortality after age 70 years relative to current smokers.

Selected Health Facts for Older Adults

- Average health care costs per year were directly related to income: those living in poverty cost \$21,033 compared with those with more means at \$12,440. Of these monies, inpatient healthcare was 25% (down from 32% in 1994) and prescription costs were 16% (up from 8% in 1997).
- Older adults paid 60% of prescription costs out-of-pocket, compared with public programs at 35% and private insurers at 38%. Finally, virtually every older person was paying some out-of-pocket expenses (95%). From 1977 to 2006 the percentage of household income that people aged 65 years and older allocated to out-of-pocket spending for healthcare services increased from 12% to 28%. Today, over 50% of monies are going toward medication (Gliklich & Dreyer, 2010).
- Compared to other age groups, older adults have the highest numbers of doctor visits, hospital stays, and prescription medication usage. In 2004 the average annual cost per older person \geq 65 years was \$3899 (Blount et al., 2007). Left unchecked, healthcare expenditures will likely rise from the current level of ~15% to 29% of gross domestic product (GDP) in 2040. As intimated, medication use is high among the elderly.
- Adverse drug reactions account for a substantial amount of emergency room use, hospital admissions, and other healthcare expenditures. Only 50% of medication is taken properly, and there have been 1.9 million drug-related injuries (Cogbill, Dinson, & Duthie, 2010).
- Several barriers have been attributed to poor medication use. These include physical illness, medication side effects, cognitive dysfunction, psychiatric conditions (mood disorders), functional loss, social loss, inability to afford medication at full dosing, and the idea that a medication is not needed now (Cogbill et al., 2010).
- While only 5% of those over 65 years reside in a nursing home, between 10% and 15% of community dwelling elders require considerable support and assistance to remain in their own homes.

Living results in problems in adjustment. Demographics (dependency ratio—percentage of nonworking people who depend on those who do), health care costs, GDP alterations, birth cohort inversion, among others,

mandate that this be addressed. This is a new era we have just not seen before. As we have said throughout this book, older adults live in the real world. There is no easy escape. Problems occur internally and externally. One's self-attitude, general health, psychological and subjective view of life, and past history conspire to dictate choices for well-being. These are the internal variables. Externally there is everything else. Here we are largely discussing "everything else." We discuss this general problem area largely through the prism of the disablement model.

Disablement Model and Personal Responsibility

The definition of health according to the World Health Organization reads: "Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (WHO, 1946). The interplay, therefore, of physical and mental health, social interactions, and environment of the individual contributes to their ability to achieve their goals and experience quality of life. Although there are many definitions of quality of life, in essence it is the ability of the individual to do what they wish to do in carrying out essential tasks or in pursuit of desired goals and to do so according to values and preferences.

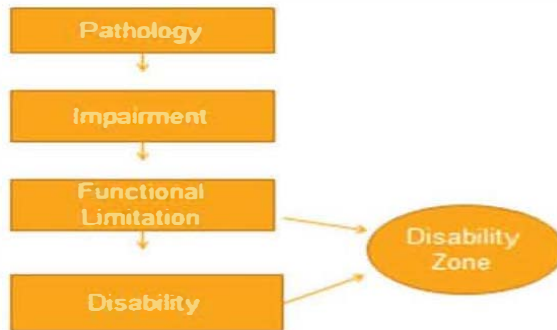
In more recent years, at least three common theories regarding older age have best articulated adjustment. These are the SOC model (Selective Optimization with Compensation; Baltes & Baltes, 1990), the Dual Process Model of Developmental Regulation (Brandstädter, Rothermund, Kranz, & Kühn, 2010), and the Motivational Theory of Life Span Development (Heckhausen et al., 2010). These have in common that older adults need to adapt across the lifespan and compensate for disability or limitations. Mobility limitations especially may restrain older adults from engaging in goals related to exercise and lead to disengagement from goals related to cultural activities. The decrease in the total proportion of women, for example, reporting goals related to exercise and cultural activity during follow-up periods in several studies is substantial, as is the increase in women reporting goals related to independent living. Higher age was associated with not engaging in goals related to cultural activity and with maintaining goals related to independent living.

This relates to the disablement process model. This model (Nagi, 1977) describes four stages from the time a disease develops until it causes daily dependence on another person due to inability to function. The stages are known as pathology, impairment, functional limitation, and, eventually, disability where limitations and performance of socially defined roles and activities within the context of physical and sociocultural environment are

evident. Evans, Rosenberg, and Thompson (1991) designated a disability zone as the period during which a chronic illness causes notable functional limitations and necessitates caregiver services and assistance.

The goal of health then should be to delay entry into the disability zone. This is usually the last zone that a person demonstrates after having a chronic disease and reaching a period of time when there is a need for more care. Another perspective on managing functioning in older adults is to view the disability as having different levels and components. There might be a fixed physical limitation because of disuse and deconditioning and excessive disability. Many have cautioned against a policy that simply promotes longer lives without considering the human dignity, personal identity, and psychological control that can be lost as a result of high physical and physiological dysfunction at these later stages.

Disablement Model



Based on the disablement process model, changes in measures of limitation of mobility difficulties and difficulties performing ADLs and IADLs were noted (Spalter, Brodsky, & Shnoor, 2013). There were four measures of impairment but also patterns of improvement. These trends imply that there is no single negative pattern of decline in function at old age. Rather there are patterns of improvement and decline, depending on the person: The greater the age, the greater the prediction of functional decline. Getting older then has consequences but they vary. The important

issue then is to have a sense about decline, to have some ability to measure it, and above all to have some interventions that matter.

Caregivers of older adults are of course involved. They actually provide appropriate daily rehabilitation care, even if it is not through a professional rehabilitative frame. In effect, they will allow older adults to maintain their abilities and may even prevent rapid functional declines. But issues can become complex. Social demographic variables add and subtract to the percentage of variance to explain IADLs. The managing of finances and medications is related to levels of education and income, as well as to cultural differences, age, and sex. On the other hand, social demographic variables contribute less to the explained variance of mobility and movement changes, which are better explained by health variables, as might be expected.

Depending on the environmental influence, core features of psychological health are altered. Health-related goals, for example, increase among older women. As the circumstances of life alter, health-related goals change, as do exercise, close relationships, cultural activities, and independent living, as well as close relationships. Control has played a crucial role in helping understand the etiology of psychological wellbeing across the life course. Many have captured the vitally important role that feelings of personal control play in society. They might affect the stress belief regarding control over one's life as a most important factor (Krause and Hayward, 2014).

It cannot be stated too strongly that this can all change for the better. As we have alluded to previously, it had been thought by the National Institute of Mental Health that the onset of clinical depression and other such diseases cannot really be prevented. In recent years this has been debunked. Risk factors for depression, such as specific and nonspecific factors, clearly have an impact. Nonspecific factors lead to high rates of depression and other disorders, such as poverty, exposure to violence, and the like. At different stages some factors, for example, parental bereavement, divorce, and having a serious medical illness, may increase the likelihood of a child developing difficulties, while others (presence of stable relationships, good schools, effective vigilant parenting) may promote the development of resilience. Integrative models about the causation of depression suggest that, for example, depression is determined in part by familial and genetic factors and in part by current life adversities. Vulnerabilities then may be expressed in different ways at different developmental epochs. Yes, the evidence clearly suggests that prevention works, and that poor lifestyle behaviors interact with poor life adjustment factors to result in problems, especially at later life.

This of course is epigenetics. This really matters in real world environments. Experience alters the predilections of choice in life. We have discussed this important life feature. Experiments on animals suggest clearly that experiences in youth can re-write the epigenetic markers in the brain, altering our behavior as adults. Depression, for example, may be in many ways an epigenetic disease. Several groups of scientists have mimicked human depression in mice by pitting animals against each other. We have plenty of reasons to be concerned about our part of the equation in epigenetics. Comprehending the ability of environmental factors to promote epigenetic inheritance that substantially promotes genetic mutations is a significant advance in our understanding of how environment impacts disease and evolution. Epigenetic effects due to a host of environmental toxins connecting pesticides, plastics, fungicides, dioxin, and hydrocarbons to diseases and abnormalities as much as three generations later have been noted. A lack of physical activity accounts for about a quarter of coronary heart disease and colon cancers, and a fifth of osteoporotic fractures. It has also become known that the percentage of older adults who show signs of silent strokes or damage of this sort is at least 25% and more likely 40%. Given a stroke, depression is a determinant of further problems, strokes, and heart disease. It is extremely important to do something about risks in general in order to slow or halt deterioration for this or memory deterioration in general.

Variables

Let's look at the core variables in Life Adjustment. There are several factors that are highly related. The number and type of variables for consideration regarding Life Adjustment is massive. This construct also interfaces with other domains, particularly health. In a general way, we are addressing stress in the environment. Social strain is a marker of the extent to which a person feels that there are too many demands on them. This involves real life as well as messy constructs like acculturation; higher acculturation generally represents a state of friendly acceptance of the modal society. We target SES/education, functional needs, safe environment, socialization/relationships, happiness/life satisfaction and meaning, and stress. We end with a special section on caregiving as it is so important.

SES/Education: SES is a problem area for older adults. As one example, nearly 45% of older people with atherosclerotic cardiovascular disease report financial hardships due to medical bills. The impact of SES starts early. As previously reported, in the United States, over 20% of children under the age of 18 are officially poor. This means that they live

in a household with incomes below the federal poverty level. Another 20% of children are near poor, living in households with incomes between 100 and 200% of the federal poverty line. Poverty is a critical risk factor for many of the mental, emotional, and behavioral disorders of children and youth. Family poverty is complex and intertwined with the large number of what most researchers refer to as poverty cofactors. These are correlates of poverty, some of which may be determinates in prior generations and some of which may be mediators of the effects of poverty and children. For example, low school attainment and teen parenting both increase adolescent chances of raising children in poverty. Education, achievement, and family structure in one generation can therefore be determinants of family poverty and then children's health and development in the next generation.

Other correlates of poverty can represent mechanisms by which family poverty affects children. These can include distressed neighborhoods, persistently low performing schools, less nutritious food supplies, and much more. These exposures to poverty risks are sometimes viewed as rival explanations for the association between poverty and children's health and development. The Kaiser health system has widely promulgated its ACES (Adverse Childhood Experiences Scale) data showing there is loose agreement that the correlates of family income, such as parenting quality, family structure, and parent psychological factors, are responsible for associations between income and children's attainment.

In this regard researchers see classes that continue to run at a fixed distinction among upper middle and lower class status. The higher in social economic status you are, the more independent-oriented you are likely to be, while the lower in status you are, the more group-minded you are likely to be. Social class, which can be defined as a social context that individuals inhabit in enduring and pervasive ways over time, is a fundamental end to which we can see ourselves in others. Because lower ranking people have fewer resources and opportunities than those of relatively high rank, they tend to believe that external uncontrollable social forces and others' power have correspondingly greater influence over their lives. Success for them, therefore, depends on how well they can lead, rely on, and help out others. By contrast, those who enjoy resources and greater social class live in a context that has enhanced their personal power and freedom, and that has bestowed upon them larger and safer living spaces, the means by which to buy high-priced goods and experiences, and educations that provide access to influential people, ideas, and venues. These conditions give rise to a more self-focused approach to life.

Interestingly, people of lower rank tend to be more emotionally attuned to others. In a 2000 paper by Krause and Haywood (2014) it is reported that less educated people are better than more educated peers in identifying emotions on faces. They are also more accurate in reading a stranger's emotions during a group job interview. Other studies find that people with less income and education are more generous, trusting, and helpful than their wealthier, more educated peers. The good news about having influence and control is intriguing: High powered people stay authentically the same person no matter the context, but people who are relatively low power change their aspects of themselves because having low power means people have to adapt and fit in to the different contexts they are in.

A necessary subsection of SES involves retirement. Often it is forced. Regardless, it changes life and the factors necessary for life satisfaction. The incidence of adjustment difficulties regarding retirement varies across dimensions. A steep upward career path is associated with fewer financial adjustment difficulties, but with more difficulties adjusting to the loss of status. Compared with continuously married retirees, divorced retirees without a partner are more likely to miss the social dimensions of work, and those who have re-partnered are more likely to miss the financial resources. The longer individuals are retired, the less likely they are to miss the work-related social context. This just makes common sense.

Achievement gaps relating to differences in the performance found between children from families with higher education versus lower SES circumstances are typically defined in the literature according to parental occupation, education, and income levels. For example, students from lower SES backgrounds, that have lower family income and parental education attainment, tend to have lower reading, math, and science scores compared with students from higher SES backgrounds. Further, children who attend high poverty schools have lower achievement scores in math and reading across the board compared with students who attend low poverty schools. SES is a robust predictor of educational outcomes and recent research suggests that the SES-based achievement gap, that is the gap between high income families at the 90th percentile of the income distribution and low income families at the 10th percentile of the income distribution, has only widened since the mid-1970s, but is now twice as large as the black-white achievement gap. Specifically, on both standardized reading and math scores, the SES achievement gap has widened from roughly 0.9 of a standard deviation to approximately 1.25 of a standard deviation, where the white-black achievement gap has narrowed

from roughly 0.9 of a standard deviation to approximately 0.75 of a standard deviation (see Lichtenberg & Mast, 2015).

The presence of additional stressors, such as those correlated with low SES, is likely to increase the overall levels of stress and an individual's faith, and may exacerbate the effects of race-based social stressors. Increased exposure to neighborhood stress, such as exposure to violence, pollution and toxins, and disturbances by neighbors, in addition to daily life stressors, such as high levels of financial strain and associated increases in family conflict, may combine additively and synergistically with race-based stressors to influence educational outcomes.

Things pile up. Even changes in stress hormones and sleep processes are important factors to consider in the understanding of ethnic disparities in at least academic achievement. Findings of disparities in stress and sleep compliment previous theoretical work that applies a stress and coping approach to understanding the consequences of social stressors. Specifically, in addition to focusing on coping responses that have been identified previously as important for educational achievement attainment, we should also consider the role of biological response, namely the HPA axis activity and sleep in the emergence of racial ethnic academic achievement gaps. Often we note that stress is experienced deeply where the person naturally pushes away conflict and seeks reward/comfort, at the expense of an overactive HPA system. This is a good definition of stress.

One other marker of SES is nutrition. Data on the prevalence of malnutrition in older adults living in the community are limited. It is estimated, however, to be between 5% and 30% and it increases with the level of care. This is of course an issue. Over-nutrition is the focus of most public health initiatives, educational efforts, and commercial ventures (the "diet industry"), and it tends to overshadow another problem: malnutrition. It's a prevalent public health issue even in developed countries, and the risk of malnutrition increases with disability and age. Weight loss and poor nutritional status in older patients are associated with increase in morbidity and mortality. Some of the adverse effects of poor nutrition and involuntary weight loss include increased risk for hospitalization, poor wound healing, decrease in function, poor surgical outcomes, and increased risk for developing pressure ulcers and even cognitive decline. As a result, early screening and detection of nutritional deficiencies are important in older patients, with the goal of improving quality of life and outcomes. Importantly, use of better nutrition programs, like the Mediterranean diet, results in positive findings such as a reduced risk of frailty and increased longevity. It does so of course not in a vacuum, as it

has a cultural context and relates to sense of community (see Lichtenberg & Mast, 2015).

There is of course a connection between a poor life experience and depression. Brinda et al. (2016), in a study from the World Health Organization on Global Aging and Adult Health, WHO-SAGE, showed that geriatric depression is reliably associated with poverty, economic insecurity, and national indicators of economic disparity across 6 low and middle income countries. Significant correlates of depression include female gender, illiteracy, poverty, indebtedness, past informal-sector occupation, bereavement, angina, and stroke, all of which increase the odds of depression, and pension support and health insurance lower the odds. An index of wealth, ownership of durable assets, house dwelling and characteristics, type of toilet access, and source of drinking water was also associated with geriatric depression. Brinda and colleagues drew 2 conclusions. First, social economic factors play an important role in the etiopathogenesis of the management and prevention of geriatric depression, and second, the key to lowering the risk for geriatric depression is social and broader efforts.

“... there are two conclusions. First, social and economic factors play an important role in the etiopathogenesis, diagnosis, management, and prevention of geriatric depression in all cultures. Second the key to lowering the risk of geriatric depression is not individual therapy but rather broader efforts to reduce economic disparity. They are critical of Western psychiatry. By medicalizing psychosocial distress, we have shifted the focus from the responsibilities of the states for poverty and structural violence, and transferred pathology and burden to individuals.”

Albert, p. 1209, 2016

We include education here. Education, not income is the better predictor of life expectancy. Clearly, the fact that a better education leads to improved cognition and, in turn, to better choices in health-related behaviors is notable. The net worth of college educated American households with children rose by almost 50% between 1981 and 2013, whereas among high school educated households, the net worth actually fell by 17%. The upshot of this, although we would like to think otherwise, is that the playing field of opportunity today has not just become uneven, it is getting worse as time goes on. That is because in the same way the stacking of one economic or educational advantage upon another amplifies the possibilities for successful lives in affluent kids,

increasing an already larger range of possibilities. Finally, we note again that the opportunity gap that Putnam identified has less to do with race than with education level obtained, which in turn is associated with socioeconomic status.

Education, then, is the gift that keeps on giving. The disparities in mortality across different levels of education have widened substantially over time. For example, mortality rates fell modestly among those with high school degrees, but mortality rates fell much more rapidly among those who had college degrees. As a result then of just encouraging high school completion among adults who had not finished high school, twice as many lives could be saved among those born in 1945 as compared with those born in 1925. These results suggest that policies and interventions that improve educational attainment could substantially improve survival in the US population.

One other important factor needs highlighting: leisure. We addressed this in Chapter 5. Verghese et al. (2003), in evaluating a cohort of 469 adults older than 75 living in the community and without dementia, followed a group for 5.1 years. Their exposure to cognitive activities (board games, reading, playing a musical instrument, crossword puzzles, writing) and physical activities (dancing, housework, walking, climbing stairs, bicycling, swimming) were rated from 0 to 7. Outcomes were onset of dementia, defined by a battery of neuropsychological tests. Board games, reading, playing a musical instrument, and dancing made a difference for people as they had a better quality of life and were less at risk for dementia. Having the opportunity to be active and use cognitive and social tasks makes a difference.

Parisi, Roberts, Szanton, Hodgson, and Gitlin (2015) assessed the issue of valued activities among individuals with and without cognitive impairments. Growing evidence suggests that continued participation in leisure activities is associated with positive health outcomes in later life. Leisure activities that are distinct from functional activities of daily living vary widely and may include those that are inherently socially and cognitively engaging, such as visiting friends and family and participation in clubs, classes, and other organized activities. Using the National Health and Aging Trend Study, NHATS, this group examined the activity preferences and participation of individuals without cognitive impairment. Respondents were classified as having no dementia, possible dementia, or probable dementia. Respondents rated the importance of an actual participation in 4 activities: visiting friends and family, religious services, clubs and classes, and going out for enjoyment. It also examined whether transportation or health limited participation. Overall, visiting friends and

family was most important, although the relative importance of activities varied with cognitive status. Compared to cognitively healthy individuals, those with possible or probable dementia were less likely to indicate activities were important and engage in valued activities. Additionally, poor health limited participation in activities for those cognitively intact or with possible dementia. This was not true for those with probable dementia. Transportation difficulty limited going out for enjoyment for a greater percentage of those with cognitive impairment than those without such impairment. The implications of this study were that, regardless of cognitive level, older adults highly value activities. However, actual participation may decrease with greater impairment in cognitive and physical health and with transportation challenges. Developing tailored interventions, therefore, makes considerable sense in health.

Whether deficits can be made up or adjusted to in later life is unknown. This especially applies to mental health (and low SES). There are significant correlations between female gender, illiteracy, poverty, indebtedness, past informal-sector occupation, bereavement, angina and stroke, and depression. Factors like pension support and health insurance influence the odds also. Depression seems to especially influence problems. An index of wealth (ownership of durable assets, house dwelling, characteristics, type of toilet access, and source of drinking water) was also associated with depression. Depression is reliably associated with lower SES, poverty, and economic insecurity. That said, we must also note that increasingly people are more upwardly mobile.

SES is then formidable in adjustment and quality of life. This construct as it applies to the person/family in the room is messy to accurately measure. In general, for the current reality of living, we ask about the objective facts of money and problems at home. Subjectively this is as valuable and one's view of their situation tends to be as reliable. Second, we also assess their occupation and education. This involves the level of education, partner's education, and occupational prestige (Kok et al., 2017). We also ask about the past, growth years: mother and father's education and occupation as well as perception of their growth situation. ●f course, these factors are noted but no real validated metric is applied.

These are both current and distal markers. We target both the economic feature and living situation as well as education. We especially look for a sense of safety and for satisfaction in living. These involve basic living requirements, life quality, and overall satisfaction. Education enhances these. This especially applies to mental health (and low SES). There are significant correlations between female gender, illiteracy, and poverty especially.

Functional Needs: Again we consider function. It is always relevant and asserts an influence, often changing. From an archive at Mercer of over 500 people of 65 or older who had been referred for a possible memory problem, the average prevalence rate of some functional problems for older adults was 64%, indicating that most had functional problems. This ran the gamut of older adults with normal cognition to dementia. On the SF-12, we showed that 60% had clear mobility problems and could not perform an activity of daily living. Many needed Meals-on-Wheels, transportation, in-home care, and other supports. The data below suggest that functional impairment is a factor, based on the MBMD markers of impairment, PAI markers of stress and support and FAQ. These are both practical measures as well as perception. These are again patients showing up with memory problems as the chief complaint. Clearly functional problems are prevalent and demand addressing.

Variable	% with Problem
Functional Concerns	52%
Social Isolation	12%
Medical Utilization Concerns	22%
Stress	22%
Non-support	25%
IADL Problems	48%
Function Rating	55%

Functional Concerns = MBMD Functional Problems;
 Social Isolation = MBMD Social Isolation;
 Medical Utilization = MBMD Health Utilization;
 Stress = PAI Stress;
 Non-support = PAI Non-support;
 IADL Problems = FAQ (>7);
 Function Rating = SF-12 total

Driving is a key marker of function. The reasons older adults stop driving are such things as age-related declines in cognition, functional and/or visual spatial capacities, or health crises such as stroke or fractures from a fall. The negative effects of driving cessation on older adults' physical, mental, cognitive, and social functioning have been extensively studied. The inability to drive and the resulting poor mobility can significantly restrict older adults' instrumental activities of daily living and social integration and can lead to further physical and functional and mental decline and reduce friendship networks. Several studies have found

that mobility restrictions affect older adults' social relationships and participation more than engagement in paid employment or other formal activities. Former drivers and those who have never driven also have higher risks of long-term care entry after adjusting for demographic and health variables than drivers, and both former and never-drivers are at increased risk of death (Meuser, 2015).

We highlight the Functional Assessment Questionnaire (FAQ; Pfeffer et al. 1982) as a marker of function, notably executive function in the adjustment realm. This marker provides a rating of one's IADLs necessary for quality of life. The FAQ had a direct relationship with the level of cognitive decline: better with normal aging, worse with subject memory impairment, and most impaired with MCI and then dementia.

We often also apply the AD-8 (Galvin et al., 2005). The family member or other informer is asked to discuss the target patient who has experienced change over the last few years in each of eight areas. These include judgment, decreased interest in activities and hobbies, repeating themselves, trouble learning about how to use tools, gadgets, and appliances, forgetting the correct month or year, trouble handling complicated financial matters, trouble remembering appointments, and daily problems of thinking and memory. For detecting milder levels of cognitive impairment, the use of standard questionnaires involves everyday cognitive function (Farias et al., 2008) and cognitive change checklists (Schinka et al., 2010).

Environment: This can take several forms. From the macro-level, the influence on life quality can be subtle but noteworthy. The WHO estimates that in 2012 household air pollution caused 43 million deaths, ambient air pollution caused 37 million deaths, and unsafe water or poor sanitation and inadequate hygiene caused 842,000 deaths. Contaminated soil at active and abandoned mines, industrial facilities, and hazardous waste sites has killed tens of thousands of people and injured hundreds of thousands of others. In recent years, the impact of a dirty environment has been causative for deaths due to cancer, lead poisoning, and COPD problems too.

From the macro-level just noted, the pervasiveness of environmental problems is very individualistic and can be lost to subtlety. Lum et al. (2014) noted that aging in place refers to the ability to live in one's own home and community safely, independently, and comfortably regardless of age and common ability level. Promoting aging in place is a policy objective in developed countries. It contributes to the wellbeing of older adults by providing a sense of attachment, connectedness, security, familiarity, identity, independence, and autonomy. Preference is central to

the concept of aging in place in distinguishing a choice to stay versus being stuck in place. Although it is recognized that older adults generally prefer to stay in their home and local community for as long as possible, the situation is more complicated for elders with low income, who lack financial resources and care support.

Their preferred mode of living alters when faced with physical decline. This requires a high level of community support and services that are currently lacking. With a rapidly aging population, the present infrastructure for healthcare will prove even more inadequate in meeting seniors' physical and mental health needs. A paradigm shift away from the sole focus on the delivery of interventions at an individual level to more prevention-focused, community-based approaches may become essential. Recent initiatives have been proposed to promote healthy lifestyles and preventive care to enable older adults to age in place. Prominent among these are the World Health Organization's Global Age-Friendly Communities (AFC) Network, with 287 communities in 33 countries, and AARP's Network of AFCs with 77 communities in the United States. In an AFC, older adults are actively involved, valued, and supported with necessary infrastructure and services. Specific criteria include affordable housing, safe outdoor spaces and built environments conducive to active living, inexpensive and convenient transportation options, opportunities for social participation and community leadership, and accessible health and wellness services. Active, culture-based approaches, supported and developed by local communities, and including an intergenerational component are important.

Jeste et al. (2016) outlined several challenges to such an effort. The core ideas are reasonable housing options, pleasant and clean environments, transportation options, respectful interface with the community, encouraging active participation in the community, and providing accessible care including preventative services. While this list is aspirational and perhaps not practical, it is relevant and important.

Challenges to Aging Friendly Environment

- Coordinating services from a national hub
- vercoming ageism
- Facilitating challenges for relevant stockholders
- Ensuring program evaluation
- Resolving conflicts with local businesses
- ptimizing financial priorities
- Promoting interpersonal activities
- Focusing on underserved communities

Expanding involvement of academic centers/schools
Developing rural senior friendly communities (SFCs)
Supporting greater use of technology
●Offering educational opportunities for older adults
Working with local media
Jeste et al., 2016

Nahemow and Lawton (1973) suggested in their ecological theory of aging that a decision to relocate can be determined by the balance between the environment of the person and the individual's competencies. Theoretically, therefore, an aging in place preference can be modified with increased neighborhood support. Although previous research had emphasized individual competencies, recent studies have directed more attention to ecological frameworks and the role of neighborhood support and aging in place. These studies focused on actual relocation or expectations to relocate. For example, community support networks are always found to be an important social, economic, and environmental element for aging in place. Perceived availability of community services is related to anticipation of aging in place or relocation. If community ties are in place, with many neighbors known by name and personal ties being important, then relocation is less appealing. Evidence for the impact on neighborhood support in aging in place preference, however, is lacking.

Finally, whatever applies to old age applies to the old-old more, especially with rural living. Adults of ages 85 or older who are rural residents have significantly higher levels of chronic disease, take more medications, and die several years earlier than their urban counterparts. Research confirms some of the special challenges facing older populations in rural or remote areas, who often have less access to physicians and long distances to travel for care, and sometimes have a lower socioeconomic and educational level and other issues. It also reflects health problems that might have been reduced if they were treated earlier or more aggressively, researchers say.

Data from several different study groups has found that rural residents measured significantly higher on the Modified Cumulative Illness Rating Scale, with about an 18 percent higher disease burden. There are fewer physicians, fewer specialists, and higher caseloads. Doctors have less support staff and patients have less public transportation. A patient sometimes might need to wait months to see a doctor, and have to drive significant distances. Adverse effects can increase from taking multiple medications. It's of particular concern that rural older adults started with

more disease burden, which significantly increased over the next five years.

For one target group, Goeres et al. (2016) summarized the problems in Oregon (below). It can be seen that there are needs and that such populations can be targets of programs for care. Additionally, the Alzheimer's Association embarked on a holistic program for older adults at risk for problems (frailty, cognition, social problems) and proposed a total care effort of wellness, yoga, physical exercise, and diet for an extended time period in the community. It will have small groups, leaders, rewards, and considerable support daily. This is an indicated prevention program at several sites in the US (see Alzheimer's Association).

Relevant Rural Oregon Study Findings:

- The rural population of Oregon contains a greater proportion of older adults than the urban population.
- Rural participants were found to use an average of 5.5 medications, compared to 3.7 for urban participants. The use of many medications can be especially risky for people in their 80s and 90s, leading to a concern ("polypharmacy") when a person takes five or more medications.
- At baseline measurements, valuable medications to aid bone mineralization were often used less in rural populations, but pain-killing opioids were used more often.
- Medication use for high blood pressure went up significantly over time for rural populations, but not urban ones, in which their use had already been higher.
- The rate of disease accumulation was significant in the rural cohort, and negligible in their urban counterparts.
- The median survival time of the rural cohort was 3.5 years, compared to 7.1 years for the urban older adults.
- Risk factors of chronic diseases were low education, poor socioeconomic status, a history of chronic disease, being female, and older age. These factors are associated with a typical rural population.
- Living with someone, and/or having a large social network are protective factors against chronic disease, and may be more common in an urban or suburban population. (Goeres et al., 2016)

Questions that target the quality, acceptability and tolerability of the overall community are important. We are interested in what the person's life looks like and how they feel about their day. What can the person do and do they enjoy this? The person is asked if they like their life now

where they are. In effect, the issue has to do with the quality of life as lived and rated, as well as where there are openings for improvement. Importantly, do they see the need for help and can they accept the recommended assistance?

