Using short-term memory measures to assess long-term memory in early-stage Alzheimer's disease

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Using Short-Term Memory Measures to Assess Long-Term Memory

In Early-Stage Alzheimer’s Disease

by

Scott O. Burkhart

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Abstract

Alzheimer’s disease (AD) impairs long-term memory of both verbal and visual information. While these impairments have been studied extensively at traditional short-term delays of 20-35 minutes, they have not been explored at long-term delays. In the current study, long-term delays of 1 day and 7 days were introduced to traditional short-term memory measures. 60 Participants were recruited for the study including; 20 individuals with Early AD, 20 individuals at risk for AD, and 20 age matched healthy controls. During an initial visit Participants were administered a self-evaluation questionnaire, the Mini Mental Status Exam, the Alzheimer’s disease Caregiver Questionnaire, 2 subtests from the Wide Range Assessment of Memory Learning, 2\textsuperscript{nd} Edition (WRAML2: Story Memory and Verbal Learning subtests), the Wechsler Adult Intelligence Scale, 4\textsuperscript{th} Edition (Information subtest), and Rey-Osterrieth Complex Figure Test (ROCFT). 1-day and 1-week later, Participants were administered the WRAML2 subtests and ROCFT as an assessment of long-term memory.
Early AD Participants performed significantly poorer at the 1-day interval than both the healthy control group and at-risk group, while performance at the 7-day interval showed less variation across groups. This pattern was evident across all measures administered, except the self-evaluation questionnaire. Results indicate adding a 1-day interval for assessment of long-term memory using a word list task (WRAML2 Verbal Learning subtest) to a clinical AD screening provides increased diagnostic sensitivity to detect impairment of Early AD individuals.
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Dedications

I would like to dedicate my journey through graduate school and development personally and professionally to my parents, Joyce and Steve Burkhart, and John Ortiz. They have continually encouraged, inspired, and believed in me while celebrating my successes. I could not have completed this journey without them.

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Chapter 1

Introduction

Alzheimer’s disease (AD) is one of the most prominent subtypes of dementia in the United States (Welsh-Bohmer & Warren, 2006). Research on AD began in the early 20th century when Alois Alzheimer reported that his patient, Auguste D., who had shown memory impairments, had neurofibrillary tangles and amyloid plaques in her brain upon autopsy. Since then, the pathology and neuropsychology of AD has been studied and much new information has been revealed (Welsh-Bohmer & Warren, 2006).

Memory and Aging

Memory is a basic cognitive function that tends to go unnoticed until it begins to fail. As individuals age, the ability to encode new memories of events or facts and working memory shows decline in both cross-sectional and longitudinal studies (Hedden & Gabrielli, 2004). Because of the cumulative effect, typically the older the individual, the more these changes are likely to negatively affect memory. In normal aging, short-term and remote memory are less negatively affected than recent memory retrieval which may be witnessed in the “forgetting” of the names of individuals recently met or retrieval of other novel information recently encountered. These and many other common memory phenomena are known because of an extensive research literature of memory.
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The first systematic study of human memory was performed in Germany by Ebbinghaus in the 1880s (Ebbinghaus, 1885). Ebbinghaus investigated a wide range of memory characteristics which would eventually be known as immediate memory, distributed practice, chunking, meaningfulness, intentional and spontaneous recall, memory decay, incremental learning and resultant savings, rehearsal, and interference (Miyake & Shah, 1999). Many of these concepts are still considered foundational and have clinical utility. Another important contribution are Ebbinghaus’ “learning” and “forgetting curves” that quantitatively display the acquisition and decay of new information over time. Surprisingly, little clinical application has been based on this acquisition and decay paradigm.

In the last 10 to 15 years, a general consensus view of memory has begun to emerge stressing the active, dynamic, and multi-systemic nature of memory, involving the interaction of such factors as long-term memory (LTM), short-term memory (STM), executive functions that control and regulate mental actions, and the knowledge and skills of the learner (Kintsch, 1999). STM is important to LTM since, in a sense, STM feeds the LTM system with new information by briefly storing the information before consolidation into LTM.

LTM research since Ebbinghaus’ initial investigations has primarily focused on and been limited to memory development and retention over extended periods of time (Howe, 2000), memory decay in association with projected rate of decline (Fajnsztejn-Pollack, 1973), and differences in LTM acquisition skills and retention (Howe & Hunter, 1986). The methods and research by which LTM has been evaluated have varied widely. Research on LTM has typically examined its change over time without using standardized measures of assessment or measures with known psychometric properties such as reliability (Howe, 2000). Storage in LTM is based
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on relatively permanent changes in brain cell structure, although there does not appear to be a single local storage site for stored memories (Lezak, 1995). When information is recalled after an hour or even a year, the information is located and retrieved from LTM (Lezak, 1995).

One consistent finding is that long-term memory is an especially vulnerable cognitive system (Howe, 2000). Many neurodegenerative diseases can cause long-term memory loss, and currently the most prevalent and intensely researched neurodegenerative disease, Alzheimer’s disease, is characterized by significant LTM loss, even in its early stages.

Alzheimer’s Disease (AD)

AD continues to become an increasing area of psychological research given the increased life expectancy growing population of individuals likely to be affected by the disease. AD is therefore a serious problem and early identification and treatment becomes the responsibility of medical and psychological professionals to diagnose accurately and develop effective treatment plans in the early stage of disease progression.

