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LOMA LINDA UNIVERSITY School of Behavioral Health in conjunction with the Faculty of Graduate Studies

Neuropsychological Correlates of Anosognosia in MCI and Dementia

by

Kyrstle Dina Barrera

A Dissertation submitted in partial satisfaction of the requirements for the degree Doctor of Philosophy in Clinical Psychology

September 2013

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, Co-Chairperson Travis G. Fogel, Director of Neuropsychology and Psychology, Assistant Professor of Physical Medicine and Rehabilitation, Assistant Professor of Psychology

Co-Chairperson

Susan A. Ropacki, Associate Professor of Psychology

Michael J. Gilweski, Associate Professor of Physical Medicine and Rehabilitation, Assistant Professor of Psychology

David A. Vermeersch, Professor of Psychology, Director of Clinical Training

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ABSTRACT OF THE DISSERTATION

Neuropsychological Correlates of Anosognosia in MCI and Dementia

by

Kyrstle Dina Barrera

Doctor of Philosophy, Graduate Program in Psychology Loma Linda University, September 2013 Drs. Travis G. Fogel and Susan A. Ropacki, Co-Chairpersons

Anosognosia is a general term used to describe a lack of awareness of a disability and is well documented in various disorders associated with neurological compromise. While anosognosia is well documented as part and parcel to Alzheimer's dementia, less research has focused on determining the presence of anosognosia in what has come to be viewed as the subclinical precursor to dementia, mild cognitive impairment. In addition, a number of different methodologies and instruments are employed in quantifying and assessing anosognosia in various populations, which make comparison across studies and diagnoses difficult. Research commonly employs a paradigm that uses the discrepancy between informant and patient reports as measures of anosognosia, using informant reports as the benchmark against which patient ratings are compared. Little research has been done, however, to investigate the accuracy of informant reports as they relate to actual patient performance.

The current study sought to investigate the accuracy of patient and informant reports as they relate to actual neuropsychological function, identify the presence or absence of anosognosia within the MCI population, and explore the diagnostic utility of anosognosia assessment in MCI and dementia populations. A total of 49 patients were included in the sample (n=24 MCI patients and n=25 dementia patients). Patients

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underwent routine neuropsychological evaluation across 6 domains of function. They were asked to predict their performance on each neuropsychological measure prior to administration, and then evaluate their actual performance subsequent to administration. Parallel prediction ratings were solicited from an informant.

Results indicated that informant predictions were often less accurate with respect to actual neuropsychological performance than patient predictions. In addition, MCI patients often demonstrated greater levels of anosognosia than their dementia counterparts, with their ratings being less favorable than their actual performance. Lastly, results indicate that anosognosia measures are reliable in predicting group membership, with anosognosia for general cognitive ability and delayed contextual memory being the most predictive of all the measures administered. Thus, the current study provides evidence for the utility of routine assessment of anosognosia in MCI and dementia neuropsychological evaluation.

CHAPTER ONE INTRODUCTION

Anosognosia Defined

Anosognosia is a general term used to describe a lack of awareness of a disability and was initially coined by Joseph Babinski in 1914 to describe patients who denied leftsided hemiplegia. The clinical syndrome that Babinski described was one in which patients professed that they were able to engage in activities that required full use of their left side, when in fact the left side of their body lay flaccid in their hospital bed. Although an unawareness of this magnitude could lead one to question the psychiatric underpinning of these beliefs, it was noted long before Babinski's involvement that this behavioral anomaly was typically a result of acute focal cerebral lesions (Prigatano & Schacter, 1991).

Unawareness of injury and its relation to denial of an injury are presumed to be two different phenomena, and careful steps are taken in the literature to distinguish between the two (Kortte & Wegener, 2004). Denial of injury is often used to describe a characterological dynamic in which a patient is unwilling to accept the presence of a medical condition and the impact that is may have on his/her health. For example, a patient diagnosed with diabetes may deny the presence of the disorder and refuse to account for the impact it may have on his/her overall well-being, continuing to eat foods repeatedly shown to exacerbate the medical condition. Denial may also be viewed as a psychological defense employed by an individual to guard against the emotional pain associated with a significant loss (Heilman & Harciarek, 2010). The clinical presentation

of denial lies in theoretical contrast to that of anosognosia, which is typically observed in conjunction with neurological dysfunction.

Facets and Clinical Relevance of Anosognosia

Flashman and McAllister (2002) discuss unawareness as a multifaceted construct, and identify three dimensions of unawareness that are important in conceptualizing the anosognostic picture across different neurologic populations. The first dimension is whether an individual is aware of a specific deficit, and this dimension is commonly a dichotomous (yes or no) distinction that can vary across different areas of deficit. For example, an individual may be aware of a deficit in memory but unaware of a deficit in social pragmatics. The second dimension is a corresponding emotional response to each area of functioning, regardless of whether or not an individual is aware of a deficit. This second dimension is best understood on a continuum related to emotional arousal, with complete indifference (anosodiasphoria) on one end of the spectrum and anger on the other. Given these two dimensions, it is possible that an individual may be aware of a deficit but display indifference to it. The third dimension relates to an individual's ability to appreciate the functional impact that a deficit may have on day-to-day life and speaks to the real-world implications of anosognosia and the obstacle it creates in meaningful rehabilitation. An individual may be aware of a deficit, have an emotional response to experiencing the deficit, but have little or no grasp of how such a deficit would impede his/her daily function, and may fail to appreciate the importance of procuring help from friends and family members. Thus, this dimension is also best understood on a continuum, with a lack of appreciation for the functional impact of a deficit on one end

and complete appreciation for the functional impact on the other (Flashman & McAllister, 2002).

Unawareness puts patients in immediate physical danger, and the impact of anosognosia on rehabilitation efforts after an acute injury is immense (Hartman-Maeir, Soroker, & Katz, 2001). For example, a patient without an awareness of his/her own hemiplegia may be inclined to get out of bed to use the restroom, which would ultimately result in a fall. Patients with anosognosia have been found to have a higher incidence of safety issues as well as a higher incidence of being deemed unsafe at discharge from acute rehabilitation (Hartman-Maeir et al., 2001). Patients lacking insight into their impairments will be unable to utilize compensatory strategies in real world situations and have the propensity to get into situations that they will subsequently have difficulty managing. They are likely to be unable to assimilate and utilize feedback about their limitations to set realistic goals, and have poorer outcome after rehabilitation (see Prigatano (2005) for a review). Due to the fact that they are unaware of their deficits, it is likely that patients exhibiting anosognosia will resist help and/or treatment offered by family and medical professionals who demonstrate concern for their overall well-being (Flashman & McAllister, 2002; McGlynn & Kaszniak, 1991). In addition, a lack of insight into their areas of difficulty will make it more difficult to engage them in consistent rehabilitation efforts that are imperative to their overall improvement. While these risks can be managed on an acute inpatient setting, once the patient is no longer in daily contact with treating medical providers or family members who may or may not be aware of their deficits, the responsibility for rehabilitation falls in the hands of the patient. Although the literature on functional outcome after acute rehabilitation for patients with

anosognosia is mixed, anosognosia has been implicated as having a negative impact on recovery in acute rehabilitation settings as well as in long term recovery (Gialanella, Monguzzi, Santoro, & Rocchi, 2005; Hartman-Maeir et al., 2001)

While it can be difficult to discuss the presence and impact of anosognosia with patients who have just endured a life changing incident, having a specific event with which to tie functional and/or cognitive changes can make these issues more tangible for patients and their families. For patients who exhibit anosognostic traits in relation to general, typically longstanding neurological dysfunction, in the absence of acute focal neurological damage, clinical concerns become increasingly difficult to broach. Often times cognitive degeneration associated with dementia, for example, is insidious and even family members who are actively involved in caregiving for the patient have difficulty pinpointing the onset of symptoms (for a review, see Levy (1994)). In the elderly population, it is often difficult for family members to discern the nature of cognitive decline associated with normal aging and abnormal cognitive decline indicative of neurological dysfunction warranting medical attention (Levy, 1994). Thus, methods of compensating for cognitive deficits and promoting healthy cognition are likely to be ignored.

Anosognosia in Various Disorders

As previously discussed, the initial body of literature describing anosognosia focused primarily on unawareness of, or lack of appreciation for, the functional impact of encapsulated deficits of gross motor or perceptual ability that resulted from acute neurological injury (Prigatano & Schacter, 1991). The nature of these deficits was such

that they were readily apparent by observers, primarily complete hemiplegia or hemianopia. Over time, the term has broadened in its application and has come to be used to describe a lack of insight into any deficit resulting from atypical neurological functioning, including focal neurologic damage such as stroke (Kortte & Hillis, 2009; Pedersen, Jørgensen, Nakayama, Raaschou, & Olsen, 1996; Starkstein, Jorge, & Robinson, 2010), cortical blindness (Prigatano & Wolf, 2010), Wernicke's aphasia (Kertesz, 2010), as well as more diffuse neurologic dysfunction, such as traumatic brain injury (Prigatano, 1991, 2005, 2010a), schizophrenia (Gilleen & David, 2010), and a variety of dementia (McGlynn & Kaszniak, 1991; O.C. Okonkwo, Spitznagel, Alosco, & Tremont, 2010).

Anosognosia and Stroke

Anosognosia for motor deficits as a consequence of right hemisphere stroke is well documented and thoroughly researched (Kortte & Hillis, 2009; Pedersen et al., 1996; Starkstein et al., 2010), although the cause of anosognosia and the neuroanatomical mechanism of action remains highly disputed. In a recent literature review on anosognosia and stroke, the incidence of anosognosia was cited to range between 20% to 40%, with the variability across studies ascribed to time since stroke, age, and operational definitions of anosognosia (Starkstein, Jorge & Robinson, 2010). According to Starkstein et al.'s (2010) review of the literature, anosognosia often resolves relatively early on in the rehabilitation trajectory and is rarely seen three months post-stroke. The occurrence of anosognosia increases with age, and its incidence varies depending on the methodology used to define anosognosia in the literature. Clinically, it is relatively

simple to diagnose using discrepancies between interviews with patients reporting their functional ability and observation of patients' actual functional ability, especially for impairments as blatant as hemiplegia. The operational definition that is necessitated by systematic research, however, requires that true anosognosia be clearly distinguished from other syndromes that are commonly observed with in clinical settings (Starkstein et al., 2010).

Anosognosia in stroke patients is typically seen as one piece of a multifaceted clinical presentation, and is often associated with a host of other peculiar symptoms, the two most common being a lack of recognition of ownership of a limb (asomatognosia) as well as attributing one's limb to another person (somatoperephrenia). Anosognosia is also observed with concomitant indifference to the affected limb (anosodiaphoria), as well as feeling automatic movement in the affected limb (kinaesthetic hallucinations) and negative feelings toward the affected limb (misoplegia) (see Starkstein et al., (2010) for review). Interestingly, sensation in the affected limb in these patients is often intact, and thus cannot be accounted for by a lack of incoming sensory stimuli. Although these symptoms are typically documented in right hemisphere damage, which complicates the explanation of the anosognostic syndrome as being "housed" in right hemisphere function (Cocchini, Beschin, Cameron, Fotopoulou, & Della Sala, 2009).

Anosognosia and Focal Lesions: Cortical Blindness

Anton's syndrome, or cortical blindness, is a loss of vision characterized by dysfunction of the occipital lobe where visual sensory information is processed. This is

in contrast to the more common forms of blindness associated with a dysfunction of sensory perception (Vighetto & Krolak-Salmon, 2007). Anosognosia for cortical blindness is also highly documented in the literature, actually predating the time of Babinski. In 1898, Anton reported cases of patients who demonstrated unawareness of visual loss incurred as a result of focal cerebral lesions (as cited in Prigatano, 2010b). Due to the fact that cortical blindness is not a focus of current anosognostic research, there has been little advancement in its understanding. According to theoretical discussion by Prigatano and Wolf (2010), the clinical presentation of anosognosia for cortical blindness deserves special attention and the authors proposed a specific set of terminology to differentiate between its subtypes. The authors argue that Anton's syndrome is a term that should be reserved for patients who experience true cortical blindness, or experience complete loss of vision secondary to bilateral primary visual cortex damage in the occipital lobe, and demonstrate an unawareness of this blindness. The term Anton-like syndrome should be ascribed to patients who are completely blind secondary to damage to the orbit or to the optic nerve, and thus not cortical in nature. Lastly, Prigatano and Wolf (2010) posit that those patients unaware of a hemianopia (UHEM) secondary to damage to visual projection areas should be differentiated as well. These patients' blindness is, by definition, limited to discernable portions of their visual field(s) as a direct result of brain lesions. A majority of the literature on Anton's and related syndromes consists of case studies, and little is known about the neuroanatomical underpinnings of how these conditions relate to unawareness and why the two often go hand in hand.

Anosognosia and Focal Lesions: Wernicke's Aphasia

An arguably more frequent clinical syndrome that results from acute neurological insult, and a prime example of anosognostic behavior, is that of jargon aphasia, also referred to as "anosognosic aphasia" (Rubens & Garrett, 1991). Jargon aphasia is traditionally associated with Wernicke's aphasia, but is also seen in transcortical sensory and global aphasia. It is colloquially referred to as "word salad", such that meaningless combinations of words wrought with phonemic, semantic, and neologistic paraphasias characterize a patient's speech (for a review of aphasia, see Abutalebi & Cappa, 2008). Of note, and the reason this behavioral pattern is associated with anosognosia, is that patients exhibiting this type of speech have no awareness that their speech is nonsensical and no insight into the fact that the listener is unable to comprehend what they are saying. The fact that these patients are so fluent in their speech, speaking without correction, pause, or hesitation, suggests that they are simply unaware that anything is in need of being corrected (Kertesz, 2010; Rubens & M. F. Garrett, 1991).