Social Variables (relationships): We often live alone or are forced to live with family or friends. An analysis of the US Census Bureau conducted by the Harvard Health Letter, by Harvard's Joint Center for Housing Studies, found that between 2001 and 2016 the number of Americans of ages 65 and older increased by about 40%. During the same period the number of people of 65 and older living in a grown child's home nearly doubled from 1.4 million to 3.4 million, as did the number living in sibling's homes, from 226,000 to 453,000. The number of older adults living with non-relatives also increased, from roughly 400,000 in 2001 to 900,000 in 2016 (Harvard Health Letter, 2018).

Additionally, older adults now have less friends and experience themselves as alone more than at any other time. Social Connectedness is important, assessing how older people use their time. To start with, at later life marriage is empowering. It results in a largely supportive environment. This is intimate connectedness. Perhaps a more important issue is social connectedness. The link between social relationships and cognitive limitations at later life has received substantial attention. Larger social networks seem to be protective against increases in cognitive limitations. In contrast, social isolation and loneliness are associated with increased cognitive limitations irrespective of education, physical and mental health status, and health behavior. However, past studies have predominantly focused on the size of social networks and/or the frequency of social engagement and do not consider how the quality of any particular social relationship might influence cognitive limitations. A significant literature, then, has established that the quality of relationships, particularly marital relationships, is important in shaping health outcomes (Krause & Hayward, 2014).

People who report feeling lonely have a 26% increased risk of early death and those with fewer or no social contacts or activities have a 29% increased risk (Yeager, Dahl, & Dweck, 2017). Cacioppo (UCLA Health, 2017) identifies 3 separate levels of connectedness: intimate (one who affirms you), relational (mutual rewarding face-to-face), and collective (part of a group). Each fulfills a lasting need for connectedness. Even a simple embrace from a loved one improves daily functioning and at times health. Social support can even lower the likelihood of experiencing excess levels of stress-related hormones, such as cortisol, epinephrine,

norepinephrine, disturbed sleep, cardiovascular disease, high blood pressure, and diabetes, as well as AD.

Whether social support is available to individuals or not makes a difference in a number of health outcomes. However, as intimated, social support should be differentiated. Available social support, that is whether the person has someone who is able to provide support, is consistently related to better health outcomes. The stress buffering model proposes that social support unfolds its beneficial effect via direct and buffering effects. There is empirical evidence for both pathways. Available social support directly influences health or physical functioning and it seems to buffer, that is, dampen age-related declines in physical health associations between stressors, depressive symptoms, blood pressure, or inflammatory processes (Wolff, Lindenberger, Brose, & Schmiedek, 2014).

Choi, Yorgason, and Johnson have applied this finding to marital quality: Negative marital quality, such as marital strain and conflict, is associated with the deterioration of functional health and higher risk of mortality over time. Researchers have shown that for some time the social relationships in different social class groups are not the same. The social forces that shape social relationships in different social economic groups are quite dissimilar. For example, a considerable number of studies have pointed out that rundown neighborhood environments play a role in determining the nature of the social relationships that residents form within them. We have alluded to this above.

There are several social support scales. Our data suggest that focused questions on the quantity and quality of connections, family and friend based, are sufficient. Living alone and recent deaths are highly relevant. The unfolding of a typical day or week often gives the necessary and sufficient information. The number of friends that one can call, if in danger, is also an important issue.

Happiness/Life Satisfaction and Meaning: In the Proceedings of the National Academy of Science (2017), researchers questioned 800 adults of age 52 or older (Nummenmaa et al., 2012). They assessed the specific personal attributes that are linked to health and wellbeing in older age. The results suggested that the life skills of optimism, conscientiousness, emotional stability, control, and determination significantly boosted the odds of wellbeing. No single attribute was more important than another. The effect was seen to be a function of the accumulation of life skills. Only 6% of those with 4 or more skills had reported poor health, relative to 33% of those in the lowest quartile.

The Million Women Study is a prospective study of UK women recruited between 1996 and 2001 and followed electronically for cause-

specific mortality. Three years after recruitment, the baseline questionnaire for the present report asked women to self-rate their health, happiness, stress, feelings of control, and whether they felt relaxed. During 10 years of follow-up, 4% of participants died. Self-rated poor health at baseline was strongly associated with unhappiness: better health, better life satisfaction.

Meaning in life or life purpose is crucial in the adaptability required to survive. Any psychological experience in the service of a person's core meaning (especially for adaptation) must also be responsive to changing environmental circumstances. Thus it needs to be recognized that research demonstrating the effects of manipulation on reports of meaning in life ought to recognize this as a factor. The role of adaptive functioning may indeed be critical for meaning and survival. Consider the types of manipulations that seem to matter in meaning in life. These are social relationships, the experience of pleasure, and the detection of a reliable pattern of coherence in the environment. Although potentially trivial seeming from an adaptive perspective, these three factors are anything but. Human beings need the social group to survive. In addition, pairing pleasure with adaptive behaviors is evolution's way of getting us to do the things we must to survive. Finally, extracting reliable associations in the environment is a survival-relevant capacity for all species.

If a person believes that what they do has a purpose and that their life will outlast their physical presence, the person feels connected to others, has a positive mood, and can deal better with difficult times. In fact, there is good evidence that meaning-constructed existence can be created and is natural to the functions of living. It may be evolutionary and one cause for existence (Heintzelman & King, 2014). A meaningful life is associated with many positive things – better self-reported health, decreased mortality, and higher quality of living. Meaning means that one has a purpose and has significance. It involves social relationships, positive mood, and environmental coherence, as well as positive illusions, response biases, and more adventurous living.

Purpose in Life (PIL) has arms that reach beyond its area. A study by Mota et al. (2016) evaluated the incidence and determinants of physical disability in a contemporary nationally representative sample of US military veterans. The focus here was on purpose in life. Two-year incidence of any physical disability, that is an IADL or ADL problem, among veterans of age 55 revealed incidences of ADL and IADL disability of 3% and 11% respectively. Older age, being married or cohabitating, and number of medical conditions, specifically diabetes, heart attack, and chronic pain, were associated with increased risk for any

potential physical disability and incident IADL disability. PIL was found to be protective for incident IADL. More importantly, a positive attitude translates into better quality of living. Optimism (“I feel confident that the rest of my life will turn out well”) makes a difference. Note that PIL is distinct from WTL (will to live). The WTL markers focus on whether one is willing to continue to live as they are. This is more depression-based and, in fact, highly related to depression.

Related, generativity refers to a concern for establishing, guiding, and contributing to the welfare of future generations. It is commonly manifested either within a family through tangible support exchanges and the transmission of values or outside the family through volunteerism and societal engagement. The provision of emotional support predicts greater self-esteem and control beliefs among elderly adults with comorbid health conditions. Provisions of practical support through volunteerism and civic engagement are associated with a higher quality of life for older adults. Psychological functions of giving each type of support are quite important.

Religion and spirituality are also relevant. They create meaning or foster it. In a review of research on some of the topics in terms of how they might be protective and produce healthier aging, Vahia et al. (2011) concluded that “of all the positive traits discussed, spirituality/religiosity may be the most extensively used to promote clinical outcomes” (page 241, cited in McFadden, 2014). Religion, spirituality, and psychotherapy do intermingle. There are several therapies involving meaning-centered counseling, spiritually sensitive psychotherapy, religious accommodative therapy, value-based psychological treatment, and religiously integrative psychotherapy. There is a sense in positive psychology that there is a limit to how much a person can control and that spirituality helps people come to terms with human limitations. Typically spirituality addresses a sense of significance. This includes a reliance on religion to conserve a sense of significance, and sometimes a radical religious change is required because of a person’s meaning and value system.

Again as with other variables in this area, this factor has many referents for possible assessment. We assess this area by addressing spirituality and religion with formal questions – do you have a church/place of worship, are you active, what bring purpose in your life, is this important to you, etc. We also question meaning of aging and how the person views/rates life now. The person is asked about how their life creates joy and meaning for them. Related issues involve volunteering, self-education, and in general the practice of positivity.

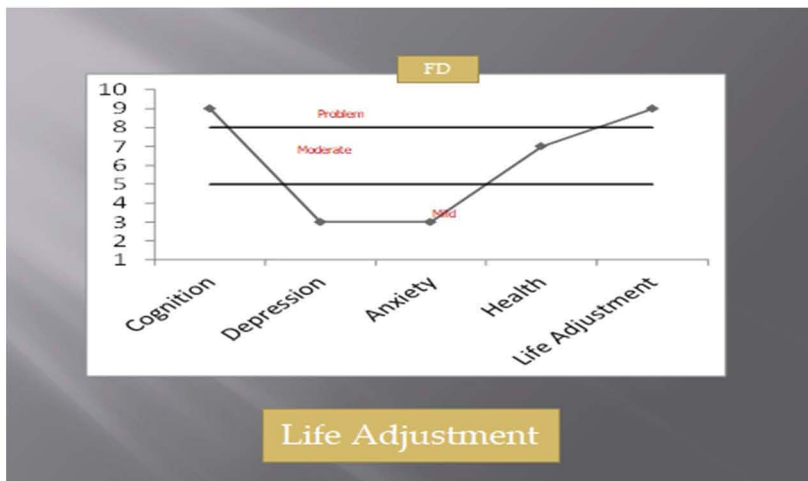
Stress: We have discussed stress in the context of health. Older persons are frequently exposed to various stressors. We have noted that about 25%

of healthy older adults experience one stressful life event within a 3-month period. Life events include acute, as well as ongoing stressors, such as the death of a close relative or relocation. Comijs et al. (2011) found differential associations for different negative life events with cognitive decline, none of which were mediated by depressive symptoms. The death of a child or grandchild, which may be considered a highly stressful event, was associated with a higher risk of cognitive decline, where more chronic stressors, such as the illness of a partner or relative or serious conflicts, were associated with better cognitive function. The associations between life events and cognitive function were stronger in ApoE4 carriers compared to non-carriers suggesting this gene plays a significant role in the association between stress and cognitive function. Negative affect or neuroticism is also a key marker reflecting problems in life. Stress in a person with high N (neuroticism) increases problems measurably. It is an indicator of the person's adjustment.

We assess stress by use of a measure of 1-10 on a stress dimension. We will also use the Perceived Stress Scale (PSS) which is longer and more accurate as it includes many aspects of living. Stress is also assessed on omnibus scales (PAI and MBMD).

Case 2

PATIENT: FD
 DOB: 1941



Life Adjustment

Measure	Healthy Intact	Mildly Problematic	Problematic or Syndromic
Description	Overall lifestyle is positive, patient engages in the core components of lifestyle without major problems	Lifestyle choices are likely to contribute to disorder or have begun to do so.	Lifestyle is compromising patient's mental and/or physical health. Self-care is lacking or not present.
Education and SES	College or professional certificate and continuing interests	HS education or extensive interests in reading/leisure	No HS education and little intellectual interests
FAQ (or AD-8)	≤10	11-18	≥19
Safe/supportive environment	Safe	Limited safety	Unsafe
Relationship Status	Married/in a Relationship	Single/divorced	Widowed in the last five years
Meaning in Life	Purpose in life/happy	Limited purpose or happiness	No purpose/unhappy
Stress (1-10)	0-3	4-7	>7

Life Adjustment

SES/Education: 10th grade and low SES

FA: 14

Environment: Mildly safe (reported problems) and lives alone

Relationship: Low support and lives alone

Meaning in life: Low PIL and poor optimism

Stress: Little. There is no self-realization or self-monitoring

Cognition

MMSE = 22/30

MoCA = 15/30

Trails A = <10%

Trails B = Could not finish

Health:

Rated poor

Parkinson's disease (MCI possible dementia)

Variable medicine adherence

Depression/Anxiety

Self report scales normal ranges

Recent widow

Case Formulation

CHIEF COMPLAINT: Evaluation for Parkinson's disease problems (cognition and adjustment).

HISTORY OF PRESENT ILLNESS: The patient was driven here by her daughter. The patient was not a good reporter of her situation, but struggled to give some answers. She has had Parkinson's disease for a four to five-year period of time. Reports of mild dementia during much of this period are increasingly evident. Her husband died six months ago and that has caused considerable problems. She lives at home alone, but has loose supervision by her daughter. She does complex IADLs (especially cooking, finances, and meds) for her. However, in her capacity of living alone, she does simple cooking, housecleaning, and laundry, and does most of her ADLs. There is no plan for her to move.

RELEVANT DEVELOPMENTAL HISTORY: The patient is a native of Georgia. She is the youngest of three children. Her father was a farmer. She indicated that she worked on a cotton farm, but that her life was positive. There were no milestone disruptions. She graduated only from the 10th grade and at age 18 married. He worked in a grocery store and

recently died, six months ago. They had three children. Their youngest child died at age 35. She herself worked at a bank and then at a church for roughly 20 years. She stopped working 10-15 years ago. She currently lives alone. Currently she is on a fixed income that barely pays the bills. Her daughter helps her.

RELEVANT MEDICAL HISTORY: The patient's medical records were provided by Dr. X. She has Parkinson's disease. She also has headaches, angiodyplasia disease, and memory loss. She is on carbidopa/levodopa 5 times a day, as well as Sinemet CR 50-200 extended release 1 tab h.s. She takes gabapentin, aspirin, multivitamin, calcium D, simvastatin, and psychiatric medications of Lexapro and Xanax. She takes fish oil. She has lost a considerable amount of weight over the last six months to where now she weighs 112 pounds. She does not drink alcohol or use tobacco. She indicates that she is not in pain. She indicates that she sleeps reasonably well. She takes a nap during the day. She relates that she is tired in the day. She is reasonably active during the day. She has been on antidepressant medications and anxiolytics for the last two years. This was precipitated by her husband's decline. She has difficulty with complex IADLs and needs help with shopping, medication management, driving, managing finances, and using the phone. In other areas she is largely independent. She cooks and does laundry and housework by herself.

MENTAL STATUS: The patient is a 75-year-old Caucasian female who was driven to this appointment. She was nicely dressed, smiled, and tried to answer but had problems. She had PD slowness and rigidity but was able to perform minor tasks. She had difficulty with questions related to her history. She was a poor reporter of her life. She deferred to her daughter. That said, she did smile and on occasion would come back to the question and surprise the interviewer with answers. She is oriented. She indicated that she is not depressed, but occasionally worries mildly. She noted that she tries to adapt to the situation. There is no evidence of perceptual anomalies or delusory thinking. Her insight appears to be lacking, but her judgment appears to be poor, but still active.

OVERALL COGNITIVE FUNCTIONING: Mrs. D has an intelligence score at below or low normal. Now she has a Shipley Verbal and Abstract score at <2%. Her Vocabulary score is at 7 (WAIS-IV). This is also well below normal. Her reading score was also poor (70, WRAT-4-Reading). Currently Mrs. D scored a RBANS index of 72 placing her at less than 10% for people her age. She is low in attention, visuospatial, new learning, and memory. Her language was low/adequate. Mrs. D's performance on EF was poor. On a task requiring her to alternate sequentially numbers and letters she was unable to complete the task (Trails B). She scored lower on

math, as well as Serial 7s. She was below average on both fluency tests. Her UPDRS was in the 2-3 range (Hoehn Yahr scale) with bilateral involvement but no obvious falls. This is being monitored.

Key Domains are Life Adjustment: There were deficits on the FAQ, a measure of executive functions in activities of daily living. She has problems on most IADLs where some problem solving is required. She was also rated by her daughter as having problems in most IADL areas especially meds, finances, cooking, and phone use. Her education is lower and she has a low SES. Her environment fit is poor as she lives alone. She is a widow. She also has poor relationships. Her stress is low as she is content. Her meaning in life is poor.

Emotional self-report scales indicated no depression or anxiety. Emotional Scales: She reported average on BDI-II, GDS-SF and PHQ-9 as well as GAD-7 and MBMD markers. Again, she has few self-reflective skills. She does have a concern about her cognition. Her pain and sleep are adequate.

Watch and Wait: This is a 75 y/o female who was referred for an evaluation for her cognitive status. She suffers from PD and is in a dementia (approximately 3 years). She is oriented and wants to be independent. She had notable problems in new learning, visuospatial, and attention, as well as in executive functioning. Her memory is quite poor. She has been living at home alone and struggling. She recently lost her husband. She has poor support at home but her daughter is attentive. She has poor motor skills and her function is suffering. Like others with PD, she shows rigidity and slowness with mild anxiety and concerns about her future. She has problems communicating clearly with other people because of speech that may tend to be confused, truncated, or tangential. She is even tempered and has a loss of insight. Sleep and pain are not issues at present.

Interventions:

Discernment: No (cooperative but marginally compliant)

Therapy Markers

Validate Problem X

Psychoeducation of Model X (caregiver)

Assessment X

Alliance X (caregiver)

Monitoring X

Case formulation X

Problem List

1. Life Adjustment: Assist in the transformation to an ALF. Her daughter is open to this now. Interface with the medical staff and

- social worker, assist the daughter in her transition caregiving role and help needed, Request more stimulation in AFL.
2. Cognition: Assure that the ALF has knowledge of her skills/limits and has CT/exercise/activity program for her needs. Encourage and train daughter in new caregiving tasks involving activity, stimulation and some CT.
 5. Health: Interact with and support ALF medical team with her care, assure that her neurologist is involved and connected to care. She is losing weight and needs her PD monitored closely.

She met minimal criteria for some Watch and Wait Therapy Markers. She is less willing to be a part of any change. Her caregiver was involved in all aspects and was very much on board. The number one aspect of her care involves caregiving and support (ALF). The first order of care then involves her life adjustment. Now she lives in a rural area and needs help with most IADLs. A plan will be suggested for her to be admitted to an ALF. Initially she was not willing. In time, her position changed. This will allow her to be reasonably independent. All IADLs can be managed here. She is taking an antidepressant as well as Xanax. While she does miss her husband, she is coping and seems to adjust. She denies depression. Her health is attended to by her PCP and neurologist. We will now interface with the ALF (especially on CT issues). We will see her bi-weekly for a period and then will see her in 4 months as follow-up.

In sum, she has a life adjustment issue that can be addressed with a change in living. Her cognition reflects a dementia and is a concern that is also handled by the ALF move. Her PD is addressed by her neurologist and her anxiety is addressed by her psychiatric meds.

Self or other monitoring: Targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling:

Environmental control: X

Contingency plans (If-then):

Apply progressive relaxation:

Concreteness training:

Imagery exercise for counter-experience:

Compassion training:

Communication and Assertiveness training: X

Behavioral experiments: X

Emotional exposure:

Homework: X

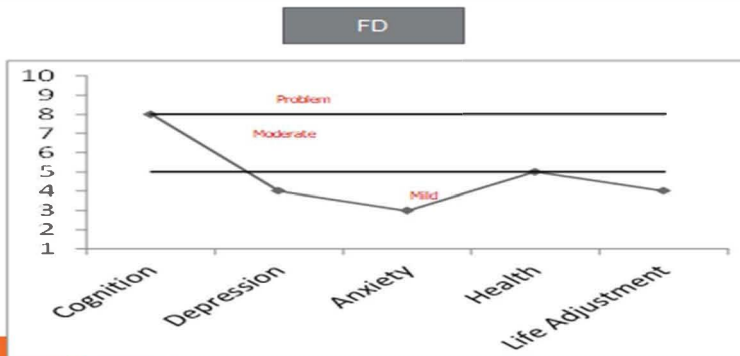
Longer Therapy processes: Interface with ALF and possible caregiving.

Lifestyles:

- Practice Spirituality/Religion (she has a church)
- Socialize: ALF activities
- Exercise: ALF activity
- Somatic softening: None on board
- Values and Leisure: ALF alerted

Follow-up: We will follow her for a period of time. We will interface with the ALF. Initiate caregiving protocol.

This is an updated model. She has been entered into an ALF and her needs are largely met. We will assure that her impaired cognition is responded to.



In many ways this is an uncomplicated case as Mrs D has Parkinson's disease dementia (PDD). She needs to have better care day-to-day. This Life Adjustment domain is now taken care of and her health and cognition take precedence. We address what we see as necessary components of this area.

Caregiving: A Special Case

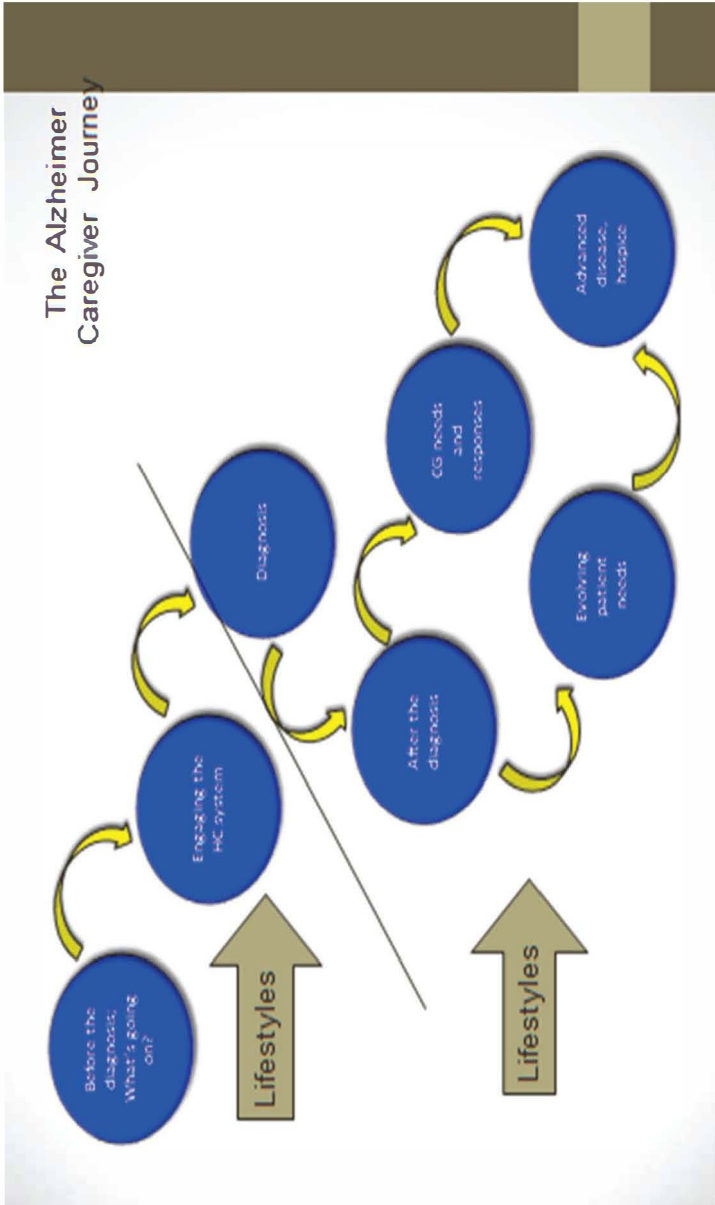
Caregiving is also an issue highly enmeshed in living problems. After a brief overview we reconsider again and address epigenetics and the disablement model as they are core elements of function and accommodation.

Common Caregiver Dilemma

An older woman fell at home resulting in a fractured hip. Surgery for hip replacement is followed by pneumonia as a consequence of low mobility during recovery, lengthening her hospital stay and interrupting rehabilitation of the hip. During her hospitalization she receives services from multiple departments with no overlapping staff, emergency surgery, rehabilitation, pulmonary, and geriatric medicine. She is discharged from the hospital to a skilled nursing facility for a few additional weeks of rehabilitation, after which she is allowed a few additional sessions of physical therapy from a home health agency. When the Medicare funding for home health service ends, she has not yet regained her previous level of functioning, so the family is left to facilitate further recovery or support of the reduced level of functioning she now shows.

Caregiving is always implied in the management of the older adult. Its importance is perhaps best seen as a cascade effect. This is real life. We have lamented the lack of health coordination and, because of this, lack of life quality. It is in this context that we have discussed caregiving (Hyer, 2014). Translating evidence-based dementia caregiving interventions into real practice has been an issue. For the more than 5 million persons in the United States living with dementia, there are 15 million family members providing ongoing support, including care coordination, transportation and accompaniment to healthcare visits, daily assistance with bathing and feeding, and end-of-life care. The consequences for families providing protracted care for this complex condition are extensive and well documented. With disease progression, families are at elevated risk for many problems, including financial, impaired quality of life, physical morbidities, and suicidal ideation, as well as dementia itself.

In recent years, interventions for caregivers have been summarized suggesting that a strong evidence base exists. There are many interventions that are effective for caregivers. A wide array of proven interventions then exist. We have previously cited Maslow (2012), Brodaty and Arasaratnam (2012), and Gitlin and Hodgson (2015) identifying more than 300 interventions. Exemplars of interventions that



could address problems include care management (Maximizing Independence at Home, Samus et al., 2016; Partners in Care, Bass et al., 2014), disease education (Savvy Caregiver, Scharf, Bell, & Smith, 2006), skills to manage functional independence (Care of Persons with Dementia in Their Environments [Cope], Gitlin et al., 2010; Skills Care, Gitlin et al., 2003), strategies to address behavioral symptoms (Advancing Caregiver Training, Gitlin et al., 2010), and activities to effectively engage (Tailor Activity Program, Gitlin et al., 2009; Counseling and Support, Mittleman et al., 2004).

The caregiver journey is formidable. It starts early in degenerative diseases with suspicions and fear. It unfurls in complicated and downward trajectories. There are challenges at every level – new information needed, new demands required, and new needs to be addressed. It is ineluctable. Importantly, problems alter across time.

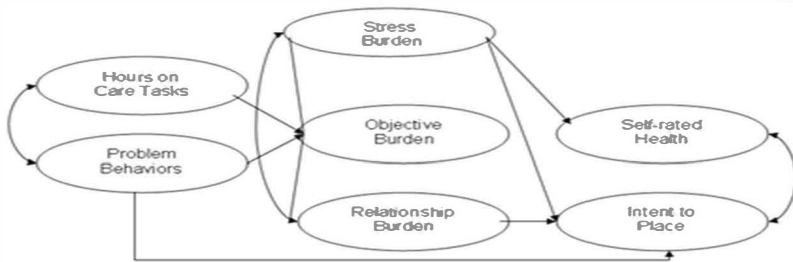
Recall too that the stress part of the equation is most telling and impactful. Burden is a multidimensional construct (Savundranayagam et al., 2011). Therefore, multiple dimensions require evaluation. The model below shows that dimensions of burden are conceptually independent constructs as well as showing two outcomes, a plan to institutionalize and caregiver rated health. This model draws attention to the manner in which the three types of burden differentially affect caregivers. The negative subjective appraisal of burden, daily care bother, and behavioral bother are key. Burden is best thought of as worry or anxiety. Daily care bother is the hassle experienced by the caregiver as a result of providing assistance with ADLs and IADLs. This results of changing dilemmas from those related to IADLs such as shopping and finances, through to those related to ADLs such as bathing and dressing. Behavioral problems involve memory related issues that can be disruptive. They also involve negative related behaviors such as repetition, being aggressive, and crying. Behavioral and psychological problems (behavioral and psychological symptoms in dementia [BPSD]) are modal: often the strain cannot be tolerated and causes long term care entry.

Results show that these subjective appraisals are highly predictive of caregiver emotional and physical health outcomes and may have a better predictive power than objective counterparts. Burden (subjective) is the only construct related to self-rated health and intent to place in a setting. Workload is not the major predictor of outcomes. The relationship between problem behaviors and intent to place make sense (problems behaving, you must go). Note the absence of a relationship between objective burden and any of the outcomes. Relationship problems do, however, mean something (intent to place increases). Stress burden, for

example, is a problem with self rated health. Care plans that stress this intervention (stress management) may work for health ratings. If the goal is to reduce the possibility of placement, problem behaviors are key, but relationship issues and stress burden are also involved.

The challenge is to manage the dance of internal turmoil and external demands. It is a balance of self-care, role acceptance, reduction of avoidance, involvement with people, and being on guard against cynical, negative attitudes. The challenge too is to avoid compassion fatigue (when you have cared for others more than yourself). The important point is that objective problems are encased in subjective ones – subjective views mediate objective issues and health. The caregiver has hope and options.

Model for Spouse Caregivers



Below is the broad view – what works, beliefs that are problematic, prototype tasks for the caregiver, and adaptive coping.

What Works

- Psychoeducation
- Counseling
- Multimodal input (team) with individual targets
- Adult Day Care as Respite
- Caregiving in Specialized Memory/Special Clinic

Problem Beliefs

- Problem will go away or is temporary
- I am not sure about this; ambivalence: forced into role
- I will make the situation worse
- I do not have the capacity for this
- I am stuck with his/her behaviors (repetition)
- My health suffers

- I have no context (psychoeducation)
- I have no help for really bad behavior

Prototype Tasks

- Acknowledging the disease,
- Making the cognitive shift,
- Developing emotional tolerance,
- Taking control,
- Establishing a realistic goal,
- Gage recipient's capacities,
- Designing opportunities for satisfying work,
- Becoming a sleuth

Adaptive Coping

- Accept condition; ask for help
- Learn to be aware
- Don't hide or run from negative feelings (fear, anger, sadness)
- Observe, don't judge
- Seek social support: turn to friends, support people
- Positive reappraisal: minimize negative, emphasize positive
- Downward comparison: compare self to less fortunate

Problem beliefs are especially noteworthy as they are reality-based but can be changed. Caregiver attributions matter then. Polenick et al. (2018) isolated 5 categories, based on focus groups, of concerns for behavioral and psychological symptoms of dementia. These were neurological disease factors, physical symptoms or comorbid health conditions, psychological reactions to dementia, shifting social ties and relationships following a dementia course, and environmental changes, such as lack of routine and medical transitions (hospitalization). Despite this seemingly multifactorial attribution to behavioral and psychological symptoms of dementia etiology, a number of respondents also indicated that these symptoms were at least partly within the care recipient's control. Family caregivers, therefore, attribute these behavioral problems to a range of care recipient and environmental factors. Caregivers' own causal beliefs about the behavioral and psychological symptoms may reflect then unmet educational needs that should be considered in the development of targeted interventions to minimize caregiver stress.