AD accounts for 50%-70% of all cases of adult dementia (Neugroschl & Sano, 2009). The incidence of AD rises exponentially with advancing age; for example, 10 % incidence is reported at age 65 with an increase to 50% reported at age 85 (Neugroschl & Sano, 2009). Men and women are equally at risk, however more women are affected since AD is a disease of the elderly and women have a longer average life span than men. These gender differences are congruent across all age groups and ethnic differences (Neugroschl & Sano, 2009).

Approximately 30% of individuals with AD have a biological family member with AD (Neugroschl & Sano, 2009). AD is a progressive disease whose clinical course varies, with some people having the disease for their last 2 to 3 years of life while others may live with AD for up
to 20 years. AD’s progressive decline is most typically characterized by memory loss, but language deterioration, visios-spatial impairment, poor judgment, novel problem solving, and an indifferent attitude are also common symptoms (First & Tasman, 2005).

In the early 1900s, Alois Alzheimer published a medical case report of a 55-year-old female patient suffering from what was described as a progressive dementia. Autopsy subsequently revealed an abnormal number of plaques and tangles not typical of traditional dementia (Albert & Moss, 2002). Alzheimer was later credited for discovering this unique form of dementia whose symptoms were not acknowledged in individuals younger than 65. However, in the 1970s, more refined diagnostic criteria were published reducing the age of suspected onset by at least a decade (Albert & Moss, 2002).

Currently the Diagnostic Statistical Manual, Fourth Edition (DSM-IV-TR) labels AD as Dementia of the Alzheimer’s Type (American Psychiatric Association [APA], 2005). An individual diagnosed with Dementia of the Alzheimer’s Type must demonstrate multiple cognitive deficits manifested by both memory impairment and at least one additional cognitive disturbance including aphasia, apraxia, agnosia, or executive functioning. Further, the DSM-IV-TR requires that the cognitive disturbances cause significant impairment in social or occupational functioning and represent a gradual onset and progressive cognitive decline (APA, 2005).

The AD deficits must cause significant impairment, be marked by gradual onset and progressive decline, and not be caused by another neurodegenerative disorder (APA, 2005). AD is classified with or without a behavioral disturbance and with early (before and up to age 65) or late-onset (after 65; (APA, 2005).
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Memory and AD

LTM is clinically significant because of its susceptibility to gradual developmental decline, especially within elderly populations (Morrison, Allardyce, & McKane, 2002). Generally LTM is defined as an individual’s ability to store new information and recall the stored information after few minutes or more. The time span when STM ends and LTM begins has not been clearly defined and is somewhat arbitrarily set, although generally STM is seldom characterized as lasting beyond 30 seconds (Morrison, Allardyce, & McKane, 2002).

LTM is of particular interest, considering the majority of measures used in assessing AD and other forms of cognitive decline focus primarily on short-term memory (Nygard, 2003). LTM struggles may exist for individuals with early-stage AD past the traditional 15-minute delay used in popular short-term memory measures. Of particular interest are the mechanisms underlying the neurocognitive processes in early onset stages of AD. Patients with AD exhibit cognitive impairment in the years preceding a clinical diagnosis (Forstl & Kurz, 1999). Memory impairments are particularly pronounced, but the relative degree to which other cognitive functions are impaired and the speed with which they decline during the pre-clinical years remain unclear (Arnaiz & Almkvist, 2003).

Fleishman and Gabriel performed a detailed study of 11 patients over three years proceeding the first year of non-normal diagnosis, or potential indication of early-stage AD. The researchers attempted to characterize and identify the neural and psychological bases of LTM failure in AD. Data were collected via convergent volumetric neuro-imaging and revealed that performance declines rapidly in all areas of cognitive functioning but abilities sub served by the medial and lateral temporal lobes (i.e., memory tract locations) appeared to be substantially more
impaired with greater loss of episodic, implicit, and semantic memories (Fleishman & Gabrieli, 1999). This loss of episodic, implicit, and semantic memories appears to be neurologically related to early-stage limbic-diencephalic pathology (Fleishman & Gabrieli, 1999). Non-mnemonic impairment was specifically related to later-stage temporal-neocortical pathology (Fleishman & Gabrieli, 1999).

Other research studies have examined pharmacological means of delaying deterioration in AD using donepezil hydrochloride (Aricept™) to treat early AD symptoms of memory loss (Coubrough, 2008). Cholinesterase inhibitors such as Aricept remain the primary pharmacological treatment for AD and are used to slow symptoms of AD by acting on the acetylcholine activity in the brain and reducing acetylcholine levels by as much as 90% in AD, inhibiting the neurotransmitter acetylcholinesterase. Common cholinesterase inhibitors prescribed for AD currently include medications with the trade names Aricept, Nameda, Exelon, and Razadyne. NMDA receptor antagonists may also be prescribed to block the excitotoxicity of the neurotransmitter glutamate at NMDA receptors, however these are less prescribed due to evidence of clinical efficacy (Gourley & Eoff, 2009).

Despite the considerable amounts of clinical and empirical attention give to AD, making an accurate diagnosis is still considered controversial. Significant effort has gone into clarifying biological and psychological indicators for AD, however none of these indicators have been widely accepted due to differences in origin and a precise primary symptomology (Jelicic, Bonebakker, & Bonke, 1995). AD is currently described as a clinical diagnosis arrived at by exclusion in order to account for the progressive change in cognitive functioning (APA, 2005). Nevertheless, there is consensus that AD consists of neurodegeneration of the brain disrupting
the ability to create and retrieve memories (APA, 2005). Further, early-stage AD is defined as the period of time in which an individual first experiences signs and symptoms of AD that interfere with daily functioning. The signs and symptoms include observable cognitive decline, becoming forgetful of recent events or details, impaired mathematical ability, and a diminished ability to carry out complex tasks (Jelicic et al., 1995).