Anosognosia and Traumatic Brain Injury

While many early accounts of anosognosia are related to focal cerebral injury, more recent inquiry into anosognosia focuses on more diffuse neurological involvement. Although detailed accounts date back to the time of Phineas Gage, anosognosia subsequent to traumatic brain injury has been the topic of scientific investigation in more recent anosognosia literature. While significant strides have been made in quantifying and systematically studying the disorder, the wide range of abilities and disabilities for which a patient could be lacking awareness as well as the variation in the degree of

severity of unawareness makes generalizable conclusions difficult to achieve (Dirette & Plaisier, 2007; Hart, Sherer, Whyte, Polansky, & Novack, 2004; Hoofien, Gilboa, Vakil, & Barak, 2004; Newman, Garmoe, Beatty, & Ziccardi, 2000; G.P. Prigatano, 2010a; Trahan, Pepin, & Hopps, 2006). While previously discussed anosognostic phenomena were related to specific deficits in awareness, traumatic brain injury can lead to impaired awareness across many areas of life. Thus, the lack of awareness in traumatic brain injury is often multifaceted, including, but not limited to, overall cognitive ability, sensorimotor functioning, general mood state and emotional lability, behavioral impulsivity, and social pragmatics.

Flashman and McAllister (2002) reported that up to 45% of individuals who have sustained a moderate to severe traumatic brain injury exhibit deficits in awareness of disabilities that are readily observable by others, and that these deficits in awareness, unlike that of anosognosia of hemiplegia post-stroke, are often permanent. Numerous questionnaires and interviews have been constructed to measure anosognosia in traumatic brain injury populations, but many of these studies are confounded by the presence of denial that is not quantifiably differentiated. In the previously discussed neurological disorders, anosognosia is often seen in the absence of any severe cognitive impairment, thus the patients presumably have the capacity for awareness. This is in contrast to traumatic brain injury, where impaired self-awareness is often seen as part of a constellation of cognitive difficulties that may, in and of themselves, render the patient cognitively incapable of grasping such an intangible construct.

Not surprisingly, the severity of the traumatic brain injury, as assessed by length of post-traumatic amnesia and admitting Glasgow Come Scale score, has been shown to

be related to severity and duration of anosognosia, such that mild traumatic brain injury patients often become aware of deficits in balance, memory, concentration, and sensory sensitivity (to light and noise) rather quickly (Prigatano, 2010a). This patient population typically experiences distress secondary to their improved awareness, which typically abates within the first 30 to 90 days post-injury with improved neuropsychological functioning (Prigatano, 2010a). Their self-assessments of functioning are typically comparable to the assessments provided by informants, which facilitates post-injury rehabilitation efforts. For patients with severe traumatic brain injury, the clinical picture is often more concerning. These patients typically have no recollection of their injury and often fail to appreciate their need for medical care (Prigatano, 2010a). Many of these individuals, even after undergoing acute inpatient neuropsychological rehabilitation, continue to exhibit anosognosia for multiple impairments even one to two years post injury. It is estimated that approximately 30-40% of patients suffering from severe traumatic brain injury demonstrate anosognosia, but it is unknown what proportion of these patients display longstanding anosognostic behavior (Prigatano, 2010a). For patients with moderate traumatic brain injury, awareness typically emerges once posttraumatic amnesia clears, but this rule of thumb is mitigated by frontal lobe involvement, such that increased frontal lobe damage is associated with more severe levels of anosognosia. Unfortunately, this group of patients has yet to be well-studied in isolation and are typically combined with patients with severe traumatic brain injury in research studies (Prigatano, 1991, 2005, 2010a).

Anosognosia and Schizophrenia

Given the complicated nature of assessing anosognosia subsequent to an acute neurological insult and the equivocal nature of conclusions from these studies, the study of anosognosia in neurological conditions characterized by generalized cognitive impairment can be expected to be increasingly complex. Schizophrenia is traditionally viewed as a psychiatric disorder characterized by a general detachment from reality, which can be conceptually linked to the unawareness inherent in anosognostic phenomena. Patients with schizophrenia are typically unaware of the presence of a mental disorder, which makes treatment and community integration notoriously difficult. Given the evidence correlating schizophrenia to frontal lobe dysfunction, and frontal lobe dysfunction to impaired self-awareness, viewing schizophrenic symptomology through the lens of anosognosia provides an opportunity to combine theoretically related clinical syndromes (Gilleen et al., 2010; McGlynn & Kaszniak, 1991; Stuss, 1991).

According to Gilleen and collegues (2010), the nature of unawareness seen in schizophrenia is best conceptualized as a deficit in judgment and reasoning, such that they interpret and attribute abnormal perceptions related to hallucinations and delusions as true. Anosognosia secondary to neurological insult, however, is argued to be a product of an inaccessibility of the information to monitoring systems responsible for modality specific perception. In current literature, three main theories seek to conceptualize unawareness in schizophrenia in the following ways: as a symptom of psychiatric symptomology in general, as a result of neuropsychological impairment inherent in schizophrenia, or as a psychologically motivated defense against negative emotions.

Although the unawareness seen in schizophrenia can theoretically be differentiated from the unawareness seen in neurologic populations, there are also commonalities between the unawareness observed in the two populations. Interestingly, patients with schizophrenia have been shown to lack awareness regarding their abnormal, involuntary motor movements (McGlynn & Kaszniak, 1991). While it is parsimonious to attribute both the presence of the motor movement and the lack of awareness of their presence to the side effects of neuroleptic medications, the presence of these motoric abnormalities has been documented long before the invention of neuroleptic medication and are seen in patients who are unmedicated (McGlynn & Kaszniak, 1991). Thus, there appears to be a connection between the neuroanatomical systems that mediate motor movement and that which mediate awareness of motor movement that would account for this dynamic, similar to that seen in right hemisphere stroke patients that demonstrate anosognosia for hemiplegia. Analogous to patients with anosognosia secondary to diffuse traumatic brain injury, and thus general neurological dysfunction, patients with schizophrenia also often experience unawareness of deficit in various spheres of functioning, including overall cognitive functioning, mood and irritability, as well as social pragmatics. This implies that awareness may be linked to intact gross systemic brain function as opposed to being localized in a specific brain area (Gilleen et al., 2010).

Anosognosia and Dementia

Similar to schizophrenia, dementia is also characterized by gross neurological involvement that is often accompanied by an impaired awareness of both cognitive and behavioral deficit. While the prevalence of anosognosia is debatable in stroke and

traumatic brain injury literature, anosognosia is consistently part and parcel to the clinical picture of dementia. It is most frequently reported in dementia of the Alzheimer's type, which is definitively diagnosed post-mortem and is colloquially distinguished by poor memory ability (Wagner, Spangenberg, Bachman, & O'Connell, 1997). Alzheimer's dementia is a degenerative disorder that is characterized by amyloid plaque build up in cortical tissue, which impedes proper functioning of neural networks and results in dramatic functional decline. The initial cause for concern in patients who are eventually diagnosed with Alzheimer's dementia is marked forgetfulness, which progresses into confusion, and ultimately into disorientation as the disease unfolds (McGlynn & Kaszniak, 1991).

Anosognosia is a common feature of dementia of the Alzheimer's type, and of particular concern to family members and caregivers who cannot understand why their loved one is unaware of their decline in functioning or is unconcerned and offers benign explanations. Barrett, Eslinger, Ballentine, and Heilman (2005) conducted a study asking patients diagnosed with probably Alzheimer's dementia (pAD) to conduct pretest and posttest estimations of their performance within various cognitive domains. The pretest ratings asked the patient to estimate how well they thought they would do on tasks assessing their ability within a certain cognitive domain, such as measures of memory or visuospatial skills. The posttest ratings were conducted after the entire battery of measures had been completed, presumably with the patient having a better understanding of how they performed on the tasks and better insight into their own capabilities. The authors created an anosognosia ratio, which allowed them to account for both

overestimations as well as underestimations, and their performance was compared to a control group.

Barrett and colleagues (2005) found that, compared to their healthy control counterparts, patients with pAD overestimated their visuospatial skills on pretesting and overestimated their memory skills on post testing. Thus, visuospatial skill estimations were amended by actual performance on tasks, whereas estimations of memory continued to be poorly estimated after testing took place. Thus, patients demonstrated anosognosia for memory even after performing poorly on memory tasks. While the authors noted that pAD patients performed significantly worse on memory measures, actual performance on neuropsychological testing was not analyzed in relation to their pretest or posttest estimations. Interestingly, these results elucidate domain specific anosognosia and differential impact of poor performance on estimations of ability (Barrett et al., 2005). Of note, it is unclear if the discrepancy in scores can more readily be accounted for by the nature of the patient population's poor memory, which would preclude participants from providing accurate estimates of their performance on measures simply due to a lack of memory for having completed them.

In a review by Agnew and Morris (1998), a number of correlates have been proposed to coincide with anosognosia and Alzheimer's dementia, and it is difficult to determine which of these facets are directly related to and/or responsible for the anosognostic phenomena seen in this population. Moreover, failure to adequately measure these facets in controlled studies puts them in the position of being potential confounds, and could account for the discrepancy in results across studies. According to the authors, anosognosia has been associated with the severity of dementia, with higher

levels of anosognosia related to more severe states of dementia, and relatively preserved awareness associated with early stages of dementia.

Current literature on the topic, however, remains inconsistent, with argument that even early stages of dementia exhibit marked anosognosia, and that the presence of early anosognosia can be used as a predictor of dementia characterized by a more rapid deterioration of overall ability (Carr, Gray, Baty, & J. C. Morris, 2000; Derouesné et al., 1999). Agnew and Morris (1998) also discuss language impairment as a correlate of anosognosia in dementia, which may be related to the anosognosia seen in aphasia patients discussed earlier. Not surprisingly, memory impairment is also correlated with anosognosia, as well as executive dysfunction and frontal lobe deficits (Agnew & Morris, 1998).

While initial anosognostic literature in Alzheimer's dementia patients focused on the lack of awareness for memory dysfunction, relatively recent literature also highlights a lack of awareness for visuospatial skills as well (Agnew & Morris, 1998; Barrett, Eslinger, Ballentine, & Heilman, 2005). Less is known about frontotemporal dementia and primary progressive aphasia in relation to anosognosia, primarily due to the fact that the literature on anosognosia and dementia is still in its infancy. Briefly, frontotemporal dementia is characterized by marked behavioral changes associated with a decline in frontotemporal brain function. A hallmark of this disorder is the lack of awareness of the behavioral changes, as well as a lack of concern when these changes are discussed with the patient (Miller & Cummings, 2006). Thus, a lack of awareness for behavioral changes is a main criterion in its diagnosis. Primary progressive aphasia is another type
of dementia associated with frontotemporal degeneration, marked by deficits in language ability (Banks & Weintraub, 2009).

Patients with frontotemporal dementia also demonstrate impaired awareness for cognitive and behavioral deficits, which is consistent with the role of the frontal lobes as being involved in self-monitoring and inhibition (Agnew & Morris, 1998). Patients with primary progressive aphasia typically do not initially demonstrate impaired awareness, but little is known about the awareness of this population because few studies have investigated primary progressive aphasia in relation to anosognosia (Agnew & Morris, 1998). A study conducted by Banks and Weintraub (2009) found that patients with frontotemporal dementia did not significantly differ from patients with Alzheimer's dementia in their level of awareness, but that patients with primary progressive aphasia demonstrated relatively intact levels of awareness. Due to the fact that the symptomatology between primary progressive aphasia and frontotemporal dementia begin to overlap as both diseases progress, assessment of awareness early on in treatment may be a beneficial tool in diagnosis and treatment planning (Banks & Weintraub, 2009)

Anosognosia and Mild Cognitive Impairment

Current conceptualization of dementia views the disorder as a continuum of impairment with mild cognitive impairment on one end of the spectrum and traditional dementia on the other, as opposed to a dichotomous diagnostic category. Mild cognitive impairment (MCI) can be viewed as a sub-clinical precursor to dementia, and factors predicting conversion to dementia are not well known or understood. The most promising area of research, and the most flourishing, is the investigation of

pathognomonic biomarkers beta-amyloid protein ($\alpha\beta$) and tau (τ), which are closely associated with the progression of Alzheimer's dementia (Albert et al., 2011). $\alpha\beta$ and τ are biological markers for the amyloid plaques and neurofibrillary tangles whose buildup are thought to be responsible for the neurodegeneration of cortical tissue. The cognitive dysfunction seen in MCI, and ultimately Alzheimer's dementia, are attributed to this neurodegeneration. The following section provides a review of the current literature relating to MCI diagnosis and its clinical presentation. MCI has received considerable attention in recent literature and the prognostic value of levels of insight into areas of impairment as a predictor of conversion to Alzheimer's dementia is gaining popularity. Patients with mild cognitive impairment demonstrate impaired awareness for memory and visuospatial skills, similar to Alzheimer's dementia patients (Galeone, Pappalardo, Chieffi, Iavarone, & Carlomagno, 2011; Vogel et al., 2004). In fact, Vogel and colleagues (2004) found that there was no statistical difference between the level of unawareness demonstrated by patients with mild cognitive impairment and that of patients with probable Alzheimer's dementia. While Clément, Belleville, and Gauthier (2008) found that there was no difference in overall cognitive complaints in both groups, numerous studies have found that patients who eventually converted to Alzheimer's dementia reported less deficits than their informants did, and that patients who did not convert had the opposite pattern (Okonkwo et al., 2008, 2009). Patients with mild cognitive impairment have also been shown to have impaired insight into functional abilities such as driving, medication management, and financial abilities, which presents grave concerns for these patients since they are typically in the beginning stages of treatment and are, by definition, functionally independent (Okonkwo et al., 2008, 2009).

While anosognosia investigation typically focuses on memory and visuospatial domains in the MCI and dementia populations, comprehensive evaluation of levels of awareness and their relationship with actual neuropsychological performance is limited. Anosognosia is conceptualized as a discrepancy score between informant and patient predictions of performance, which is problematic in many respects and presents challenges to the validity of the conclusions produced by these studies (Derouesné et al., 1999; Okonkwo et al., 2009; Vogel et al., 2004). The current study seeks to explore the level of awareness in the mild cognitive impairment population using both informant ratings and actual neuropsychological performance as means of determining anosognosia in a comprehensive neuropsychological battery.

Mild Cognitive Impairment and Dementia

Diagnostic Criteria for Mild Cognitive Impairment and Dementia

Mild cognitive impairment is a relatively new diagnosis, and was initially created to give attention to patients who did not meet criteria for dementia but displayed clinically significant impairment that deserved focused medical care. While the MCI diagnosis has been discussed in prior literature, it was streamlined by Petersen (2004). His model proposed four subtypes of MCI that differentiate patients based on the domain(s) of impairment. The most widely researched and arguably most common subtype of MCI is the amnestic type single domain, whose hallmark symptom is impairment in memory and is thought to convert to dementia of the Alzheimer's type. In contrast, the MCI non-amnestic type single domain is characterized by impairment in a non-memory cognitive domain such as language, visuospatial skills, attention and

concentration, or executive function. This subtype of MCI is the least common and is thought to convert into Lewy Body dementia or fronto-temporal dementia. The last two subtypes of MCI both involve multiple areas of impairment, one of which includes memory impairment and one that does not. They are labeled multiple domain amnestic type and multiple domain non-amnestic type, and are thought to convert to Alzheimer's dementia and Lewy Body dementia, respectively (Petersen, 2004).