Dementia caregivers are also more likely to experience depression, social isolation, obesity, and increased levels of cortisol and reduced antibodies. A particular interest is whether a spouse who handles dementia

accelerates their own cognitive decline. This was evaluated by Dassel, Carr, and Vitaliano (2015). Vitaliano and colleagues (2011), in their Chronic Stress Model, proposed that spousal dementia caregivers were at additional risk for cognitive problems over demographically similar non-caregivers because of: psychosocial factors – that is depression, poor sleep, and social isolation; behavioral factors – that is sedentary lifestyle and poor diet; and physiological factors – that is increased inflammation and metabolic abnormalities associated with caring for a loved one with dementia. These could also place spousal dementia caregivers at increased risk for cognitive decline themselves, a phenomenon the authors referred to as Irony Tragedy. Additional factors that place spousal dementia caregivers at risk for cognitive decline include their advanced age and associated increased risk of dementia. Caregivers with fewer vulnerabilities and more resources may be more likely to experience positive benefits on aging.

Finally, a real connection with life or the identified patient does something curative. Cheng et al. (2014) assessed the effects of benefit-finding interventions, which are a key feature being used in gain-focused reappraisal strategies, to find positive meanings and benefits in caring for someone who has a dementia. Although the risk of dementia caregiving is very stressful, the level of stress felt, as noted, is not solely determined by the objective stressor, such as the care of the recipient's behavior problems, but also by the way the caregiver evaluates the situation. Benefit-finding involves key intervention activities such as exercises where the caregiver would nominate situations they had found stressful and attempt to provide alternative positive reappraisals. Training for caregivers in this context involved work to reframe positive issues to downplay physiological symptoms of dementia, recognizing emotional reactions in caregiver situations, stress management and muscle relaxation, caregiver self-care, activity scheduling for self and care recipient, and tactics to manage psychological behavioral problems. Importantly, the caregiver who loves or is committed to the care-receiver has a substantial emotional and psychological advantage because problems that result are seen as loving tasks. Positive growth might even come about as a result of a meaningful and acceptable challenge.

Life adjustment is the only domain that is not followed up with any formal testing. The core issues are followed but in general the life situation is handled or not. Interventions are of course made over time. Life domain issues are practically addressed. Follow-up entails a monitoring of real life problems.

Conclusion

We have discussed many variants of life adjustment. We have prosecuted the position that the real world is a challenge at later life. In this context little things matter, a reasoned program of living can make a difference, and prevention counts, a lot. These “little things” are the very variables that cause a problem or make it worse. If someone is trapped in a home, has few resources, is poor, well.... A practical accounting of the case is a necessity, left not to the discharge nurse but to the health care provider in the clinic. Little things – from stress, living, socialization, supports, the environment, ability to function, meaning in life, and ambient living – matter. Real living problems translate to real problems and poorer quality of life. It takes a village...

Life adjustment is hard to measure, but its value is easy to see. Just having one’s license taken or losing health can matter to such a degree that one’s life changes measurably. Interventions in this domain are rather impactful. In the context of day-to-day living, prevention works and information is power. But, living long enough, all will require help. Tritely, the older adult lives somewhere day-to-day. “Where” is matters. An awareness of the life adjustment variables allows for interventions even at the first level of prevention. The implementation of a collaborative care model among community-dwelling older people using life adjustment risk assessment instruments can result in better health behaviors and can increase the use of holistic care.

References

- Baltes, P. B., & Baltes, M. M. (1990). Psychological perspectives on successful aging: The model of selective optimization with compensation. *Successful aging: Perspectives from the behavioral sciences*, 1(1), 1-34.
- Bass, D. M., Judge, K. S., Snow, A., Wilson, N. L., Morgan, R. ●., Maslow, K., ... & Elbein, R. (2014). A controlled trial of Partners in Dementia Care: veteran outcomes after six and twelve months. *Alzheimer’s research & therapy*, 6(1), 9.
- Blount, A., Schoenbaum, M., Kathol, R., Rollman, B. L., Thomas, M., ●’donohue, W., & Peek, C. J. (2007). The economics of behavioral health services in medical settings: A summary of the evidence. *Professional Psychology: Research and Practice*, 38(3), 290.

- Brandtstädter, J., Rothermund, K., Kranz, D., & Kühn, W. (2010). Final decenterations: Personal goals, rationality perspectives, and the awareness of life's finitude. *European Psychologist, 15*(2), 152.
- Brinda, E. M., Rajkumar, A. P., Attermann, J., Gerdtham, U. G., Enemark, U., & Jacob, K. S. (2016). Health, social, and economic variables associated with depression among older people in low and middle income countries: world health organization study on global AGEing and adult health. *The American Journal of Geriatric Psychiatry, 24*(12), 1196-1208.
- Brodaty, H., & Arasaratnam, C. (2012). Meta-analysis of nonpharmacological interventions for neuropsychiatric symptoms of dementia. *American Journal of Psychiatry, 169*(9), 946-953.
- Cheng, S. (2014). Double compression: A vision for compressing morbidity and caregiving in dementia. *The Gerontologist, 54*(6), 901-908.
- Choi, H., Yorgason, J. B., & Johnson, D. R. (2015). Marital quality and health in middle and later adulthood: Dyadic associations. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 71*(1), 154-164.
- Cogbill, E., Dinson, K., & Duthie, E. (2010). Considerations in prescribing medication to the elderly. In V. Hirth, D. Weiland, & M. Dever-Bumba (Eds.), *Case-based geriatrics: A global approach*. New York, NY: McGraw-Hill.
- Comijs, H. C., van den Kommer, T. N., Minnaar, R. W., Penninx, B. W., & Deeg, D. J. (2011). Accumulated and differential effects of life events on cognitive decline in older persons: Depending on depression, baseline cognition, or ApoE ε4 status?. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 66*(suppl_1), i111-i120.
- Dassel, K. B., Carr, D. C., & Vitaliano, P. (2015). Does caring for a spouse with dementia accelerate cognitive decline? Findings from the Health and Retirement Study. *The Gerontologist, 57*(2), 319-328.
- Diehr, P.H., Thielke, S.M., Newman, A.B., Hirsch, C., & Tracy, R. (2013). Decline in health for older adults: Five-year change in 13 key measures of standardized health. *Journals of Gerontology Series A: Biological Sciences and Medical Sciences, 68*(9), 1059-1067.
- Douma, L., Steverink, N., Hutter, I., & Meijering, L. (2017). Exploring subjective well-being in older age by using participant-generated word clouds. *The Gerontologist, 57*(2), 229-239.
- Heckhausen, J., Wrosch, C., & Schulz, R. (2010). A motivational theory of life-span development. *Psychological review, 117*(1), 32.

- Evans, W., Rosenberg, I. H., & Thompson, J. (1991). *Biomarkers: the 10 determinants of aging you can control*. Simon & Schuster.
- Family Caregiver Alliance. (2006). *Caregiver Assessment: Voices and Views from the Field. Report from a National Consensus Development Conference (Vol.II), II*.
- Farias, S. T., Mungas, D., Reed, B. R., Cahn-Weiner, D., Jagust, W., Baynes, K., & DeCarli, C. (2008). The measurement of everyday cognition (ECog): scale development and psychometric properties. *Neuropsychology, 22*(4), 531.
- Galvin, J.E., Roe, C.M., Powlishta, K.K., Coats, M.A., Muich, S.J., Grant, E. Miller, J.P., Storandt, M., & Morris, J.C. (2005). The AD8 a brief informant interview to detect dementia. *Neurology, 65*(4), 559-564.
- Gitlin, L. N., & Hodgson, N. (2015). Caregivers as therapeutic agents in dementia care: the context of caregiving and the evidence base for interventions. In *Family caregiving in the new normal* (pp. 305-353).
- Gitlin, L. N., Winter, L., Corcoran, M., Dennis, M. P., Schinfeld, S., & Hauck, W. W. (2003). Effects of the home environmental skill-building program on the caregiver-care recipient dyad: 6-month outcomes from the Philadelphia REACH initiative. *The Gerontologist, 43*(4), 532-546.
- Gitlin, L. N., Winter, L., Dennis, M. P., Hodgson, N., & Hauck, W. W. (2010). A biobehavioral home-based intervention and the well-being of patients with dementia and their caregivers: the COPE randomized trial. *Jama, 304*(9), 983-991.
- Gitlin, L. N., Winter, L., Dennis, M. P., Hodgson, N., & Hauck, W. W. (2010). Targeting and managing behavioral symptoms in individuals with dementia: a randomized trial of a nonpharmacological intervention. *Journal of the American Geriatrics Society, 58*(8), 1465-1474.
- Gitlin, L. N., Winter, L., Vause Earland, T., Adel Herge, E., Chernet, N. L., Piersol, C. V., & Burke, J. P. (2009). The Tailored Activity Program to reduce behavioral symptoms in individuals with dementia: feasibility, acceptability, and replication potential. *The Gerontologist, 49*(3), 428-439.
- Gliklich, R. E., & Dreyer, N. A. (2010). *Registries for Evaluating Patient Outcomes: A User's Guide*. 2nd edn (Prepared by Outcome DECODE Center [Outcome Sciences, Inc. d/b/a Outcome] under Contract No. HSA29020050035I T03.) AHRQ Publication No. 10-EHC049 Agency for Healthcare Research and Quality: Rockville, MD, USA.
- Goeres, L. M., Gille, A., Furuno, J. P., Erten-Lyons, D., Hartung, D. M., Calvert, J. F., ... & Lee, D. S. (2016). Rural-Urban Differences in

- Chronic Disease and Drug Utilization in Older Oregonians. *The Journal of rural health*, 32(3), 269-279.
- Harvard Health (2018). The health benefits of shared living. *Harvard Health Letter*, 43(7), 1-8.
- Heintzelman, S. J., & King, L. A. (2014). Life is pretty meaningful. *American Psychologist*, 69(6), 561.
- Hyer, L. (2014). *Psychological treatment of older adults: A holistic model*. Springer Publishing Company.
- Jeste, D. V., Blazer, D. G., Buckwalter, K. C., Cassidy, K. L. K., Fishman, L., Gwyther, L. P., ... & Vega, W. A. (2016). Age-friendly communities initiative: public health approach to promoting successful aging. *The American Journal of Geriatric Psychiatry*, 24(12), 1158-1170.
- Kok, A. A., Aartsen, M. J., Deeg, D. J., & Huisman, M. (2017). Capturing the diversity of successful aging: an operational definition based on 16-year trajectories of functioning. *The Gerontologist*, 57(2), 240-251.
- Krause, N., & Hayward, R. D. (2014). Church-based social support, functional disability, and change in personal control over time. *Journal of religion and health*, 53(1), 267-278.
- Lichtenberg P. & Mast, B. (2015). *APA Handbook of Clinical Psychology (Vol 2): Assessment, Treatment and Issues of Later Life*. APA Press; Washington, DC.
- Lum, T. Y., Lou, V. W., Chen, Y., Wong, G. H., Luo, H., & Tong, T. L. (2014). Neighborhood support and aging-in-place preference among low-income elderly Chinese city-dwellers. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 71(1), 98-105.
- Martin, K. L., Blizzard, L., Srikanth, V. K., Wood, A., Thomson, R., Sanders, L. M., & Callisaya, M. L. (2013). Cognitive function modifies the effect of physiological function on the risk of multiple falls—a population-based study. *Journals of Gerontology Series A: Biomedical Sciences and Medical Sciences*, 68(9), 1091-1097.
- Maslow, K. (2012). Translating innovation to impact: Evidence-based interventions to support people with Alzheimer's disease and their caregivers at home and in the community. *A white paper*.
- Meuser, T. (2015). Driving and the transition to nondriving mobility: Change process and opportunities for intervention (499-521), In P. Lichtenberg and B. Mast *APA Handbook of Clinical Psychology (Vol 2): Assessment, Treatment and Issues of Later Life*. APA Press; Washington, DC.
- Mittleman, M. S., Roth, D. L., Haley, W. E., & Zarit, S. H. (2004). Effects of a caregiver intervention on negative caregiver appraisal of behaviors

- problems in patients with Alzheimer's disease: Results of a randomized trial. *The Journal of Gerontology Series B: Psychological Sciences and Social Sciences*, 59(1), 27-34.
- Mota, N. P., Tsai, J., Kirwin, P. D., Sareen, J., Southwick, S. M., & Pietrzak, R. H. (2016). Purpose in life is associated with a reduced risk of incident physical disability in aging US military veterans. *The American Journal of Geriatric Psychiatry*, 24(9), 706-714.
- Nagi, S. Z. (1977). The disabled and rehabilitation services: A national overview. *Am Rehabil*, 2(5), 26-33.
- Nahemow, L., & Lawton, M. P. (1973). Toward an ecological theory of adaptation and aging. *Environmental design research*, 1, 24-32.
- Nash, S. H., Liao, L. M., Harris, T. B., & Freedman, N. D. (2017). Cigarette smoking and mortality in adults aged 70 years and older: Results from the nih-aarp cohort. *American journal of preventive medicine*, 52(3), 276-283.
- Nummenmaa, L., et al. (2012). Emotions promote social interaction by synchronizing brain activity across individuals. *Proceedings of the National Academy of Sciences of the United States of America*, 109(24), 9599-9604.
- Parisi, J. M., Roberts, L., Szanton, S. L., Hodgson, N. A., & Gitlin, L. N. (2015). Valued activities among individuals with and without cognitive impairments: Findings from the National Health and Aging Trends Study. *The Gerontologist*, 57(2), 309-318.
- Pfeffer, R. I., Kurosaki, T. T., Harrah Jr, C. H., Chance, J. M., & Filos, S. (1982). Measurement of functional activities in older adults in the community. *Journal of gerontology*, 37(3), 323-329.
- Polenick, C. A., Struble, L. M., Stanislawski, B., Tumwald, M., Broderick, B., Gitlin, L. N., & Kales, H. C. (2018). "The Filter is Kind of Broken": Family Caregivers' Attributions About Behavioral and Psychological Symptoms of Dementia. *The American Journal of Geriatric Psychiatry*, 26, 548-556.
- Putnam, R. D. (2016). *Our kids: The American dream in crisis*. Simon and Schuster.
- Samus, Q. (2016). Maximizing independence at home for people with dementia: Design, outcomes, and future directions. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 12(7), P216-P217.
- Savundranayagam, M. Y., Montgomery, R. J. V., & Kosloski, K. (2011). A dimensional analysis of caregiver burden among spouses and adult children. *Gerontologist*, 51(3), 321-331.
<http://doi.org/10.1093/geront/gnq102>

- Scharf, L., Bell, P. A., & Smith, S. A. (2006). Number of training sessions does not change program effectiveness: Evaluation of formats of one, two, or three sessions for the Alzheimer's Savvy Caregiver Training Program. *Dementia, 5*(4), 559-569.
- Schinka, J. A., Raj, A., Loewenstein, D. A., Small, B. J., Duara, R., & Potter, H. (2010). The cognitive change checklist (3CL): cross-validation of a measure of change in everyday cognition. *International journal of geriatric psychiatry, 25*(3), 266-274.
- Spalter, T., Brodsky, J., & Shnoor, Y. (2013). Improvements and decline in the physical functioning of Israeli older adults. *The Gerontologist, 54*(6), 919-929.
- Stawski, R. S., Sliwinski, M. J., & Smyth, J. M. (2006). Stress-related cognitive interference predicts cognitive function in old age. *Psychology and aging, 21*(3), 535.
- Vahia, I. V., Chattillion, E., Kavirajan, H., & Depp, C. A. (2011). Psychological protective factors across the lifespan: implications for psychiatry. *Psychiatric Clinics, 34*(1), 231-248.
- Verghese, J., Lipton, R. B., Katz, M. J., Hall, C. B., Derby, C. A., Kuslansky, G., ... & Buschke, H. (2003). Leisure activities and the risk of dementia in the elderly. *New England Journal of Medicine, 348*(25), 2508-2516.
- Vitaliano, P. P., Murphy, M., Young, H. M., Echeverria, D., & Borson, S. (2011). Does caring for a spouse with dementia promote cognitive decline? A hypothesis and proposed mechanisms. *Journal of the American Geriatrics Society, 59*(5), 900-908.
- Wolff, J. K., Lindenberger, U., Brose, A., & Schmiedek, F. (2014). Is available support always helpful for older adults? Exploring the buffering effects of state and trait social support. *Journals of Gerontology Series B: Psychological Sciences and Social Sciences, 71*(1), 23-34.
- World Health Organization. (1946, June). Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity. In *International Health Conference, New York* (pp. 19-22).

CHAPTER 11

PERSONALITY

Personality is a friendly construct that allows for an understanding of the consistency of a person's behavior through stressful and non-stressful times. This occurs across the lifespan. It provides clarity of treatment action in managed care formulations. Personality traits in particular represent one non-genomic area of consideration in the formation of consistent behaviors based on a broad swath of genetic and environmental factors. Personality traits can also be assessed inexpensively and noninvasively. They may also indicate susceptibility to cognitive dysfunction long before noticeable clinical signs. In sum, they provide a window into the logic of the patient's actions and patterns of behavior. It is indeed a deep phenotype of intra-personal and extra-personal activity. Knowing personality is a way of shining a light on a person process and helps that person validate their own ways of being and belonging in the world.

Simplistically, personality can be divided into traits and personality disorders (PDs). We will discuss both. Both are heuristic constructs that inform and often direct; they discover and unfold the person in subtle and loud ways, ones to be followed. They are not diseases. They are complex and intra- and inter-active, and are understandable reflectors of the person, and habits that are often unduly rigid. Given the models, the patterns are also logical. They are persistent, but decipherable. Health care providers seem to know when someone has a particular trait but they certainly have a good idea when someone has a PD; often PD patients are seen as willful and unpleasant, often just rigid. In some sense it is the health care provider that is reactive and thereby takes note of the "personality" of the identified patient.

Personality at later life alters in slight ways with age. Unfortunately, the aging process has many other positive or problem components. The reality is that personality bends but does not break (heterotypic continuity). Personality is a mediator of the cohort-specific issues of living long – love, care, wisdom – and the unpacking of this gift is forever similar but new, and (even better) understandable. Personality is also

responsive to the ravages of living – health declines of all sorts. It often exaggerates its core trait features. Again, the sketching of decline in aging is evident but the painting lusters with the construct of personality. What's more, the construct of PD has many miles to go before its final understanding. In time, research and wisdom will reveal this. For now, it is a presence that allows us as health care providers to take as good a glimpse into personhood and behavior.

Dan McAdams, a leading personality theorist, asks a critical question regarding personality: "What do we know when we know a person?" We believe that personality is the most efficient and economical path to answering this question. It provides an understandable and human template for both clinical reasons (treatment) and human reasons (meaning making). At late life there may be no more important construct. Hopefully, future research will provide better measures and models for its understanding.

In this chapter we first provide an overview of personality at late life with lifespan implications. We consider the typology of personality, as a trait and disorder. We emphasize the five factor model. We assess according to the DSM-IV model, however. We consider the improved state of analysis for personality. We then address personality disorders and consider their value at late life. We also consider resilience as a distinct feature of a person connected to personality. Emotionality as an aspect of resilience is also addressed. We end with a discussion on assessment and treatment. Three cases specific to personality are provided. At the end a Watch and Wait model case is presented.

Overview

Personality psychology has long exhibited a tension between, on the one hand, trying to uncover commonalities and differences among people and, on the other, understanding the intricacies and motives within which specific individual lives are formed. The former approach is sometimes identified as nomothetic and the latter idiographic. Given the different focus of each approach and the broad nature of people's behaviors, it is not surprising to find a vast array of personality theories. In the past decade, however, the emergence of personality psychology has been characterized by arguments based more on empirical findings than abstract theory. These empirical findings have involved consistencies in traits, as well as more in-depth components regarding the individual nature of personality.

Typologies have had several starts. Some 60 years ago Eysenck (1967) proposed an endogenous model of personality. Early on, the focus was on

how the trait dimensions of extroversion and neuroticism were rooted in biological substrates, such as the limbic system and the subsequent effects of arousal and behavior. Later, with developments and the understanding of genetics, DNA became the distal antecedent of personality traits (Eysenck, 1997). In other words, DNA and other biological factors determine the brain structures to which personality traits are linked. The primary genetics are integral to the biological essentialist perspective – for good reason given the strong evidence for its effects. Across studies, usually about 40-60% of the variance in personality is attributed to genetics (Kreuger, Johnson, and Kling, 2006). This is often considered to be temperament. Indeed, temperamental structures have also been found to have strong genetic influences, but not all the genetic variance between traits and temperament is shared. In contrast, shared environmental effects (for example living in the same household) have been found to account for almost none of the variation of these traits.

Dan McAdams (again) argued for a richer model. He has proposed a multilevel theory of personality that involves a contextually sound characteristic adaptation level consisting of diverse sets of constructs, such as goals, schemas, and values. Similarly, Hooker and McAdams (2003) outlined an aging research agenda that specified several issues pertinent to personality in later life. They proposed a structure and process model noting six foci of personality. This includes traits (dispositional signatures), personal action constructs (goals or motivations), and life stories (narrative reflections of self) as structural components, as well as short term states, such as within-person changes (emotions), self-regulation (primary and secondary control processes or assimilative or accommodative procedures), and self-narration (remembering) as the parallel process constructs. These constructs are distinct from those identified in most research on younger adults and allow the clinician to assess how persons at late life “become increasingly like themselves” (Neugarten, 1964).

Perhaps the most widely known of all these is the five-factor model or the Big 5. This has a long history but has been made popular and operational by McCrae and Costa (2003). The traits that compose this model are neuroticism, extroversion, openness to experience, agreeableness, and conscientiousness. Wilson et al. (2007) found that higher levels of conscientiousness, one of the classic Big 5 personality traits characterized by organization, self-discipline, dependability, and a drive to achieve, also afforded protection in life. Participants in a religious order who were assessed and scored in the 90th percentile on conscientiousness had an 89% reduction in the risk for developing AD. This was whether or not they had plaques and tangles. Importantly, lifestyle factors, education especially,

contaminated this finding as this variable has an impact on the decline process. Clearly, conscientiousness leads to more education. These variables interface with personality traits to allow what appears to be a unique expression of behaviors and emotions by the person.

This model of personality has a special application for older adults. Mroczek and Spiro (2003), for example, followed a sample of 1600 men from the Normative Aging Study for 12 years and found curvilinear slopes for neuroticism (N), which declined up to age 80, and an overall linear trajectory for extraversion (E) indicating no average change. Extraversion declined in subjects older than 80 years. As with many such studies, there were considerable intra-individual differences. In one other study, Terracciano, McCrae and Costa (2006) examined personality traits over a 42 year period by using data from the Baltimore Longitudinal Study on Aging (N = 2,359; individuals aged 17-98 collected between 1958 and 2002). Hierarchical linear modeling analysis revealed cumulative mean-level changes averaging about .5 SDs across adulthood. Scores on E showed distinct developmental patterns; activity declined from ages 60-90 years, restraint increased, 'ascendancy' peaked at 60, and sociability declined slightly. Scales related to N showed curvilinear declines up to age 70 and increased thereafter. Agreeableness (A) and Openness to Experience (O) changed little. Masculinity declined linearly. Once again, there was significant individual variability in change. Men and women were similar in variability and death rates, as well as attrition slopes.

Major research on the association between personality and cognition has focused on neuroticism and openness to experience, as well as anxiousness. The association with cognition is only apparent in midlife and old age. Relationships between personality and cognition are more likely to be significant and consistent in middle age and older adults than in younger adults. Studies reporting on positive association between openness to experience and cognitive ability include the Normative Aging Study and the Baltimore Longitudinal Aging Study, among others. Booth et al. (2006) showed a significant positive relationship between measures of verbal memory and general cognitive ability and openness in a sample of older adults. The association between neuroticism and depression has been dialogued for many years, especially as it applies to cognition. Among depressed subjects, those with higher neuroticism showed even lower cognitive scores than depressed individuals with average neuroticism scores. Longitudinal research has suggested that dementia risk is elevated in those who have higher neuroticism.

The life course epidemiological approach also suggests that personality may have life-long pathways of influence on risk of dementia and MCI.

There is a growing literature showing that individual differences in personality traits, such as openness, conscientiousness, neuroticism, and self-efficacy beliefs, impact cognitive intervention effectiveness in both younger and older adults. For example, self-efficacy beliefs about memory ability in older adulthood predict individual differences in cognitive training outcomes. Personality should be considered also as a predictor. In a recent meta-analysis that linked traits such as conscientiousness, a disposition favoring self-control, organization, and industriousness, there resulted a reduced risk of incidence for AD. This may hold particular importance for predicting patterns of cognitive aging because conscientious individuals may engage in more memory recall and long-term planning.

Low, Harrison, and Lackersteen (2013) assessed 12 longitudinal and 3 case-controlled studies and looked for personality affect markers in dementia. Five of the nine studies found that higher neuroticism was associated with greater dementia risk and two studies showed that it increased the risk of MCI. Three of four studies showed that higher conscientiousness was protective against dementia and one of two studies suggested it reduced the risk of MCI. Three of four studies found no association between openness and dementia. However, pooled data suggested openness was protective. Seven studies indicated that extraversion was not linked with dementia and four found no association between agreeableness and dementia. In sum, neuroticism increases the risk of dementia and conscientiousness seems to reduce the risk.

Dar-Nimrod noted the moderating effect of the five factors in the relationship between genetic risk factors and cognitive decline in aging populations (Dar-Nimrod et al., 2012). They noted that lower levels of openness as well as neuroticism have been shown to be related to cognitive decline and AD, where extraversion and conscientious subjects are more connected to positive affect. The Terracciano group added that neuroticism and lower levels of conscientiousness have been associated with aggregate morbidity, HIV, and self-rated health and depression, as well as AD (Sutin, Zonderman, Ferricci & Terracciano, 2013). Of course the existence of chronic disease itself can have an impact on personality. Sutin et al. (2013) showed that personality traits confer a risk for disease, in large part through health behaviors.

Roberts and DelVecchio (2000) examined the rank-order stability of traits from childhood to older adulthood. While aggregating a variety of traits, including the Big 5, results showed a linear increase in consistency across lifespan. A pronounced shift in trait consistency occurred between childhood and young adulthood, followed by an increasing consistency until middle age, and similar stability for those aged 60 and older. Roberts,

Walton, and Viechtbauer (2006) conducted another meta-analysis, in this case focusing on mean-level of change among the Big 5. The trait of extraversion was broken into two facets of social dominance and social vitality; social dominance reflects independence and self-confidence an increase from young adulthood to age 40, thereafter remaining stable. In contrast, social vitality, which reflects sociability and positive affect, showed a slight decrease in young adulthood from its peak in adolescence, followed by stability in middle adulthood and again declining in later adulthood. There are other changes with agreeableness and conscientiousness, as well as openness. These meta-analyses provide then a useful framework by which to consider how personality exhibits significant stability and can be broken down, while also demonstrating some change.

Generally speaking, neuroticism goes down across middle adults, although evidence has suggested that it begins to rise again in individuals who are 70 and older. Most studies have found a rise in agreeableness and conscientiousness until at least middle age, though thereafter the results are less conclusive. In contrast, there appears to be a slight increase in openness to experience from younger middle adults. Trajectories of extraversion reveal how adults often vary between stability and slight decline. No doubt any differing results are a function of different facets of extraversion and the manner in which they change over the course of a life. Finally, neuroticism has been associated with global as well as prefrontal and medial temporal cortical atrophy but is unassociated with B-amyloid and tau (Chapman, 2017). It is most associated with vulnerability to stress as a risk factor for poor depression treatment response and for cognitive decline.

FF Model

●verall

The five factors involved are neuroticism, openness to experience, extraversion, agreeableness, and conscientiousness. These represent the combined influence of genetics and of social and physical environments. They are reasonable predictors of health-related behaviors and morbidity and seem to be most influential when the gene-disease relationship is multi-determined.

- penness to experience: Appreciation for art, adventure, unusual ideas, curiosity, and variety of experience. ●penness reflects the degree of intellectual curiosity and creativity, and the preference for novelty and variety a person has. High openness can be perceived as unpredictability or lack of focus. Moreover, individuals with high openness are said to pursue more high risk pursuits. Conversely, those

with low openness seek to gain fulfillment through perseverance, and are characterized as pragmatic and data-driven – sometimes even perceived to be dogmatic and closed-minded.