A review of the neuropsychological literature on long-term memory deficits in AD suggests that individuals demonstrate a significant deficit in explicit memory, a less severe deficiency in implicit memory for both verbal and visuo-spatial information, and relatively preserved implicit memory for visuo-motor skills (Carlesimo & Oscar-Berman, 1992). The majority of recent long-term memory and AD research has focused on the decline of previously learned information and impairment in short-term and long-term implicit memory (Jelicic et al., 1995). The PsychInfo search engine reveals six studies that deal with AD and long-term memory, three of which using a similar context as the present study. These three previous studies highlighted results and findings relating AD and impairment in LTM (Backman, Jones, Berger, Laukka, & Small, 2005; Mickes, Wixted, Fennema-Notestine, & Galasko, 2007; Rogers et al., 2006). These three studies focused primarily on early detection of early long-term memory defects via behavioral observations and visual-spatial assessment.

In the first study, 1,207 preclinical AD cases were examined in order to determine the degree of impairment across various cognitive domains (Backman et al., 2005). Preclinical AD was defined in the study as the early signs of impairment in global cognitive ability. Participants for the study were obtained as volunteers from an aging resource center in Stockholm, Sweden. The results revealed larger preclinical effect size deficits in global cognitive ability, episodic
memory, perceptual speed, and executive functioning, and smaller effect size deficits in verbal ability, visual-spatial skill and attention. Individuals 75 years and younger with shorter follow-up intervals of three years or less revealed larger effect sizes for both global cognitive ability and episodic memory. Episodic memory differences were evidenced in pre-identified clinical individuals in terms of baseline cognitive impairment. Within episodic memory, both delayed testing and recall-based assessment also resulted in larger effect sizes. The authors concluded that deficits in multiple cognitive domains (including memory) were evident several years prior to AD diagnosis, and multiple brain structures and functions were affected long before the AD diagnosis is made (Backman et al., 2005). The deficits identified in multiple cognitive domains were more characteristic of LTM than STM based on episodic memory decline (Backman et al., 2005).

In the second study, the decline of semantic memory as a symptom in persons eventually diagnosed with AD was also researched (Rogers, Ivaniou, Patterson, & Hodges, 2006). Participants used in the study included 36 individuals who participated on a volunteer basis, with concerns regarding noticeable cognitive decline. The authors focused on semantic dementia as a point of emphasis to assess AD using category fluency and letter fluency. Semantic memory was defined as the conscious recollection of factual knowledge and general information about the world. Assessment measures used included the Booklet Category Test, the Information and Comprehension subtests from the WAIS-III, and a letter fluency measure designed for the study. Results revealed that early on individuals eventually diagnosed with AD were more impaired in category fluency than letter fluency. Results also revealed that individuals with AD performed worse on tasks associated with naming (information subtest) than with comprehension tasks.
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(comprehension subtest). Individuals eventually diagnosed with AD also revealed deficits in semantic and verbal comprehension suggesting a semantic decline (Rogers et al., 2006). The decline in semantic memory suggests impairment in LTM rather than STM in the individuals eventually diagnosed with AD (Rogers et al., 2006).

The third study that examined long-term memory in those with AD was a longitudinal investigation that focused on impairment in neuropsychological tasks using a preclinical AD group. The authors defined AD as a decline in cognitive functioning served by the medial and lateral temporal lobes (episodic and semantic memory, respectively). Patients were obtained from an Alzheimer’s disease research center. The authors examined the degree of impairment using an evaluation of 11 individuals until an AD diagnosis was made. Individual test scores over time suggested decline in semantic and episodic memory more than frontal lobe functioning (Mickes et al., 2007). The decline in both episodic and semantic memory indicates LTM impairment as the 11 individuals progressed towards an eventual AD diagnosis (Mickes et al., 2007).

Based on the three studies just described, a gap in the literature exists related to the assessment of LTM in individuals with early signs of AD. While previous research has focused on components of LTM including verbal, episodic, semantic, and visual memory, the studies have failed to examine potential decline at intervals longer than the traditional 15- to 30-minute delay. Based on Ebbinghaus’ research noted earlier, more pronounced declines might be especially obvious as the client approached a 24-hour delay.

Despite the fact that LTM is a cognitive process worthy of investigation, none of the current clinical measures evaluate memory decay beyond intervals of 15-30 minutes. Measuring
memory decay over a longer period (e.g., hours or days) may be crucial in assessing early cognitive decline in aging adults eventually diagnosed with AD. It is important to detect potential cognitive decline in explicit and implicit LTM; this may be more accurately done using intervals longer than the traditional 15-30 minutes. It may also be important to monitor the nature and pattern of LTM decline in early-stage AD. This study will compare traditional memory decay intervals with more extended intervals, specifically delays of one day and one week. Commonly used short-term measures will be adapted to measure LTM loss in early-stage Alzheimer’s patients.