A majority of the current literature focuses on the type of MCI due to Alzheimer's dementia. These patients exhibit memory impairment but remain functionally independent, and are seen as the subclinical form of what will eventually progress into clinical Alzheimer's dementia (Albert et al., 2011). Dementia and related disorders can also be viewed along a continuum of functionality, with functionally independent MCI patients at one end of the spectrum and dementia patients on the other, such that patients that meet criteria for a diagnosis of dementia are no longer capable of functional independence (Albert et al., 2011).

While Petersen made great strides in the conceptualization and categorization of MCI and its subtypes, further exploration of the topic deemed step-by-step diagnostic criteria that would guide overall MCI diagnosis necessary. Recent efforts have been put forth to do just that in order to create a standard set of diagnostic criteria for MCI due to Alzheimer's dementia. In order to be diagnosed with MCI, a patient must meet the following four criteria: (1) Have a concern about a change in cognitive status, either from the patient, family members, caregivers, or a treating clinician, obtained through thorough clinical evaluation by a skilled clinician. If serial assessments of patients' cognitive status are available, objective measurement of the changes is helpful. (2)

Objective cognitive impairment in one or more cognitive domains, one of which being episodic memory, as obtained by formal clinical neuropsychological evaluation. Patients with MCI typically score between 1 and 1.5 standard deviations below their same age and educated peer group, with estimates of premorbid functioning taken into consideration. (3) Functional independence is the distinguishing criterion between MCI and dementia, such that MCI patients are still able to complete their activities of daily living and instrumental activities of daily living with minimal assistance. Activities of daily living include activities such as dressing, bathing, caring for personal hygiene, and cooking, whereas instrumental activities of daily living refer to higher-level activities required for independent living. Examples of instrumental activities of daily living are paying bills on time, writing checks, or shopping. Patients may demonstrate decreased efficiency on these tasks, but in order to meet criteria for an MCI diagnosis, they must be able to complete them with minimal assistance. (4) A lack of a dementia diagnosis (Albert et al., 2011; McKhann et al., 2011; Petersen, 2004).

With advances in technology come efforts at obtaining biologically driven, objective markers that are useful in determining etiology and prognosis for various disorders. As discussed briefly earlier, the area of dementia research is no exception. A complementary research paradigm for studying MCI has set forth recommendations for research protocols studying MCI in an effort to guide the field of study into a more standardized assessment across research groups (Albert et al., 2011). The authors propose that tracking beta-amyloid protein ($\alpha\beta$) and tau (τ), both of which are biological markers for Alzheimer's dementia, can help definitively reflect the progression of Alzheimer's dementia and provide support for the contention that MCI due to

Alzheimer's dementia is a predementia stage along the continuum of Alzheimer's disease (Albert et al., 2011).

Neuropsychological Profiles for Mild Cognitive Impairment and Dementia

Due to the fact that mild cognitive impairment (MCI) can be viewed as a catch-all diagnostic entity encompassing multiple types of sub-clinical dementia, the neuropsychological profiles that accompany each variant of MCI correlate closely with their dementia counterparts. Petersen (2004) proposed a stepwise dichotomous model of categorizing the variants of MCI, with the first dichotomous distinction being the presence of a memory impairment. If memory impairment is present, it follows the amnestic MCI trajectory with further classification accounting for single domain impairment or multiple domain impairment. Thus, a patient performing in the impaired range on verbal or non-verbal measures of memory and within normal limits on all other neuropsychological domains, using Petersen's model, would be diagnosed with single domain amnestic MCI. Amnestic patients demonstrating impairment in multiple domains can be seen as being closer to the dementia threshold than their single domain MCI counterparts, as more cognitive impairment is associated with lower functional independence. Of the patients along this trajectory that demonstrate further cognitive decline and eventually meet criteria for a diagnosis of dementia, they typically convert to dementia of the Alzheimer's type (Albert et al., 2011; Looi & Sachdev, 1999; Pachana, Boone, Miller, Cummings, & Berman, 1996; Petersen, 2004)

For diagnosis of patients with intact verbal and visual memory but impairment in a non-memory cognitive domain, the non-amnestic MCI trajectory is most appropriate. Non-amnestic MCI can also be further categorized into single domain or multiple domain, depending on how many impairments are seen on neuropsychological testing (Petersen, 2004). Similar to the amnestic MCI trajectory, the non-amnestic, multiple domain MCI diagnosis is best viewed as closer to the threshold for a diagnosis of dementia than its single domain counterpart, with impairments in multiple cognitive domains indicative of lower levels of functionality. Of the patients that eventually decline to the point of functional dependence, these patients may convert to one of a number of different types of dementia (Looi & Sachdev, 1999; Petersen, 2004).

Vascular dementia is the second most commonly diagnosed type of dementia, but unlike Alzheimer's dementia, it is less widely studied and lacking clear neuropsychological profiles consistently produced across studies. Current diagnostic criteria for vascular dementia include the following: (1) Impaired memory, including difficulty learning and/or recalling learned information, or the presence of aphasia, apraxia, agnosia, or impaired executive function, (2) impairment in social or occupational function, (3) focal neurological signs related to cerebrovascular disease and (4) a lack of delirium. Due to the diffuse nature of the vasculature in the brain, there is a lack of consensus regarding the typical neuropsychological profile seen in these patients, most likely due to the fact that different brain areas can be affected by cerebrovascular disease, which would lead to heterogeneity of symptoms (Garrett et al., 2004; Looi & Sachdev, 1999; McPherson & Cummings, 1996; Sachdev et al., 2004).

Many of the non-amnestic dementia diagnoses are still under investigation, and research regarding the neuropsychological profiles of each is ongoing. Many of the defining factors that contribute to a diagnosis of a non-amnestic dementia are clinical in nature, such that behavioral observation and interpretation become equally as important as neuropsychological test performance. The behavioral variant of frontotemporal dementia is often associated with cognitive impairment in one or multiple non-memory domains accompanied by dramatic changes in personality, reduced social pragmatics, and often times language impairment in the face of intact visuospatial skills (Banks & Weintraub, 2009; Neary & Snowden, 1996; Pachana et al., 1996). Lewy body dementia, which can also be seen in conjunction with Alzheimer's dementia, is associated with relatively characteristic delusions and hallucinations, with evidence for executive dysfunction and reduced visuospatial skills (Galasko, Katzman, Salmon, & Hansen, 1996; Salmon et al., 1996)

Neuroanatomical Bases of Anosognosia

While early research on anosognosia for left sided hemiplegia lent itself to a simplistic neuroanatomical explanation for the localization of anosognosia in right hemisphere function, the broadening of the anosognostic picture to include different diagnoses with diverse neuroanatomical involvement has made neuroanatomical correlations for anosognosia exceedingly complex. Indeed, the accumulation of anatomical theories used to make sense of the different neurological and neuropsychiatric populations discussed in the current study would implicate almost every gross brain area possible in a bilateral fashion. Anosognosia for hemiplegia and hemianopia would

implicate right temporal and right occipital involvement, while anosognosia for aphasia would implicate left temporal involvement. Anosognosia for deficits incurred from traumatic brain injury typically discuss right frontal or bifrontal involvement. Similarly, anosognosia in schizophrenia is usually associated with bilateral frontal functioning in the literature (Banks & Weintraub, 2009).

Given that research on anosognosia as a result of focal injury has been unsuccessful at localizing awareness, it is not surprising that research on disorders characterized by general cognitive decline such as MCI and dementia have also been unsuccessful at accounting for anosognostic symptoms in a parsimonious neuroanatomically based theory. In general, researchers have resigned to the fact that anosognosia is a complex, multifaceted neurological syndrome that is not well understood and is still in the beginning stages of research (Gilleen et al., 2010; Kertesz, 2010; McGlynn & Kaszniak, 1991; Prigatano, 1991; Prigatano & Wolf, 2010; Stuss, 1991). Several studies have, however, begun to form the foundation for scientific inquiry regarding neuroanatomical correlates of dementia. Functional imaging studies in Alzheimer's dementia patients revealed hypoperfusion in the prefrontal pathway, which includes the right prefrontal cortex, inferior parietal lobe, anterior cingulate gyrus, and limbic system. Further investigation implicated the right post-central gyrus, the right parietotemporal-occipital association cortex, and the rostral prefrontal cortex (Cutting, 1978; Prigatano & Schacter, 1991). Voxel-based morphometry imaging revealed right ventromedial prefrontal cortex involvement in patients with a mixed group of individuals with neurodegenerative disease (Rosen et al., 2010).

Neuropsychological studies have also attempted to make predictions regarding anatomical correlates of anosognosia, which not surprisingly point to frontal dysfunction (Rosen et al., 2010). In general, these studies point to the involvement of prefrontal, temporal, and limbic structures in anosognosia and Alzheimer's dementia, but the variability in protocols used across studies make generalizable conclusions difficult to obtain (Michon, Deweer, Pillon, Agid, & Dubois, 1994). It is clear that more research needs to be done on this topic in order to fully understand the neuroanatomical underpinnings of awareness. To date, a single study has attempted to use imaging to investigate the neurological correlates of awareness in the MCI population. The authors utilized functional imaging technology to quantify brain activity while the patient engaged in a self-appraisal task where they were asked to make yes or no determinations as to whether a given characteristic (e.g. calm, obnoxious) was self-descriptive. The researchers found reduced activation in cortical midline structures during tasks of selfappraisal, which they argued attested to the role of cortical midline structures in awareness of the self, and thus related to anosognosia. More research is necessary in order to come to meaningful conclusions about the relation of MCI and awareness as it relates to cortical and subcortical function (Ries et al., 2007).

Methodological Issues Related to Measuring Awareness

An integral first step towards systematic, thorough investigation of anosognosia is the development of is a robust measure of awareness. Unfortunately, no such measure currently exists in anosognosia literature. Instead, multiple measures have been implemented and none are used consistently across studies. The following is a discussion

of the current methodologies used to quantify awareness, the difficulties associated with them, and the proposed methodology used in the current study that seeks to address these issues (Fleming, Strong, & Ashton, 1996).

Clinician Diagnosis

Clinical diagnosis of the presence or absence of anosognosia is relatively simple and relies solely upon the clinician's observation of patient behavior. The more objective the facet of behavior the patient is lacking awareness of, the more straightforward the diagnosis. Thus, for diagnosis of easily observable deficits or deficits that can readily be tested bedside, such as hemiplegia and cortical blindness, this method of diagnosis is reliable and effective. As assessment moves from more concrete to more abstract domains of cognitive skills or functional activities, however, clinician diagnosis becomes less helpful simply because clinicians do not have the luxury of spending extended periods of time with patients. As the deficits for which an individual could potentially be unaware become less tangible in nature, reliable measurement becomes increasingly difficult. While it is possible that the presence or absence of a lack of awareness is more readily detectable, the level of awareness becomes more difficult to quantify (Orfei, Caltagirone, & Spalletta, 2010).

Structured interviews have been created to address the variability in information collected during clinician interviews, but still lead to inconsistent results (Cocchini et al., 2009; Simmond & J. Fleming, 2003; Trudel, Tryon, & Purdum, 1998). Clinicians, inevitably, will require the input of an informant regarding the patient's everyday level of functioning, which can be assessed in relation to the patient's perception of his/her

abilities, as a measure of overall awareness. This implies that the informant is the most accurate rater of the patient's true ability, which has not been thoroughly investigated or validated in the literature. An additional problem in this methodology is that the patient may acknowledge having difficulty in a certain area of functioning, but have no insight into when the deficit is actually occurring, thus making compensation for the area of deficit difficult. Thus, when a clinician is questioning the patient and using this as a measure of awareness, the clinician is assuming that awareness of a deficit will lead to appropriate compensation for the deficit, which is not always the case. To address this issue, Crosson and collegues (1989) proposed a theoretical framework that accounted for various facets of awareness, including intellectual awareness, or the verbal acknowledgment of a disorder, emergent awareness that becomes evident when the patient has difficulty completing a task in real time, and anticipatory awareness, in which the patient is able to appreciate and compensate for the functional impact of a deficit. These facets of awareness are difficult to account for by clinician interview alone, which may account for the discrepancy in results across studies employing various measures of awareness.

Discrepancy between Self and Informant-Reported Ability

To account for the difficulties inherent in clinician diagnosis of anosognosia, a number of rating scales have been created to assess patient levels of awareness, and parallel versions have been created to allow for corresponding ratings by an informant. The patient him/herself, an informant/caregiver, and/or a treating clinician typically complete these rating scales, and awareness on the part of the patient can be assessed in two ways. Discrepancy between patient and informant ratings can be used, as well as discrepancy between patient and clinician ratings (Crosson et al., 1989). A discrepancy score is typically created by subtracting the patient's ratings from that of the informant, the absolute value of which is used to quantify the patient's level of awareness (Evans, Sherer, Nick, Nakase-Richardson, & Yablon, 2005; Flashman & McAllister, 2002; Hart, Seignourel, & Sherer, 2009; Sherer et al., 1998).

Using this type of comparison as a measure of anosognosia implies that the rating of the informant or the clinician is unbiased and accurate, because their ratings serve as the basis of comparison for the patient ratings. Depending on the amount of interaction the informant has with the patient and how involved he or she is in the patient's care, using this method of quantifying levels of awareness is problematic. Levels of concern in relation to caregiver response bias and has not been addressed in any of the literature to date, so there is no method of accounting for how this may impact caregiver ratings. In addition, response bias and recent experience with the patient have the tendency to color the manner in which both the patient and the informant answer these measures, which pose a threat to their validity and may unjustly impact the quantity of the discrepancy seen between informant and patient scores. Also, taking absolute values of the discrepancy between patient and informant ratings does not account for both over and under estimation of ability, which artificially restricts the range of scores and can, in turn, impact the robustness of the statistical results.