- **Conscientiousness:** A tendency to be organized and dependable, show discipline, aim for achievement, and prefer planned rather than spontaneous behavior. High conscientiousness is often perceived as stubbornness and obsession. Low conscientiousness is associated with flexibility and spontaneity. This trait has an impact on mortality and quality of life. Hauser and Palloni (2014) noted that rank and high school class is a marker of this. This also correlated with both IQ and academic performance and is implicated in later life health and survival. Conscientiousness represents the personal characteristics, habits, tendencies, and behaviors that lead to academic success. Conscientiousness would appear to be a key factor in this regard and ideally this would represent a longitudinally better response to life.
- **Extraversion:** Energy, positive emotions, assertiveness, sociability, and the tendency to seek adventure in the company of others, and talkativeness. High extraversion is often perceived as attention-seeking, and domineering. Low extraversion causes a reserved, reflective personality, which can be perceived as aloof or self-absorbed.
- **Agreeableness:** A tendency to be compassionate and cooperative. It is also a measure of one's trusting and helpful nature. High agreeableness is often seen as naive or submissive. Low agreeableness personalities are often competitive or challenging to people, which can be seen as argumentative or untrustworthy.
- **Neuroticism:** The tendency to experience unpleasant emotions easily, such as anger, anxiety, depression, and vulnerability. Neuroticism also refers to the degree of emotional instability and poorer impulse control. A low score reflects a need for stability and manifests itself as a stable and calm personality. A high N reflects a low need for stability that results in a reactive and excitable personality. The neurotic can be unstable or insecure. There is growing evidence that neuroticism moderates the magnitude of physiological responses to stressors. Neuroticism is most connected to vulnerability to stress ("I feel as if I am going to pieces") and impulsiveness ("I have trouble resisting my cravings"). Several studies suggest that individuals high in neuroticism have larger and more prolonged sympathetic responses to stressors, greater cardiovascular reactivity, and higher morning levels of cortisol. Neuroticism is also related to the disruption of circadian rhythms and is correlated with abnormalities of the immune system. Higher neuroticism is seen as being associated with more prolonged depression

of secretory immunoglobulin following a stressor, a typical response of natural distress, diminished antibody response to vaccination, and higher leukocyte counts. Persons high in neuroticism are markedly more likely to smoke, to become dependent on alcohol and other drugs, and to get into difficulty with life with issues such as cancer, asthma, emphysema, cardiovascular disease, and other diseases. Neuroticism seems to be unrelated to B-amyloid, tau protein, and Lewy Body pathology.

Finally, after we reach our 50s and 60s most of us become more conscientious, less competitive, more socially inhibited, and better able to cope with challenges. Most personality changes are beneficial. The sharp edges also seem to mellow over time. Changes also have a strong impact on one's sense of satisfaction, more so than income, job status, and marital status.

Age-Related Personality Tendencies or Changes

1. Greater agreeableness
2. Greater emotional stability
3. Greater conscientious
4. Better able to cope with negative information
5. Greater self-transcendence
6. Increased harm avoidance
7. Greater social inhibition
8. Greater spirituality
9. Less open to new experience
10. Decreased neuroticism

Personality Disorders

Personality disorders (PDs) are another matter. They are best represented as polythetic, as prototype markers of core traits with additional sequelae. They assert a rather strong influence over Axis I problems. PDs (or rigidity in traits) “suggest” the form of psychopathology as a result of stress, implying that Axis II and Axis I (DSM-IV terms) are cohabitators and intermingle in predictable ways. Stress makes this influence especially noteworthy.

The relationship in later life between Axis II and Axis I disorders is unknown. Several studies have suggested that co-morbidity with Axis I disorders is high. For example, Thompson, Gallagher, and Czirr (1988) reported that 33% of depressed older adults met criteria for a PD using

structured interviews. Thompson, Gallagher, & Czirr (1988) determined that 33% of depressed older adults being treated with psychotherapy in a geropsychiatric outpatient clinic also met DSM-III PD criteria. Fogel and Westlake (1990) included 2,332 inpatients with major depression and found that 15.8% also met criteria for a PD. Kunik et al. (1993) evaluated depressed older adults as inpatients and found 24% had a comorbid PD. Even higher rates have been reported. The high comorbidity is problematic for at least two reasons: (1) comorbidity causes even more complications with treatment; and (2) at least ¼ of individuals with one PD will have other PDs (Segal et al., 2006). PD co-morbidity is the rule among younger adults (generally > 50%) but how this applies to older adults is less well known.

From a broader perspective older people with PDs have significant problems with relationships in particular. They have difficulty with family members and friends. As noted, people with PDs are also more likely than those without to develop Axis I problems, especially depression and anxiety. Research also identifies personality pathology as a risk factor for adverse physical (heart rate, arthritis, obesity) and other mental health problems and outcomes (Segal, Marty, Meyer, & Coolidge, 2012), especially for middle age and older samples. Recent research has begun to link PDs in older samples with hospital admissions, increased healthcare use, and reduced health quality of life (Powers and Oltmanns, 2012).

Despite the importance of PDs, the research has been relatively light regarding older adults. The results of many cross-sectional studies indicate that PDs are less prevalent in older adults than they are in younger adults (Casey & Schrodt, 1989; Gutierrez et al., 2012.), suggesting that PDs diminish or burn out with age. In each of these studies, either PDs or personality traits were found to be less prevalent or less severe in older adults compared with younger adults. There is other evidence that PDs may actually increase with age. This is because PDs can emerge or become functionally problematic for the first time in later adulthood. This is not to suggest that older people possess latent personality pathologies that now meet criteria for the diagnosis for the first time in later life. Late life obviously presents different stressors. This would apply especially to people who have narcissistic needs, people who are avoidant, and people who are more schizoid, as well as people who are dependent.

Gould and Hyer (2004) have suggested that patients with dementia assert their pathology by extending premorbid personality traits (e.g., neuroticism). These have an impact on their behavior profile during the course of the illness. They assessed the influence of premorbid personality on the expression of behavioral disturbances among individuals with

dementia. A total of 68 outpatients with a diagnosis of dementia were assessed for current cognitive functioning, premorbid personality traits, and new behavioral disturbances since dementia onset. The results of this study showed some evidence for the influence of personality on the expression of both irritability and withdrawal. Specifically, increased withdrawal and irritability after dementia onset were associated with an inhibited premorbid personality style. In addition, a premorbid independent personality style was associated with less withdrawal. Results suggested that personality may be a value-added construct in the explanation of problem behaviors for those who are dementing.

Caselli et al. (2018) further noted that behavioral problems with individuals with AD present management challenges. The current prevention strategies are usually anchored to cognitive outcomes, but behavioral outcomes may provide another clinically relevant opportunity for preemptive therapy. This was sought in this study regarding personality changes that predispose to behavioral disorders. Members of the APOE-4 genetically enriched cohort at the Maricopa County Residence who were neuropsychologically healthy at entry were included. Over an interval of 7 years the NEO-PI-R neuroticism T-scores increased significantly more in MCI transitioners than in non-transitioners. Also included, openness was less in the MCI transitioners than nontransitioners. Concurrent subclinical but statistically significant changes in behavioral scores worsened more in MCI transitioners than nontransitioners for measures of depression, somatization, irritability, anxiety, and aggressive attitude. In sum, this group saw that personality and subclinical behavioral changes began during the transition from preclinical states to MCI and beyond.

Prevalence: Previously we reviewed PDs in community settings where there was considerable variability (Hyer, 2014). Older adults have been found to score lower than younger adults on scales of antisocial, histrionic, narcissistic, borderline, and paranoid PDs, while scoring higher on obsessive-compulsive and schizoid PDs (Cohen, Nestadt, Samuels, Romanoski, McHugh, & Rabins 1994; Segal, Hook, & Coolidge, 2001). In a study of 810 community-dwelling participants, Cohen et al. (1994) administered the semi-structured Standardized Psychiatric Examination to determine levels of PD in younger and older adults. Prevalence rate for the older adults (55 years and older) was 6.6% versus 10.5% for the younger adults, with older participants significantly less likely to have PDs. Segal and Coolidge (1998) found much higher prevalence rates in a study of 189 community-dwelling older adults (mean age = 76.2 years) attending a community center. More recently, Coolidge, Segal, and Rosowsky (2006) conducted a study of 681 community dwelling participants ranging in age

from 18 to 89. Prevalence was measured utilizing the CATI self-report and participants were compared in two groups; older ($n=114$, ages 60-89) and younger ($n=567$, ages 18-59). The study found PDs in 11% of the older sample and 20% of the younger group.

Schuster et al. (2013), using a National Epidemiologic Survey on Alcohol and Related Conditions, addressed a subsample of over 8,000 adults of 65 or older. A total of 8.07% of American adults had at least one PD. These adults tended to have less robust quality of life. Personality traits, on the other hand, may represent one non-genomic area of consideration in predictive models because they are the result of a swath of genetic and environmental factors and index a wide range of risk behaviors. They may also reflect susceptibility to cognitive dysfunction long before noticeable clinical signs.

In mental health settings, PDs in older adults show relatively low prevalence rates compared to younger groups. In one cross-sectional study of changes in personality with age for individuals with PD, Molinari, Kunik, Snow-Turek, Deleon, and Williams (1999) administered a battery of tests to 392 psychiatric inpatients diagnosed with a PD. The battery included: the Minnesota Multiphasic Personality Inventory, Millon Clinical Multiaxial Inventory, California Psychological Inventory, and Psychological Inventory of Personality Styles. The study concluded that older adults with PDs were consistently less impulsive, paranoid, high-energy, antisocial, and irresponsible compared to their younger counterparts. These findings support previous literature indicating a mellowing of PDs with age.

In studies conducted in nursing homes results have determined that 11% to 15% of that population meets criteria for a PD diagnosis (Margo, Robinson, & Corea, 1980; Teeter, Garetz, Miller, & Heiland, 1976). A reason for this dearth of information is both the absence of an appropriate assessment tool, and the absence of prototype markers of PD behavior in such a setting.

Two Expressions of Personality

The DSM-5 has of course altered the structure of personality: No more Axis II. Nonetheless, the interface between Axis II (personality) and Axis I (disorders) are worth understanding. One reason is that the interactions are multi-determined and varied, even if there is a consistency and “psycho”-logic to the consistency. There are in addition several related, orthogonal person variables that impact on the actual behavior. They are related to personality but represent features that allow expression in more

dramatic or obvious ways. Regardless, they represent behaviors that allow for personality expression.

While there are others, two seem to stand out. One is resilience. Reviews of aging literature identified resilience as a personality trait. It has been variously described as a process, or a phenomenon that develops over time and that relies on the integration of biological, psychological, and social cultural factors. Reviews in developmental psychology on resilience indicate that it fosters inner strength and meaning, as well as the capacity to learn from positive and negative outcomes. Resilience is then described as the individual's level of tolerance and ability to adapt positively to adversity and other challenging life situations. However, there is no universal agreement on the definition of the construct of resilience. Others have even speculated on a moral dimension, noting Personality H as one with core features of honesty, trust and competence (Lee et al., 2012).

One approach to resilience is more holistic and incorporates psychosocial cultural factors, including temporal, multidimensional, and multilevel processes, such as social competency and spiritual strength. This approach embraces a wider influence of local community, ecological, and macrosocial level factors. One study assessed the life narrative interviews of participants demonstrating a continuum of self-identity with an integrated task present and anticipated future (Browne-Yung, Walker, and Luszcz, 2017). There are many examples of redemptive episodes through the life course where participants reconvered negative events with positive outcomes. For example, self-mastery and self-reliance developed at times of grief provided coping skills for dealing with loss while maintaining continuity of self-identity. Life experiences resulting in redemptive events appeared to equip individuals with varying adaptive coping and instrumental skills and an awareness of personal capabilities. This suggests that resilience may increase following successful coping with adverse events at different stages of the lifecycle. Additionally, interventions that foster resilience (building optimism, emotional intelligence, acceptance, mindfulness, fostering positive relationships and health) appear especially to make a difference among older adults (Resilience, 2018).

One other variable is emotionality, especially emotional intelligence. On the down side, emotionality involves the penchant to poorly handle stress and emotions when present. Emotions are read less well and experienced more negatively, and outcomes are more problematic. Research typically shows that the emotion system maintains itself into old age, with the capacity to experience and express emotions (both positive and negative) remaining intact. Older adults report greater effort to control

the experience of emotion in their daily lives (Gross, Carstensen, Pasupathi, Tsai, Skorpen, & Hsu, 1997; Lawton, Kleban, & Rajagopal, 1992). Research with community-dwelling older adults consistently finds mood profiles to be positive (i.e., similar or better levels of positive affect and lower levels of negative affect) compared to younger and middle-aged adults (Gueldner, Loeb, Morris, Penrod, Bramlett, Johnston, & Schlotzhauer, 2001). These findings represent the outcome of the transaction between an individual's ability to actively select, optimize, and develop adequate compensatory strategies. When frailty occurs, however, the lower levels of generally positive mood states for older residents may reflect the increased need for security and consequent reduction in the ability to proactively shape their environment. It would seem that, with age, emotional signatures tend to mellow.

Assessment

A personality inventory is part of a good treatment. It should be administered at the onset of an intervention or treatment to assist with person-centered approaches to promote cognitive resilience and to better understand which individuals are more likely to gain from the treatment. It is typically done as part of an omnibus evaluation with other major psychiatric disorders. After doing so, clinicians should focus on understanding the multiple pathways that link personality constructs to patterns of cognitive decline or other psychological problems, as well as whether these associations hold across adulthood. Clinicians should also encourage consideration of the possibility that cognitive interventions hold transfer effects on dispositional characteristics, such as traits and self-efficacy beliefs, which may ultimately influence the long-term effect of the methods to prevent cognitive decline. Personality, therefore, is seen as a precursor and in this way personality traits predict the likelihood of the onset of several chronic conditions that a person may develop and their tendency to react to stress, engage in physical activity, or show a proclivity toward adherence to medical regimens.

There are two sides to assessment here. Improved analyses have allowed for more sophisticated profiles of later life patterns. Statistical analysis in the past two decades has enabled investigators to more closely measure between and within individual change (Mroczek & Spiro, 2007). In so doing, they have aligned personality research with a basic premise of lifespan developmental theory, the concept of individual differences, and intra-individual change (Baltes & Nesselrode, 1973). As applied to personality, this is the idea that individuals differ between one another not

only in the level of a given personality trait, but also in the direction and rate at which they change. Some people change quickly, some less so, and some change in different directions.

Clearly people change. This is an undeniable developmental fact of life. What has remained in dispute in personality research is how much change takes place and what it means. Even if there is agreement for some degree of plasticity in personality, there remains strong disagreement about the mechanisms for such change. Beyond the biological, important research remains to be done at the phenotypic level. Increasing use of longitudinal studies in the past several decades has consistently improved researchers' understanding of personality change. As extended studies continue to follow individuals across time and new studies emerge, individuals will be better able to refine their understanding of these trajectories.

Among the many issues, several questions remain. One issue is that, as life expectancy increases for much of the populous, what is meant by late adulthood will again have to be redefined and so too will our understanding of personality development. Clearly more attention will need to be paid to intra-individual differences, not just to the patterns themselves, but to what these changes might portend. Also, another issue involves how clinicians can assist individuals in trait development. More research is needed to assess which traits are amenable to change and which interventions are most successful in exacting each change. This is related more to the academic side of assessment.

The second type of assessment is related to clinical care. Assessment of PD at later life is far from simple. Psychologists who work with older adults in varied clinical settings often observe a familiar paradox and frequently healthcare professionals confronted with a difficult behavior in an older adult attribute this to the individual's personality rather than to the environment or other causes, engaging in what has been labeled the Fundamental Attribution Error (Balsis, Zweig, & Molinari, 2015). These authors have developed a reasonable taxonomy for looking for and assessing personality pathology. They advocate several conceptual markers. The first involves aligning the assessment approach to a conceptual model in measurement selection. When an older adult is referred for the assessment of personality pathology, the selection of appropriate assessment methods and measures depends in part on the conceptual model of neural personality and personality pathology that is fundamental to the assessment endeavor. We have already discussed McAdams' concept of the individual's unique variation and general evolutionary design for human nature versus more trait-based assessment.

Balsis et al. (2015) also argue for aligning the assessment approach with guidelines for evaluating older adults. In this case, the assessment of a PD in an older adult, which includes selection, administration, interpretation of measures, and communication of findings, is framed by the general principles that undergird the psychological assessment of older adults. This requires first and foremost the selection of methods and measures that, ideally, have been developed, validated, and shown to be psychometrically appropriate for older populations. Next, the authors identified the need for considering the relevance of social history in clinical judgment because most conceptualizations of personality and personality pathology emphasize characteristic inner experiences, behaviors, adaptations, and interpersonal patterns that have an enduring and pervasive quality. The individual's social history or life narrative is an essential source of information. For older adults the approach to assessing personality pathology begins. Balsis et al. (2015) also identified several key markers: identify PDs versus a persisting Axis I diagnosis, PD versus a context-dependent role in behavior, PD versus personality change resulting from neurological illness, and PD versus difficult doctor-patient relationships. All of these can be considered major problems.

That said, there are very few specific measures used for older adults when assessing personality. The Gerontological Personality Disorder Scale (Van Alphen, Engelen, Kuin, Hoijsink, & Derksen, 2004) is the sole measure developed for the use of older adults with personality pathology. Several of the scales have been subjected to empirical enquiry and thus have evidence of validity for use with older populations. These include self-report measures of personality and personality pathology, such as the MMPI-2 (Butcher et al., 1989) and NEO-PI (Costa & McCrae, 1992). They also include multi-scale self-reported inventories, such as the Coolidge Axis-II Inventory (Coolidge & Merwin, 1992), the Millon Clinical Multiaxial Inventory (MCMI-3 [now MCMI-4]; Millon, Davis, & Millon, 1997), the Personality Assessment Inventory (PAI; Morey, 2007), and the Schedule for Non-adaptive and Adaptive Personality (SNAP-2; Clark, 2009). There are also structured clinical interviews, which involve the Personality Disorder Interview-IV (Widiger et al., 1995), and the Structured Clinical Interview for the DSM-IV Personality (SCID-P; Pfohl, Bloom, & Zimmerman, 1995). In addition, there are domain-specific self-measures, such as the Inventory for Interpersonal Problems (IIP; Horowitz et al., 1988) and prototypes for matching clinical report instruments, such as the Personality Assessment Form (PAF; Pilkonis & Frank, 1988) or the Shedler-Westen Assessment Procedure (SWAP-200; Westen & Shedler, 2007).

Personality pathology then is generally seen according to the DSMs as an enduring pattern of inner experience and behavior that deviates markedly from the expectations of the individual culture. A dimensional approach is also addressed in the DSM-5. It subserves the five-factor model derived from the study of normal personality. The Millon batteries have been designed for the measurement of Axis II traits, PDs, or related coping patterns. The Millon Behavioral Medical Diagnostic (MBMD) is a reliable personality measure that addresses these variables. The MBMD was developed as a personality inventory that addresses issues of physically ill people and medical-behavioral decision issues of importance. The MBMD has 165 items with 29 clinical scales. It is based on Millon's belief that personality-mediated coping styles are powerful influences on the genesis and course of illness. This scale has not been applied to older adults to any degree, but it is especially relevant for this group because of their high preponderance of medical problems.

In like fashion to other Millon instruments, this test assesses personality patterns, as well as validity items, response patterns (Disclosure, Desirability, and Debasement), psychiatric factors, stress moderators that increase (e.g. spirituality) or decrease (e.g. lack of social supports) coping, factors that impact treatment prognostics, such as a tendency to abuse medications or poor compliance with physician orders, and lifestyle habits that have a negative effect on health, such as cigarette smoking. On the MBMD, raw scores are transformed into prevalence rate (PR) scores which serve as the basis for selecting optimal cutting scores and ensure that the proportions of various MBMD cut-off scores will be compatible with clinically judged indices found in diverse medical populations. For personality and psychiatric problems, elevations greater than 74 suggest that that variable asserts an influence on the person (presence), and at 84, that the variable is prepotent in its influence (prominence). For Treatment Prognostics and Stress Moderators, scores are bi-directional, low scores (<PR 36) reflect relative strengths, and high scores (>74) relative problems. We use this measure liberally in this book.

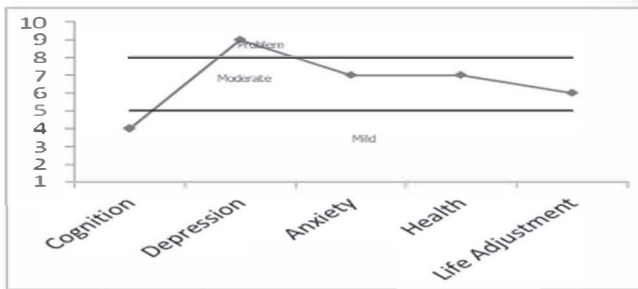
Below is an accounting of personality traits for older adults who presented for memory problems. This is based on the PR rates from the MBMD. Not surprisingly the most common type or personality is cooperation, with the more externalized, independent ones less frequent. These numbers provide data on PRs of 80 or more. Many other older adults have PR rates greater than 60 or 75 suggesting that these can be in play.

Mean and Percentage of Older Adults with PR > 79

Personality	Mean	PR 75 or > (%)
Introversion	54.7	8%
Inhibition	48.6	5%
Dejected	34.3	8%
Cooperation	60.0	12%
Social	45.4	2%
Confident	46.8	3%
Nonconforming	39.3	1%
Forceful	35.5	1%
Respectful	56.0	8%
Opposition	53.7	4%
Denigrate	52.5	6%
Valid N = 394		

Personality Cases: Here we present three brief cases of older adults presenting for memory. The personality profile is provided by the MBMD. It reflects the process of adjustment for three patients presenting for memory problems. We do not present the full Watch and Wait model.

Case 1



PATIENT: RB

OB: 1946

CHIEF COMPLAINT: Evaluation for memory problems.

Psychiatric Indications	AA	3	50			ANXIETY-TENSION
	BB	0	15			DEPRESSION
	CC	5	52			COGNITIVE DYSFUNCTION
	DD	4	50			EMOTIONAL LABILITY
	EE	3	25			GUARDEDNESS
Coping Styles	1	2	15			INTROVERSIVE
	2A	1	25			INHIBITED
	2B	1	15			DEJECTED
	3	16	92			COOPERATIVE
	4	6	35			SOCIABLE
	5	6	35			CONFIDENT
	6A	4	25			NONCONFORMING
	6B	5	25			FORCEFUL
	7	18	50			RESPECTFUL
	8A	4	45			OPPOSITIONAL
8B	5	70			DENIGRATED	

HISTORY OF PRESENT ILLNESS: The patient drove to this interview. She lives locally and has been here in this area for 30 years. She lives with her husband of over 50 years. She is retired from teaching and has had memory problems since breast surgery in 2011. She had chemotherapy and radiation subsequent to that. She currently is a caregiver for her mother and to some extent her husband and feels like she is overwhelmed. She indicates that memory has been an issue, as well as depression.

MEDICAL HISTORY: She is a victim of breast cancer. The surgery that she had was a radical mastectomy in 2011 and she also had chemotherapy and radiation. She indicates that physical problems and memory problems have evolved since that time. She has been depressed for over 30 years. She has been on medication for that period of time and on a few occasions has seen a therapist. She takes temazepam, an estrogen blocker, Effexor, Wellbutrin, levothyroxine, Benicar, Crestor, Imitrex, Lyrica, Restasis, and melatonin. Indications are that she does not drink alcohol or use tobacco. She is not in pain. Indications are that she sleeps poorly. Memory problems started roughly seven years ago and have increased. She feels that she is at least 25% lower than she was seven years ago before the procedure on her breasts.

MENTAL STATUS: The patient is a 71-year-old Caucasian female who drove to this appointment. She was dressed appropriately, answered questions with specificity, smiled, and was pleasant. There was on occasion some delay in her responses. She is oriented. She is depressed. She is also anxious about her situation. There is no evidence of perceptual anomalies or delusory thinking. Her insight and judgment appear to be quite good.

PREMORBID FUNCTIONING and OVERALL COGNITIVE FUNCTIONING: She has a college and master's degree. She taught for 24 years and also has a counseling certificate. She has been married for 50 years and lives locally with her husband. She is a caregiver to her mother and husband. She has a 25/30 MoCA, below normal for her age and education. She had visuospatial problems, math problems, and executive function issues on the MoCA.

She is above normal on general intelligence (Full Scale = 113; Verbal = 121 and Performance = 103). She scored a RBANS index of 100, placing her at the 50% level for other people her age. Based on these scores, she is responding at her pre-morbid abilities, given some variability. Her memory and new learning were low average. Her performance on EF tasks was variable. On a task requiring her to alternate sequentially numbers and letters (Trails B) she had scored lower. She was

lower on the Wisconsin Card Sort, a test of executive function. She scored average on arithmetic but had problems on Serial 7s. She scored low average on WASI-Block Design and above average on WASI-Matrix Reasoning. She was average on fluency tests. There were mild deficits on the FAQ, a measure of executive functions in activities of daily living.

Emotional self-report scales indicated mild depression and general anxiety. She is very concerned about her life. Her MINI reflects these problems. Her BDI-II was 9; her PHQ-9 was 4; her Mood Scale was 4; all non-depressed ranges. Her anxiety is slightly higher but normal (GAD-7 = 5).

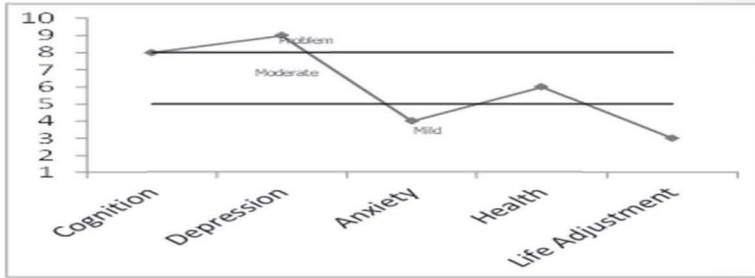
The MBMD indicated no Axis I problems. She has a personality pattern reflective of cooperative traits. She is not likely to take the initiative and is inclined to downplay problems. She may become somewhat dependent on her health care workers. She is inactive. She has no problems with stress moderators and treatment prognostics. She tends to put others above her needs. She is of course a caregiver.

SUMMARY: This is a 71 y/o female who was referred for an evaluation for depression and memory. She has been retired for a few years as a teacher and has had breast CA with radiation and chemotherapy. She has a master's degree and has a therapy certificate. She is married and lives locally with her husband who has health issues. She is a caregiver for her mother and at times her husband.

Cognitively she is above average pre-morbidly. She has a master's degree. She has good intelligence skills, especially in verbal areas. She has reasonable skills in all areas (with some problems), including visuospatial, attention, new learning, and memory. She has some problems with sustained attention and complex executive functioning. In sum, she is functioning adequately in these areas.

She has a history of depression. Her self-report scales reflect very mild perturbation in worry and depression. She has a personality pattern that is simple – passivity and dependence. She is intrinsically cooperative. This is an ideal personality for compliance and better life habits. Sleep is an issue; pain is not. Her treatment for depression and needs in life (some cognitive problems and stress from caregiving) will unfold from her passive dependent style, making her compliant. Support and reward as well as psychoeducation and direction in her life will help.

Case 2



Life Adjustment

PATIENT: KS
DOB: 1948
CHIEF COMPLAINT: Evaluation for memory.

KS

	AA	19	75								
Psychiatric Indications	BB	15	70							ANXIETY-TENSION	
	CC	23	79							DEPRESSION	
	DD	17	61							COGNITIVE DYSFUNCTION	
	EE	24	71							EMOTIONAL LABILITY	
											GUARDEDNESS
											INTROVERSIVE
Coping Styles	1	22	103								INHIBITED
	2A	15	69								DEJECTED
	2B	5	60								COOPERATIVE
	3	20	92								SOCIAL
	4	3	5								CONFIDENT
	5	11	44								NONCONFORMING
	6A	17	61								FORCEFUL
	6B	15	55								RESPECTFUL
	7	22	55								OPPOSITIONAL
	8A	32	103								DENIGATED
	8B	17	79								

HISTORY OF PRESENT ILLNESS: She indicated that roughly six months ago she started having difficulty with memory. This was largely word-finding problems and difficulty remembering events in her life. Indications are that she has lived with a significant other for roughly five years. She is retired having had several jobs, the last of which was jewelry sales.

DEVELOPMENTAL HISTORY: She is the middle child of three. She describes herself as “the middle child” and was described by her siblings as mean. She graduated from high school and went on and got a hairdresser’s certificate. She worked in hairdressing and then drove a truck. She has been retired or not working for the last six years. She was married three times, at age 18, age 20, and in her 30s. She has been cohabitating with a male friend for the last five years.

MEDICAL HISTORY: She is taking cyclobenzaprine, desvenlafaxine, Restasis, a statin, levothyroxine, meloxicam, gabapentin, lorazepam, tramadol, and Plavix. Indications are that she has been on a psychiatric medication given through her primary care largely for the last 10 years. She has also been on lorazepam for roughly that period. Indications are that she has put on weight (20 pounds in 6 months). She does not drink alcohol or use tobacco. She indicates that she is not in pain, but that her legs hurt. She does not sleep well. She describes it as a problem and she is taking trazodone, as well as Ativan. She does not have an exercise program. She is somewhat inactive during the day, but remains in the house and does housework. She does all of her ADLs and IADLs.

MENTAL STATUS: The patient is a 69-year-old female who drove to this appointment. She was less than 5 feet tall and was heavy. She was dressed quite nicely and interviewed quite well. She was pleasant, smiled, and answered questions. It was also evident that she has a strong independent streak in her. She is oriented. She indicated that she is mildly depressed and anxious. She is concerned about her cognition. There was no evidence of perceptual anomalies or delusional thinking. Her insight and judgment appear to be quite good.

PREMORBID FUNCTIONING and OVERALL COGNITIVE FUNCTIONING: Mrs. S has an intelligence score at average, based on her education and occupation. Her MoCA was low normal (25/30). She had 4/5 on memory. Her executive functioning was low. Her WASI is low normal with little variation. Currently Mrs. S also scored a RBANS index of 83, placing her at the 13% level for other people her age. Her memory and new learning were low normal; visuospatial is average as was language. She scored 4 on the FAQ, indicating no problems on this measure of executive functions in activities of daily living. Mrs. S’s

performance on several tests of EF was largely below average. On a task requiring her to alternate sequentially numbers and letters she was impaired (Trails B). She scored adequately on arithmetic. She was very poor on the WCS with no categories. Her WASI-Matrix Reasoning was lower, as was WASI-Block Design. She was average on both fluency tests. There were deficits on the FAQ, a measure of executive functions in activities of daily living.