A study in which current STM measures including the Wide Range Assessment of Memory and Learning, Second Edition (WRAML2) Story Memory and Verbal Learning subtests, along with the Rey Complex Figure Test (ROCFT) were used to measure performance at 24-hour (T3) and 7-day (T4) intervals with non-clinical older adults. The sample of older adults included 30 individuals between the ages of 55 and 75, recruited on a volunteer basis for participation in the study. Results revealed continuing psychometric viability for these subtests when used to assess retention well beyond the existing immediate (T1) and 15-minute delay (T2) intervals (Frise & Adams, 2009); that is, enough performance variability remained in order to detect varying levels of deficit. Claims for continued psychometric viability were based on the fact that scaled scores were found 2.0 standard deviations or more below the respective subtest T2 means. Scaled scores on the Story Memory subtest revealed possible scaled scores 3.0 standard deviations or more below the subtest’s mean at T3 and T4 (Frise & Adams, 2009). Scaled scores on the Verbal Learning subtest revealed scoring possible scaled scores of 2.0 standard deviations or more below the subtest’s mean at T3 and T4 (Frise & Adams, 2009).
Scaled scores on the ROCFT also revealed performance more than three standard deviations below the mean at T3 and T4 (Frise & Adams, 2009). This study provides support for the potential use of current STM measures in the assessment of LTM at delays, especially in clinical groups for which longer-term retention is especially impaired.

As already noted, current memory assessments use measures of immediate recall followed by 15 to 30 minutes later with measures of delay recall. This paradigm may provide a less robust early estimate of impairment in older persons who will eventually manifest more convincing symptoms that currently justify the diagnosis of AD. The purpose of this proposed study is to assess the relative diagnostic sensitivity of LTM measures compared to currently utilized measures using a group of individuals with early-stage Alzheimer’s disease.

This study is the first that examines the diagnostic utility of using longer-term memory measures created from modified short-term neuropsychological memory measures to detect early-stage AD. Because accurate diagnosis and early treatment can lead to interventions that can slow the progression of the disease, early detection in AD is critical. In this study, the sensitivity of several categories of measures will be evaluated in their sensitivity to distinguish between those suspected of being in the early stages of AD from those who are considered to be functioning within normal limits for age. The general domains chosen for this study include screening measures commonly used in clinical settings for detecting early signs and symptoms of AD, a self-evaluation measure, and both verbal and visual memory tests.

AD diagnostic accuracy might be improved if longer-term memory assessment was performed in conjunction with current clinical screening measures. Three brief cognitive screenings used to identify early-stage AD were also included in this study given their
practicality in clinical/medical settings and reported detection of early AD symptoms. Because it is relatively unknown how non-AD individuals living in a retirement home would perform on the specific neuropsychological assessments included in this study, 20 non-AD controls were recruited in this study. It was important to compare the two diagnostic groups to a non-AD group in a similar environment.
Chapter 2

Methods

Participants

Participants in this study were 60 people ranging from ages 55 to 75 residing in residential care facilities or nursing homes in Oregon. The sample was comprised of three subgroups: a non-AD subgroup (n = 20), an at-risk for AD subgroup (n = 20), and an early-stage AD subgroup (n = 20). Inclusion criteria for each group are listed below. Using 60 Participants allowed an effect size (Cohen’s $d$) of .80 or higher across the three groups. Using a significance level of $p \leq .05$ (Cohen, 1998) increased the likeliness of statistical significance across groups given a sample size of 60. For each hypothesis in the current study, power was established at .80 in order to increase the probability of rejecting the null hypotheses and avoid type II errors.

Care facilities and contact information for the facilities willing to participate in the study are included in Appendix A. Participating care facilities were selected on the basis of existing relationships with the author through participation in an AD Caregiver Support Group. Of the two care facilities approached, both agreed to participate. Each was located in Oregon and had specialized units for cognitive-related disorders. However, both of the care facilities also provided residential care to spouses of individuals with cognitive-related disorders, as well as an array of chronic medical problems. All eligible Participants for this study were required to be residing within one of the two selected residential care facilities at the time of participation, and were required to not have a current diagnosis of a cognitive-related disorder. Participants were
representative of individuals residing at the two care facilities without a cognitive-related disorder diagnosis. The Participants’ reasons for residing in the two care facilities was a result of various medical conditions estimated as cardiovascular (15%), musculoskeletal (5%), infectious (5%), or respiratory disease (20%) or as spouses or family members of residents with cognitive-related disorders such as dementia, AD, and Parkinson’s disease (55%). Eligibility was determined based on the criteria listed above as well as a review of each of the potential Participant’s medical record by facility coordinators with a master’s degree or higher at both care facilities.

Participants’ records were screened based on the criteria above to assure they did not have a previous AD diagnosis made by a physician or psychologist within 12 months prior to eligibility for this study. Potential eligibility for the current study was performed using a thorough medical record review to assure eligible Participants did not have an existing or previous diagnosis of a cognitive-related disorder that staff at the care facilities may not have been aware of. Medical record review was performed by the respective care facility coordinating staff members. Eligibility for the current study was determined by care facility coordinating staff review of medical records and observations from residential staff members regarding residents whom staff members had expressed concerns about possible early cognitive decline but lacked a pre-existing AD diagnosis. “Expert” status was based on an education level of a master’s degree or higher in nursing (Country Side Coordinator) or medical administration (Edgewood Downs Coordinator) along with at least three years working the in a field of geriatric care, and currently employed as a patient “Coordinator” at the two residential care facilities that participated in the study. Each coordinator (one per facility) identified and classified potential Participants into one
of the three subgroups (non-AD, at-risk AD, and early-stage AD) based on direct observation of residents, discussion with residential care facility staff, signs and symptoms of AD, and their knowledge of individuals residing in the respective residential care facilities.