Another set of issues that must be addressed when using any type of questionnaire is the measure's validity. Measures of validity in neurologic populations are difficult given their inherent cognitive compromise, and relying on informant measures of

reliability imposes the same issues of bias previously discussed in using these measures for quantifying awareness. Thus, these measures are poorly validated in general and are difficult to validate in the patient populations for which they are intended to measure. Due to the fact that patients with neurological impairments are completing these measures, it is assumed that they have the cognitive ability to understand what is being asked of them and answer the questions accurately. Given their overall cognitive deficits, this is not always the case. In addition, this is not taken into account in the literature, which can artificially impact the results derived from studies employing these methodologies.

Discrepancy between Self-Reported Ability and Actual Performance

Another way of quantifying anosognosia is by comparing patient ratings to actual neuropsychological performance, which removes the bias associated with using non-participant ratings (Fleming, Strong, & Ashton, 1996). Unfortunately, this method of quantification is less often used in the literature. The variability in rating scales employed across studies utilizing the discrepancy method, and the reliance upon potentially inaccurate informant ratings may account for the varied results and lack of consensus regarding mechanisms of action in the literature.

Barrett and collegues (2005), as discussed earlier, conducted a studying asking MCI patients to provide estimations of ability before and after a battery of tests were completed. They found that visuospatial skills were overestimated prior to testing, and memory skills were overestimated after testing. Thus, the patient's pretest and posttest ratings were used in relation to their actual performance on neuropsychological testing to

determine awareness levels. Although this methodology removes the bias associated with informant ratings, it does pose a rather pragmatic problem. Patients who have memory difficulties, as those with MCI due to Alzheimer's dementia do, may not remember the battery administered, and thus may not be able to accurately estimate their performance after the battery is completed. This is particularly problematic for measures that are earlier in the battery, due to the fact that more time has elapsed when posttest ratings were collected. Thus, the overestimation of memory complaints may be, in part, accounted for by memory ability in and of itself. In addition, memory measures are often multistep in nature, differentiating between types of memory function, such as immediate and delayed recall, as well as free recall and cued recall or recognition. Without clearly differentiating between which type of memory the evaluation is referring to, it is difficult to gain a more fine grained understanding of the specific areas of memory the patient views him/herself as having difficulty. Lastly, the ratings solicited from the patients were not tied explicitly to any given measure in the assessment battery, and were instead broad questions regarding general cognitive domains, such as memory, attention, etc. If patients are unaware of how these constructs were tested, as well as which tasks measured each cognitive domain, it may be difficult for the patient to provide accurate pretest and posttest estimations of ability. For example, the Wisconsin Card Sorting Test may not be viewed by the patient as a measure of executive function, and may be misinterpreted as a visuospatial task. Thus, they may rate themselves poorly on posttest ratings of visuospatial functioning but rate themselves better on executive function tasks. Thus, while this methodology is moving the body of research in the right direction, it still leaves room for improvement (Barrett et al., 2005).

Discrepancy between Self-Predicted, Self-Evaluated, Informant-Predicted, and Actual Performance

The current study employs a novel paradigm to assess anosognosia that incorporates aspects of the previously discussed methodologies and allows for assessment of the accuracy of each. The current study utilizes a prediction and evaluation model of assessing anosognosia in a standard neuropsychological evaluation, by obtaining participant predictions of performance a priori and evaluations of performance a posteriori before and after each test is administered. Thus, before administering each measure, the participant is read a brief explanation of the task and asked to predict how well he/she will do on each measure. Then the given task is completed. After the task is completed, the patient is asked to evaluate his/her performance on the measure on the same scale that the prediction was provided on, as a way to remind the patient how he/she thought he/she would perform prior to engaging in the task. Informant ratings for each neuropsychological measure are also obtained, using the same description of each measure given to the patient during the assessment. This will allow for investigation as to the accuracy of informant ratings in comparison to actual neuropsychological performance, as well as provide an unbiased measure of anosognosia by comparing patient prediction of performance to actual performance on neuropsychological measures. By obtaining these prediction and evaluation ratings immediately before and after each neuropsychological measure, the impact of memory of the battery is removed, as well as the ambiguity associated with trying to understand what each measure is measuring. In addition, it allows for the exploration of the possibility of emergent awareness as a result

of poor performance by obtaining evaluations of performance immediately after the measure is completed.

CHAPTER TWO

HYPOTHESES

Given the current state of the literature on anosognosia in mild cognitive impairment, the following hypotheses are proposed:

Hypothesis 1 – Patient Versus Informant Ratings

Given that anosognosia has been documented in the literature in both the MCI and dementia populations, it is hypothesized that patient and informant predictions of patient performance will be significantly different (Dekkers, Joosten-Weyn Bannignh, & Eling, 2009; Vogel et al., 2004), regardless of their diagnostic group. The magnitude of discrepancy and direction of discrepancy will serve as indicators of the degree to which anosognosia is present.

Hypothesis 2 – Accuracy of Ratings

Research has yet to compare the accuracy of patient's prediction of their own performance to that of informant predictions as they relate to true neuropsychological performance. As it stands, informant predictions are presumed to accurately reflect the patient's actual level of ability. It is hypothesized that informant predictions will be more accurate predictors of the patient's actual performance than patient predictions. In addition, informant predictions of patient ability will be more accurate for MCI patients than dementia patients. Given that dementia patients are less functional, it is likely that informants will underestimate their actual ability across domains of function that may, in fact, be less impacted by the severity of their diagnosis. Given that MCI patients are at

what could be considered a "turning point" in their cognitive function, it is likely that they will underestimate their actual ability, which can be seen as a reflection of their concern regarding their changes in cognitive status.

As discussed earlier, domain specific anosognosia has been found for memory ability (Cosentino, Metcalfe, Butterfield, & Stern, 2009). The current study also hypothesized that both patient and informant predictions will demonstrate poorer visuospatial awareness in relation to memory awareness.

Hypothesis 3 – Emergent Awareness by Cognitive Domain

Emergent awareness has not been systematically studied in formal neuropsychological assessment paradigms. The current study hypothesized that emergent awareness will vary across cognitive domains, with more emergent awareness seen for tasks allowing for physical manipulation of objects and/or verbal feedback from the examiner (i.e. Block Design, Wisconsin Card Sorting Test). It is additionally hypothesized that this emergent awareness will be greater for MCI patients than dementia patients.

Hypothesis 4 – Diagnostic Utility of Anosognosia Assessment

As previously stated, the diagnostic utility of anosognosia evaluation has yet to be researched in the field. As such, the current study hypothesizes that ratios of predicted performance relative to actual performance (anosognosia prediction ratios) will demonstrate diagnostic utility, such that diagnostic group membership can be predicted based on the anosognosia ratios generated throughout the course of the evaluation. In

addition, it is hypothesized that measures of memory will be most predictive of diagnostic group than other cognitive measures collected.

CHAPTER THREE

METHOD

Study Information

Research activity took take place in Loma Linda University Medical Center's East Campus Rehabilitation Center's Department of Neuropsychology and Rehabilitation Psychology. The data that was used for the current analysis was archived in nature, as the data is routinely collected as a part of the standard neuropsychological evaluation that Travis Fogel, Ph.D. conducts for persons with suspected dementia.

Inclusion/Exclusion Criteria

Males and females ages 18-100 who were referred to Loma Linda University Medical Center's Rehabilitation Center Department of Neuropsychology and Rehabilitation Psychology for neuropsychological evaluation were included in the sample.

Exclusion criteria were as follows: (1) Individuals younger than 18 years of age or older than 100 years of age, as normative data for the neuropsychological tests administered in the standard evaluation range from ages 18 to 100, thus precluding the evaluators from attaining standardized scores for patients falling outside that age range. (2) Patients who were not fluent in the English language were excluded due to the impact that unfamiliarity with the language may have on patient performance. (3) Patients who were hearing impaired or vision impaired were excluded due to the auditory and visual demands needed to engage in the neuropsychological evaluation in a meaningful way. (4) Lastly, patients with motor impairment in their dominant hand were also excluded due to the manual dexterity needed for stimulus manipulation and writing demands needed for a portion of the neuropsychological measures administered.

Subject Recruitment and Screening

Medical charts of patients who were referred for neuropsychological evaluation and meet inclusion/exclusion criteria were examined from Loma Linda University Medical Center's East Campus Department of Neuropsychology and Rehabilitation Psychology. The data collected were a part of the standard care practices for the Department of Neuropsychology and Rehabilitation Psychology; thus an informed consent waiver and HIPAA privacy waiver was requested.

Participant Characteristics

A total of 49 participants were included in the study, with relevant demographic characteristics presented in Table 1. As displayed in Table 2, a total of 24 participants were diagnosed with MCI and 25 participants were diagnosed with dementia using criteria outlined previously (Albert et al., 2011; McKhann et al., 2011; Petersen, 2004). Diagnoses were provided by Dr. Travis Fogel, Ph.D. and corroborated by Kyrstle Barrera, M.A. A total of 23 male and 26 female participants were included in the sample. A total of 24 patients were excluded from the sample, twenty due to unclear or complicated clinical presentations or diagnostic rulings, one from reduced effort, two due to diagnosis not warranted (i.e. "worried-well"), and one due to prominent psychiatric overlay superimposed on compromised neuropsychological function.

Table 1

	MCI		Der	Dementia		Total	
Characteristic	n	%	n	%	n	%	
Age (<i>m</i> , <i>SD</i>)	(71, 9.63)		(77,	(77, 7.85)		(74, 9.33)	
40-49	1	4.2	0	0.0	1	2.0	
50-59	1	4.2	1	4.0	2	4.1	
60-69	5	20.8	3	12.0	8	16.3	
70-79	14	58.3	11	44.0	25	51.0	
80-89	3	12.5	9	36.0	12	24.5	
90+	0	0.0	1	4.0	1	2.0	
Total	24		25		49		
Gender							
Male	13	54.2	10	40.0	23	46.9	
Female	11	45.8	15	60.0	26	53.1	
Education							
< 12 years	0	0.0	3	12.0	3	6.1	
12 years	6	25.0	5	20.0	11	22.4	
13-15 years	7	29.2	7	28.0	14	28.6	
16+ years	11	45.8	10	40.0	21	42.9	
Race							
White/Caucasian	15	62.5	20	80.0	35	71.4	
Black/African	2	8.3	2	8.0	4	8.2	
Other	7	29.2	3	12.0	10	20.4	
Current Occupation							
Not in Labor Force	20	83.3	23	92.0	43	87.8	
Unskilled Labor	0	0.0	0	0.0	0	0.0	
Semiskilled Labor	1	4.2	0	0.0	1	2.0	
Skilled Labor	0	0.0	0	0.0	0	0.0	
Managerial/Clerical/Sales	2	8.3	2	8.0	4	8.2	
Professional/Technical	1	4.2	0	0.0	1	2.0	
Previous Occupation							
Not in Labor Force	0	0.0	4	16.0	4	8.2	
Unskilled Labor	0	0.0	2	8.0	2	4.1	
Semiskilled Labor	3	12.5	1	4.0	4	8.2	
Skilled Labor	2	8.3	1	4.0	3	6.1	
Managerial/Clerical/Sales	7	29.2	7	28.0	14	28.6	
Professional/Technical	12	50.0	9	36.0	21	42.9	

Participant demographic characteristics

Table 2

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Diagnoses	n	% of Subgroup	% of Diagnosis Group	% of Sample
Mild Cognitive Impairment (MCI)				
Amnestic				
Single Domain	1	6.3	4.2	2.1
Multiple Domain	15	93.8	62.5	30.6
Total	16	100.0	66.7	32.7
Non-Amnestic				
Single Domain	3	37.5	12.5	6.1
Multiple Domain	5	62.5	20.8	10.2
Total	8	100.0	33.3	16.3
MCI Total	24		100.0	49.0
Dementia				
Alzheimer's Dementia				
Mild	12	85.7	48.0	24.5
Moderate	2	14.3	8.0	4.1
AD Total	14	100.0	56.0	28.6
Vascular Dementia				
Mild	2	66.7	8.0	4.1
Moderate	1	33.3	4.0	2.0
VD Total	3	100.0	12.0	6.1
Mixed AD/VD	6		24.0	12.2
Frontotemporal Dementia				
Nonbehavioral Variant	1	50.0	4.0	2.0
Behavioral Variant	1	50.0	4.0	2.0
FTD Total	2	100	8.0	4.1
Dementia Total	25		100.0	51.0
Total	49	100.0	100.0	100.0

Study Design

The following procedures are part of the standard of care for the Department of Neuropsychology and Rehabilitation Psychology neuropsychological evaluation procedure for patients referred to Dr. Travis Fogel, Ph.D.

Background

The patients were initially seen by their primary care physician and were referred to Dr. Travis Fogel, Ph.D. for a comprehensive neuropsychological evaluation. At the initial point of contact, a Frequently Asked Question sheet was sent to the patient (see Appendix A), which included a recommendation to bring a family member or friend to the evaluation (informant). In instances when an informant was not present, informant paperwork was given to the patient, who was instructed to have a family member or friend complete. Each evaluation took approximately 2 - 2.5 hours in its entirety, which

Table 3

Informant characteristics

	MCI		Dementia		Total	
Characteristic	n	%	n	%	n	%
Relationship to patient						
Spouse	14	58.30	13	52.00	27	55.10
Child	8	33.30	9	36.00	17	35.40
Caregiver	0	0.00	0	0.00	0	0.00
Other Relative	2	8.30	2	8.00	4	8.30
Friend	0	0.00	0	0.00	0	0.00
Declined to respond	0	0.00	1	4.00	1	2.00
Total	24	100.00	25	100.00	49	100.00
Years of acquaintance (m, SD)	(39	, 15.07)	(46	, 15.62)	(46	, 42.97)
Reported familiarity w/patient						
Very familiar (provides daily care)	23	95.80	18	72.00	41	83.70
Somewhat familiar (often cares for)	0	0.00	4	16.00	4	8.20
Not very familiar (has minimal contact)	1	4.20	0	0.00	1	2.00
Declined to respond	0	0.00	3	12.00	3	6.10
Total	24	100.00	25	100.00	49	100.00

included a 30-minute clinical interview with the patient and family member/caregiver, and a 90 to 120-minute assessment session conducted with the patient individually. Table 3 presents relevant informant characteristics.