Emotional self-report scales indicated depression and anxiety. She scored 24 on the BDI-II, 15 on the PHQ-9, and 5 on the GDS-SF, all depressed. She also scored as one with mild anxiety on the GAD-7 (5).

The MBMD indicates that her personality is one of an introversive, cooperative, oppositional style. This is a complex pattern. She wants to keep the peace in life but has issues with authority. She can be toughminded and be her own worst enemy. She may also be distrustful. She is also one who prefers to keep more to herself. She has a significant other and it is predictable that there is conflict. She has had previous marriages. She is concerned about her cognitive status. She has no problems with stress moderators. Treatment prognostics are normal except for compliance.

SUMMARY: This is a 69 y/o female who was referred for an evaluation for her memory. She has also reported a slight loss of cognitive ability in the last 6 months. She has been married three times and has a mild psychiatric history (outpatient). She is treated for depression. She now lives with her significant other and relates to cognitive problems. She can do all ADLs and IADLs. She is discontent with her life.

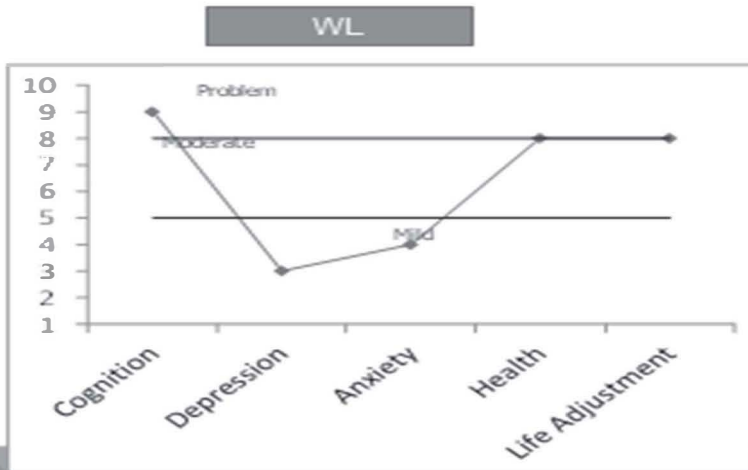
Cognitively she has mild difficulties now. Her intelligence is low normal and her composite scores for the RBANS were lower. She has an equal score for verbal and performance areas. Her attention is poor, as is new learning. She has real distractibility problems. Her executive functioning is also poor in several areas including problem solving and planning. She is adequate in language, visuospatial and motor areas. In sum, she is variable cognitively, but is able to manage.

At base, she shows signs of depression and anxiety. She relates to experiencing cognitive problems with anxiety. Her personality is multi-trait with patterns for being a loner, cooperative, and oppositional. This is a pattern in which she desires to keep more to herself, not to make waves in life, but to assert herself. Compliance may be an issue. She may have limited social skills, with particular difficulty interpreting the normal nuances of interpersonal behavior that provide the meaning to personal relationships. Her social isolation and detachment may serve to decrease a sense of discomfort that interpersonal contact fosters. It can also be a

problem. Her cognitive status is an issue but less so in light of her simple life, support, and more important affect problems. They need addressing, however. Her thought processes are likely to be marked by confusion, distractibility, and difficulty concentrating, and she may experience her thoughts as being somehow blocked or disrupted. Her treatment will involve targets based on her depression but her cooperative, isolative and independent personality pattern will have to be supported and respected.

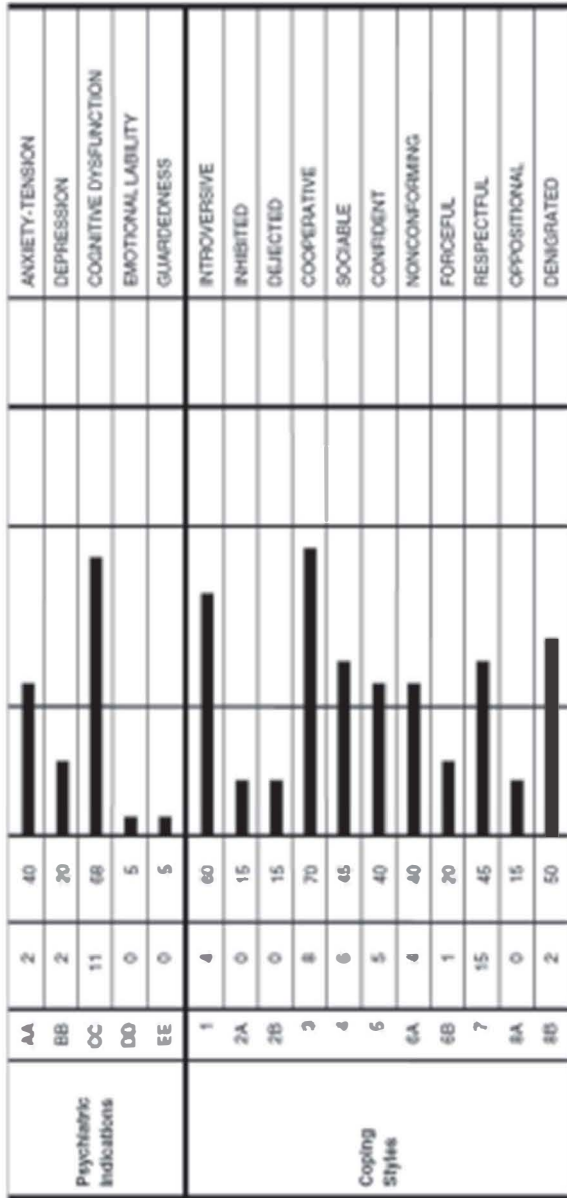
Case 3

PATIENT: WL
DOB: 1941



Cognition/
Life Adjustment

WL



HISTORY OF PRESENT ILLNESS: The patient and wife were interviewed. Concerns were that memory has been a problem for him in the last year or so and he has been increasingly shutting down and doing less. He indicates that this has extended roughly over a six-month period. He had heart surgery three years ago and his wife indicated that this is the point in which problems started to evolve. He is a retired colonel and has also been a teacher for many years in secondary education.

RELEVANT DEVELOPMENTAL HISTORY: He is an only child. He went to college. He then went into the Air Force and became a pilot, flying B-52s and later helicopters. He flew 50 missions in Vietnam. He was in the Air Force for 26 years. He is a retired officer. He also had other degrees from three universities and had a doctoral degree from a Georgia university. He was married in 1968 to his now wife and they have two daughters. The daughters both have mental problems and have been a source of distress for the couple. The daughters are now on disability and are assisted by their parents.

RELEVANT MEDICAL HISTORY: He is a long-time diabetic who is taking Lantus and Humalog. He is also taking metoprolol, aspirin, B12, levothyroxine, omeprazole, and Zocor. Indications are that he does not drink and he does not use tobacco. He does have minor lower back pain that he accommodates. He also indicates that he sleeps roughly 7-8 hours a day and takes naps, but his sleep is disrupted at night. He does not have a formal exercise program, but he does walk. He does all of his ADLs and IADLs, although increasingly he is giving more and more tasks over to his wife. There is no psychiatric history.

PREMORBID FUNCTIONING and OVERALL COGNITIVE FUNCTIONING: Currently Mr. L shows evidence of cognitive problems. He is facile socially, is motivated, and is socially aware of politics and issues in the military. He has goals for himself. His MoCA is 22/30 and suggests cognitive problems. He did poorly with memory (0/5) and had problems with more complex problem solving. He has good attention skills. He has no problems with simple abstraction but has visuospatial problems. He is less active and is now living at home with his wife and mother-in-law. His intelligence is:

WASI	Scale Score
Verbal	87 (19%)
Performance	106 (73%)
Full Scale	99 (47%)

He has an RBANS at 76, 5%. He was very poor on memory and new learning. His CPT-2 was largely normal and suggested reasonable attention skills. On scales of EF he was poor. On Trials B he was 2 standard deviations low. In arithmetic, he was average. He was not able to do the WCS, a negative sign. He was below average in WASI-Similarities. WAIS-IV Letter Number Sequencing was poor (6). He was average on WASI-Matrix Reasoning (53) and WASI-Block Design (53). There were no deficits on the FAQ, a measure of executive functions in activities of daily living, due to abilities.

Mr. L was given several tasks reflective of emotional status. On the MINI, he indicated that he is depressed. That said, his MBMD was free of Axis I problems, as was the PAI. He had a normal score on the GDS-SF (3) and on the BDI-II (6), both normal. His PHQ-9 was also normal (3). He scored in the normal anxiety range on the GAD-7 (1).

The MBMD indicated that he has a personality pattern reflective of a cooperative style. This pattern reflects a desire to comply and be accepting. He is inclined to overlook or deny problems. Given that he lives with several strong-willed women, this is not a problem unless he strongly desires to be heard. He is concerned about his thinking. He did not endorse depression and anxiety on the MBMD. He also has stress moderators that cause problems – spiritual absence. He showed two problems with treatment prognostics. Interestingly, one of these is compliance, suggesting that he did not want to be in this evaluation.

Stress Moderators: Intrapersonal and extrapersonal characteristics that affect medical problems. They target cognitive appraisals, resources, and context factors. He is concerned about her frailty, functioning, and is reflecting on her spirituality.

STRESS MODERATOR

		Weakness
Illness apprehension	vs Illness Acceptance	X
Functional Deficits	vs Functional competence	X
Pain Sensitivity	vs Pain Tolerance	
Social Isolation	vs Social Support	
Future Pessimism	vs Future Optimism	
Spiritual Absence	vs Spiritual Faith	X

Treatment Prognostics: Behaviors and attitudinal aspects that may complicate or enhance treatment efficacy. He is concerned about what is going on with him as well as interestingly compliance.

TREATMENT PROGNOSTIC	Weakness
Interventional Fragility vs Interventional Resilience	
Medication Abuse vs Medication Conscientiousness	
Information Discomfort vs Information Receipt	X
Utilization Excess vs Appropriate Utilization	
Problematic Compliance vs Optimal Compliance	X

SLEEP: Patient indicates that he has no problems sleeping. He notes that he receives 7-8 hrs/day. He is not in pain at night. He endorsed no pain sensitivity on the MBMD. His average pain is 1/10. He is mildly tired during the day. He does take naps. He has a normal ESS (6).

SUMMARY: This 75 y/o male was referred for memory. He lives at home with his wife and has been retired for 6 years. Also, he is in a house with 3 women including his two daughters. He is a retired AF officer and pilot. He is a diabetic and has had memory problems for at least a year. He is inactive. He seems to function to a reasonable level given his support but is increasingly concerned about his life.

Cognitively he was average premorbidly but has problems now. He did poor especially in areas with memory, new learning, executive function, and language. He reads adequately. Complex problem solving is also poor. In sum, it appears that he has average intelligence and now has clear problems with selective deficits in memory and executive function, and new learning.

He is depressed. He has a cooperative personality. He has a friendly way about him. He does not want to be a bother. He is open to suggestions, however. That said, he is in turmoil and is fearful (depressed) about his future. He requires support and direction.

In sum, he scores as one with MCI (MCD). He is clearly having problems and is probably in a mild dementing state or will segue to one in the coming years. He has problems with memory and EF especially. He is in mild stress but is supported by his wife. Increasingly he is showing problems with adjustment as he must have help with some IADL tasks. Health of course is an issue as he is a diabetic. He is open to suggestions for help. He has good cognitive reserve also.

Post Script: He has continued to decline and has shown more problems day-to-day. He has also missed medication markers for his diabetes. His wife is increasingly concerned.

Two of the cases show a more accepting and accommodating personality in the context of some cognitive or depressive features. Both will require individual attention and the profile will change over time. The personality will be stable. The personality is influential and will influence the process of care. In the other case (Case 2), a more complex therapy will ensue due in part to a more varied background and complex personality.

Treatment

How does personality fit in here? Perhaps it is best to consider personality as a body of descriptive material collated across several perspectives. This formulation allows for modal criteria to define and diagnose a PD, but looser criteria to treat. It is therefore not so much an explanatory model as a framework for guidance in understanding the person. The guiding principle, as we indicated before, is that treatment should start not from a narrowly focused disorder, but from a detailed analysis or deconstruction of the patient's life or, if needed, from breaking apart psychopathology into domains of dysfunction. As previously noted, treatment methods are selected on the basis of what works for the specific problem and domain that are the focus of the therapeutic intervention. A global diagnosis based on current diagnostic categories is insufficient. In order to select appropriate interventions, then, a psychiatric entity needs to be decomposed into different functional domains. This reveals an additional benefit of integration (holistic care) and accommodates the considerable heterogeneity among patients with a given disorder and permits treatment to be tailored to the individual.

In the treatment of older adults at later life with PDs, there is not much empirically supported information. Given the frequent necessity of mental health consultation with older adults, especially because of the havoc caused by institutional settings, Sadavoy and Fogel (1992) proposed some basic principles for working with older adults with PDs. These include containing and limiting pathological behavior, establishing a working alliance between the client, family, and staff, utilizing a coordinated team approach to prevent splitting and unwarranted counter-transferential reactions, and in the long term helping the client reduce interpersonal strain triggering the personality disorder behavior. In a like manner, Mordekar and Spence (2008) have outlined principles for treating PDs also, including fostering of the therapeutic relationship, treating comorbid Axis-I disorders, being consistent using supportive CBT, developing good ties with other professionals, and involving family and friends.

Dirksen et al. (2012) developed a method with Dutch and Belgian experts on how to treat older adults. Four different treatment levels were differentiated based on treatment, indications, and exclusions. Treatments directed at personality change are lengthy (for example, DBT) and only suitable for motivated individuals who are capable of self-reflection and who do not have significant cognitive impairment. Treatments emphasizing adaptation enhancements (interpersonal psychotherapy) are aimed at those motivated individuals who may have less of the internal resources needed for change. Treatments that provide structure and support are for those with severe cognitive disorders who require a surrogate support system. Examples include geriatric daycare and the like. Finally, experts noted that pharmacotherapy should be considered with proper precautions for those with serious behavioral problems who are not motivated to change.

One other framework to guide the PD treatment of older adults has been proposed. Segal et al. (2006) employed a goodness-of-fit model in planning interventions for older adults with PDs. The goodness-of-fit model is undergirded by four basic premises: each personality trait a) lies along a continuum, b) results in a composite of traits that establish a profile, c) has care providers that like or dislike different personality traits, and d) is given personality-based treatment based on whether or not a personality trait is labeled as a problem or disorder. In essence this last feature depends on whether the personality trait is favored or not by the provider or the setting. Interventions based on this goodness-of-fit model would be categorized broadly as a) changing the demand on the client with the PD so that the PD trait is not exacerbated, b) promoting traits favored by the care provider, c) reducing non-therapeutic responses by the staff that can exacerbate maladaptive behaviors, and d) helping to replace the loss that precipitated the increase of these maladaptive defenses in the first place.

The Axis I and personality interaction then makes treatment more difficult than for just a simple Axis I problem. Many of the specific interventions evolving from empirically supported treatments can be used. It is perhaps best to have the freedom to select bits and pieces from these and apply them as needed in individual cases. This will be a function of the knowledge of the therapist, the client's preferences, and the nature of the client's problem, as well as the empirically supportive treatment itself. For over 4 decades, various hypotheses related to this interaction have been espoused – predisposition, subclinical or prodrome, life events, scarring, pathoplasty, or forme frust. Additionally, most “rules” of therapy addressing therapy type, severity, case mix issues, and interventions are

just guides, as the complexity of cases explodes in most therapy rooms. Some general “rules” are given below.

Each person with a PD has a unique biopsychosocial etiology. We are treating a person, not a disease. The consequences of failing to identify and attend to PDs can be troubling indeed. When PDs are not addressed up front, the result is lengthier therapy, more frequent treatment failures, and unnecessary complications to the therapeutic relationship (Sadavoy, 1999). As noted above, those with a PD develop Axis I disorders earlier in life, have more symptoms, display longer episodes of the problem, and have more frequent relapses. After treatment, those with a PD reveal greater residual problems, have greater levels of relapse, and experience a lower quality of life. It is believed that the PD pattern is always present but becomes especially noisy in times of stress.

PD Treatment Axioms at Late Life

1. Ensure unconditional acceptance where the PD patient will be validated as a person, but negative consequences will be established for unacceptable behaviors.
2. Elucidate expectations for reasonable and responsible behavior and the protection of personal boundaries for both you and the PD patient.
3. Consistency must be established so PD patients are aware that, despite their demands and behaviors, you will not give up on them. In order to allow gradual change, structure can be provided via a “holding environment.”
4. Countertransference may determine treatment options by understanding patients and how they affect others. One must be careful not to respond impulsively or to “act-out” against the patient in terms of unwarranted or premature assumptions or interpretations.
5. Approach PD patients with a coordinated team approach. Communication and regular meetings are necessary to prevent the patient from potentially dividing the team, causing members to “take sides,” and destroying the necessary consistency, integration, and structure of the biopsychosocial approach the patients need.

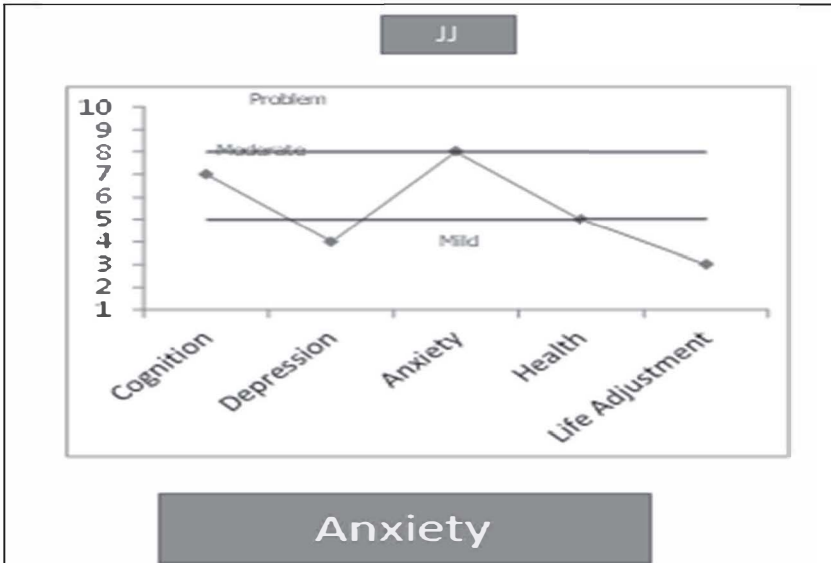
Given this, the distinct components of treatment, delivery, receipt, and enactment require modification when a PD is present. The client must be presented with materials and tasks, receive them openly, and carry out assignments. Distinct goals can be designed on the basis of Axis I with input regarding the influence of the personality pattern. The problems that the person has dictate the intervention. The personality provides data for the process of care and its ease of therapy. Above all, the patient with a PD

arrives to therapy with problems that are externalized or ego-syntonic and closed to change. In treatment, there are core features characteristic of all PDs and those unique to each one separately. As with therapy at any age, the more impaired the older adult, the more the intervention involves therapeutic support, family support, and environmental support. Behavioral activation, too, is a primary consideration. For the higher functioning person, more interventions can be problem directed at the “inner level” of the person.

Therapy (notably personality therapy) is best defined not by techniques but by conceptualization. In older clients with PDs their “head” changes in therapy before their gut. The person must experience the “sensation of the PD” and see that it is okay before change takes place. As a general rule, the clinician attacks the overall rigidity in the PD, to increase flexibility and to decrease self-defeating patterns. Zweig and Agronin (2006) outline several treatment rubrics. The core rubric involves a common factors-integrative approach that strives to employ a combination of therapeutic modalities tailored to individual treatment goals, uses both somatic and psychosocial approaches, and appreciates the psychodynamic influence of a PD. In effect, this involves a case formulation with a PD element in the treatment plan. Additionally, the clinician applies therapeutic components as specified in the CBT, as well as utilizing the family and the treatment team, to advance treatment goals.

Case formulation is always involved for older adults. This allows for hypotheses about the case, as well as a structured plan for intervention. As with the American Psychological Association’s emphasis on EBPP (Evidence Based Practice in Psychology), the idea is to formulate EBTs (empirically based therapies) on the basis of a person with a defined disorders and allow the trait-based information to provide structure and plans for care. The case is, then, carefully formulated and the PD rubric applied to components of the person. Also, reasonable rules of therapy, especially as related to older adults, are applied. Millon’s model is a good place to start.

We now present a personality case based on the Watch and Wait model.



Anxiety

- MINI = Anxiety
- GAD-7 = 12
- BAI = 21
- STAI = 43

Cognition

- MMSE = 24/30
- MoCA = 19/30
- Trails A = 45%
- Trails B = 40%

Depression

- BDI-II = 10
- PHQ-9 = 7
- GDS-SF = 4

Health

- ETOH
- Minor HTN and cholesterol issues

Life Adjustment

- Currently in ALF

HISTORY OF PRESENT ILLNESS: The patient was driven to this appointment. In October of 2015 he fell while drinking. He has been adjusting since this period of time. He broke his hip and had problems

with his knee and also had a hematoma. He was in the hospital in Jacksonville and in Macon and was also in a rehabilitation center and is now in an assisted living facility. He has given his power of attorney over to his sister-in-law who is married to his brother who has Alzheimer's disease. They also live in Macon. He is a retired accountant who had been living in Houston.

On a later date his sister-in-law (POA holder) indicated that he has always been idiosyncratic and adjusted marginally in life. She indicated that he lived in a hovel in Texas and then was drunk and in a hospital in Florida when she caught up with him. He is now adjusting at the ALF and is content with his setting. He manages his money, does not drive, and has all his IADLs met.

RELEVANT DEVELOPMENTAL HISTORY: The patient indicates that he was born in South Carolina and migrated quickly to the coast of Georgia. He went to high school and later went to Notre Dame. He was the second of four children. His father was in management and his mother was a college graduate. He worked in accounting where he got a degree from college and worked at several places most recently in Texas. He retired in 2009. He had been living as a retiree since 2009 in Texas when he went to a high school reunion in 2015 and fell. He never married.

RELEVANT MEDICAL HISTORY: He is taking a statin, famotidine, losartan, Senexon, and tamsulosin, as well as Remeron and sertraline. He has a long history of psychiatric problems that involve OCD and depression. Indications are that he has been treated for these on and off through his life. He had the accident in 2015 and has been in rehabilitation ever since. He indicated that he is mildly depressed and that the OCD features he feels are an undertow. He indicated that alcohol has been a part of his life, but never a problem for him. He has no DUIs, nor has he ever been in rehabilitation. He has not had a drink in many months. He is not in pain. He indicates that he sleeps well. He is not tired during the day. He does not have a formal exercise program, but does go to OT and PT.

MENTAL STATUS: The patient is a 68-year-old Caucasian male who was driven to this appointment. He was rather thin, but was dressed appropriately and answered questions with some degree of specificity, although he could be obsessive in his answers. He seemed to be rather sharp in his reflection. He is well oriented and well versed in current events. He indicated that he is mildly depressed and that he has had OCD in his life. He was unable to provide a distinct definition of his OCD, whether it was more compulsions or obsessions. There was no evidence of perceptual anomalies or delusionary thinking. His insight appears to be reasonable and his judgment appears to be good.

OVERALL COGNITIVE FUNCTIONING: Mr J was given the WASI and is normal (IQ =99). Currently Mr. J scored an RBANS index of 78 placing him at below average for other people of his age and education (12%). This is certainly below his intelligence. His MoCA was 19/30, impaired. He especially had problems with memory and executive function tasks. Noteworthy too, his CPT-2 was poor, suggesting that he has attentive problems with commissions and sustained attention. Interestingly, he did well on 4/5 EF tests including Trails B, WCS, WAIS-4 Block Design, and fluency. In general, scores are lower or below average and suggest difficulty on overall cognitive functioning (with some exceptions). He has no problems adjusting day-to-day. He is in an ALF with support. There were, however, mild deficits on the FAQ, a measure of executive functions in activities of daily living. His IADLs are suspect as he receives some help. In sum, he scores cognitively at a level where he is below average but his functioning is controlled.

















EMOTIONAL ASSESSMENT: Emotional self-report scales were evaluated. He was administered the MINI showing problems with depression. On the Mood Scale he scored 4, non-depressed; on the BDI-II, 10, mildly depressed; on the PHQ-9, 7, mildly depressed. On the GAD-7 he was very anxious (18).

The MBMD indicated that he has anxiety. He also has a respectful personality style. This is a personality pattern reflective of a person who is prone to be controlled, conforming and responsible. He is serious minded and appears diligent. This trait suggests that he is compliant, rule oriented, and has a pattern that has fears internally, and has needs for conformity and guarded respect. Given a possible dementing process, he is likely to hide problems but be obedient and confused emotionally. He may have problems with compliance and is non-religious.

PAIN: His average pain is 2/10. He rated pain at present as 2/10. He did not endorse pain sensitivity on the MBMD.

SLEEP: Patient indicates that he has no problems sleeping. He sleeps well at night. He is taking Remeron. He has a 1 on the ESS, suggestive of no fatigue during the day.

SUMMARY: This is a 68 y/o male referred for an evaluation for cognitive problems and depression. He has had a more isolative life with OCD and alcohol as a problem. Alcohol is not a problem now. He graduated from a prestigious university and was an accountant for many years. He retired in 2009 and had been living in Houston until an accident in 2015. He has been in recovery/rehabilitation ever since. His sister-in-law is the POA. He has never married. He does ADLs and most IADLs. He is in an ALF.

		SCORE		PROFILE OF PREVALENCE SCORES				CLINICAL SCALES
		RAW	PS	0	35	75	85	
Psychiatric Indications	AA	8	81					ANXIETY-TENSION
	BB	3	50					DEPRESSION
	CC	9	70					COGNITIVE DYSFUNCTION
	DD	6	65					EMOTIONAL LABILITY
	EE	4	40					GUARDEDNESS
Coping Styles	1	1	10					INTROVERSIVE
	2A	0	5					INHIBITED
	2B	0	5					DEJECTED
	3	4	40					COOPERATIVE
	4	13	65					SOCIABLE
	5	14	69					CONFIDENT
	6A	2	10					NONCONFORMING
	6B	5	25					FORCEFUL
	7	25	80					RESPECTFUL
	8A	4	45					OPPOSITIONAL
	8B	1	10					DENIGRATED

Cognitively he is as average premorbidly. Now he has some problems in memory, attention, and visuospatial areas. He scores reasonably well in EF and in language. He has abused ETOH over the years and has had depression on and off.

Emotionally he is anxious. He has considerable obsessions that freeze him from any decision. He cannot make decisions. Now these are the main issue in his life. He has been alone and confused about his future. He has been concerned about how he can make an impact on his life. He is also a loner who has no options now. He appears to want direction and then to subvert it. He is in an ALF.

WATCH and WAIT: He has cognitive and emotional problems. His personality also suggests his style of interaction. He is structured and awkward socially. He is likely to be compliant but hide his symptoms. He may actually like the patient role. He has had OCD. He is rather anxious. He now has memory problems and has been having problems with attention also. Alcohol has caused problems in his life including a TBI. He has always been idiosyncratic. He is in an ALF and seems to be doing well. He needs direction and some support. His sister-in-law is very committed but has her hands full with her dementing husband.

The protocol calls for a slow psychoeducation process, motivational interviewing, some behavioral activation, and targets for his goals that can involve the new strategy for him to adjust day-to-day, given his cognitive problems. His personality suggests that he will not develop a substantial behavioral problem but will require some support and reframing in the process. This will take persistence, commitment and patience. His sister-in-law will require care also as she feels like a caregiver.

Checklist

Discernment: X

Core markers

- Validate Problem X
- Psychoeducation of Model X
- Assessmen X
- Alliance X
- Monitoring X
- Case formulation X

Problem List

1. ANXIETY: Monitor anxiety daily with SUDS. Have him in a CBT group weekly, teach relaxation, apply worry control, counseling focusing on self-image, coping and assertive needs, HRV biofeedback. Address his procrastination and rumination.

2. **COGNITION**: CT training, socialization in the ALF, exercise, periodic follow-up

Self or other monitoring: Small targets of behavior over time

Activity scheduling: X

Mastery-Pleasure activities: X

Graded task assignments: X

Verbal rehearsal of tasks: X

Managing situational contingencies:

Role playing and modeling: X

Environmental control:

Contingency plans (If-then):

Apply progressive relaxation: X

Concreteness training:

Imagery exercise for counter-experience:

Compassion training: X

Communication and Assertiveness training: X

Behavioral experiments:

Emotional exposure:

Homework: X

Longer Therapy processes:

CBT: Use of group and 3-6 column evidence record; moderate
●CD profile

ACT: use of acceptance interventions

■PT: Interpersonal role play

Lifestyles:

- Practice happiness
- Gratitude exercises
- Socialize: Less decline and live longer
- Develop resilience
- Exercise: Even housework
- Diet
- Somatic softening: Meditate and Mindfulness
- Values and Leisure

Follow-up: Re-evaluate every 6 months

Anxiety

MINI = non-Anxiety (subclinical ●CD)

GAD-7 = 5

BAI = 12

STAI = 23

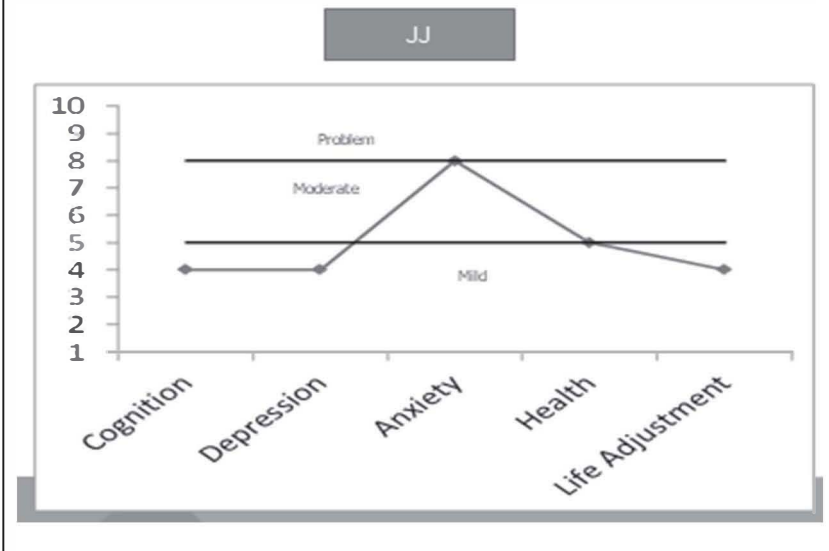
MBMD: Anxiety/Tension PR =50 (low)

Cognition

MoCA = 21

Relapse Issues: none noted

Updated domain model. He has been safely placed in an ALF, is aware of his situation, and has support. Anxiety is still a marker.