The two residential care facility coordinators were given an example of the Participant informed consent form (found in Appendix B) prior to the administration of any test measures. In a similar fashion, the two care facilities were provided with an example of the caregiver informed consent form (found in Appendix C). Caregiver consent forms were provided to facility staff responsible for daily direct care of potential Participants. Caregiver consent forms were not given to family members of research Participants due to Participants’ legal status to sign their own individual consent forms. Participants were provided with Participant Consent Forms (found in Appendix B) and staff members were provided with a Caregiver Consent Form (found in Appendix C) prior to their research participation, which was required to be completed prior to data collection. Participant and Caregiver Consent Forms were reviewed by this writer prior to the start of assessment to assure consent on behalf of the Participant and Caregiver for research participation.

Measures

Immediately following the completion of consent forms, Participants were administered a list of seven self-evaluation questions designed to identify each Participant’s perceived memory abilities. This short list of questions provided a self-evaluation of memory functioning prior to the administration of memory tasks. The list of self-evaluation questions can be found in Appendix D. Administration of the list of self-evaluation questionnaire took approximately three
minutes. These questions were included in order to obtain a self-evaluation of memory functioning.

Immediately thereafter, the Mini Mental Status Exam (MMSE) was administered. The MMSE is a commonly used screening tool for psychologists and a brief quantitative measure of gross cognitive status, used to screen for severity of impairment at a given point in time (Folstein, Folstein, & McHugh, 1975); the content of the MMSE is found in Appendix E. Administration of the MMSE took approximately eight minutes. Scores on the MMSE can range from 0 to 30; a MMSE score between 20 and 25 suggests potential early cognitive decline based on MMSE norms for ages 55-75 (Folstein et al., 1975). Scores between 26 and 30 suggest normal cognitive functioning, while scores below 25 are indicative of varying degrees cognitive impairment based on norms for ages 55-75 (Folstein et al., 1975). Research has shown that clinical MMSE AD cut scores were established using 2 standard deviations based on a non-clinical sample of individuals between the ages of 55 and 75 (Folstein et al., 1975). The MMSE has shown near 80% specificity in the diagnosis of AD with a clinical sensitivity between 80-90% for AD when scores between 20 and 25 are achieved (Folstein et al., 1975). Previous research has not defined a mild or early AD range for MMSE scores.

The Alzheimer’s Disease Caregiver Questionnaire (ADCQ) was also administered to the care facility staff identified as direct caregivers of Participants with a frequency of one ADCQ administered per caregiver per participant (found in Appendix F). Administration of the ADCQ took approximately 10 minutes. The ADCQ is a computer-based assessment measure that reports pre-screening success at a level of 83-91% accuracy for AD (Solomon & Murphy, 2002). These pre-screening success rates were determined when individuals were administered the ADCQ.
prior to a full battery of cognitive measures resulting in positive diagnoses for AD (Solomon & Murphy, 2002). The ADCQ and MMSE provided useful information in the identification of typical AD symptoms and behaviors. Both of the selected screening measures were chosen for this study due to clinical relevance, ease of administration, common utilization by physicians when formulating an AD diagnosis (Kaufman, Solomon, & Salisberry, 2003), and use in previous related research (Kaufman et al., 2003).

Memory was assessed using portions of a common memory measures, the Wide-Range Assessment of Memory and Learning, Second Edition (WRAML2; Sheslow & Adams, 2003). The WRAML2 is designed to provide estimates of verbal and non-verbal memory functioning from 5 to 90 years of age. For the purpose of this research, Participants were administered two WRAML2 subtests, Story Memory and Verbal Learning. A description of the subtest procedure and the directions are included in Appendix G (Story Memory) and Appendix H (Verbal Learning). The WRAML2 was selected based on accessibility and attention to the specific needs of this current study to measure verbal memory deficits perceived in early-stage AD. Given the importance memory plays in AD (First & Tasman, 2005), the WRAML2 subtests provided an appropriate measure of verbal memory.

The WRAML2 Story Memory subtest consists of the narration of a short story of a few paragraphs, followed by the Participant retelling the story. This is followed by a second story that is also narrated and immediately followed by a retelling. The WRAML2 Story Memory subtest was used since it utilizes memory demands similar to those found in everyday conversation or passive listening. The WRAML2 Story Memory subtest Immediate Recall task consisted of narration of both stories and lasted approximately 8 minutes. The scaled score from
the Immediate Recall task performance represented the Participant’s performance for the Immediate Recall phase.

Approximately 15 minutes later, and without forewarning, the Participant is asked to recall both stories (without the stories being repeated). This is called the Story Memory Delay Recall phase. Administration of the delay recall phase took approximately 4 minutes. The scaled score from the Delay Recall task performance represented the Participant’s performance on the Delayed Recall phase. For both the Immediate and Delay Recall phases, the number of story items identified was totaled for a raw score, which was then converted to a scaled score \(M = 10, SD = 3\), using age-based norms found in the *WRAML2 Test Manual* (Sheslow & Adams, 2003).

The WRAML2 Verbal Learning subtest was also used. With this procedure, Participants were asked to recall a list of 16 nouns over four learning trials. This common learning and recall format was useful since it required new learning and recall of seemingly unrelated information, such as found with a grocery list or details within a set of directions. At the end of each learning trial the Participant was asked to recall as many words as could be remembered. The total number of words remembered over the four learning trials yielded a raw score that was then converted to an age-related scaled score using the WRAML2 norms from the *Test Manual* \(M = 10, SD = 3\). Test-retest correlation coefficients for the WRAML2 subtests of Story Memory and Verbal Learning subtests are shown in Table 1.