Clinical Interview

The clinical interview typically lasted approximately 30 minutes, and Dr. Travis Fogel, Ph.D., the patient, and the informant(s) were present. Dr. Fogel interviewed the patient and informant, and gathered clinically relevant information (see Appendix B for details regarding information collected). The information collected fell within four domains: (1) demographic information (i.e. age, ethnicity, handedness, years of education, occupation, etc.), (2) history of cognitive complaints and current cognitive status, (3) personal medical/psychiatric history, and (4) family medical history. Upon completion of the clinical interview, the patient began the neuropsychological evaluation. The informant was asked to wait in the waiting room and was given two forms to complete; the patient history form and the Neuropsychiatric Inventory (see Appendix C and D, respectively). These forms were collected from the informant at the conclusion of the evaluation.

Neuropsychological Evaluation

The standard neuropsychological evaluation was approximately 90-120 minutes long. Either Dr. Fogel, Ph.D. or Kyrstle Barrera, M.A., administered the neuropsychological measures individually with the patient in a quiet, distraction-free testing office. The evaluation included administration of the following neuropsychological measures (see Appendix E):

- Modified Mini Mental Status Exam (3MS): A 100 point, global screening of cognitive function assessing temporal and spatial orientation, attention and working memory, language, immediate and delayed recall, abstract reasoning, and verbal fluency (Teng & Chui, 1987).
- Trail Making Test Form A (Trails A): A speeded measure of attention, scanning, sequencing and psychomotor speed requiring the connection of numbered circles in consecutive order (Reitan & Wolfson, 1985).
- Trail Making Test Form B (Trails B): A speeded measure of attention, scanning, sequencing, psychomotor speed, mental flexibility, and set shifting requiring the connection of numbered and lettered circles in order, alternating between consecutive numbers and letters (i.e. 1-A-2-B-3-C etc.) (Reitan & Wolfson, 1985).
- 4. Controlled Oral Word Association Test (FAS and Animals): A timed measure of phonemic and category verbal fluency requiring the generation of as many words as possible beginning with a given letter of the alphabet (F, A, and S) and as many items belonging to a given category (animals) in 60 seconds (Benton, Hasmsher, & Sivian, 1994).
- California Verbal Learning Test 2nd edition, Short Form (CVLT): List learning verbal memory test comprised of a 9-item word list containing words from three categories (fruit, clothing, and tools) tested in free recall, cued recall, recognition (CVLT Rec), and forced choice (CVLT FC) paradigms at short (CVLT IR) and long delays (CVLT DR) (Delis, Kramer, Kaplan, & Ober, 2000).
- 6. Wechsler Adult Intelligence Scale 3rd edition subtests (Wechsler, 1997)

- a. Block Design (BD): A timed measure of visuospatial and visuoconstructive skill requiring the use of bicolored blocks (red and white) to reconstruct a given picture.
- b. Digit Span (DSF, DSB): A measure of attention, concentration, and working memory requiring the repetition of verbally presented stings of numbers of increasing length in both forward and backward sequences.
- Symbol Digit Modalities Test (SDMT; written version): a timed measure of complex visual scanning, tracking, perceptual speed, divided attention, and psychomotor processing requiring the transcription of symbols into numbers given a key (Smith, 1991)
- 8. Geriatric Depression Scale (GDS): A 30-item self-report inventory assessing feelings of depression over the past week (Yesavage et al., 1983).
- 9. Wechsler Memory Scale 3rd edition subtests (Wechsler, 1997b)
 - a. Logical Memory I (LMI): A contextual verbal memory measure requiring the immediate memory of two short stories, the second of which is repeated twice
 - b. Logical Memory II (LMII): A contextual verbal memory measure requiring the memory of two short stories after a delay.
- 10. Judgment of Line Orientation (JLO): A spatial perception and orientation measure requiring the estimation of the orientation of two lines presented concurrently using a set of reference lines, similar to a protractor (Benton, Varney, & Hamsher, 1978).
- Boston Naming Test (BNT): A measure of confrontational naming requiring the naming of simple, line drawn pictures of common objects and animals (Kaplan, Goodglass, & S Weintraub, 1983).

- 12. Wechsler Test of Adult Reading (WTAR): A measure of premorbid intelligence requiring the reading and pronouncing of 50 words that have irregular grapheme-tophoneme translations (Wechsler, 1997a).
- 13. Wisconsin Card Sorting Test-64 Card Version, Live administration (WCST): A test of abstract concept formation, mental set maintenance and shifting, and the ability to utilize feedback by properly sorting a deck of 64 cards differing in the color, shape, and number of symbols (Heaton, 1993)
- 14. Dot Counting Test (DC): A timed measure of effort requiring the accurate and timely counting of randomly placed dots on cards (Boone, 2002)

Each neuropsychological assessment measure was administered according to the standard administration procedures outlined by its respective publisher. Routinely before the administration of each measure, the examiner verbally presented a brief overview of the upcoming measure and asked the patient to predict how well he/she would do on the measure (see Appendix F for script). The patient was given a blue pen and a scale with "Extremely Well" at the top with a smiling face and "Extremely Poorly" at the bottom with a frowning face (see Appendix G for sample scales). The patient was asked to base his/her prediction in comparison to his/her same aged and educated peers, and draw a line on the scale where he/she expected his/her performance to fall. Once the measure was administered, the same scale was presented to the patient, which now contained his/her performance prediction. The patient was given a green pen and asked to draw a line indicating an evaluation of how well he/she actually performed. Different colored pens were used to differentiate between the two (prediction and evaluation) values. In addition, the patient was asked how concerned he/she was about his/her cognitive ability, as well

as how concerned his/her family was about his/her cognitive ability. The patient provided this assessment on a similar scale, with "Very Concerned" at the top and "Not Concerned" at the bottom. The patient provided this assessment both before the assessment process began and after the entire assessment process was concluded. At the conclusion of the session, the patient was also asked to provide a rating of how well he/she thinks he/she performed on the measures administered as a whole using the same scale used for the individual assessment measures.

At the conclusion of the evaluation, the patient was informed that the information gleaned from the interview, neuropsychological evaluation, and forms completed by the informant would be used to compile a chart note, which would be included into the patient's medical record. They were informed that the information was available for review by their referring physician and would be discussed with the patient at their next follow up appointment, or should the patient desire, via a feedback session with Travis Fogel, PhD.

Informant Data Collected

Within the patient history form (see Appendix C), informant predictions of patient performance were collected. Ratings were provided along a 10-point Likert scale such that lower values indicated less favorable predictions (i.e. 1 = "Relatively Poorly) and higher values indicated more favorable predictions (i.e. 10 = "Relatively Well"). Items corresponded exactly to the predictions collected from the patients for each neuropsychological measure and relevant subsections of measures.

Variables Captured

Basic demographic information was obtained, as well as relevant historical information and psychiatric symptomatology, as provided by the informant. Ratings of concern regarding patient's cognitive ability on behalf of the family and the patient, from the perspective of the patient, was obtained before and after the assessment battery was administered. Patient predictions of performance and evaluations of performance were obtained before and after each neuropsychological assessment measure, as well as at different steps on multistep measures when appropriate (see Appendix F for a complete list of neuropsychological prediction and evaluation measures collected and the script used to solicit ratings). Actual neuropsychological performance scores were obtained on each neuropsychological measure administered.

CHAPTER FOUR

STATISTICAL ANALYSIS

Data Preparation

Prediction and Evaluation Percentages

Prediction and evaluation scores provided by the patient were quantified by measuring the distance from the bottom of the scale to the response point, rounded down to the nearest quarter of an inch. Each value was then divided by the total length of the scale, and then multiplied by 100 to produce a percentage value of each response, such that:

For example, a prediction response for the Judgment of Line Orientation placed in the center of the scale, or a response 6 cm from the bottom of a 12 cm scale, would result in the following percentage:

Prediction Percentage =
$$\frac{6}{12}$$
 X 100 = 50.00

Of note, prediction values were also calculated for informant predictions, using the same mathematical calculation, save for the fact that the scales used for informant predictions were based on a 10 point Likert scale, with larger values indicated more favorable predictions. Since informants were not actually present for the neuropsychological evaluation, evaluation of performance responses were not collected.

Actual Performance Percentages

Actual performance percentages were calculated using the observed raw score value for each measure. These scores were then divided by the total possible raw score and multiplied by 100 to produce a percentage value for each measure, such that:

For example, a raw score of 25 on the Judgment of Line Orientation, which has a total of 30 items, would result in the following percentage:

 $\begin{array}{rcl} \text{Actual Performance} \\ \text{Percentage} \end{array} = \begin{array}{rcl} \frac{25}{30} & \text{X} & 100 \end{array} = 83.33 \end{array}$

Anosognosia Prediction and Evaluation Ratios

Anosognosia ratios were calculated by subtracting the predication or evaluation percentage from the actual performance percentage, and dividing by the sum of the two values (as seen in Barrett et al., 2005), such that:

Given the prediction percentage and actual performance percentage for the two examples above, the anosognosia prediction ratio would be calculated as follows:

 $\frac{\text{Anosognosia}}{\text{Prediction Ratio}} = \frac{50.00 - 83.33}{50 + 83.33} = -0.25$

This analysis allows for a range between -1 and +1, thus accounting for both overestimations and underestimations of performance. As such, values closer to 0 indicate perfect awareness of actual performance, such that prediction or evaluation values are commensurate with actual performance. Negative values indicate under prediction or evaluation of performance relative to actual performance, with increased discrepancy as values near -1. Positive values indicate over prediction or evaluation of performance relative to actual performancy as values near +1. As such, this also takes into account the actual cognitive ability of the patient.

Power Analysis

Given the number of data points being collected on each patient, the required sample size in order to obtain the appropriate power to detect a meaningful difference in ratings with a medium effect size (Cohen's d = 0.05, α =0.05, β =0.8), is a total of 30 participants.

Missing Data

The neuropsychological assessments were clinical in nature, conducted by Kyrstle Barrera, M.A., or Travis Fogel, Ph.D., and were typically unrestrained by time. Thus, the only reason a patient would be missing neuropsychological test data would be due to their inability to perform the measure. This was most commonly seen with the Judgment of Line Orientation test, which requires the patient to estimate the orientation of lines based on a reference set, similar to estimating angle orientation given a protractor as a reference. In order to keep these patients in the analysis, the lowest possible raw score was inputted (i.e. a score of zero).

Data Cleaning

Data was cleaned to ensure that the data meet the assumptions required to complete multivariate analysis of covariance (MANCOVA) and logistic regression analyses. A wide range of abilities was expected given the nature of mild cognitive impairment and mild dementia. As such, data that could represent the wide range of ability seen in these diagnoses was not removed from the sample. Normality was assessed using boxplots and histograms. Scatterplots and Pearson's *R* were used to assess linearity of the dependent variables. Lastly, homogeneity of variance and covariance were assessed using Box's *M*. Literature regarding significant *Box's M* results indicate that MANCOVA analyses are robust to violations of the assumption of homogeneity of variance when sample sizes are equal between comparison groups (Field, 2005). As such, in instances when Box's *M* values were significant, data interpretation continued, given that data was deemed normally distributed via the ancillary measures employed.

Data Analyses

Data was compiled using SPSS version 19 for the Macintosh operating system (Mac OSX) and analyzed using SPSS version 20.

Hypothesis 1 – Patient Versus Informant Ratings

In order to investigate the effect of information source on predictions of patient performance, a one way multivariate analysis of covariance was performed using information source as the independent variable with two levels, patient and informant prediction percentages across all 20 neuropsychological measures as the dependent variable. Pillai's trace results were analyzed and the effect of age was removed by using age as a covariate in the analyses. It is important to note that this analysis is unadjusted for actual performance.

Hypothesis 2 – Accuracy of Ratings

In order to investigate the accuracy of information source and diagnosis on predictions of patient performance, a two-way multivariate analysis of covariance was performed using two independent variables: information source with three levels (patient prediction percentage, informant prediction percentage, and actual performance percentage) and diagnosis with two levels (MCI and dementia). Dependent variables included prediction percentages for all 20 neuropsychological measures. Pillai's trace results were analyzed and the effect of age was removed by using age as a covariate in the analyses. It is important to note that this analysis is unadjusted for actual performance.

Hypothesis 3 – Emergent Awareness by Cognitive Domain

In order to investigate the presence of emergent awareness by diagnosis, a doubly multivariate analysis of covariance was performed using diagnosis group as the betweensubjects independent variable with two levels (MCI and dementia) and time as the
within-subjects independent variable with two time points (prediction and evaluation). Dependent variables included prediction and evaluation anosognosia ratios all 20 neuropsychological measures. Pillai's trace results were analyzed and the effect of age was removed by using age as a covariate in the analyses. It is important to note that this analysis is adjusted for actual performance by incorporating actual performance in the anosognosia ratios for each measure.

Hypothesis 4 – Diagnostic Utility of Anosognosia Assessment

In order to assess the diagnostic utility of anosognosia assessment, a logistic regression was used on patient prediction anosognosia ratios across all 20 neuropsychological measures to predict diagnostic group membership (MCI versus dementia). Anosognosia ratios were multiplied by 100 to adjust the data range, changing the range from -1 to +1 to -100 to +100 to account for the impact of restricted range on the odds ratio results and interpretability. Initially an exploratory logistic regression was run by using the Enter method in SPSS, to see if predictive anosognosia ratios, as a group, reliably predicted diagnostic group membership. Then, a forward logistic regression was run to elucidate which combination of anosognosia ratios were reliably predictive of diagnostic group membership.

CHAPTER FIVE

RESULTS

Hypothesis 1 – Patient Versus Informant Ratings

General Cognitive Ability, Premorbid Function, and Effort

A one-way MANCOVA was conducted to determine the effect of information source (patient vs. informant) on predictions of patient performance in the domain of general cognitive ability, premorbid function, and effort, including the 3MS, WTAR, and DC, while controlling for age (see Table 4 and Figures 1-3). The main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.441, F(3,79)=20.799, p=.000, multivariate $\eta^2=.441$). The covariate did not significantly influence the combined DV (Pillai's Trace=.033, F(3,79)=.910, p=.440, multivariate $\eta^2=.033$). Multivariate results indicated that both 3MS predictions and DC predictions were significantly effected by information source (F(1,81)=29.022, p=.000, multivariate $\eta^2=.264$; F(1,81)=8.090, p=.006, multivariate $\eta^2=.091$, respectively), while the WTAR predictions were not (F(1,81)=.560, p=.456, multivariate $\eta^2=.007$). Comparison of prediction means indicated that patients provided more favorable predictions than informants on the 3MS, while patients provided less favorable predictions than informants on the DC.