Conclusion

Recall that the value of personality is that it represents a profile that is unique to the person and also reflects traits like some others. This is the old personality dictum applied across the years. It suggests that the construct is most informative when indicating the patterns of the individual but that there are some commonalities across people. Importantly, the value of this construct is that it provides information on the processes of the person. Individual expressions of the person, however, unfold from goals, passions, and interests. This is ultimately what makes a person unique. It is not accessible.

Personality is a tried and true construct with one big advantage: It provides a loose model for understanding and treatment. Personality at later life seems to fill in the gaps of problems presented and suggests a

way for understanding issues and providing direction for change. It may not provide one intervention but it does wonders for the process, types of strategy and day-to-day intervention types. The cases throughout the book have been anchored in and allowed to unfold upon a personality base. The behavior and to some extent function of the person are (well) served by the dictates of their personality and coping program. If personality does not assist with treatment (it may well not), it at least provides cover for a plan and best considered interventions.

Concurrent treatments of mood and anxiety disorders are riddled with trial and error. Studies show that only about half of patients respond to their first course of treatment, whether it is psychotherapy or medication. Two people, clearly, can both be diagnosed with major depression and yet not have a single symptom in common. People differ due to personality and life -- the interactive power and influence of life events, mood, attitudes, and health. Again, the construct of personality provides some knowledge and heuristic help in the parsing of plans for care.

Watch and Wait provides the infrastructure for change. Personality allows for an explanation of the probable decline process and how this can be altered with time, plans, and feedback. It takes into account the whole field of problems and gives the subtext for change. Perhaps above all, knowledge of a personality profile allows the clinician to anticipate problems, structure care, and avoid excessive problems.

References

- Abrams, R. C. & Horowitz, S. V. (1996). Personality disorders after age 50: A meta-analysis. *Journal of Personality Disorders*, 10(3), 271-281.
- Abrams, R. C., & Horowitz, S. V. (1999). Personality disorders after age 50: A meta-analytic review of the literature. In E. Rosowsky, R. C. Abrams, & R. A. Zweig, *Personality disorders in older adults: Emerging issues in diagnosis and treatment* (pp. 55-68). Mahwah, NJ: Laurence Erlbaum Associates.
- Agronin, M. & Maletta, G. (2000). Personality disorders in late life: Understanding and overcoming the gap in research. *Journal of the American Geriatrics Society*, 8, 4-18.
- Alexopoulos, G. S., Raue, P., & Arean, P. (2003). Problem-solving therapy versus supportive therapy in geriatric major depression with executive dysfunction. *American Journal of Geriatric Psychiatry*, 11(1), 46-52.

- Ames, A. & Molinari, V. (1994). Prevalence of personality disorders in community-living elderly. *Journal of Geriatric Psychiatry and Neurology*, 7, 189-194.
- Arean, P. A., Perri, M. G., Nezu, A. M., Schein, R. L., Christopher, F., & Joseph, T. X. (1993). Comparative effectiveness of social problem-solving therapy and reminiscence therapy as treatments for depression in older adults. *Journal of Consulting and Clinical Psychology*, 61(6), 1003-1010.
- Bagby, R. M. & Farvolden, P. (2004). Personality Diagnostic Questionnaire-4 (PDQ-4). In M. Hersen (Editor-in-Chief) & M. Hilsenroth & D.L. Segal (Vol. Eds.), *Comprehensive handbook of psychological assessment: Vol. 2. Personality assessment* (pp. 122-133). Hoboken, NJ: Wiley.
- Balsis, S., Zweig, R. A., & Molinari, V. (2015). Personality disorders in later life. In P. A. Lichtenberg, B. T. Mast, B. D. Carpenter, & J. Loebach Wetherell (Eds.), *APA handbook of clinical geropsychology, Vol. 2: Assessment, treatment, and issues of later life.* (pp. 79-94). Washington, DC: American Psychological Association.
- Baltes, P. B., & Nesselroade, J. R. (1973). The developmental analysis of individual differences on multiple measures. In J. R. Nesselroade & H. W. Reese, *Life-span developmental psychology: Methodological issues.* Oxford, England: Academic Press.
- Benjamin, L. S. (1993). Every psychopathology is a gift of love. *Psychotherapy Research*, 3(1), 1-24.
- Beck, A. T. (1999). Cognitive aspects of personality disorders and their relation to syndromal disorders: A psychoevolutionary approach. In Cloninger, C. R. (Ed.), *Personality and Psychopathology* (pp. 411-429). Washington, D.C.: American Psychiatric Press
- Beutler, L. E., Mahoney, M. J., Norcross, J. C., Prochaska, J. O., Sollod, R. M., & Robertson, M. (1987). Training integrative and eclectic psychotherapists II. *Journal of Integrative and Eclectic Psychotherapy*, 6, 296-332.
- Booth, J. E., Schinka, J. A., Brown, L. M., Mortimer, J. A., & Borenstein, A. R. (2006). Five-factor personality dimensions, mood states, and cognitive performance in older adults. *Journal of Clinical And Experimental Neuropsychology*, 28(5), 676-683.
- Browne-Yung, K., Walker, R. B., & Luszcz, M. A. (n.d.). An Examination of Resilience and Coping in the Oldest Old Using Life Narrative Method. *GERONTOLOGIST*, 57(2), 282-291.

- Burish, T. G. & Carey, M. P. (1986). Conditioned aversive responses in cancer chemotherapy patients: Theoretical and developmental analysis. *Journal of Consulting and Clinical Psychology, 54*(5), 593-600.
- Burish, T. G., Carey, M. P., Krozely, M. G., & Greco, F. A. (1987). Conditioned side effects induced by cancer chemotherapy: Prevention through behavioral treatment. *Journal of Consulting and Clinical Psychology, 55*(1), 42-48.
- Burish, T. G., Carey, M. P., Wallston, K. A., Stein, M. J., Jamison, R. N., & Naramore Lyles, J. (1984). Health locus of control and chronic disease: An external orientation may be advantageous. *Journal of Social and Clinical Psychology, 2*(4), 326-332.
- Burish, T. G., Snyder, S. L., & Jenkins, R. A. (1991). Preparing patients for cancer chemotherapy: Effect of coping preparation and relaxation interventions. *Journal of Consulting and Clinical Psychology, 59*(4), 518-525.
- Burish, T. G., Vasterling, J. J., Carey, M. P., Matt, D. A., & Krozely, M. G. (1988). Posttreatment use of relaxation training by cancer patients. *The Hospice Journal, 4*(2), 1-8.
- Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegen, A., & Kaemmer, B. (1989). *Minnesota Multiphasic Personality Inventory-2 (MMPI-2): Manual for administration and scoring*. Minneapolis, MN: University of Minnesota Press.
- Carey, M. P. & Burish, T. G. (1985). Anxiety as a predictor of behavioral therapy outcome for cancer chemotherapy patients. *Journal of Consulting and Clinical Psychology, 53*(6), 860-865.
- Carey, M. P. & Burish, T. G. (1987). Providing relaxation training to cancer chemotherapy patients: A comparison of three delivery techniques. *Journal of Consulting and Clinical Psychology, 55*(5), 732-737.
- Carey, M. P. & Burish, T. G. (1988). Etiology and treatment of the psychological side effects associated with cancer chemotherapy: A critical review and discussion. *Psychological Bulletin, 104*(3), 307-325.
- Carstensen, L. L. (1988). The emerging field of behavioral gerontology. *Behavior Therapy, 19*, 259-281.
- Casey, D. A. & Schrodt, C. J. (1989). Axis II diagnoses in geriatric inpatients. *Journal of Geriatric Psychiatry and Neurology, 2*, 87-88.
- Caselli, R. J., Langlais, B. T., Dueck, A. C., Henslin, B. R., Johnson, T. A., Woodruff, B. K., ... Locke, D. E. C. (2018). Personality Changes During the Transition from Cognitive Health to Mild Cognitive

- Impairment. *Journal of the American Geriatrics Society*, 66(4), 671–678.
- Caspi, A., Roberts, B. W., & Shiner, R. L. (2005). Personality development: Stability and change. *Annual Review of Psychology*, 56, 453–484.
- Chapman, B. P. (2017). Implications of differential associations of neuroticism facets with cognitive function in late-life depression. *The American Journal of Geriatric Psychiatry*, 25(3), 230–232.
- Chatterjee, A., Strauss, M., Smyth, K., & Whitehouse, P. (1992). Personality changes in Alzheimer's disease. *Archives of Neurology*, 49(5), 486–491.
- Clark, L. A. (1993). *Schedule for Nonadaptive and Adaptive Personality*. Minneapolis, MN: University of Minnesota Press.
- Cohen, B. J., Nestadt, G., Samuels, J. F., Romanoski, A. J., McHugh, P. R., & Rabin, P. V. (1994). Personality disorder in later life: A community study. *British Journal of Psychiatry*, 165, 493–499.
- Coolidge, F. L. (2000). *Coolidge Axis II Inventory: Manual*. Colorado Springs, CO: Author.
- Coolidge, F. L., & Merwin, M. M. (1992). Reliability and Validity of the Coolidge Axis II Inventory: A New Inventory for the Assessment of Personality Disorders. *Journal of Personality Assessment*, 59(2), 223.
- Coolidge, F. L., Segal, D.L. & Rosowsky, E. (2006). *Prevalence and comorbidity of personality disorders among older persons*. Manuscript submitted for publication.
- Coon, D. W. & Thompson, L. W. (2003). The relationship between homework compliance and treatment outcomes among older adult outpatients with mild-to-moderate depression. *American Journal of Geriatric Psychiatry*, 11, 53–61.
- Costa, P. T., Jr. & McCrae, R. R. (1992). *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO-FFI) Professional Manual*. Odessa, FL: Psychological Assessment Resources.
- Dar-nimrod, I., Hansen, I. G., Proulx, T., Lehman, D. R., Chapman, B. P., & Duberstein, P. R. (2012). Coolness: An Empirical Investigation, 33(3), 175–185.
- Digman, J. M. (1990). Personality structure: Emergence of the five-factor model. *Annual Review of Psychology*, 41, 417–440.
- Dobson, K. S. (1989). A meta-analysis of the efficacy of cognitive therapy for depression. *Journal of Consulting and Clinical Psychology*, 57, 414–419.

- Eysenck, H. J. (1967). *The biological basis of personality*. New Brunswick, NJ: Transaction Publ.
- Eysenck, H. J. (1997). Personality and experimental psychology: The unification of psychology and the possibility of a paradigm. *Journal of Personality and Social Psychology*, 73(6), 1224-1237.
- Finkel, S. I. (1998). *Behavioral and psychological symptoms of dementia (module 2)*. UK: Gardiner-Caldwell Communications Limited.
- First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. S. (1997). *Structured Clinical Interview for DSM-IV Axis I Personality Disorders (SCID-II)*. Washington, DC: American Psychiatric Press.
- Fogel, B. S. & Westlake, R. (1990). Personality disorder diagnoses and age in inpatients with major depression. *Journal of Clinical Psychiatry*, 51(6), 232-235.
- Gallagher, D., & Thompson, L. W. (1981). *Depression in the elderly: A behavioral treatment manual*. Los Angeles: University of Southern California Press.
- Gallagher, D. L., & Thompson, L. W. (1982). Treatment of major depressive disorders in older adult outpatients with brief psychotherapies. *Psychotherapy Theory, Research, and Practice*, 19(4), 482-490.
- Gallagher-Thompson, D. & Coon, D. W. (1996). Depression. In J. I. Sheikh & I. D. Yalom (Eds.), *Treating the Elderly* (pp. 1-44). San Francisco: Jossey-Bass.
- Gallagher-Thompson, D., Hanley-Peterson, P., & Thompson, L. W. (1990). Maintenance of gains versus relapse following brief psychotherapy for depression. *Journal of Consulting and Clinical Psychology*, 58(3), 371-374.
- Gatz, M., Fiske, A., Fox, L. S., Kaskie, B. Kasl-Godley, J. E., McCallum, T. J., & Wetherell, J. L. (1998). Empirically validated psychological treatments for older adults. *Journal of Mental Health and Aging*, 4, 9-46.
- Gerson, S., Belin, T. R., Kaufman, A., Mintz, J. & Jarvik, L. (1999). *Harvard Review of Psychiatry*, 7(1), 1-28.
- Gould, S. L. & Hyer, L. A. (2004). Dementia and behavioral disturbance: Does premorbid personality really matter? *Psychological Reports*, 95(3), 1072-1078.
- Gross, J. J., Carstensen, L. L., Pasupathi, M., Tsai, J., Skorpen, C. G., & Hsu, A. Y. C. (1997). Emotion and aging: Experience, expression, and control. *Psychology and Aging*, 12(4), 590-599.

- Grossberg, G. & Desai, A. (2003). Management of Alzheimer's Disease. *Journal of Gerontology*, 58A(4), 531-553.
- Guedner, S. H., Loeb, S., Morris, D., Penrod, J., Bramlett, M., Johnston, L., & Scholtzhauer, P. (2001). A comparison of life satisfaction and mood in nursing home residents and community-dwelling elders. *Archives of Psychiatric Nursing*, 15(5), 232-240.
- Gutiérrez, F., Vall, G., Maria Peri, J., Bailiés, E., Ferraz, L., Gárriz, M., & Caseras, X. (2012). Personality disorder features through the life course. *Journal of Personality Disorders*, 26(5), 763-774.
- Hanley-Peterson, P., Futterman, A., Thompson, L., Zeiss, A. M., Gallagher, D., & Ironson, G. (1990). Endogenous depression and psychotherapy outcome in an elderly population. *Gerontologist*, 30, 51A.
- Hayflick, L. (1988). Aging in cultured human cells. In B. Kent & R. N. Butler (Eds.), *Human aging research: Concepts and techniques*. New York: Raven Press.
- Helson, R., Kwan, V. S. Y., John, O. P., & Jones, C. (2002). The growing evidence for personality change in adulthood: Findings from research with personality inventories. *Journal of Research in Personality*, 36(4), 287-306.
- Hinrichsen, G. A. (1999). Treating older adults with interpersonal psychotherapy of depression. *Journal of Clinical Psychology*, 55, 949-960.
- Hooker, K. & McAdams, D. (2003). Personality reconsidered: A new agenda for aging research. *Journal of Gerontology*, 58B(6), 296-304.
- Hauser, R. M., & Palloni, A. (2011). Adolescent IQ and Survival in the Wisconsin Longitudinal Study. *JOURNALS OF GERONTOLOGY SERIES B-PSYCHOLOGICAL SCIENCES AND SOCIAL SCIENCES*, 66, 91-101.
- Horowitz, L. M., Rosenberg, S. E., Baer, B. A., Ureno, G., & Villasenor, V. S. (1988). Inventory of interpersonal problems: Psychometric properties and clinical applications. *Journal of Consulting and Clinical Psychology*, 56, 885-892.
- Hyer, L. (2014). *Psychological Treatment of Older Adults: A Holistic Model*. New York: Springer Press
- Hyer, L., Hilton, N., Friedman, M., & Sacks, A. (in press). GIST: An Efficient and Effective Cognitive Behavioral Therapy In Long Term Care *Clinical Gerontologist*.
- Hyer, L., Sohnle, S., Miller, M., & Hamer, R. (2003) Cognitive testing battery: Differences among groups with dementia, Huntington's Disease, and controls. *Psychological Reports*, 93, 497-504.

- Hyer, L., Swanson, G. & Lefkowitz, R. (1990). The use of a cognitive schema model with stress groups at later life. *Clinical Gerontologist. Special Issue: Group Therapy in Nursing Homes*, 9(3/4), 145-190.
- Hyer, L., Swanson, G., Lefkowitz, R., Hillesland, D. Davis, H., & Woods, M. G. (1990). The application of the cognitive behavioral model of two older stressor groups. *Clinical Gerontologist*, 9, 145-190.
- Hyler, S. E. (1994). *Personality Diagnostic Questionnaire, 4th edition, PDQ-4+*. New York: New York State Psychiatric Institute.
- Jamison, C. & Scogin, F. (1995). The outcome of cognitive bibliotherapy with depressed adults. *Journal of Consulting and Clinical Psychology*, 63(4), 644-650.
- Jeste, D. V., Alexopoulos, G. S., Bartels, S. J., Cummings, J. L., Gallo, J. J., Gottlieb, G. L., et al. (1999). Consensus statement on the upcoming crisis in geriatric mental health. *Archives of General Psychiatry*, 56(9), 848-853.
- Jones, C. J. & Meredith, W. (1996). Patterns of personality change across the life span. *Psychological Aging*, 11(1), 57-65.
- Karel, M. C., & Hinrichsen, G. (2000). Treatment of depression in late life: Psychotherapeutic interventions. *Clinical Psychology Review*, 20, 707-729.
- Kunik, M. E., Mulsant, B. H., Rifai, A. H., Sweet, R. A., Pasternak, R., Rosen, J., & Zubenko, G. S. (1993). Personality disorders in elderly inpatients with major depression. *The American Journal of Geriatric Psychiatry*, 1(1), 38-45.
- Lawton, M. P., Kleban, M. H., Rajagopal, D., & Dean, J. (1992). Dimensions of affective experience in three age groups. *Psychological Aging*, 7(2), 171-184.
- Lee, K., & Ashton, M. C. (2012). *The H Factor of Personality: Why Some People Are Manipulative, Self-Entitled, Materialistic, and Exploitive—And Why It Matters for Everyone*. Waterloo, Ontario, Canada: Wilfrid Laurier University Press.
- Lenzenweger, M. F., Johnson, M. D., & Willett, J. B. (2004). Individual growth curve analysis illuminates stability and change in personality disorder features. *Archives of General Psychiatry*, 61, 1015-1024.
- Lichstein, K. L. & Johnson, R. S. (1993). Relaxation for insomnia and hypnotic medication use in older women. *Psychological Aging*, 8(1), 103-111.
- Livesley, W. J. (1998). Suggestions for a framework for an empirically based classification of personality disorder. *Canadian Journal of Psychiatry*, 43, 137-147.

- Loranger, A. W. (1999). *International Personality Disorder Examination: DSM-IV and ICD-10 Interviews*. Odessa, FL: Psychological Assessment Resources.
- Low, L.-F., Harrison, F., & Lackersteen, S. M. (2013). Does Personality Affect Risk for Dementia? A Systematic Review and Meta-Analysis. *AMERICAN JOURNAL OF GERIATRIC PSYCHIATRY*, 21(8), 713–728.
- Lynch, T. R., Morse, J. Q., Mendelson, T., & Robins, C. J. (2003). Dialectical behavior therapy for depressed older adults: A randomized pilot study. *American Journal of Geriatric Psychiatry*, 11(1), 33-45.
- Magai, C. & Passman, V. (1997). The interpersonal basis of emotional behavior and emotion regulation. In M. P. Lawton & K. W. Schaie (Eds.), *Annual Review of Gerontology and Geriatrics: Vol. 17*. New York: Springer.
- Margo, J. L. Robinson, J. R., & Corea, S. (1980). Referrals to a psychiatric service from old people's homes. *British Journal of Psychiatry*, 136, 396-401.
- McCarthy, P. R., Katz, I. R. and Foa, E. B. (1991) Cognitive-behavioural treatment of anxiety in the elderly: A proposed model. In C. Salzman & B. D. Lebowitz (Eds.), *Anxiety in the Elderly*. New York: Springer.
- McCrae, R. R. (2002). The maturation of personality psychology: Adult personality development and psychological well-being. *Journal of Research in Personality*, 36(4), 307-317.
- McCrae, R. R., Costa, P. T., Jr. (2003). *Personality in adulthood: A five-factor theory perspective* (2nd ed.). New York: Guilford Press.
- McCrae, R. R., & Costa, P. T., Jr. (2008). The five-factor theory of personality. In O. P. John, R. W. Robins, & L. A. Pervin (Eds.), *Handbook of personality: Theory and research* (pp. 159-181). New York, NY, US: Guilford Press.
- McReynolds, C. J., Garske, G. G., & Turpin, J. O. (1999). Psychiatric rehabilitation: A survey of rehabilitation counseling education programs. *The Journal of Rehabilitation*, 65(4), 45-49.
- Mega, M., Cummings, J., Fiorello, T., & Combein, J. (1996). The spectrum of behavioral changes in Alzheimer's disease. *Neurology*, 46(1), 130-135.
- Mezzich, J. E., Fabrega, H., Coffman, G. A., & Glavin, Y. (1987). Comprehensively diagnosing geriatric patients. *Comprehensive Psychiatry*, 28, 68-76.
- Miller, M. D., Frank, E., Comes, C., Houck, P. R., & Reynolds, C. F. 3rd (2003). The value of maintenance interpersonal psychotherapy (IPT) in

- older adults with different IPT foci. *American Journal of Geriatric Psychiatry*, 11(1), 97-102.
- Millon, T. (1969). *Modern psychopathology: A biosocial approach to maladaptive learning and functioning*. Philadelphia: Saunders.
- Millon, T. (1981). *Disorders of personality: DSM-III, Axis II*. New York: Wiley.
- Millon, T. (1983). *Millon clinical multi-axial inventory manual (3rd ed.)*. Minneapolis: National Computer Systems.
- Millon, T. (1988). Personologic psychotherapy: Ten commandments for a post-eclectic approach to integrative treatment. *Psychotherapy*, 25, 209-219.
- Millon, T. (1990). *Toward a new personology: An evolutionary model*. New York: Wiley-Interscience.
- Millon, T. & Davis, R. D. (1996). *Disorders of personality: DSM-IV and beyond*. New York: Wiley.
- Millon, T., Davis, R., & Millon, C. (1997). *MCMI-III manual (2nd ed.)*. Minneapolis: National Computer Systems.
- Millon, T., Antoni, M. H., Millon, C., Meagher, S., & Grossman, S. (2001). *Test manual for the Millon Behavioral Medicine Diagnostic (MBMD)*. Minneapolis, MN: National Computer Services.
- Moffitt, T. E., Caspi, A., Harrington, H., Milne, B. J. (2002). Males on the life-course-persistent and adolescence-limited antisocial pathways: Follow-up at age 26 years. *Development and Psychopathology*, 14, 179-207.
- Molinari, V., Ames, A., & Essa, M. (1994). Prevalence of personality disorders in two geropsychiatric inpatient units. *Journal of Geriatric Psychiatry and Neurology*, 7, 209-215.
- Molinari, V., Kunik, M. E., Snow-Turek, A. L., DeLeon, H. & Williams, W. (1999). Age-related personality differences in inpatients with personality disorder: A cross-sectional study. *Journal of Clinical Geropsychology*, 5, 191-202.
- Molinari, V. & Marmion, J. (1993). Personality disorders in geropsychiatric outpatients. *Psychological Reports*, 73, 256-258.
- Molinari, V. & Marmion, J. (1995). Relationship between affective disorders and Axis II diagnoses in geropsychiatric patients. *Journal of Geriatric Psychiatry and Neurology*, 8, 61-64.
- Mordekar, A., & Spence, S. A. (2008). Personality disorder in older people: how common is it and what can be done? *Advances in Psychiatric Treatment*, 14(1), 71-77.

- Mroczek, D. K., Spiro, A. III. (2003). Modeling intraindividual change in personality traits: Findings from the Normative Aging Study. *Journal of Gerontology*, 58B, P153-P165.
- Mroczek, D. K., & Spiro, A. III. (2007). Personality change influences mortality in older men. *Psychological Science*, 18(5), 371-376.
- Neugarten, B. (1964). *Personality in middle and later life*. New York: Atherton Press.
- Nezu, A. M., Nezu, C. M., Friedman, S. H., Faddis, S., & Houts, P. S. (1998). *Helping cancer patients cope: A problem-solving approach*. Washington, D.C.: American Psychological Association Press.
- Nussbaum, P. (1997). *Handbook of the neuropsychology of aging*. New York: Plenum Press.
- Paris, J. (2003). *Personality disorders over time*. Washington, DC: American Psychiatric Press.
- Persons, J. B. (2005). Empiricism, mechanism, and the practice of cognitive-behavior therapy. *Behavior Therapy*, 36, 107-118.
- Pfohl, B., Blum, N & Zimmerman, M. (1997). *Structured Interview for DSM-IV Personality (SIDP-IV)*. Arlington, VA: American Psychiatric Publishing.
- Pilkonis, P. A., & Frank, E. (1988). Personality pathology in recurrent depression: Nature, prevalence, and relationship to treatment response. *The American Journal of Psychiatry*, 145(4), 435-441.
- Powers, A. D., & Oltmanns, T. F. (2012). Personality disorders and physical health: A longitudinal examination of physical functioning, healthcare utilization, and health-related behaviors in middle-aged adults. *Journal of Personality Disorders*, 26(4), 524-538.
- Resilience: Boost Your Ability to Bounce Back. (cover story). (2018). *Mind, Mood & Memory*, 14(10), 1.
- Reynolds, C. F. & Kupfer, D. J. (1999). Depression and aging: A look to the future. *Psychiatric Services*, 50, 1167-1172.
- Roberts, B. W., & DelVecchio, W. F. (2000). The rank-order consistency of personality traits from childhood to old age: A quantitative review of longitudinal studies. *Psychological Bulletin*, 126(1), 3-25.
- Roberts, B. W., Walton, K. E., & Viechtbauer, W. (2006). Patterns of mean-level change in personality traits across the life course: A meta-analysis of longitudinal studies. *Psychological Bulletin*, 132(1), 1-25.
- Rosowsky, E. & Gurian, B. (1991). Borderline personality disorder in late life. *International Psychogeriatrics*, 3, 39-52.
- Rubinstein, R. L. & Lawton, M. P. (1997). *Depression in long-term and residential care: Advances in research and treatment*. New York: Springer Publishing Company.

- Ruegg, R. & Frances, A. (1995). New research in personality disorders. *Journal of Personality Disorders*, 9(1), 1-8.
- Rybarczyk, B., DeMarco, G., DeLaCruz, M., & Lapidus, S. (1999). Comparing mind-body wellness interventions for older adults with chronic illness: Classroom versus home instruction. *Behavioral Medicine*, 24(4), 181-190.
- Sadavoy, J. (1996). Personality disorder in old age: Symptom expression. *Clinical Gerontologist*, 16(3), 19-36.
- Sadavoy, J. (1999). The effect of personality disorder on axis I disorder in the elderly. In M. Duffy (Ed.), *Handbook of Counseling and Psychotherapy with Older Adults* (pp. 397-413). New York: Wiley.
- Sadavoy, J. & Fogel, F. (1992). Personality disorders in old age. In J. E. Birren, R. B. Sloane, & G. D. Cohen (Eds.), *Handbook of mental health and aging* (2nd ed.). San Diego, CA: Academic Press.
- Schuster, J.-P., Hoertel, N., Le Strat, Y., Manetti, A., & Limosin, F. (2013). Personality Disorders in Older Adults: Findings From the National Epidemiologic Survey on Alcohol and Related Conditions. *AMERICAN JOURNAL OF GERIATRIC PSYCHIATRY*, 21(8), 757-768.
- Scogin, F. & McElreath, L. (1994). Efficacy of psycho-social treatments for geriatric depression: A qualitative review. *Journal of Consulting and Clinical Psychology*, 62, 69-74.
- Scogin, F., Rickard, H. C., Keith, S., Wilson, J., & McElreath, L. (1992). Progressive and imaginal relaxation training for elderly persons with subjective anxiety. *Psychological Aging*, 7(3), 419-424.
- Scogin, F., Welsh, D., Hanson, A., Stump, J., & Coates, A. (2005). Evidence-based psychotherapies for depression in older adults. *Clinical Psychology: Science and Practice*, 12(3), 222-237.
- Segal, D. L. & Coolidge, F. L. (1998). Personality disorders. In A.S. Bellack & M. Hersen (Eds.), *Comprehensive clinical psychology: Vol. 7. Clinical geropsychology* (pp. 267-289). New York: Elsevier Science.
- Segal, D. L., Coolidge, F. L., & Hersen, M. (1998). Psychological testing of older people. In I. H. Nordhus, G. R. VandenBos, S. Berg, & P. Fromholt (Eds.), *Clinical geropsychology*. Washington, DC: American Psychological Association.
- Segal, D. L., Coolidge, F. L., & Rosowsky, E. (2006). *Personality disorders and older adults: Diagnosis, assessment, and treatment*. New York: Wiley.
- Segal, D. L., Hersen, M., Kabacoff, R. I., Falk, S. B., Van Hasselt, V. B., & Dorfman, K. (1998). Personality disorders and depression in

- community-dwelling older adults. *Journal of Mental Health and Aging*, 4, 171-182.
- Segal, D. L., Hersen, M., Van Hasselt, V. B., Silberman, C. S., & Roth, L. (1996). Diagnosis and assessment of personality disorders in older adults: A critical review. *Journal of personality disorders*, 10(4), 384-399.
- Segal, D. L., Hook, J. N., & Coolidge, F. L. (2001). Personality dysfunction, coping styles, and clinical symptoms in younger and older adults. *Journal of Clinical Geropsychology*, 7, 201-212.
- Segal, D. L., Marty, M. A., Meyer, W. J., & Coolidge, F. L. (2012). Personality, suicidal ideation, and reasons for living among older adults. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, 67(2), 159-166.
- Shedler, J., & Westen, D. (2007). The Shedler-Westen Assessment Procedure (SWAP): Making Personality Diagnosis Clinically Meaningful. *Journal of Personality Assessment*, 89(1), 41-55.
- Small, B. J., Hertzog, C., Huлтsch, D. F., & Dixon, R. A. (2003). Stability and change in adult personality over 6 years: Findings from the Victoria Longitudinal Study. *Journal of Gerontology*, 58B, P166-P176.
- Snow, L., Powers, D., & Liles, D. (2006). Cognitive-Behavioral Therapy for Long-Term Care Patients with Dementia (pp. 210-241). In L. Hyer and R. Intrieri (Eds.) *Geropsychological interventions in Long Term Care*. New York: Springer.
- Strack, S. (1991). *Manual for the Personality Adjective Check List (PACL), Revised Edition*. Pasadena, CA: 21st Century Assessment.
- Steunenberg, B., Twisk, J. W. R., Beekman, A. T. F., Deeg, & Kerkhof, A. J. F. M. (2005). Stability and change of neuroticism in aging. *Journal of Gerontology*, 60B, P27-P33.
- Sutin, A. R., Zonderman, A. B., Ferrucci, L., & Terracciano, A. (2013). Personality traits and chronic disease: Implications for adult personality development. *The Journals of Gerontology: Series B: Psychological Sciences and Social Sciences*, 68(6), 912-920.
- Teeter, R. B., Garetz, F. K., Miller, W. R., & Heiland, W. F. (1976). Psychiatric disturbances of aged patients in skilled nursing homes. *American Journal of Psychiatry*, 133, 1430-1434.
- Teri, L., Curtis, J., Gallagher-Thompson, D., & Thompson, L. (1994). Cognitive-behavioural therapy with depressed older adults. In L. S. Schneider, C. F. Reynolds, B. D. Lebowitz, & A. J. Friedhoff (Eds.), *Diagnosis and treatment of depression in late life: Results of the NIH Consensus Development Conference*. American Psychiatric Press, Washington DC.