Approximately 15 minutes later, and without forewarning, the Participant was again asked to recall the words (without the words being repeated). This was called the Delay Recall phase. For the Delay Recall phase, the number of the 16 words identified was totaled and converted to an age-related scaled score using the WRAML2 norms \(M = 10, SD = 3\).
Table 1

*Test-Retest correlation coefficients for WRAML2 Story Memory and Verbal Learning subtests, and ROCFT.*

<table>
<thead>
<tr>
<th>Procedures</th>
<th>Test-Retest Reliability (Immediate Recall)</th>
<th>Test-Retest Reliability (Delay Recall)</th>
</tr>
</thead>
<tbody>
<tr>
<td>WRAML2 Story Memory</td>
<td>.75</td>
<td>.78</td>
</tr>
<tr>
<td>WRAML2 Verbal Learning</td>
<td>.78</td>
<td>.73</td>
</tr>
<tr>
<td>ROCFT</td>
<td>.76</td>
<td>.89</td>
</tr>
</tbody>
</table>

*Note: WRAML2 = Wide Range Assessment of Memory Learning, Second Edition; ROCFT = Rey Osterrieth Complex Figure Test.*

In this investigation, two additional phases were added to both the Story Memory and Verbal Learning procedures in order to create a longer-term measure of verbal memory. Specifically, after approximately 24 hours following the administration of the Immediate and Recall phases, each Participant was contacted by telephone and asked again to recall the stories and list of words without forewarning. A telephone call was used rather than an office visit since this would be a practical procedure for most practitioners, plus it did not create undue hardship on the Participant having to schedule an additional appointment. This 24-hour recall phase will be referred to as the T3 phase (with T1 being the Immediate Recall phase and T2 the Delay Recall phase).
A final recall trial occurred seven days following the T1 and T2 phases. This was labeled the T4 phase. Again, the Participant was telephoned and asked to recall each of the stories and list of words without forewarning and without the stories or words being repeated. However, each Participant was notified that there would be two follow-up telephone calls when obtaining the Participant’s consent. Time required for each test component as well as total time for all aspects of T1 – T4 phases is found in Table 2.

The Rey Osterrieth Complex Figure Test [ROCFT] (Meyers & Meyers, 1995) was also administered to Participants. The ROCFT is another widely used neuropsychological procedure; it is designed for the assessment of spatial processing, visual memory, and executive functioning. It was used in this study to assess visual memory decay over time in reference to long-term memory. The ROCFT consists of three test conditions: Copy, Immediate Recall, and Delayed Recall. The ROCFT requires Participants to draw one complex figure consisting of eleven interconnected and nested components, each one given one point for being drawn accurately and one point for being drawn in the correct location, yielding a maximum total score of 36. A third qualitative score, ranging from 1 (low) to 7 (high) is given for overall organization. Osterrieth defined the average adult score on the Copy production to be 32, and on the Recall production to be a 22 (Lezak, 1983). Age-related norms across ROCFT age groups indicate average ranges based on raw scores for a copy score between 27.0 and 36.0, an immediate recall score between 6.5 and 20.5, and a delayed recall score between 6.5 and 20.5 (Lezak, 1983).

The directions and procedures for the ROCFT can be found in Appendix I. and were followed in this study. The examiner first showed the Participant the ROCFT complex figure and asked the Participant to copy it. Performance on this copying was scored and constituted the
Using STM Measures to Assess LTM in Early AD

Table 2

*Content of test administration of Trial 1 through Trial 4 (T1 – T4), with estimates of the amount of time each procedure required.*

<table>
<thead>
<tr>
<th>Time of Tasks</th>
<th>Order of Procedures</th>
<th>Time (minutes)</th>
</tr>
</thead>
<tbody>
<tr>
<td>T1 – Immediate</td>
<td>Self-Evaluation Questionnaire</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>MMSE</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>ADCQ</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>WRAML2 Story Memory</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>WRAML2 Verbal Learning</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>ROCFT Copy and Immediate Recall</td>
<td>10</td>
</tr>
<tr>
<td>T2 – 30 minutes delay</td>
<td>WRAML2 Story Memory Recall</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>WRAML2 Verbal Learning Recall</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>WAIS-IV Information</td>
<td>8</td>
</tr>
<tr>
<td></td>
<td>ROCFT Recall</td>
<td>7</td>
</tr>
<tr>
<td><strong>TOTAL VISIT 1</strong></td>
<td></td>
<td><strong>69</strong></td>
</tr>
<tr>
<td>T3 – 24 hours delay</td>
<td>WRAML2 Story Memory Recall</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>WRAML2 Verbal Learning Recall</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>ROCFT Recall</td>
<td>7</td>
</tr>
<tr>
<td>T4 – 7 day delay</td>
<td>WRAML2 Story Memory Recall</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>WRAML2 Verbal Learning Recall</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>RCFT Recall</td>
<td>7</td>
</tr>
<tr>
<td><strong>TOTAL TME</strong></td>
<td></td>
<td><strong>95</strong></td>
</tr>
</tbody>
</table>

*Note: MMSE = Mini Mental Status Exam; ADCQ = Alzheimer’s Disease Caregiver Questionnaire; WRAML2 = Wide Range Assessment of Memory Learning, Second Edition; RCFT = Rey Complex Figure Test; WAIS-IV = Weschler Adult Intelligence Scale, Fourth Edition.*
Copy phase score. Three minutes after copying the figure, the Participant was asked to draw the figure from memory. The score derived constituted the Immediate Recall score. Administration of both the Copy and Immediate Recall phases of the ROCFT lasted approximately 10 minutes. Approximately 30 minutes later, and without forewarning, the Participant was again asked to draw the figure from memory. The score derived was called the Delay Recall score, the Delay Recall phase took most Participants about 7 minutes. For Copy, Immediate and Delay Recall phases, points earned were determined by a scoring rubric provided by the test authors (Meyers & Meyers, 1995). Accordingly, raw scores were converted to T scores, using age-based norms ($M = 50, SD = 10$).