Attention, Concentration, and Processing Speed

A one-way MANCOVA was conducted to determine the effect of information source (patient vs. informant) on predictions of patient performance in the domain of attention, concentration, and processing speed, including Trails A, DSF, DSB, and the SDMT, while controlling for age (see Table 5 and Figures 4-7). The main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.216, F(4,85)=5.843, p=.000, multivariate $\eta^2=.216$). The covariate did not significantly influence the combined DV (Pillai's Trace=.079, F(4,85)=1.811, p=.134, multivariate $\eta^2=.079$). Multivariate results indicated that Trails A predictions were significantly effected by information source (F(1,88)=13.560, p=.000, multivariate $\eta^2=.134$), while DSF, DSB, and SDMT predictions were not (F(1,88)=2.876, p=.093, multivariate $\eta^2=.032$; F(1,88)=.064, p=.801, multivariate $\eta^2=.001$; F(1,88)=.089, p=.767, $\eta^2=.001$). Comparison of prediction means indicated that patients provided more favorable predictions than informants on Trails A.

Table 4

Mean scores and standard deviations for the domain of general cognitive ability, premorbid function, and effort for patient and informant predictions of performance percentages, and multivariate analysis of covariance

Information		Predicti	on	MA	NCOVA	1
Source	n	Mean	SD	<i>F</i> (1,81)	р	η^2
3MS				29.02	0.000	0.26
Patient	45	63.03	22.46			
Informant	39	39.74	16.46			
Total	84	52.22	22.98			
WTAR				0.56	0.456	0.01
Patient	45	43.04	27.89			
Informant	39	38.97	20.36			
Total	84	41.15	24.62			
DC				8.09	0.006	0.09
Patient	45	38.27	22.78			
Informant	39	52.56	22.91			
Total	84	44.90	23.81			
Note: $F(3,79)=2$	0.79	9, <i>p</i> =.00	$0, \eta^2 = .441$			



Figure 1. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Modified Mini Mental Status Examination (3MS)



Figure 2. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Wechsler Test of Adult Reading (WTAR)



Figure 3. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Dot Counting Test (DC)

Mean scores and standard deviations for the domain of attention, concentration, and processing speed for patient and informant predictions of performance percentages, and associated multivariate analysis of covariance

Information		Predicti	on		Μ	1	
Source	n	Mean	SD	-	<i>F</i> (1,88)	р	η^2
Trails A					13.56	0.000	0.13
Patient	49	57.47	21.89				
Informant	42	41.19	20.02				
Total	91	49.96	22.47				
DSF					2.88	0.093	0.03
Patient	49	39.89	22.62				
Informant	42	32.62	17.95				
Total	91	36.54	20.81				
DSB					0.06	0.801	0.00
Patient	49	26.44	22.26				
Informant	42	27.62	19.10				
Total	91	26.98	20.76				
SDMT					0.09	0.767	0.00
Patient	49	37.15	23.14				
Informant	42	35.71	21.43				
Total	91	36.49	22.26				
Note: $E(1.95) = 5$	5 9/2	n = 0.00	$m^2 - 216$				

Note: F(4,85)=5.843, p=.000, $\eta^2=.216$



Figure 4. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Trail Making Test Part A (Trails A)



Figure 5. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for Digit Span Forward (DSF)



Figure 6. Patient and informant prediction of performance percentages and actual performance percentage $(\pm SD)$ for MCI and dementia patients for Digit Span Backward (DSB)



Figure 7. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Symbol Digit Modalities Test (SDMT)

Language

A one-way MANCOVA was conducted to determine the effect of information source (patient vs. informant) on predictions of patient performance in the domain of language, including FAS, Animals, and the BNT, while controlling for age (see Table 6 and Figures 8-10). The main effect of information source did not indicated a significant effect on the combined DV (Pillai's Trace=.041, F(3,87)=1.241, p=.300, multivariate $\eta^2=.041$).

Table 6

Mean scores and standard deviations for the domain of language for patient and informant predictions of performance percentages, and associated multivariate analysis of covariance

Information		Predicti	ion	MA	NCOVA	4
Source	п	Mean	SD	<i>F</i> (1,92)	р	η^2
FAS				0.06	0.808	0.00
Patient	48	48.05	21.75			
Informant	44	47.05	18.25			
Total	92	47.57	20.05			
Animals				0.81	0.370	0.01
Patient	48	48.00	22.19			
Informant	44	52.27	23.61			
Total	92	50.04	22.86			
BNT				2.42	0.123	0.03
Patient	48	54.42	23.17			
Informant	44	62.05	23.78			
Total	92	58.07	23.65			
Note: $E(2.97) - 1$	241	n = 200	$m^2 - 0.41$			

Note: F(3,87)=1.241, p=.300, $\eta^2=.041$



Figure 8. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for FAS



Figure 9. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for Animals



Figure 10. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Boston Naming Test (BNT)

Visuoperception and Visuoconstruction

A one-way MANCOVA was conducted to determine the effect of information source (patient vs. informant) on predictions of patient performance in the domain of visuoperception and visuoconstruction, including the JLO and BD, while controlling for age (see Table 7 and Figures 11-12). The main effect of information source did not indicate a significant effect on the combined DV (Pillai's Trace=.08, F(2,88)=2.692, p=.073, multivariate $\eta^2=.058$).

Memory

A one-way MANCOVA was conducted to determine the effect of information source (patient vs. informant) on predictions of patient performance in the domain of memory, including the CVLT IR, CVLT DR, CVLT Rec, CVLT FC, LMI and LMII, while controlling for age (see Table 8 and Figures 13-18). The main effect of information

Mean scores and standard deviations for the domain of visuoperception and visuoconstruction for patient and informant predictions of performance percentages, and associated multivariate analysis of covariance

Information	-	Predicti	on	MA	Ι	
Source	п	Mean	SD	<i>F</i> (1,89)	р	η^2
JLO				1.50	0.224	0.02
Patient	49	49.81	24.76			
Informant	43	43.49	24.48			
Total	92	46.85	24.70			
BD				1.32	0.254	0.01
Patient	49	44.89	23.93			
Informant	43	50.47	22.25			
Total	92	47.50	23.20			

Note: F(2,88)=2.692, p=.073, $\eta^2=.058$



Figure 11. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Judgment of Line

Orientation (JLO)



Figure 12. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for Block Design

source indicated a significant effect on the combined DV (Pillai's Trace=.252, F(6,77)=4.321, p=.001, multivariate $\eta^2=.252$). The covariate did not significantly influence the combined DV (Pillai's Trace=.110, F(6,77)=1.590, p=.162, multivariate $\eta^2=.110$). Multivariate results indicated that the CVLT FC and LMII predictions were significantly effected by information source (F(1,82)=6.039, p=.016, multivariate $\eta^2=.069$; F(1,82)=3.999, p=.049, multivariate $\eta^2=.047$, respectively), while CVLT IR, CVLT DR, CVLT Rec, and LMII predictions were not (F(1,82)=.764, p=.385, multivariate $\eta^2=.009$; F(1,82)=.047, p=.829, multivariate $\eta^2=.001$; F(1,82)=.026, p=.872, multivariate $\eta^2=.000$; F(1,82)=3.218, p=.077, multivariate $\eta^2=.038$, respectively). Comparison of prediction means indicated that patients provided more favorable predictions than informants on CVLT FC, but less favorable predictions than informants on LMII.

Information		Predicti	on	MA	NCOVA	A
Source	n	Mean	SD	<i>F</i> (1,82)	р	η^2
CVLT-IR				0.76	0.385	0.01
Patient	48	29.29	20.01			
Informant	37	33.24	19.30			
Total	85	31.01	19.68			
CVLT-DR				0.05	0.829	0.00
Patient	48	24.75	22.10			
Informant	37	23.78	16.39			
Total	85	24.33	19.71			
CVLT-Rec				0.03	0.872	0.00
Patient	48	33.60	24.67			
Informant	37	32.70	20.09			
Total	85	33.21	22.67			
CVLT-FC				6.04	0.016	0.07
Patient	48	46.96	23.82			
Informant	37	35.68	17.72			
Total	85	42.05	21.99			
LMI				3.22	0.077	0.04
Patient	48	44.46	23.37			
Informant	37	36.22	17.22			
Total	85	40.87	21.20			
LMII				4.00	0.049	0.05
Patient	48	21.13	20.24			
Informant	37	29.46	16.99			
Total	85	24.75	19.24			
Note: $E((77))$	1 201	001	2 252			

Mean scores and standard deviations for the domain of memory for patient and informant predictions of performance percentages, and associated multivariate analysis of covariance

Note: F(6,77)=4.321, p=.001, $\eta^2=.252$



Figure 13. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the California Verbal Learning Test Immediate Recall (CVLT IR)



Figure 14. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the California Verbal Learning Test Delayed Recall (CVLT DR)



Figure 15. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the California Verbal Learning Test Recognition (CVLT Rec)



Figure 16. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the California Verbal Learning Test Forced Choice Recognition (CVLT FC)



Figure 17. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for Logical Memory I (LMI)



Figure 18. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for Logical Memory II (LMII)

Executive Function

A one-way MANCOVA was conducted to determine the effect of information

source (patient vs. informant) on predictions of patient performance in the domain of

executive function, including the WCST and Trails B, while controlling for age (see Table 9 and Figures 19-20). The main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.156, F(2,88)=8.147, p=.001, multivariate η^2 =.156). The covariate did not significantly influence the combined DV (Pillai's Trace=.004, F(2,88)=.182, p=.834, multivariate η^2 =.004). Multivariate results indicated that both the WCST and Trails B predictions were significantly effected by information source (F(1,92)=616.384, p=.000, multivariate η^2 =.155; F(1,92)=4.845, p=.030, multivariate η^2 =.052, respectively). Comparison of prediction means indicated that patients provided more favorable predictions than informants on both the WCST and Trails B.

Table 9

Mean scores and standard deviations for the domain of executive function for patient and informant predictions of performance percentages, and associated multivariate analysis of covariance

Information		Predicti	ion	MA	NCOVA	OVA	
Source	n	Mean	SD	<i>F</i> (1,92)	р	η^2	
WCST				16.38	0.000	0.16	
Patient	49	50.37	24.92				
Informant	43	30.93	20.21				
Total	92	41.28	24.72				
Trails B				4.85	0.030	0.05	
Patient	49	45.80	26.42				
Informant	43	34.88	19.93				
Total	92	40.70	24.11				
$\mathbf{N}_{\text{stars}} = \mathbf{E} \left(2.99 \right)$	0 1		1 .2 15	C			

Note: F=(2,88)=8.147, p=.001, $\eta^2=.156$



Figure 19. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Wisconsin Card Sorting Test (WCST)



Figure 20. Patient and informant prediction of performance percentages and actual performance percentage (\pm SD) for MCI and dementia patients for the Trail Making Test Part B (Trails B)

Hypothesis 2 – Accuracy of Ratings

General Cognitive Ability, Premorbid Function, and Effort

A two-way MANCOVA was conducted to determine the effect of information source (patient vs. informant vs. actual performance) and diagnosis group (MCI vs. dementia) on predictions of patient performance (or actual performance, when applicable) in the domain of general cognitive ability, premorbid function, and effort, including the 3MS, WTAR, and DC, while controlling for age (see Table 10 and Figures 1-3). As presented in Table 11, the main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.74, F(6,238)=23.39, p=.000, $\eta^2=.37$). The main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.09, F(3,118)=3.69, p=.014, $\eta^2=.09$). The interaction effect between information source and diagnosis group also indicated a significant effect on the combined DV (Pillai's Trace=.18, F(6,238)=3.93, p=.001, $\eta^2=.09$). The covariate did not significantly influence the combined DV (Pillai's Trace=.03, F(3,118)=1.15, p=.333, multivariate $\eta^2=.03$).

Univariate ANOVA results are presented in Table 12. Results indicated that all three DVs (3MS, WTAR, and DC) predictions/performances were significantly effected by information source (F(2,120)=52.18, p=.000, partial $\eta^2=.47$; F(2,120)=6.86, p=.002, partial $\eta^2=.010$; F(2,120)=21.33, p=.000, partial $\eta^2=.026$, respectively). Results indicated that the 3MS and DC were also significantly effected by diagnosis group (F(1,120)=5.06, p=.026, partial $\eta^2=.04$; F(1,120)=10.05, p=.002, partial $\eta^2=.08$, respectively), but the WTAR was not (F(1,120)=1.47, p=.227, partial $\eta^2=.01$). In addition, the interaction effect between information source and diagnosis group was significant for the 3MS

Mean scores and standard deviations for the domain of general cognitive ability, premorbid function, and effort for patient and informant predictions of performance percentages and actual performance percentages for MCI and dementia patients

		Information Source							
Diagnosis		Patier	nt			Informa	nt		
Group	n	Mean	SD		п	Mean	SD		
3MS									
MCI	22	55.58	19.87		19	45.79	17.74		
Dementia	23	70.16	22.88		20	34.00	13.14		
Total	45	63.03	22.46		39	39.74	16.46		
WTAR									
MCI	22	39.96	28.30		19	43.68	23.62		
Dementia	23	45.99	27.79		20	34.50	16.05		
Total	45	43.04	27.89		39	38.97	20.36		
DC									
MCI	22	39.27	22.17		19	62.11	21.49		
Dementia	23	37.31	23.80		20	43.50	20.84		
Total	45	38.27	22.78		39	52.56	22.91		
	I	Performa	ance			Total			
	n	Mean	SD		п	Mean	SD		
3MS									
MCI	21	86.33	6.94		62	63.00	23.33		
Dementia	22	68.14	14.26		65	58.35	23.80		
Total	43	77.02	14.47		127	60.62	23.59		
WTAR									
MCI	21	62.86	16.45		62	48.86	25.17		
Dementia	22	50.82	21.32		65	44.09	23.17		
Total	43	56.70	19.83		127	46.42	24.19		
DC									
MCI	21	73.37	7.09		62	57.82	23.13		
Dementia	22	59.80	20.99		65	46.83	23.71		
Total	43	66.43	17.07		127	52.19	23.98		

Multivariate analysis of covariance for the domain of cognitive ability, premorbid function, and effort for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

			Hypothesis	Error		
Effect	Value	F	df	$d\!f$	Sig.	η^2
Age	0.03	1.15	3	118	0.333	0.03
Source	0.74	23.39	6	238	0.000	0.37
Diagnosis	0.09	3.69	3	118	0.014	0.09
Source * Diagnosis	0.18	3.93	6	238	0.001	0.09

Table 12

Univariate analysis of covariance for the domain of general cognitive ability, premorbid function, and effort for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

	Main Effect of Source		Main . Dia	Main Effect of Diagnosis			Source x Diagnosis Interaction		
Measure	F(2, 120)	р	η^2	<i>F</i> (1,120)	р	η^2	F(2,120)	р	η^2
3MS	52.18	.000	0.47	5.06	.026	0.04	12.08	.00 0	0.17
WTAR	6.86	.002	0.10	1.47	.227	0.01	1.95	.14 7	0.03
DC	21.33	.000	0.26	10.05	.002	0.08	1.88	.15 7	0.03

Note: *ABC*= Significant Multivariate Analyses

 $(F(2,120)=6.86, p=.000, \text{ partial } \eta^2=.017)$ but not the WTAR or DC $(F(2,120)=1.95, \eta^2=.017)$

p=.147, partial $\eta^2=.010$; F(2,120)=1.88, p=.157, partial $\eta^2=.03$, respectively).