- Teri, L., Logsdon, R. G., Uomoto, J., & McCurry, S. M. (1997). Behavioral treatment of depression in dementia patients: A controlled clinical trial. *Journal of Gerontology*, 52B(4), P159-P166.
- Teri, L., & McCurry, S. M. (2000). Psychosocial therapies with older adults. In Coffey CE, Cummings JC (eds.), *Textbook of geriatric neuropsychiatry* (2nd ed.), pp. 861-890. Washington DC: American Psychiatric Press.
- Terracciano, A., McCrae, R. R., & Costa, P. T. Jr. (2006). Longitudinal trajectories in Guilford-Zimmerman Temperament Survey Data: Results from the Baltimore Longitudinal Study of Aging. *Journal of Gerontology*, 61B, P108-P116.
- Thompson, L. W. (1996). Cognitive-behavioral therapy and treatment for late-life depression. *Journal of Clinical Psychiatry*, 57(5), 29-37.
- Thompson, L. W., Gallagher, D., & Breckenridge, J. S. (1987). Comparative effectiveness of psychotherapies for depressed elders. *Journal of Consulting and Clinical Psychology*, 55 (3), 385-390.
- Thompson, L. W., Gallagher, D., & Czirr, R. (1988). Personality disorder and outcome in the treatment of late-life depression. *Journal of Geriatric Psychiatry*, 21, 133-146.
- Thompson, L. W., Gallagher, D., Hanser, Gantz, & Steffen (1991, November). Comparison of desipramine and cognitive/behavioral therapy in the treatment of late-life depression. Paper presented at the meeting of the Gerontological Society of America, San Francisco.
- Thompson, L. W., Gallagher-Thompson, D., Dick, L. P. (1995). *Cognitive-Behavioral Therapy for Late Life Depression: A Therapist Manual*. Palo Alto, CA: Older Adult and Family Center, Veterans Affairs Palo Alto Health Care System.
- Tracey, T. J. & Ray, P. B. (1984). Stages of successful time-limited counseling: An interactional examination. *Journal of Counseling Psychology*, 31(1), 13-27.
- van Alphen, S. P. J., Engelen, G. J. J. A., Kuin, Y., Hoijsink, H., & Derksen, J. J. L. (2004). [Construction of a scale to signal personality disorders in the elderly]. *Tijdschrift Voor Gerontologie En Geriatrie*, 35(5), 186-195.
- Wallston, K. A., Smith, R. A., King, J. E., Smith, M. S., Rye, P., & Burish, T. G. (1991). Desire for control and choice of antiemetic treatment for cancer chemotherapy. *Western Journal of Nursing Research*, 13(1), 12-23.
- Westen, D., & Shedler, J. (1999a). Revising and assessing Axis II, part 1: Developing a clinically and empirically valid assessment method. *American Journal of Psychiatry*, 156, 258-272.

- Westen, D., & Shedler, J. (1999b). Revising and assessing Axis II, part 2: Toward an empirically based and clinically useful classification of personality disorders. *American Journal of Psychiatry*, 156, 273-285.
- Widiger, T. A., Mangine, S., Corbitt, E. M., Ellis, C. G., & Thomas, G. V. (1995). *Personality Disorder Interview-IV: A semistructured interview for the assessment of personality disorders* (Professional manual). Odessa, FL: Psychological Assessment Resources.
- Wilson, R. S., Schneider, J. A., Arnold, S. E., Bienias, J. L., & Bennett, D. A. (2007). Conscientiousness and the incidence of Alzheimer disease and mild cognitive impairment. *Archives of General Psychiatry*, 64(10), 1204-1212.
- Zanarini, M. C., Frankenburg, F. R., Sickel, A. E., & Yong, L. (1996). *The Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV)*. Belmont, MA: McLean Hospital.
- Zeiss, A. M. & Steffen, A. (1996). Treatment issues with elderly clients. *Cognitive and Behavioural Practice*, 3, 371-389.
- Zeiss, R. A., Delmonico, R. L., Zeiss, A. M., & Dombrand, L. (1991). Psychologic disorder and sexual dysfunction in elders. *Clinical Geriatric Medicine*, 7(1), 133-151.
- Zweig, R. A. & Agronin, M. E. (2006). Personality disorders. In M. E. Agronin & G. J. Maletta (Eds.), *Principles and Practice of Geriatric Psychiatry* (pp. 449-470). Philadelphia: Lippincott, Williams, & Wilkins.

CHAPTER 12

PERSPECTIVES ON HEALTH CARE

We have alluded to these facts in many of the previous chapters: Federal government spending tends to break down according to the following markers: 25% for health, medicine, Medicaid, and CHIP; 24% for Social Security, 16% for defense, 13% for other mandatory, and 16% non-defensive discretionary. Interest is 6%. In addressing the social determinates of health and health inequities it has become evident that the United States has large avoidable differences in health across groups. For example, the life expectancy of 40-year-old men in the poorest 1% of the income distribution lives 14.6 years shorter than for men in the richest 1%; and for women the difference is 10.1 years. Yes, there is more current mobility in SES levels these days, but not enough to wipe out the disparity of the SES factor. Health disparities are found also by education, race, ethnicity, sex, sexual orientation, and place of residence. It is true also that policies have reduced social disadvantage and have reduced health inequities. The health gap between blacks and whites, for example, has narrowed in the decades since the Civil Rights Act. But, based on measures of cost, quality, efficiency, and equity, the US Health System is substantially underperforming: a failure unlikely to respond to incremental modifications in the prevailing system. That the US Health System ranks only 37th in performance among 191 nations and that the United States has the poorest health of the rich nations are distinct markers. Failing health grades begin in childhood (Clark et al., 2016; Hero et al., 2017).

We are living longer and we have more of everything – time, leisure, money, health care, choices, etc. –but with more disabilities. Reports are that, although longevity has reached an upper limit, 96% of infants born in developed nations will live to 50, 84% will survive to 65, and 75% of deaths will occur between 65 and 95 (Olshansky, 2018). Although it is widely thought that as people grow older their functioning largely deteriorates, studies in recent years have shown that there can be a reversed trend. That is, their functioning improves or decline is deferred. At the least, there is more variability. In several studies, functional

limitations, largely mobility and movement with ADLs and IADLs, are markers and they can vacillate as up-and-down markers.

Changes in mobility, changes in movement, changes in ADL function, and changes in IADL function, therefore, vary. In 2015, low back pain and major depression ranked among the top 10 greatest contributors to disability in virtually every country in 20 studied, according to The Lancet (Voss et al., 2016). This causes more health loss than diabetes, chronic obstructive pulmonary disease, and asthma combined. As the world population grows and the proportion of elderly people increases, the number of people living with suboptimum health is set to rise rapidly over the decades. The rates of diabetes, for example, have been substantial, rising over 40% in the past 23 years. Death rates from diabetes only increased by 9% over that same period. The fact that mortalities are declining faster than non-fatal disease is further evidence of the importance of paying attention to health. We have arrived at the fruits of our success; but what success.

Increases in late life health further increase economic toxicity and quality of life. Chronic health problems related to the unprecedented aging of the human population in the 21st century threaten to disrupt economies and degrade the quality of later life throughout the developed world. Fundamental aging processes can be targeted by nutritional, genetic, and pharmacologic interventions to enhance both health and longevity in animals at least. Biological aging can now be slowed. The change processes of biology (metabolism, proteostasis, inflammation, adaptation to stress, epigenetics, stem cells, and macromolecular change) are in play. Geroscience is the science that links biological aging processes to health and longevity. The tie to disease, while inevitable, is now more manageable. The US government annually publishes the rate of death from individual diseases stratified by age. The rate of death increases logarithmically with advancing age in all major causes of death. Modally, the prevention of one disease will have limited effect on population health as one disease will be replaced by another. The replacement is often more insidious and problematic.

Below we end our journey in this chapter with some ideas on the health care industry. We start with cost and meander through issues of the medical model and, especially, mental health. We provide suggestions and end with some ideas about ethics in this process.

Cost

Cost and problems at later life abound and are getting worse. The connection to poverty, use of prescriptions, health care use, compliance, adverse drug reactions, and LTC use is palpable. According to the 2000 US census, the most prevalent type of limitation or disability among Americans over 65 years was physical (28.6%), followed by limitations that affect leaving the house (20.8%), then sensory limitations (14.2%), followed by cognitive (10.6%) and self-care limits (Freedman et al., 2002). The overall prevalence of disability among older adults was 41.9%, and was even higher for elders living in poverty (Hung et al., 2011). Take diabetes again: At 73, there is a twenty-four percent chance someone will have diabetes, and she (55% are female) will also be ill-equipped to manage her health; 56% have a high school education or less, 45% live on under 200% of the Federal poverty line, and 29% live alone. The average older adult makes just 3 visits to a primary care physician provider per year, each lasting a mere 17 minutes. There are considerably more with diabetes. The mismatch is even worse for older low-income adults and for those in under-deserved urban neighborhoods where access to healthcare is often poor.

In a large study in JAMA by Dieleman et al. (2016), US healthcare spending was noted to be on the increase and to account for more than 70% of the US economy. This group systematically and comprehensively estimated US spending on personal healthcare and public health according to conditions, age, sex group, and type of care. From 1996 to 2013, over 30 trillion dollars in personal healthcare spending was disaggregated by 155 conditions, age and sex group, and type of care. Among these 155 conditions, diabetes had the highest rate of health spending in 2013 with an estimated 101 billion being spent. In some modeled estimates in the US, spending on personal healthcare and public health showed a substantial increase from 1996 to 2013 with spending on diabetes, ischemic heart disease, low back pain, and neck pain accounting for the highest amount of spending by disease category. The rate of change in annual spending varies considerably among different conditions and types of care. This clearly needs better targeting or fixing.

Medical System

The American medical system has created false expectations. All cancers cannot be cured, surgery is not the only answer to heart disease, and screening for everything does postpone problems but serious diseases

persist. Early detection is one answer. Diet, nutrition, healthy lifestyle, nonsmoking, and moderation of alcohol consumption are simply not adequately emphasized. Even more problematic, when the medical model is combined with the current US reimbursement system based on volume care, the clinical focus and incentives are directed toward doing more in terms of interventions, such as tests, medications, and hospitalizations, but with little understanding of the distortions or overuse of services created by this care. For instance, patients with an increase in falls are often admitted to hospitals and evaluated extensively for cardiac and neurological conditions, but then may be discharged without sufficient long-term rehabilitation or social service support. When physicians or psychologists encounter older persons with cognitive problems, lethargy, mood disorders, or frailty, the response is often to perform an extensive diagnostic evaluation and then prescribe medication rather than facilitating solutions and support for patients that deal with social isolation, physical activity limitations, or family support needs. Medication tests for older adults can expand excessively as drugs are added for individual organ system fixes when a more multifactorial approach is needed. Older adults and their families can then spend excessive amounts of their time traveling to and from disparate clinical offices and other sites of care. The current burdensome complexity of the International Statistical Classification system, as well as the current procedural terminology code, is clear evidence that the medical model is flawed.

There is no exit here as patients with complex diseases have a higher risk of developing another one. Multi-morbidity represents a huge problem in everyday clinical practice, because it makes it more difficult to provide successful treatment. We say again: one range of diseases are genetic, while others are caused by environmental factors, or may result from a combination of both. With a new set of phenotypic data of diseases in combination with molecular biological data and modern complex system mathematics, it is possible to understand the interaction between genetic and environmental disorders in the development of complex diseases. The science of complex systems aims, amongst other things, to identify relevant information within the “big data ocean” that will lead to clinically relevant knowledge and hence to better treatment options (Klimek et al., 2016).

If there is a consensus, it is that we exist in a chronic disease society and one largely unfit but living much longer. Chronic diseases are long-term conditions with slow disease progression and without an effective cure; 38 million people die from chronic diseases each year. In addition, 16 million of these deaths occur before the age of 70 years. Chronic

diseases may lead to alterations in brain structure and function and are associated with types of dementia or just cognitive decline. Strategies are needed to reduce disease-related cognitive impairment in chronic disease patients. Unfortunately we have also lost about a year in longevity in the past two years.

Limited Foresight

So, why is the US performing so poorly relative to both potential and size of investment? The answer lies in the fissure between the current reality and understanding that multiple factors interact to determine health status and the long-standing allocation of attention and resources to the acute short-term and the limited treatment of illnesses and injuries that present for care. The persistent and increasing gap between current scientific understanding about determinates of health and the ability to move beyond fragmented and episodic patterns of care is reinforced by outdated concepts of disease and causation, and measurement systems that often continue to count what was once thought to be important and easy to measure, but that in fact offers scant perspective for health gains. And, prevention matters: no prevention, more cost.

In 2004, Blockbuster Video had a market capitalization of more than \$5 billion dollars with 9,000 stores nationally staffed by 60,000 employees. In 2010 the company declared bankruptcy before being dissolved. Like many once proud firms before, Blockbuster was a victim of its inability to recognize that emerging technologies had enabled a seismic shift in what consumers were demanding – in Blockbuster's case the convenience of being able to pick movies from the comfort of one's own home via Netflix. Many companies in many industries have made the same mistake: focusing on what they can easily produce instead of what the customers really want. Healthcare providers tend to focus on providing health services as opposed to producing health. Engaging patients will be easier if we give them more of what they want: better health rather than more health services. Healthcare in the US is at an important juncture. We have seen amazing scientific advances in the past century, but despite spending far more than any other country on healthcare services, outcomes in the United States for many portions of the population remain poor.

The enactment of the Affordable Care Act (ACA) in 2010 was the most monumental change in US healthcare policies since the passing of Medicaid and Medicare in 1965. Despite a bill that was more than 900 pages long, the primary goals of the legislation were clear: increase the number of individuals with health insurance, improve the quality of care,

and tame the seemingly inexorable increases in the cost of care. Viewpoints that address this have been rather caustic. Lavizzo-Mourey (2015) emphasizes the broader culture of health suggesting that because of the changing epidemiology of disease from acute to chronic, many more of the solutions rest in collaboration with the medical care system and communities of patients. Perhaps the US healthcare system is smart to move toward bundle payments, accountable care institutions, and reimbursement based on performance outcomes. Physicians and medical care professionals will increasingly find themselves in collaboration with partnerships with those who work outside the traditional spaces of the healthcare system. These include urban planners, legal people, and others.

Yes, we have come a long way on behalf of better care and better cost systems. Hippocrates wrote that the natural healing force within each one of us is the greatest force to getting well. While the link between physical and emotional health has been recognized since antiquity, the advent of highly effective curative somatic treatment medicine ignored the link between the mind and the body. Clinical practice changes to improve health in the community consistently rate investment in behavioral health/mental health services alongside physician health services as number one. Building interdisciplinary teams and creating community partnerships using APNs and physician extender roles, as well as investing in tools for physicians such as EMRs, also rate highly. In addition to physical and mental health, financial health has a significant impact on patients. The person has holistic needs. The lack of a source of income and access to health insurance are fundamental elements of a hierarchy of needs (such as Maslow's: food and shelter have substantial effects on health outcomes). Social needs are always important. They are best represented by employment, then access to healthy food, then housing, and then utilities.

Medical Model Changes

Maybe there are solutions in process. Care has been broadened. Accountable care organization models provide hospitals with financial incentives to implement innovative care coordination to ensure that patients get the right care at the right time. Such care coordination models include improving the use of patient-centered medical home care, clinician-patient continuity, team-based approaches, use of community hospital base support, specialized outpatient programs for high-risk patients, home visits, patient navigation to link primary care and specialty care, and referral-tracking systems. Evidence of the cost effectiveness of

such care coordination of individuals with mental illness and coexisting physical conditions is clear, but also needs more commitment as well as growth and specificity.

Medicine itself as a profession is transitioning from the initial era of ascendancy, a period of professional trust and prerogative, through a period of accountability, scrutiny, measurement, incentives, and markets, to perhaps the 3rd era of restraint and greater moral direction. Several specific directions are given by Berwick (JAMA, 2016). These include reducing mandatory measurement, stopping complex individual incentives, shifting the business strategy from revenue to quality, giving up professional prerogative when it hurts the whole system, using improvement science, ensuring complete transparency, protecting civility, hearing the voices of the people served, and rejecting greed. As we have seen, this has many markers.

Regarding depression, Hoefft, Hinton, Liu, and Unutzer (2015) showed the results of a selective review, supplemented by more current recommendations from national experts, and highlighted three priority research areas to improve health services at late-life. These include focusing on the unique needs of the patient through patient-centered care and culturally sensitive care, involving caregivers outside the traditional clinical care team, and involving alternate settings of care. This allows for a change in the way older adults are impacted by real life.

McClintock and colleagues (2016) from the University of Chicago show that the limitations of the medical model are evident. In the study that they conducted the investigators evaluated data on a community cohort of 3,000 elderly individuals, of 57-85 years of age, living at home. This was taken from the National Social Life, Health, and Aging Project. The traditional medical model characterized two-thirds of those studied as being in robust health. A comprehensive model, on the other hand, looked at more health issues from a broader perspective. The study investigators used 54 health variables consistent with the medical model to identify discrete classes and disease clusters in the cohort. Each of these was statistically distinct. They included the most prevalent causes of death, such as heart disease, cancer, stroke, and diabetes, along with arthritis and hypertension, common diseases, but with less of an immediate mortality risk. They found that the medical model did not register the range and complexity of the health concerns in this cohort, including physical function and psychological wellbeing. The comprehensive model of health included five additional functional dimensions (health behaviors, psychological health, sensory ability, neuro-immune functioning, and mobility, with variables such as depression, memory loss, and poor

vision). Findings across six different classes of health ranging from robust to multimodal suggested that diagnoses, such as cancer and hypertension, were less important predictive factors of overall health status than mental health, sensory and physical function, and previous health history. This might help develop a more clinically appropriate taxonomy to describe the health status of older adults and to reimburse for value in healthcare. Helping to facilitate the evolution of this new patient-centered approach is a challenge.

Several other systems have tried to provide ways in which to improve patient care using team-based approaches for medical conditions. On several of these, the data allow teams to easily view trending average costs and overall outcomes of patients by a specific condition. The University of Utah does this, for example, and allows for a deep dive into specific costs of treatment supplies, implants, drugs, and diagnostic tests, like imaging procedures. Informed across the patient journey, healthcare providers can easily see the cost for the same procedures performed by other healthcare providers and learn what accounts for the differences. Healthcare is not like manufacturing computers or cars. Patients are not the same shape and size and, on top of that, healthcare providers treat patients differently based on their training and experience. There are complex and fascinating solutions in data that are mined and that can provide information. Value transformation is a long journey of incremental improvements. Progress is slow. One just has to survey the current medical TV shows (e.g., *New Amsterdam*) to see the shift into challenges with elegant new medicine and culture practicality.

Psychiatry and Mental Health

“Clients are changing, and yet not in the ways they desire...virtually all problems brought to psychotherapy can be understood as vicious cycles that are initiated and driven by well-intentioned, failed solutions. In a strange way, the attempt to restore a desired state of stability escalates the problem pattern. We find ourselves in a contradictory situation in which the problem-producing process unintentionally stabilizes the undesirable condition. In other words, the more we attempt to resolve the problem, the more stable the problem becomes. Therefore, the effective interventions are targeted as changing solution patterns. The essence of problem resolution is a shift in the pattern of failed solutions....second order change is the catalyst for the problem resolution that runs through all effective psychotherapies”

Fraser & Solovey, 2007, p 40.

The efficacy of mental health care is less than it needs to be and is often just symptom-based. The “science of psychiatry” is especially plagued with failure. There are few to no biomarkers for any disease/disorder entity. Disorders share symptoms. Previously we discussed the 4Cs of understanding a problem. This elucidates the complexity. The 4Cs are *cause*, what the causative agent of the problem is and what needs to be done, then *consequences*, as the person responds idiosyncratically to an intervention, then *compensation*, as adaptation becomes its own problem, and last a *confound*. Now the cycle repeats.

In a most discouraging assessment in 2013 and beyond from an in-depth analysis of the US Burden of Disease Collaborators, investigators gathered data on 291 diseases and injuries between 1990 and 2010 (SSI Annual Statistical Report: 2013; cited by Higgins, 2017). Combining premature death and disability to calculate the burden of each disease, they found that the toll of mental disorders had grown in the past two decades, even as other serious conditions became more manageable. Explanations pointed to differing reasons. One explanation points to the vague nature of psychiatric diagnosis.

Social Security awards for disability have exploded in two areas, musculoskeletal and mental health disorders, both of which are often diagnosed on the basis of the patient’s subjective reports rather than hard measures, such as scans and blood tests. It is also possible that we are hampered by not having new treatments for patients seeking help. As it turns out, drugs developed in the past 20 years perform like older medications. Peter Kramer, in his book *Ordinarily Well* (2017), noted that the advent of today’s antidepressants has largely eliminated the immobilizing and melancholy that was once all too common. This is not near sufficient. Based on clinical experience, mental health treatment does improve symptoms and quality of life by about 20-40% in most patients. That is better than nothing, but leaves much to be desired. The chronic weaknesses of the human condition now need to be made more tolerable.

Many changes are incorporated into the latest addition of the DSM-5, but it too relies on the same general approach as earlier additions. Well-known problems with this approach are excessive comorbidity, the proliferation of hundreds of putative different pathological entities, and the lack of knowledge regarding the biology underlying the pathological syndromes. The science of psychopathology, however, has not yet advanced far enough to indicate an alternative approach. The National Institute of Mental Health was well aware of these problems when it launched research from a criteria project in 2008 to research relationships between dysfunctional behavior and neurobiological systems because that

and other research projects have not yet advanced scientifically. We need biomarkers. The DSM-5 is widely regarded as the best available classification in mental disorders despite a limited scientific foundation. As we have said, it is not scientific and is not statistical: It is a flawed taxonomy. It has poor validity.

We are, however, making progress. Despite the truly extraordinary complexity of human behavior, research has advanced dramatically in recent years and a new scientific understanding of human development has emerged that is fundamentally different from what had previously existed. The establishment of neuro and behavioral sciences as paradigmatic science disciplines has seen the intension and use of more powerful and precise scientific tools that have allowed the experimental testing of hypotheses that had to remain as speculations in the past. These include new statistical procedures, as well as technical procedures such as genetic sequencing, for example. These involve the usage of scans, MRIs and PET. A very large body of replicated and well-controlled research has examined a wide range of psychological processes from micro to macro levels; from epigenetics and neural plasticity to infant attachment, to the nature of consciousness and moral reasoning, to the impacts of social context and culture. But, the clinical rubber for real world patients has not met the road where psychiatry is in play.

Let's look at some numbers. Mental health and substance abuse disorders are prevalent, affecting about 20% of the US population. Abuse disorders are highly disabling, representing about 7.4% of disability adjusted life-years worldwide and ranking first in years lost to disability. AS many as one in four adults now, approximately 61.5 million people, experience mental illness in a given year. Fifteen million people in the US live with major depression, 42 million suffer anxiety disorders, and sadly many never seek treatment, which is one reason why suicide is the 10th leading cause of death in America according to the National Alliance on Mental Illness.

Psychotropic medication resistance has doubled every 5 years since the early 1980s and there has been a 3,000% increase in the reference literature that addresses treatment resistant psychiatric circumstances, particularly antidepressants during this timeframe. Affected people are increasingly being viewed as a complex series of neurobiological pathways that need to be untangled, activated, inhibited, or massaged in some way.

Given these resistance numbers, it is hard to call the evidence we have as "real evidence" of people being helped. Objectifying patients as mostly a complication of the signs, symptoms, and complaints is one way.

Psychiatry is overwhelmed and meds are the easy response. They are often too costly or falter in efficacy. Health is, however, about helping patients achieve self-mastery, organize their lives, and take charge of themselves. It is not merely reaching into the medications grab-bag to find yet another drug to tame a pesky or emerging symptom. Health is about promoting self-reliance and encouraging patients to explore what they need to do to get the engine of change moving steadily forward instead of subjecting them to the medication merry-go-round. Regardless of severity, every depressed individual with the intension of getting better needs to act for him or herself. Circumstances, of course, will dictate progress. Interestingly, in a 1974 book, *The Depressive Spectrum*, Dean Schuyler writes “most depressive episodes will run their course and terminate with virtually complete recovery without specific intervention.” We need to find a better balance between disorders and real health.

Anne Harrington in her book *Mind Fixers: Psychiatry's Troubled Search for the Biology of Mental Illness* (2019) laments about the field of psychiatry. It is in freefall, failing to find a brain-based causal connection to disorders, having a massive reduction of drug companies abandoning the field, having a new DSM with little validation, and with key thought leaders abandoning its core mission in favor of newer and better models. A medication-only commitment and a bankrupt taxonomy have had their toll on the lives of older people. Psychiatry further is a field where rhetoric far outstrips substance and the path forward is suspect.

Harrington anguishes whether psychiatry as a discipline can give up its specious zealotry on the mental health market and share meaningfully with other disciplines. There is a real gap between the seriously mentally ill (real mental illness) and the vast majority of the worried well. Those who are not mentally ill in any meaningful medical sense need better care. Older adults are clearly better served with an apartment or a social sense of purpose than a medicament. Do older groups really require a diagnosis, a medication, and a mental health label for their situation? Harrington again places a clarion call for psychiatry to see the bigger picture, to not be proprietary where mental health is concerned.

Fortunately today's groups (e.g. AARP) see the needs in a wider scope and have visions that are more health-based and set in positivity. The ethics of care in the twenty-first century mandates transparency and reasoned truth, not jaded manuals and get-through-the-night medications. Already the salutary ethical effect has led to big pharma being more transparent. For the most part we are not correcting biochemical deficiencies caused by neurotransmitter anomalies. Mental illness is clearly present and takes many forms, but only some is caused by disease.

Even given the possible disease “truth,” the way through the door is the biopsychosocial gate. Adolf Meyer long ago promulgated mental hygiene, the notion that mental problems have their roots in poor habits, not bad brains. How to get people to react to the demands of life is key: Mental disorders are problems of poor maladjustment mixed with the demands of living and the aging process.

Suggestions

Technology: Experts have estimated that 10% of premature mortality in the US may be due to the suboptimal quality of the health services. A much larger share, perhaps as much as 40%, is due to behavioral determinants of health. Healthcare providers traditionally regarded health behaviors as being out of their sphere of influence in medical training. However, recent improvements in technology, advances in behavioral science, and shifts in health financing create exciting new possibilities to change this. A new ecosystem of wearable and wireless technologies, patient engagement strategies, and provider feedback could allow us to manage chronic disease far more efficiently than our current approach of using episodic clinic visits.

There are problems, but feedback loops can be created that are effective in keeping patients and providers alike engaged. For example, alerting a physician’s office when a patient has markedly abnormal blood sugar or blood pressure really makes a difference. The diabetes community has gotten rather good at this. Telemedicine has also become doable, profitable, and effective. The key to designing better healthcare systems is to recognize that what patients want is to be healthy, not consume health services. Providers will improve the chances of improving the health of the United States through technology that centers care provision around the convenience of patients rather than providers, simplified and improved choice environments, and incentives designed to keep people healthy rather than care for them only when they are sick.