In this investigation, two additional phases were added to the ROCFT procedure in order to create a longer-term measure of visual memory. Specifically, after approximately 24 hours, the Participant was contacted by telephone and asked to draw the figure on paper included within a pre-addressed and postage paid envelope addressed to the author; that envelope was provided during the first research visit and the Participant was told not to open it until the follow-up phone call. The request to again draw the figure was included in the same telephone interaction used to obtain the longer-term T3 Story and Verbal Learning recall tasks. The Participant was asked to insert the T3 drawing into the envelope and mail it within a day of the telephone call. A T4 ROCFT recall trial also occurred seven days following the T1 and T2 phases. As part of the T4 telephone call requests associated with the Story and Verbal Learning recall tasks already described, the Participant was again asked to draw the figure from memory on paper that was provided in a different envelope that also contained a pre-addressed and postage paid envelope addressed to the author. This envelope had also been given with instructions at the end of the
T1/T2 session. The Participant was asked to insert the T4 drawing into the envelope and mail it within a day of the telephone call. Each Participant was notified of the T3 and T4 telephone calls during consent administration, but was not told about their content. Test-Retest coefficients for the ROCFT are shown in Table 1. Test-retest reliability coefficients for the ROCFT Immediate and Delay Recall tasks are also found in Table 3.

Table 3

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Test-Retest Coefficient</th>
<th>Scaled Score Point Difference Between Testings</th>
</tr>
</thead>
<tbody>
<tr>
<td>WAIS-IV Information</td>
<td>.93</td>
<td>.05</td>
</tr>
</tbody>
</table>

Note: WAIS-IV = Wechsler Adult Intelligence Scale, Fourth Edition.

The WAIS-IV Information subtest was also administered to each Participant as a commonly-used measure of long-term verbal memory. A series of orally presented questions that tapped the general knowledge of common events, objects, places, and people was administered to each Participant. This procedure was administered in the standardized manner, with the directions found in Appendix J. Administration of the WAIS-IV Information subtest lasted approximately 8 minutes. The test-retest reliability coefficient for the WAIS-IV Information subtest can be found in Table 3.
Procedure

The researcher was blind to the group assignment made by the “expert” site coordinators. Blindness was maintained by having the facility coordinators select individuals to participate in the study, without this information being relayed to the researcher. Site coordinators kept a list of individuals selected and their classified group on a separate spreadsheet document that was not given to the researcher until data collection was complete. Participants were classified and selected for the study until 60 Participants had been administered the research measures for a total of 20 non-AD Participants, 20 at-risk Participants, and 20 early-stage AD Participants. Facility coordinators monitored the potential Participants being selected and kept track of how many individuals had been selected for each group.

Neither Participants nor Care Facilities received financial compensation for their participation in this study. The primary researcher gained approval from the George Fox University Internal Review Board prior to beginning the research, and proceeded with an informed consent process. Care facility caregivers were asked to participate in the informed consent process and Caregiver consent forms for their participation in the current study.

Following the informed consent process, Participants were asked to complete the list of seven self-evaluation questions related to memory found in Appendix C. Following the administration of the self-evaluation questionnaire, Participant’s were administered the Self-Evaluation, MMSE, ADCQ, WRAML2 subtests (Story Memory and Verbal Learning), ROCFT, and WAIS-IV Information subtest in a randomized order. The measures administered were divided into 3 separate randomized forms (A, B, & C) which organized the measures mentioned above to allow for optimal Participant performance across measures. Randomized orders
consisted of Form A (Self-Evaluation, MMSE, WRAML2 subtests, ROCFT, and WAIS-IV Information), Form B (Self-Evaluation, MMSE, WRAML2 subtests WAIS-IV Information, and ROCFT), and Form C (Self-Evaluation, MMSE, ROCFT, WRAML2 subtests, and WAIS-IV Information).
Chapter 3

Results

Demographic information of Participants including mean age, standard deviation of age, and gender can be found in Table 4. A one-way ANOVA using age as the dependent variable yielded results that showed no significant differences across the three groups \( F(2,57) = .96, p > .40 \). Differences in gender proportions across groups were found to be non-significant \( \chi^2(1) = .27, p > .60 \).

Table 4

*Age, Size and Gender Distribution for Each Sample*

<table>
<thead>
<tr>
<th>Group</th>
<th>Mean Age and (SD)</th>
<th>N</th>
<th>Gender</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-AD</td>
<td>69.7 (0.71)</td>
<td>20</td>
<td>11 F 9 M</td>
</tr>
<tr>
<td>At-Risk</td>
<td>70.6 (4.95)</td>
<td>20</td>
<td>7 F 13 M</td>
</tr>
<tr>
<td>Early-Stage AD</td>
<td>71.4 (4.24)</td>
<td>20</td>
<td>14 F 6 M</td>
</tr>
</tbody>
</table>

*Note: AD = Alzheimer’s disease; F = female; M = male.*
To organize the results of the analyses, each hypothesis will be re-stated in terms of the dependent variables analyzed in order to address the respective hypothesis related to the 3 groups consisting of a non-AD subgroup (n = 20), an at-risk for AD subgroup (n = 20), and an early-stage AD subgroup (n = 20). To assess performance on the Self-Evaluation Questionnaire, MMSE, and ADCQ across the three groups of Non-AD, At-Risk, and Early AD group mean scores on each measure were used. Mean score performance was based on raw number of Self-Evaluation items endorsed, MMSE raw scores (up to 30 points possible), and ADCQ hits (items endorsed by caregivers). Mean score results for each group across the three measures can be found in Figure 1.