Pairwise comparison data are presented in Table 13. Comparison of

prediction/performance means for the 3MS indicated that both patients and informants

predicted lower performance than the patients actually performed, informants predicted lower performance for dementia patients than MCI patients, and dementia patients performed worse than MCI patients. In general, predictions/performances were lower across all sources of information for the dementia group than the MCI group. Comparison of prediction/performance means for the WTAR indicated that informants and patients provided similar predictions of performance, and both predicted lower performance than patients actually performed. In addition, informants predicted that dementia patients would perform worse than MCI patients. Comparison of prediction/performance means for the DC indicated that informants and patients predicted that patients would perform worse than they actually did, with patients predicting lower performance than informants

Table 13

percentages for MCI and dementia patients									
Sour	rce	Diagno	sis	Source x Diagnosis					
	p		р		р				
3MS									
Pt x Inf	0.000	MCI x Dem	0.026	Pt x Diag	ns				
Pt x AP	0.000			Inf x Diag	0.004				
Inf x AP	0.000			AP x Diag	0.000				
WTAR									
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns				
Pt x AP	0.017			Inf x Diag	0.047				
Inf x AP	0.002			AP x Diag	ns				
DC									
Pt x Inf	0.004	MCI x Dem	0.002	Pt x Diag	ns				
Pt x AP	0.000			Inf x Diag	0.014				
Inf X AP	0.008			AP x Diag	0.010				

Pairwise comparisons for the domain of general cognitive ability, premorbid function, and effort for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

Note: Pt= Patient, Inf = Informant, AP = Actual Performance, Dem = Dementia, Diag = Diagnosis, *ABC*= Significant Multivariate Analyses, *ABC*= Interpretable Pairwise Comparisons

did. Informants predicted than dementia patients would perform worse than MCI patients, and dementia patients did, in fact, perform worse than MCI patients.

Attention, Concentration, and Processing Speed

A two-way MANCOVA was conducted to determine the effect of information source (patient vs. informant vs. actual performance) and diagnosis group (MCI vs. dementia) on predictions of patient performance (or actual performance, when applicable) in the domain of attention, concentration, and processing speed, including Trails A, DSF, DSB, and the SDMT, while controlling for age (see Table 14 and Figures 4-7). As presented in Table 15, the main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.38, F(8,262)=7.71, p=.000, $\eta^2=.19$). The main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.13, F(4,130)=4.79, p=.00001, $\eta^2=.13$). The interaction effect between information source and diagnosis group also indicated a significant effect on the combined DV (Pillai's Trace=.11, F(8,262)=1.98, p=.049, $\eta^2=.06$). The covariate did not significantly influence the combined DV (Pillai's Trace=.05, F(4,130)=1.74, p=.146, multivariate $\eta^2=.05$).

Univariate ANOVA results are presented in Table 16. They indicated that Trails A and DSF predictions/performances were significantly effected by information source $(F(2,133)=16.77, p=.000, \text{ partial } \eta^2=.20; F(2,133)=11.47, p=.000, \text{ partial } \eta^2=.15,$ respectively) while DSB and SDMT were not $(F(2,133)=.36, p=.696, \text{ partial } \eta^2=.01;$

Mean scores and standard deviations for the domain of attention, concentration, and processing speed for patient and informant predictions of performance percentages and actual performance percentages for MCI and dementia patients

			Inf	formation So	ource	;	
Diagnosis		Patier	nt			Informa	nt
Group	п	Mean	SD		n	Mean	SD
Trails A							
MCI	24	56.80	23.84		22	47.27	19.56
Dementia	25	58.12	20.32		20	34.50	18.77
Total	49	57.47	21.89		42	41.19	20.02
DSF							
MCI	24	37.46	24.63		22	38.18	20.85
Dementia	25	42.23	20.73		20	26.50	11.82
Total	49	39.89	22.62		42	32.62	17.95
DSB							
MCI	24	23.30	19.42		22	30.91	18.49
Dementia	25	29.45	24.71		20	24.00	19.57
Total	49	26.44	22.26		42	27.62	19.10
SDMT							
MCI	24	37.28	24.88		22	40.91	23.08
Dementia	25	37.02	21.87		20	30.00	18.35
Total	49	37.15	23.14		42	35.71	21.43
	I	Performance				Total	
	п	Mean	SD		п	Mean	SD
Trails A							
MCI	24	81.23	7.03		70	62.18	23.01
Dementia	25	54.45	34.46		70	50.06	27.47
Total	49	67.56	28.29]	140	56.12	25.97
DSF							
MCI	24	54.69	16.51		70	43.59	22.15
Dementia	25	47.00	12.38		70	39.44	17.70
Total	49	50.77	14.91]	140	41.52	20.09
DSB							
MCI	24	35.42	14.82		70	29.84	18.14
Dementia	25	23.71	13.63		70	25.84	19.74
Total	49	29.45	15.27]	140	27.84	19.00
SDMT							
MCI	24	51.88	18.42		70	43.42	22.83
Dementia	25	24.60	18.38		70	30.58	20.12
Total	49	37.96	22.83	1	140	37.00	22.39

actual performance p	actual performance percentages for MCI and dementia patients												
Effect	Value	F	Hypothesis df	Error df	Sig.	η^2							
Age	0.05	1.74	4	130	0.146	0.05							
Source	0.38	7.71	8	262	0.000	0.19							
Diagnosis	0.13	4.79	4	130	0.001	0.13							
Source * Diagnosis	0.11	1.98	8	262	0.049	0.06							

Multivariate analysis of covariance for the domain of attention, concentration, and processing speed for patient and informant prediction percentages and

F(2,133)=.20, p=.821, partial $\eta^2=.00$, respectively). Results indicated that the Trails A and SDMT were significantly effected by diagnosis group (F(1,133)=15.05, p=.000,partial η^2 =.10, F(1,133)=11.97, p=.001, partial η^2 =.08, respectively), while DSF and DSB were not $(F(1,133)=1.73, p=.191, \text{ partial } \eta^2=.01; F(1,133)=.92, p=.340, \text{ partial } \eta^2=.01,$ respectively). In addition, the interaction effect between information source and diagnosis group was significant for Trails A and SDMT (F(2,133)=4.92, p=.009, partial $\eta^2=.07$; $F(2,133)=5.09, p=007, \eta^2=.07)$ but not DSF or DSB (F(2,133)=2.50, p=.86, partial $\eta^2=.04$, F(2,133)=2.94, p=.056, partial $\eta^2=.04$, respectively).

Pairwise comparison data are presented in Table 17. Comparison of prediction/performance means for Trails A indicated that patients provided less favorable predictions than informants, and that informants predicted that patients would perform worse than they actually did. In general, predictions/performances were lower across all sources of information for the dementia group than the MCI group. In addition, informants predicted that dementia patients would perform worse than MCI patients, and dementia patients actually did perform worse than MCI patients. Comparison of prediction/performance means for DSF indicated that patients and informants provided

processing	ce percenta	r pail iges for	MCI and	d dementia p	oatients	s S	percentages	апа	асниан
	Main S	Effect ource	of	Main Dia	Effect Ignosis	of	Source : Inte	x Diag ractio	nosis n
	<i>F</i> (2,133			<i>F</i> (1,133	0		<i>F</i> (2,133		
Measure)	p	η^2)	p	η^2)	р	η^2
Trails A	16.77	.000	0.20	15.05	.000	0.10	4.92	.009	0.07

1.73

0.92

11.97

.191

.340

.001

0.01

0.01

0.08

2.50

2.94

5.09

.086

.056

.007

0.04

0.04

0.07

Univariate analysis of covariance for the domain of attention, concentration, and processing speed for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

Note: ABC= Significant Multivariate Analyses

.000

.696

.821

0.15

0.01

0.00

11.47

0.36

0.20

Table 17

DSF

DSB

SDMT

Pairwise comparisons for the domain of attention, concentration, and processing speed for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

Sour	rce	Diagno	osis	Source x Di	agnosis
	р		р		р
Trails A					
Pt x Inf	0.002	MCI x Dem	0.000	Pt x Diag	ns
Pt x AP	ns			Inf x Diag	0.018
Inf x AP	0.000			AP x Diag	0.001
DSF					
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns
Pt x AP	0.012			Inf x Diag	0.033
Inf x AP	0.000			AP x Diag	0.046
DSB				-	
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns
Pt x AP	ns			Inf x Diag	ns
Inf x AP	ns			AP x Diag	0.006
SDMT				-	
Pt x Inf	ns	MCI x Dem	0.001	Pt x Diag	ns
Pt x AP	ns			Inf x Diag	ns
Inf x AP	ns			AP x Diag	0.000

Note: Pt= Patient, Inf = Informant, AP = Actual Performance, Dem = Dementia, Diag = Diagnosis, ABC= Significant Multivariate Analyses, ABC= Interpretable Pairwise Comparisons

similar predictions, and both predicted that patients would perform worse than they actually did. Comparison of prediction/performance means for SDMT indicated that, in general, predictions/performances were lower for the dementia group than the MCI group. In addition, dementia patients actually did perform worse than MCI patients.

Language

A two-way MANCOVA was conducted to determine the effect of information source (patient vs. informant vs. actual performance) and diagnosis group (MCI vs. dementia) on predictions of patient performance (or actual performance, when applicable) in the domain of language, including FAS, Animals, and the BNT, while controlling for age (see Table 18 and Figures 8-10). As presented in Table 19, the main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.26, F(6,266)=6.51, p=.000, $\eta^2=.13$). The main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.07, F(3,132)=3.29, p=.023, $\eta^2=.07$). The interaction effect between information source and diagnosis group did not indicate a significant effect on the combined DV (Pillai's Trace=.08, F(6,266)=1.89, p=.083, $\eta^2=.04$). The covariate did not significantly influence the combined DV (Pillai's Trace=.01, F(4,130)=1.74, p=.146, multivariate $\eta^2=.05$).

Univariate ANOVA results are presented in Table 20. The results indicated that Animals and BNT predictions/performances were significantly effected by information source (F(2,134)=4.80, p=.010, partial $\eta^2=.07$; F(2,134)=6.93, p=.001, partial $\eta^2=.09$, respectively) while FAS was not (F(2,132)=.46, p=.632, partial $\eta^2=.01$). Results indicated that Animals and BNT predictions/performances were significantly effected by diagnosis

Mean scores and standard deviations for the domain of language for patient and informant predictions of performance percentages and actual performance percentages for MCI and dementia patients

	Information Source								
Diagnosis	_	Patier	nt	_		Informa	nt		
Group	n	Mean	SD	-	п	Mean	SD		
FAS									
MCI	23	45.56	24.42		22	54.55	17.11		
Dementia	25	50.35	19.19		22	39.55	16.47		
Total	48	48.05	21.75		44	47.05	18.25		
Animals									
MCI	23	45.07	23.55		22	62.27	23.69		
Dementia	25	50.69	20.99		22	42.27	19.26		
Total	48	48.00	22.19		44	52.27	23.61		
BNT									
MCI	23	54.26	25.40		22	68.18	22.18		
Dementia	25	54.57	21.45		22	55.91	24.23		
Total	48	54.42	23.17		44	62.05	23.78		
	I	Performa	ance			Total			
	n	Mean	SD		п	Mean	SD		
FAS									
MCI	24	47.25	16.55		69	49.01	19.77		
Dementia	25	41.12	24.76		72	43.84	20.86		
Total	49	44.12	21.15		141	46.37	20.43		
Animals									
MCI	24	46.67	20.04		69	51.11	23.41		
Dementia	25	32.00	15.87		72	41.63	20.14		
Total	49	39.18	19.32		141	46.27	22.24		
BNT									
MCI	24	78.06	18.99		69	66.98	24.10		
Dementia	25	63.33	16.18		72	58.02	20.81		
Total	49	70.54	18.95		141	62.40	22.85		

group (F(1,134)=8.57, p=.004, partial $\eta^2=.06$, F(1,134)=6.36, p=.013, partial $\eta^2=.05$, respectively), while FAS was not (F(1,134)=3.12, p=.080, partial $\eta^2=.02$). In addition, the interaction effect between information source and diagnosis group was significant for

Animals (F(2,134)=5.04, p=.008, partial $\eta^2=.07$) but not FAS or BNT (F(2,134)=2.80, p=.064, partial $\eta^2=.04$, F(2,134)=1.69, p=.189, partial $\eta^2=.02$, respectively). Pairwise comparisons are presented in Table 21. Comparison of prediction/performance means for FAS indicated that informants predicted worse performance for MCI patients than dementia patients. Comparison of prediction/performance means for Animals indicated that dementia patients, in general, received less favorable predictions than MCI patients,

Table 19

Multivariate analysis of covariance for the domain of language for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

			Hypothesis	Error		
Effect	Value	F	$d\!f$	$d\!f$	Sig.	η^2
Age	0.01	0.33	3	132	0.800	0.01
Source	0.26	6.51	6	266	0.000	0.13
Diagnosis	0.07	3.29	3	132	0.023	0.07
Source * Diagnosis	0.08	1.89	6	266	0.083	0.04

Table 20

Univariate analysis of covariance for the domain of language for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

	Main Effect of Source			Main Dia	Effect gnosis	of	Source : Inte	Source x Diagnosis Interaction		
Measure	<i>F</i> (2,134)	р	η^2	<i>F</i> (1,134)	р	η^2	F(2,134)	р	η^2	
FAS	0.46	.632	0.01	3.12	.080	0.02	2.80	.064	0.04	
Animals	4.80	.010	0.07	8.57	.004	0.06	5.04	.008	0.07	
BNT	6.93	.001	0.09	6.36	.013	0.05	1.69	.189	0.02	

Note: *ABC*= Significant Multivariate Analyses

regardless of source. In addition, informants rated dementia patients less favorably than MCI patients, and dementia patients did, indeed, perform worse than MCI patients. Comparison of prediction/performance means for the BNT indicated that informants predicted lower performance for patients than patients as a whole actually performed. Dementia patients, overall, received lower predictions than MCI patients, regardless of source. Lastly, dementia patients predicted less favorable performance than their MCI counterparts, and dementia patients did, in fact, perform worse than MCI patients.