Primary Care Change: A substantial investment in primary care services can have a positive health return for practices. This may be quixotic but easily doable. These services can allow for the building of relationships with patients that include transportation between home and primary care visits, substantially longer primary care visits averaging over 30 minutes, and in-house care management that helps patients coordinate their care across multiple providers. The economic model fuels the care model. Patients can be triaged, for example, into 1 of 4 tiers based upon inputs of age, comorbidities, recent utilization patterns, and degree of

social support. The patient's tier helps to determine a variety of parameters in his/her care, including primary care visit cadence and allocation of care management resources. For example, the sickest 5% of patients are identified as critical and a clinic should work to see these people in their clinic once every three weeks. Conversely the healthiest 30%, classified as good, are scheduled far less frequently. A patient's tier is consistently re-evaluated. As part of this iterative triage, patients undergo regular structured geriatric assessments that include evidence-based screenings for depression, fall risk, and adverse drug interactions. Lifestyle is also critical. These assessments feed into the population health function that captures the need for indicated preventative testing, such as colorectal and cancer screening. Again, telemedicine can be an effective friend here.

Payment: A comprehensive model and reimbursement system would help clinicians to take a broader and more balanced approach to the provision of care for older persons and could drive a long-needed re-engineering of care systems. Less emphasis could be placed on fragmented and episodic organ system care and more focus could be placed on continuous team care for physical, cognitive, and psychosocial function. Clinical guidelines could be revised to integrate multi-morbidity, functional status, and poly-pharmacy as crucial components in making management decisions. Less emphasis, then, would be placed on expensive procedures and more time would be spent on assessing and advising patients and their families. Care teams could be redesigned to provide the type of medical, physical, and counseling services a comprehensive model would require. Sites of care would evolve to a broader model of care delivering more efficiently through a combination of the clinical site and electronic or home-based care. Clearly the big medical companies (e.g., Humana) are becoming more health oriented and payment savvy.

Something needs to be done. American spending on health care is expected to be just shy of 20% in 2027. By 2035, U.S. health-care providers believe they will face a collective shortage of about 500,000 home health aides, 100,000 nursing assistants, and 29,000 nurse practitioners. There is also a projected shortfall for doctors at 122,000 by 2032. Recall too that for every 10% growth in the proportion of people over 60, economic growth falls 5.5%. The "dependency ratio" (percentage of non-workers who rely on the employed) will soon exceed 30%, having a profound effect on Social Security and Medicare (The Week, p. 11, August 23, 2019).

Key Problems of Cost of Health Care in US

1. High administrative costs (~8%)
2. High Drug costs (~\$1500/person per year)
3. Defensive Medicine (malpractice litigation)
4. Cost of specialists (excessive pay rates relative to PCPs)
5. Specialist scans (e.g., MRI costs)
6. Branding (patients influence by brands)
7. No insurance (no preventative care and ER care)

Kenneth Zapp (August 29, 2019), "Why Health care is so expensive in America," B7, savamahnow.com

Unmet needs: This is a special concern for older adults. They are often an after-thought or a non-entity in care. Community living older adults have more than 17 million admissions to the emergency department (ED) annually. Older adults at greater risk for frequent ED admissions have multiple comorbidities and functional disabilities and characteristics that are also associated with unmet ADL needs. A study of older adults admitted to the ED concluded that nearly 40% of ED visits are potentially preventable, including visits for injuries or skin breakdown. These reasons for ED utilization are similar to older adult, self-reported consequences of unmet ADL needs. They are not managed and problems occur.

A framework provided by Anderson (Anderson and Newman, 2005) guides the methodology for determining whether unmet ADL needs increase the risk for ED admissions in the context of characteristics that predispose, enable, or create needs for medical care. Age is a predisposing characteristic that increases the risk of ED utilization. Chronic diseases, such as heart disease and diabetes, create a need for emergency care. Unmet needs for ADL assistance are considered an enabling characteristic that increases the risk for future healthcare utilization. Often, delirium is the result (or cause). It compounds both a hospital discharge and readmission, as well as community confusion, regarding effective interventions. In summation, a primary hypothesis is that older adults who report unmet ADL needs have more future ED admissions than older adults who report their ADL needs as being met (Haas et al., 2017). Unmet needs lead to "met" problems.

Class and Culture matter: We noted this above. Researchers see classes that continue to run at a fixed distinction between upper, middle and lower class status. This distinction creates different life perspectives: the higher in social economic status you are, the more independent-oriented you are likely to be, while the lower in status you are, the more group-minded you

are likely to be. Social class, which can be defined as the social context that an individual inhabits in enduring and pervasive ways over time, is a fundamental end to which we can see ourselves in others. Because lower ranking people have fewer resources and opportunities than those of relatively high rank, they tend to believe that external uncontrollable social forces and others' power have correspondingly greater influence over their lives. Success for them, therefore, depends on how well they can be lead and how they can rely on others. By contrast, those who enjoy resources and greater social class live in a context that has enhanced their personal power and freedom, and that has bestowed upon them larger and safer living spaces, the means by which to buy high-priced goods and experiences, and an education that provides access to influential people, ideas, and venues. These conditions give rise to a more self-focused approach to life. We need to access the variables for understanding the community. Culture is persuasive and always relevant, even as applies to the brain.

“A true cultural neuropsychology must go beyond simply assessing cross-cultural or ethnic groups to understand an individual’s brain as completely culturally constituted. This integrated approach thereby suggests that greater fidelity in understanding the functional human brain requires an uncompromising integration of cultural practices, cognition, and behavior. Cultural neuropsychology, therefore, challenges us to begin all assessment and inquiry into brain-behavior relationships with diversity as a point of departure, with a direct examination of a person’s cultural practices, which provide the scaffolding for the development and continued execution of cognition in people’s everyday lives.”

Cagigas & Manley, 2014 (p.147), Health and Life Span

Lifestyle: Yes, this again, as it provides more bang for the buck than any other factor. Dhalwani et al. (2017) studied the association between lifestyle factors and the incidence of multi-morbidity in an older English population. There was clearly evidence of a temporal association between the combination of different unhealthy lifestyle factors and multi-morbidity. Population level interventions, therefore, were encouraged to include the reinforcing of positive lifestyle changes in a population to reduce the risk of developing multiple comorbidities. The multiple comorbidities included such things as diabetes, hypertension, stroke, myocardial infarction, congestive heart failure, angina, lung disease, chronic obstructive pulmonary disease, asthma, arthritis, osteoporosis, cancer, hearing problems, Parkinson’s and AD, other dementias, macular

degeneration, and glaucoma. This group found a dose-response association between unhealthy lifestyle factors and multi-morbidity. Physical inactivity increased the risk of multi-morbidity by 32% on its own and inadequate fruit and vegetable intake increased the risk by 65% in women. When physical inactivity was combined with obesity or smoking, the risk increased by 2 or 3 times and more than 4 times when combined with both smoking and obesity.

Exercise rules! It is difficult to tout the value of exercise without it seeming like a “cure all.” Physical activity reduces the risk of myriad diseases, including dementia and the cardiovascular diseases as well as diabetes. In *The Physical Activity Guidelines for Americans, 2nd Edition* (PAG, 2018) it is estimated that in the US nearly 117 billion dollars in annual health care costs and 10% of premature mortality are associated with a failure to meet the recommended levels of physical activity. This recommendation involves 150 minutes of physical activity and 2 days/week with muscle activity. In fact, these activities can be made simple with apps. In sum, there is no better suggestion than this one for health and better quality of life.

Step approach: Watch and Wait depends on a slow walk through the case. There is still limited knowledge regarding the cause and cure of many medical conditions. As a result, ameliorating symptoms caused by such conditions often becomes the treatment goal, and even for diseases that are better understood existing knowledge may be insufficient to indicate specific treatments. Plans, time, teams and evidence are in play. Take for example heart disease. Several medications are available to treat heart disease: anticoagulants, beta-blockers, and diuretics, as well as a variety of surgeries, angioplasty, bypasses, stents, and implantable devices, pacemakers and defibrillators. Behavioral changes are often prescribed as well. Predicting a patient’s response to treatment can be very imprecise. Consequently a stepped approach is often used, where less intensive, invasive, and risky treatments are tried first. Physicians should not base their evaluation and treatment recommendations on the dictates of a personally selected theoretical orientation, but instead on the best available evidence, their prior experience, and patient values. Watch and Wait again...

Comprehensive Health: A new respect for mental health and function matters especially. A comprehensive model better characterizes the health status of a sample of community-dwelling older persons. This involves a shift toward a focus on the functional status of older persons and clinical care. Such an approach would focus on the patient’s functional outcomes in terms of physical, cognitive, and social status more than organ system

or chronic disease results. Rather than assessing quality of care exclusively on the basis of disease-oriented process measures (for example, proportion of patients taking types of medication or surrogate measures, lipoprotein and hemoglobin A1c levels), a new taxonomy could assess outcomes according to more patient-centered variables such as a function-first approach. Watch and wait makes this a priority.

The environment will need tweaking. Cities will have to rethink their rules; crosswalks, transportation for the isolated, car-oriented suburbs, and general focus on foods, clothing, etc. It seems clear to say that we are moving into a new era beyond LTC facilities and ALFs.

Will and Community Perspective: We know now that technology has become the helpmate to clinical care. To function, individuals rely not only on knowledge stored within our skulls, but also on knowledge stored elsewhere: in our bodies, in the environment, and especially in other people. When you put it all together, human thought is incredibly impressive, but it is a product of a community, not of any individual alone (Slomann-Fernback 2016). Technology is a logical and meaningful extension. The emphasis on the media world and community has its downsides but many more strengths regarding health.

But, we are bigger than technology. We are also bigger than our parts. The human mind is not like a desktop computer designed to hold reams of information. The mind is a flexible problem solver that has evolved to extract only the most useful information to guide decisions in new situations. As a consequence, individuals store very little detailed information about the world in their heads. In that sense, people are like bees in the society of a beehive: our intelligence resides not in individual brains, but in the collective mind. Finding the right mix of self and community (and technology) is the challenge for the future.

Related, as part of comprehensive healthcare, one of the greatest challenges in improving healthcare quality is the slow speed with which scientific innovation reaches practice. Only 14% of scientific discoveries reach practice at all, and when a practice is adopted the process takes an average of 17 years (Westfall et al., 2007). Despite the strong emphasis on translation at the National Institute of Health and other funding agencies, a leaky pipeline still exists in the trajectory from scientific discovery to wide-spread implementation of evidence-based healthcare practices. This can be corrected with incentive, commitment, and political will.

Value-Based Costs: If health status continues to be measured on the basis of medical model taxonomy, payment for value will have a constraining effect on the health of older adults. Fortunately, the U.S. healthcare finance system is slowly evolving to a value-based reimbursement

system, one in which payment is based on health status and functional outcomes. The Medical Access and CHIP Reauthorization Act of 2015 is focused on value and not volume. For these new payment systems to be maximally effective, a more comprehensive taxonomy is needed to describe and quantitate health.

Ethics

Osler recognized that it is more important to know the person that has the disease than to know what sort of disease the person has. Most problems, then, are not simple algorithms but represent an ethical challenge. Ethics requires contextualized thinking and competent knowledge. In fact, it can be argued that the practice of medicine or even the dissemination of health care data is at base an ethical enterprise. The development of an informed decision requires cognitive skills, emotional preparation, and a reflective capacity.

The “ethics of care” fosters an emotional commitment and willingness to join patients/citizens in a loose empathic association rather than a declaration for individual rights. In fact, agreed upon rubrics include the management of care largely in PCC, assessment and monitoring, and a placement of vision that allows for holistic and step care. This is a true “ethics of care.” Health problems are emergent properties of the person, the living situation, agreed upon targets, and a host of medical/psychological variables from amount of comorbidities to compliance. Perhaps we should argue for an increased role for ethics in this process. In the formulation of a treatment plan, ethical principles apply.

We have discussed this before and briefly redress it here. Four principles guide the management of older adults in health care. They are the usual suspects: autonomy, non-maleficence, justice, and beneficence. These principles can be applied to any medical condition and any patient. They will not change regardless of the issue. The moral mandate for care is then central and requires some deliberation, some validation/listening, and some suspension of hard medical tenets. That said, these may not always serve the core issues of older adults.

In chapter 4 we noted another model: Speilman (1986) stressed the principles of sociality (recognizing the need for relationships), temporality (recognizing limitations), and embodiment (respecting the change of aging) as being more important for elder ethics than autonomy. Issues regarding religion, consent, late life care, and advanced directive, as well as a competency in various areas, will always involve the ethical principles

of beneficence and non-maleficence, law and equity, and paternalism and autonomy.

●lder adults require complex deliberative thinking. Ethics provides a reasonable template for discussions. Dialogue is a two way street. Peter Salovey, the president of Yale University, compellingly makes the point that people are both influenced by and sensitive to how information is framed (2011). We influence people, even subtly. Responsibility and accountability are always micro (individual, health care professional), meso (inter-professional, teams, education and professional organizations), and macro (health care system, education systems, and policies). So, while we cannot be responsible for the data received by any person, we need to “appreciate and own the responsibility” of having a connection and loose ethical obligation for care with regard to the complex issues.

Socrates was the first to show that ethics is born of human conflict. An ethical dilemma is one in which one is breaking some ethical norm or contradicting some ethical value in making a decision. Whichever way one chooses in a given situation is an ethical challenge. Ethics tries to clarify right and wrong or good and bad. At base, however, it attempts to clarify the meanings of and justifications for decisions, and ultimately how we should act. While there are many variants of ethics (deontologists instruct one to do their duty; consequentialists require one to consider the outcome; virtue ethics advocates opine for being a person of excellence regardless of circumstances; feminist ethics seek relationship justice, etc.), one thing is certain: The role of clarification is subject to care, reason, error, and humanity.

In most cases regarding older adults the health care provider is considering the moral issue from the perspective of individuals who are less privileged. The health care provider should avoid snap judgements and provide a voice of care for others. This always involves having sufficient background information: knowing more about the identified person at issue makes harsh judgements less likely. Considering the standpoints of the marginalized is particularly noteworthy: are some perspectives silenced because of lack of power or prestige.

Watch and Wait attempts to place this philosophy front and center. Medicine and ethics cohabitate. Behavioral principles matter. Making haste slowly and validating strongly matter. ●lder adults are presenting new challenges. There is no exit: We are in the throes of the gerontology revolution. We owe a change of perspective and care to older adults now.

It was Wilber Penfield who pioneered surgery on the brains of over a thousand patients, who noted that man is a composite of material and immaterial mental abilities (see Rooney, 2019). Neuroscience provides

strong evidence for mind-brain dualism, and science confirms this. For Penfield, it dovetails nicely with the compelling philosophical demonstrations of the reality of free will. For him, there is no credible evidence in neuroscience that would lead an objective scientist or reader to conclude that free will is not real. This makes us who we are. Importantly, he noted that he practiced his trade because it will improve and be different even in his lifetime. Free will and science will triumph. We should all take notice.

Conclusion

Aging has become the next frontier in medicine. At present we are experiencing two important global trends. First, the pace with which technology is advancing is more rapid than at any other point in human history. Second, the number and proportion of older adults in the population is/will be larger than at any other time in human history. How will these two trends intersect in the near future? Chronological age, the passage of time one spends on this planet, simply cannot be reversed. But, biological age (one's health relative to the age of one's person) can be elongated or in some cases turned back! Healthy life habits are the method for aging well. This is fast becoming the number one intervention in primary care.

In this book we are not talking about human enhancement technologies, though they do open up tremendous new possibilities (and raise questions). We do not just embellish performance. We optimize a theory of self-determination which divides well-being into three parts: autonomy (the ability to make one's decisions); competence (the capacity to act and contribute to society); and social relations (the network of relationships we can count on). These are the meta-constructs that set the stage for change in real life. The biopsychosocial model fosters them at all junctions. We are on our way.

It is a different world. Older is not necessarily older. The numbers of healthy older adults are increasing, but problems (like substance abuse, number of morbidities) are also increasing. In 2035 there will be 78 million Americans of 65 or older. There will be 21.1 million minority older adults in 2030. There has been a 70% increase in older immigrants in the past 20 years. There are an estimated 2-4 million LGBT people due to be over 65 in the next two decades (Novotney, 2018). In sum, the majority of research on older adults may very well now need to be redone because this and the next generation especially are quite different.

Now, aging-friendly health systems with respect for the 4Ms (mobility, medications, mentation, and what matters) may hold the day. Clinicians who care for older adults have long been frustrated by the limitations of the prevalent disease-oriented approach; this involves the medical model as a taxonomy for defining health, disease, and payment for care. The medical model tends to focus on individual organ systems, but is inadequate for the characterization of persons with a combination of multiple diseases, limitations of function, and cognitive and psychosocial problems. Conditions such as cognitive impairment, delirium, falls, dizziness, syncope, urinary incontinence, depression, and frailty are often multifactorial in etiology. They may involve more than one organ system and the medical model is insufficient in characterizing the complexity of these disorders. Specialists are really not the answer, at least as they currently practice, as integration is poor and cost is prohibitive. The medical model then is inconsistent with the emerging concept of patient-centered outcomes, particularly in older adults.

We address several topics in each domain. We do this because the construct (function, pain, and sleep as examples) are related uniquely to the various domains. It needs to be noted (restated) that each person is unique. So, we each have a profile and scores on tests, but the phenomenology of the problem for a particular person is their issue and is totally distinct – each therapy needs to be unfolded differently.

That said, we are really on our way. Progress is being made. At the patient level, patients have entered a new age of autonomy with less dependence on physicians and more information available to them. The FDA approved 23andMe in 2017. Physicians are now consultants. At the societal level the health care system is undergoing vast changes that continue to unfold. As of November 2018 we have progress to cite. The schema below clearly portrays the progress and the areas of focus. We have gone from no caregiver interest to significant caregiver interest, and from few clinical trials previously to early and late stage clinical drug trials now. Clinical non-pharma studies are also very much in the mix. It is also now becoming evident that AI (Artificial Intelligence) can predict diseases (like AD) years before the actual diagnosis. Technology and science is nuancing our understanding of aging and disease.

Ongoing NIA AD/ADRD and Related Intervention and Prevention Trials (140+)



And so it goes...

References

- Berwick, D. M. (2016). Era 3 for Medicine and Health Care. *JAMA*, 315(13), 1329–1330.
- Cagigas, X. E., & Manly, J. J. (2014). Cultural neuropsychology: The new norm. In M. W. Parsons, T. A. Hammeke, & P. J. Snyder (Eds.), *Clinical neuropsychology: A pocket handbook for assessment* (pp. 132–156). Washington, DC, US: American Psychological Association.
- Clark, C. R., Ommertorn, M. J., Coull, B. A., Do Quyen Pham, & Haas, J. S. (n.d.). Income Inequities and Medicaid Expansion are Related to Racial and Ethnic Disparities in Delayed or Forgone Care Due to Cost. *MEDICAL CARE*, 54(6), 555–561.
- Dhalwani, N. N., Zaccardi, F., Donovan, G., Carter, P., Hamer, M., Yates, T., ... Khunti, K. (2017). Association between lifestyle factors and the incidence of multimorbidity in an older english population. *Journals of Gerontology - Series A Biological Sciences and Medical Sciences*, 72(4), 528–534. <http://doi.org/10.1093/gerona/glw146>
- Dieleman, J. L., Baral, R., Birger, M., Bui, A. L., Bulchis, A., Chapin, A., ... Murray, C. J. L. (2016). US spending on personal health care and public health, 1996–2013. *JAMA - Journal of the American Medical Association*, 316(24), 2627–2646. <http://doi.org/10.1001/jama.2016.16885>
- Fraser, J. S., Solovey, A. D., & American Psychological Association. (2007). *Second-order change in psychotherapy: The golden thread that unifies effective treatments*. Washington, DC: American Psychological Association.
- Freedman, V. A., Martin, L. G., & Schoeni, R. F. (2002). Recent Trends in Disability and Functioning Among Older Adults in the United States: A Systematic Review. *JAMA: Journal of the American Medical Association*, 288(24), 3137.
- Harrington, A. (2019) *Mind Fixers: Psychiatry's Troubled Search for the Biology of Mental Illness*. New York, W.W. Norton and Company.
- Hass, Z., DePalma, G., Craig, B. A., Xu, H., Sands, L. P., & Pruchno, R. (2017). Unmet need for help with activities of daily living disabilities and emergency department admissions among older medicare recipients. *Gerontologist*, 57(2), 206–210. <http://doi.org/10.1093/geront/gnv142>

- Hero, J. ●., Zaslavsky, A. M., & Blendon, R. J. (2017). The United States Leads ●ther Nations In Differences By Income In Perceptions ●f Health And Health Care. *Health Affairs*, 36(6), 1032–1040.
- Higgins, E. S. (2017). Is Mental Health Declining? *Scientific American Mind*, 28(1), 20–21.
- Hoelt, T. J., Hinton, L., Liu, J., & Unuetzer, J. (2015). Directions for Effectiveness Research to Improve Health Services for Late-Life Depression in the United States. *American Journal of Geriatric Psychiatry*, 24(1), 18–30.
- Hung, W. W., Ross, J. S., Boockvar, K. S., & Siu, A. L. (2011). Recent trends in chronic disease, impairment and disability among older adults in the United States. *BMC GERIATRICS*, 11.
- Klimek, P., Aichberger, S., & Thurner, S. (2016). Disentangling genetic and environmental risk factors for individual diseases from multiplex comorbidity networks. *Scientific Reports*, 6 (●ctober), 1–10. <http://doi.org/10.1038/srep39658>
- Kramer, P. D. (2017). *●rdinarily Well: The case for antidepressants*. Farrar, Straus, & Giroux.
- Lavizzo-Mourey, R. (2012). Why Health, Poverty, and Community Development are Inseparable. *●pen Forum: Voices and ●pinions from Leaders in Policy, the Field, and Academia*, 215–225.
- McClintock, M. K., Dale, W., Laumann, E. ●., & Waite, L. (2016). Empirical redefinition of comprehensive health and well-being in the older adults of the United States. *PNAS Proceedings of the National Academy of Sciences of the United States of America*, 113(22), E3071–E3080. <https://doi-org.medlib-proxy.mercer.edu/10.1073/pnas.1514968113>
- National Alliance on Mental Health. (2015). *Mental Health Facts in America*. Retrieved from www.nimh.nih.gov
- National Institute of Mental Health. (2015). Strategic Plan for Research. Retrieved from <http://www.nimh.nih.gov/about/strategic-planning-reports/index.shtml>
- Novotney, A. (2018). Working with older adults. *Monitor on Psychology*. December, 60-66.
- lshansky, S. J. (2018). From Lifespan to Healthspan. *JAMA: Journal of the American Medical Association*, 320(13), 1323–1324. <https://doi-org.medlib-proxy.mercer.edu/10.1001/jama.2018.12621>
- Rooney, A (2019). *How the World Works, Neuroscience: Unlocking the Mysteries of the Brain and Consciousness*. China, Arcturus Holdings Limited.

- Salovey, P. (2011). Framing health messages. In M. A. Gernsbacher, R. W. Pew, L. M. Hough, & J. R. Pomerantz (Eds.), *Psychology and the real world: Essays illustrating fundamental contributions to society*. (pp. 214–223). New York, NY: Worth Publishers.
- Schuyler, D. (1974). *The depressive spectrum*. New York: Aronson.
- Slooman, S. A., & Fernbach, P. (2017). *The knowledge illusion: Why we never think alone*.
- Spielman, A. J. (1986). Assessment of insomnia. *Clinical Psychology Review*, 6(1), 11–25. [https://doi-org.medlib-proxy.mercer.edu/10.1016/0272-7358\(86\)90015-2](https://doi-org.medlib-proxy.mercer.edu/10.1016/0272-7358(86)90015-2)
- The Medicare Access & Chip Reauthorization Act of 2015 Path to Value*. (2015). <https://www.cms.gov/medicare/quality-initiatives-patient-assessment-instruments/value-based-programs/macra-mips-and-apms/macra-lan-ppt.pdf>
- The graying of America (2019), *The Week*, August 23.
- US Department of Health and Human Services, *Physical Activity Guidelines for Americans*, 2nd ed. Washington, D.C.: US Department of Health and Human Services, 2018. .2
- U.S. Social Security Administration Office of Retirement and Disability Policy. (2013). SSI Annual Statistical Report: 2013, (13). Retrieved from www.ssa.gov/policy/docs/statcomps/ssi_asr/2009/sect04.html#table17
- Vos, T., Allen, C., Arora, M., Barber, R. M., Brown, A., Carter, A., ... Zuhlke, L. J. (2016). Global, regional, and national incidence, prevalence, and years lived with disability for 310 diseases and injuries, 1990–2015: a systematic analysis for the Global Burden of Disease Study 2015. *The Lancet*, 388(10053), 1545–1602. [http://doi.org/10.1016/S0140-6736\(16\)31678-6](http://doi.org/10.1016/S0140-6736(16)31678-6)
- Westfall, J. M., Mold, J., & Fagnan, L. (n.d.). Practice-based research - “Blue Highways” on the NIH roadmap. *JAMA-Journal of the American Medical Association*, 297(4), 403–406.

APPENDIX A

MBMD Indicators and Variables

Psychiatric Indications	AA Anxiety/Tension BB Depression CC Cognitive Dysfunction DD Emotional Liability EE Guardedness
Coping Styles (Personality)	1 Introversive 2A Inhibited 2B Dejected 3 Cooperative 4 Sociable 5 Confident 6A Nonconforming 6B Forceful 7 Respectful 8A Oppositional 8B Denigrated
Stress Moderators	A Illness Apprehension B Functional Deficits C Pain Sensitivity D Social Isolation E Future Pessimism F Spiritual Absence
Treatment Prognostics	G Interventional Fragility H Medication Abuse I Information Discomfort J Utilization Excess K Problematic Compliance

APPENDIX B

Step 1:

Discernment Issues

Patient

Thank you for coming, I would like to get to know you.

What is your reason for being here?

Will you commit to questions and a period of assessment?

Will you allow your caregiver to make input and have access to your information?

Will you commit to some home tasks and challenges?

Your coming here means something important in this new stage of your life.

Caregiver

Will you assist in the assessment process?

Will you allow for some openness in this process, even trying tasks not initially seen as helpful?

Are you open to caregiving help – support, psychoeducation, strategies?

Are you agreeable to the slower Watch and Wait Process?

Demoralization

- Patients seek therapy when they become demoralized from failed change methods. Their efforts at coping is failed.
- Demoralization is a cause, consequence or both of the presenting symptoms that patients bring to therapy.
- Loss of consensual validation: People need to sense validation.
- Positioning is key: Pro-change, Neutral (ambivalent), Don't-Change, Restraining, Normalizing and acceptance, Reframing, Prescribing.

Hope:

- Agency thinking (I can do this) and Pathway thinking (This is what I do).

Step 2:

Necessary Validation Process of Watch and Wait

Validate Problem	X
Psychoeducation of Model	X
Assessment	X
Alliance	X
Monitoring	X
Case formulation	X

Step 3:

Assessment is provided for each domain.

Step 4:

Mini-Module Targets of Behavior over time

- Activity scheduling
- Mastery-Pleasure activities
- Graded task assignments
- Verbal rehearsal of tasks
- Managing situational contingencies
- Role playing and modeling
- Environmental control
- Contingency plans (If-then)
- Apply progressive relaxation
- Concreteness training
- Imagery exercise for counter-experience
- Compassion training
- Communication and Assertiveness training
- Behavioral experiments: Plan experiment
- Emotional exposure
- Homework
- Worry control
- Quick cognition (autonomic negative thoughts (ANTs))
- Relaxation (breathing, slow deliberate deep breaths at a selected rhythm)
- Simple hypnosis (breathe, focus on a target and close eyes)
- Easy nutraceuticals (e.g., omega 3's)
- Acceptance and willingness (simple targets)
- Flood senses with positivity (each sense with positive experiences)

Step 5:

Empirically supported Treatment Methods

- Motivational Interviewing: What do you want for you?
- ACT: Acceptance and Commitment
- CBT: Focus on simple cognitive interference
- Behavioral activation
- Mindfulness: Somatic quieting
- Eastern Philosophy: Being with discomfort, excessive "selfing," mind creates suffering.

Step 6: Follow-up: Focus on relapse prevention

Additional Psychotherapy Helpers

Communicate control and care: “We.”

Use comments in treatment (“You have noticed that you can...”)

Reward liberally (“You have been a star in life, now...”)

Reveal: “I respect your struggle. Just yesterday, you...”

Communicate hope: “Yes, your scores are a concern, but you can hold the line or even change this with...”

Get patient stories: “What made you go back and get your GED?”

Use past judiciously: “Yes, you did this, that was the ‘you’ then; now...”

Seduce with the better ideas: Socialization, positivity, altruism, commitment, love of family.

Encourage to envision a self in three months.

Law of little things: Tilt toward the positive.

Social chitchat: “It is really nice to see you”

Reflection in moment: “How are you doing now?”

Express immediate feelings: “I am feeling good about your skills.”

Draw parallel with therapy: “I wonder if you find it easier to be here.”

Make the covert overt: “You are rather quiet today.”

Get on board: “I see you want to take an antidepressant. Great but...”

Make a deal: “Let’s make a deal here. I will... You agree to... at least for one month”

Get early positive treatment outcome expectation/early gains.

Additional Helpers:

1. Use brain pathology as a metaphor: This allows for a shared vulnerability and understanding of what can be addressed from a more medical/physical perspective.
2. Link problems to achievable reality: “This is where we are going and what we can do.”
3. Make haste slowly: “We need a little time to position ourselves for the best chance for change. So, we will take 2-3 weeks and see about our options and make plans for best care. This is a very active period and you will be in the most active stage of treatment, the planning stages. Most treatment fails because this treatment part is missing and you are thrown into an intervention too quickly.”
4. Provide a plan: “Here are the steps for change.” This is based on the case-based model that has been formulated.