Figure 1. Mean performance by group on the Self-Evaluation Questionnaire, MMSE, and ADCQ (hits), X axis = measures and Y axis = raw scores. AD = Alzheimer’s Disease, MMSE = Mini Mental State Exam, ADCQ = Alzheimer’s Disease Caregiver Questionnaire.
To determine whether the three subgroups performed differently on the Self-Evaluation Questionnaire, a one-way ANOVA was conducted to compare the total raw score means for each subgroup. Mean total raw scores and standard deviations (found in parentheses) for each group were: the Non-AD group 10.9 (1.4), for the At-Risk group 10.8 (3.3), and for the Early AD group 10.3 (2.7). A one-way ANOVA revealed no differences between groups ($F(2, 57) = .53, p < .05$), indicating that self-evaluation of memory across the three groups did not differ.

To determine whether there were differences between MMSE scores across the three groups, a one-way ANOVA was conducted using MMSE raw scores. Mean scores and standard deviations (in parentheses) of MMSE raw scores for the three groups were: Non-AD group 27.7 (1.3), At-Risk group 25.2 (2.2), and Early AD group 21.2 (1.5). MMSE scores were found significantly different across the three groups $F(2, 57) = 72.7, p < .05$, eta squared = .936). Post-hoc comparisons indicate the Non-AD and At-Risk group means did not differ, however the Early AD group mean differed from both the Non-AD and At-Risk groups.

To determine whether if there was a difference between the three subgroups on ADCQ scores, a one-way ANOVA was conducted using total ADCQ hits (items endorsed). Mean scores and standard deviations (in parentheses) of ADCQ total scores for the three groups were: Non-AD group 0.3 (0.6), At-Risk group 5.3 (1.7), and Early AD group 11.4 (1.2). ADCQ scores for the three groups were found significantly different ($F(2, 57) = 415.9, p < .05$, eta squared = .864). Post-hoc comparisons indicate meaningful ADCQ differences in the Non-AD, At-Risk, and Early AD groups.

Traditionally, clinicians and practitioners use MMSE raw scores and ADCQ hits to make decisions regarding potential symptoms of AD and the need for further diagnostic clarification.
To assess the consistency between AD screening measures, correlations were calculated using raw scores achieved on the MMSE and ADCQ hits. Inverse correlations of moderate magnitude were expected, due to the expectation that lower MMSE raw scores would be associated with more ADCQ hits and vice versa. Table 5 shows the correlations between the MMSE and ADCQ for the Non-AD, At-Risk, and Early AD groups. MMSE and ADCQ correlations for the Early AD group showed a moderate inverse correlation of $r = -.31$. Correlations for the MMSE and ADCQ among the Non-AD ($r = -.08$) and At-Risk ($r = -.07$) groups were non-significant. These findings show that when administered for AD screening purposes, the results generated by the MMSE and ADCQ in the Early AD group show inverse correlations, and are therefore diagnostic when used appropriately.

Table 5

*Pearson and Spearman Correlations between the MMSE Raw Scores and ADCQ Hits for Non-AD, At-Risk, and Early AD Subgroups.*

<table>
<thead>
<tr>
<th></th>
<th>ADCQ</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-AD MMSE</strong></td>
<td></td>
</tr>
<tr>
<td>Pearson r</td>
<td>-.08</td>
</tr>
<tr>
<td>Spearman r</td>
<td>-.13</td>
</tr>
<tr>
<td><strong>At-Risk MMSE</strong></td>
<td></td>
</tr>
<tr>
<td>Pearson r</td>
<td>-.07</td>
</tr>
<tr>
<td>Spearman r</td>
<td>-.04</td>
</tr>
<tr>
<td><strong>Early AD MMSE</strong></td>
<td></td>
</tr>
<tr>
<td>Pearson r</td>
<td>-.31</td>
</tr>
<tr>
<td>Spearman r</td>
<td>-.37</td>
</tr>
</tbody>
</table>

*Note:* AD = Alzheimer’s Disease; ADCQ = Alzheimer’s Disease Caregiver Questionnaire; MMSE = Mini Mental Status Exam.
To assess the relationship between memory variables, as well as stability for each memory measure over time, correlations were calculated among scaled scores obtained at each of the four trials for each memory measure. Correlations of moderate magnitude were expected. All correlations across T1-T4 within given tasks were significant and of moderate magnitude. Correlations based on scaled scores for all three groups on each memory measure (WRAML2: Story Memory, WRAML2: Verbal Learning, and ROCFT) across the four trials can be found in Table 6. Correlations based on scaled scores for the Non-AD group on each memory measure (WRAML2: Story Memory, WRAML2: Verbal Learning, and ROCFT) across the four trials can be found in Table 7. Correlations based on scaled scores for the At-Risk group on each memory measure (WRAML2: Story Memory, WRAML2: Verbal Learning, and ROCFT) across the 4 trials can be found in Table 8. Correlations based on scaled scores for the Early AD group on each memory measure (WRAML2: Story Memory, WRAML2: Verbal Learning, and ROCFT) across the 4 trials can be found in Table 9. These findings demonstrate that performance on a memory tasks (WRAML 2 subtests and ROCFT) are relatively stable over time across the three subgroups in this study despite anticipated declines in performance on the basis of forgetting over time.