Table 21

Sou	rce	Diagno	Diagnosis		
	p			р	
FAS					
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns
Pt x AP	ns			Inf x Diag	0.001
Inf x AP	ns			AP x Diag	ns
Animals					
Pt x Inf	ns	MCI x Dem	0.004	Pt x Diag	ns
Pt x AP	ns			Inf x Diag	0.001
Inf x AP	0.009			AP x Diag	0.010
BNT					
Pt x Inf	ns	MCI x Dem	0.013	Pt x Diag	0.010
Pt x AP	0.001			Inf x Diag	ns
Inf x AP	ns			AP x Diag	0.010

Pairwise comparisons for the domain of language for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

Note: Pt= Patient, Inf = Informant, AP = Actual Performance, Dem = Dementia, Diag = Diagnosis, *ABC*= Significant Multivariate Analyses, *ABC*= Interpretable Pairwise Comparisons

Visuoperception and Visuoconstruction

A two-way MANCOVA was conducted to determine the effect of information source (patient vs. informant vs. actual performance) and diagnosis group (MCI vs. dementia) on predictions of patient performance (or actual performance, when applicable) in the domain of visuoperception and visuoconstruction, including the JLO and BD, while controlling for age (see Table 22 and Figures 11-12). As presented in Table 23, the main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.37, F(4,268)=15.34, p=.000, $\eta^2=.19$). The main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.13, F(2,133)=9.47, p=.000, $\eta^2=.13$). The interaction effect between information source and diagnosis group also indicated a significant effect on the combined DV (Pillai's Trace=.13, F(4,268)=4.53, p=.001, $\eta^2=.06$). The covariate did not significantly influence the combined DV (Pillai's Trace=.02, F(2,133)=1.16, p=.316, multivariate $\eta^2=.02$).

Univariate ANOVA results are presented in Table 24. They indicated that BD predictions/performances were significantly effected by information source $(F(2,134)=22.89, p=.000, \text{ partial } \eta^2=.25)$ while the JLO was not $(F(2,134)=2.12, p=.124, \text{ partial } \eta^2=.03)$. Results indicated that both the JLO and BD predictions/performances were significantly effected by diagnosis group $(F(1,134)=13.31, p=.000, \text{ partial } \eta^2=.09, F(1,134)=14.66, p=.000, \text{ partial } \eta^2=.10, \text{ respectively})$. In addition, the interaction effect between information source and diagnosis group was significant for the JLO and BD as well $(F(2,134)=7.20, p=.001, \text{ partial } \eta^2=.10; F(2,134)=4.81, p=.010, \eta^2=.07)$.

Pairwise comparisons are presented in Table 25. Comparison of prediction/performance means for the JLO indicated that dementia patients, overall,

Mean scores and standard deviations for the domain of visuoperception and visuoconstruction for patient and informant predictions of performance percentages and actual performance percentages for MCI and dementia patients

	_	Information Source							
Diagnosis		Patier	nt	_		Informa	nt		
Group	n	Mean	SD	_	п	Mean	SD		
JLO									
MCI	24	46.22	24.27		22	52.73	25.67		
Dementia	25	53.24	25.23		21	33.81	19.36		
Total	49	49.81	24.76		43	43.49	24.48		
BD									
MCI	24	44.50	25.70		22	61.82	18.93		
Dementia	25	45.26	22.63		21	38.57	19.31		
Total	49	44.89	23.93		43	50.47	22.25		
	I	Performa	ance		Total				
	n	Mean	SD	-	п	Mean	SD		
JLO									
MCI	24	66.94	16.65		70	55.37	23.81		
Dementia	25	39.33	27.55		71	42.60	25.56		
Total	49	52.86	26.59		141	48.94	25.44		
BD									
MCI	24	32.78	9.71		70	45.93	22.41		
Dementia	25	17.00	13.72		71	33.33	22.38		
Total	49	24.73	14.24		141	39.58	23.19		

Table 23

Multivariate analysis of covariance for the domain of visuoperception and visuoconstruction for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

			Hypothesis	Error		
Effect	Value	F	df	$d\!f$	Sig.	η^2
Age	0.02	1.16	2	133	0.316	0.02
Source	0.37	15.34	4	268	0.000	0.19
Diagnosis	0.13	9.47	2	133	0.000	0.13
Source * Diagnosis	0.13	4.53	4	268	0.001	0.06

Univariate analysis of covariance for the domain of visuoperception and visuoconstruction for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

	Main Effect of Source		Main Effect of Diagnosis			Source x Diagnosis Interaction			
Measure	F(2,134)	р	η^2	<i>F</i> (1,134)	р	η^2	F(2,134)	р	η^2
JLO	2.12	.124	0.0 3	13.31	.000	0.09	7.20	.001	0.10
BD	22.88	.000	0.2 5	14.66	.000	0.10	4.81	.010	0.07

Table 25

Pairwise comparisons for the domain of visuoperception and visuoconstruction for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

Source		Diagno	osis	Source x Diagnosis		
	p		р		р	
JLO						
Pt x Inf	ns	MCI x Dem	0.000	Pt x Diag	ns	
Pt x AP	ns			Inf x Diag	0.013	
Inf x AP	ns			AP x Diag	0.000	
BD						
Pt x Inf	ns	MCI x Dem	0.000	Pt x Diag	ns	
Pt x AP	0.000			Inf x Diag	0.000	
Inf x AP	0.000			AP x Diag	0.000	

Note: Pt= Patient, Inf = Informant, AP = Actual Performance, Dem = Dementia, Diag = Diagnosis, *ABC*= Significant Multivariate Analyses, *ABC*= Interpretable Pairwise Comparisons

received less favorable predictions than MCI patients regardless of information source. In addition, informants rated dementia patients less favorably than MCI patients, and dementia patients did, in fact, demonstrate lower performance than MCI patients. Comparison of predication/performance means for BD indicated that, although patient and informant predictions were similar, patients predicted more favorable performance than they were actually able to perform. Dementia patients, as a whole, received less favorable predictions than their MCI counterparts, regardless of source of information. Lastly, informants predicted lower performance for dementia patients than MCI patients, and dementia patients did, in fact, performed lower than MCI patients did.

Memory

A two-way MANCOVA was conducted to determine the effect of information source (patient vs. informant vs. actual performance) and diagnosis group (MCI vs. dementia) on predictions of patient performance (or actual performance, when applicable) in the domain of memory, including the CVLT IR, CVLT DR, CVLT Rec, CVLT FC, LMI and LMII, while controlling for age (see Table 26 and Figures 13-18). As presented in Table 27, the main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=1.02, F(12,246)=21.38, p=.000, $\eta^2=.51$). The main effect of diagnosis group did not indicate a significant effect on the combined DV (Pillai's Trace=.06, F(6,122)=1.31, p=.259, $\eta^2=.06$). The interaction effect between information source and diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.25, F(12,246)=2.96, p=.001, $\eta^2=.13$). The covariate did not significantly influence the combined DV (Pillai's Trace=.02, F(6,122)=1.05, p=.396, multivariate $\eta^2=.05$).

Univariate ANOVA results are presented in Table 28. They indicated that all predictions/performance values (CVLT IR, CVLT DR, CVLT Rec, CVLT FC, LMI, and LMII) were significantly effected by information source (F(2,134)=22.89, p=.000, partial $\eta^2=.25$). Results indicated that both the CVLT IR and CVLT FC

Mean scores and standard deviations for the domain of memory for patient and informant predictions of performance percentages and actual performance percentages for MCI and dementia patients

	Information Source							
Diagnosis		Patier	nt		Informa	ant		
Group	n	Mean	SD	n	Mean	SD		
CVLT-IR								
MCI	23	27.04	18.83	19	36.84	21.10		
Dementia	25	31.36	21.20	18	29.44	16.97		
Total	48	29.29	20.01	37	33.24	19.30		
CVLT-DR								
MCI	23	21.93	20.06	19	27.37	19.68		
Dementia	25	27.35	23.93	18	20.00	11.38		
Total	48	24.75	22.10	37	23.78	16.39		
CVLT-Rec								
MCI	23	34.40	25.06	19	33.68	19.21		
Dementia	25	32.87	24.81	18	31.67	21.49		
Total	48	33.60	24.67	37	32.70	20.09		
CVLT-FC								
MCI	23	50.54	24.61	19	38.95	16.96		
Dementia	25	43.67	23.07	18	32.22	18.33		
Total	48	46.96	23.82	37	35.68	17.72		
LMI								
MCI	23	42.51	27.17	19	39.47	15.45		
Dementia	25	46.26	19.64	18	32.78	18.73		
Total	48	44.46	23.37	37	36.22	17.22		
LMII								
MCI	23	16.91	14.09	19	35.79	18.65		
Dementia	25	25.01	24.24	18	22.78	12.27		
Total	48	21.13	20.24	37	29.46	16.99		

Table 26 continued

Mean scores and standard deviations for the domain of memory for patient and informant predictions of performance percentages and actual performance percentages for MCI and dementia patients continued

	Information Source							
Diagnosis	F	Performa	ance		Total			
Group	n	Mean	SD	n	Mean	SD		
CVLT-IR								
MCI	24	54.63	21.21	66	39.89	23.32		
Dementia	25	24.00	20.71	68	28.15	19.96		
Total	49	39.00	25.87	134	33.93	22.39		
CVLT-DR								
MCI	24	51.85	20.64	66	34.37	24.00		
Dementia	25	31.56	23.06	68	26.95	21.20		
Total	49	41.50	23.98	134	30.61	22.84		
CVLT-Rec								
MCI	24	85.49	10.06	66	52.77	31.18		
Dementia	25	72.00	12.70	68	46.94	27.68		
Total	49	78.61	13.25	134	49.81	29.49		
CVLT-FC								
MCI	24	98.15	5.35	66	64.52	31.19		
Dementia	25	91.11	14.70	68	58.08	31.90		
Total	49	94.56	11.59	134	61.25	31.60		
LMI								
MCI	24	36.11	15.07	66	39.31	20.09		
Dementia	25	19.68	9.61	68	32.92	19.80		
Total	49	27.73	14.96	134	36.07	20.13		
LMII								
MCI	24	25.08	18.72	66	25.32	18.57		
Dementia	25	9.20	9.92	68	18.61	18.35		
Total	49	16.98	16.78	134	21.91	18.69		

predictions/performances were significantly effected by diagnosis group (F(1,127)=6.53, p=.012, partial $\eta^2=.05$, F(1,127)=4.01, p=.047, partial $\eta^2=.03$, respectively) while the CVLT DR, CVLT Rec, LMI and LMII were not (F(1,127)=2.49, p=.117, partial $\eta^2=.02$, F(1,127)=2.97, p=.087, partial $\eta^2=.02$, F(1,127)=2.59, p=.110, partial $\eta^2=.02$,
F(1,127)=3.43, p=.06, partial $\eta^2=.03$, respectively). In addition, the interaction effect between information source and diagnosis group was significant for the CVLT IR, CVLT DR, LMI and LMII (F(2,127)=9.40, p=.000, partial $\eta^2=.13$; F(2,127)=4.72, p=.011, $\eta^2=.07$, F(2,127)=3.64, p=.029, partial $\eta^2=.05$; F(2,127)=6.83, p=.002, $\eta^2=.10$, respectively), but not for the CVLT Rec or CVLT FC (F(2,127)=1.39, p=.253, partial $\eta^2=.02$; F(2,127)=0.00, p=.999, $\eta^2=.00$, respectively).

Pairwise comparison data are presented in Table 29. Comparison of prediction/performance means for the CVLT IR indicated that patients provided less favorable predictions than they were actually able to perform, and dementia patients received less favorable ratings regardless of the source of information. In addition, dementia patients did, in fact, perform more poorly than MCI patients. Comparison of prediction/performance means for the CVLT DR indicated that patients provided lower predictions than they were actually able to perform, and that dementia patients displayed lower performance than their MCI counterparts. Comparison of prediction/performance means for the CVLT Rec indicated that patients provided less favorable predictions than they were actually able to perform, and that dementia patients performed worse than MCI patients. Comparison of prediction/performance means for the CVLT FC indicated that both patients and informants provided less favorable predictions than patients were actually able to perform, and that dementia patients received less favorable ratings regardless of information source. In addition, dementia patients demonstrated a lower level of performance than their MCI counterparts. Comparison of prediction/performance

Multivariate analysis of covariance for the domain of memory for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

			Hypothesis	Error		
Effect	Value	F	$d\!f$	$d\!f$	Sig.	η^2
Age	0.05	1.05	6	122	0.396	0.05
Source	1.02	21.38	12	246	0.000	0.51
Diagnosis	0.06	1.31	6	122	0.259	0.06
Source * Diagnosis	0.25	2.96	12	246	0.001	0.13

Table 28

Univariate analysis of covariance for the domain of memory for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

	Main So	Effect ource	of	Main Dia	Effect agnosis	of	Source x Diagnosis Interaction			
Measure	F(2, 127)	р	η^2	<i>F</i> (1,127)	р	η^2	F(2, 127)	р	η^2	
CVLT-IR	3.11	.048	0.05	6.53	.012	0.05	9.40	.000	0.13	
CVLT-DR	11.26	.000	0.15	2.49	.117	0.02	4.72	.011	0.07	
CVLT-Rec	83.06	.000	0.57	2.97	.087	0.02	1.39	.253	0.02	
CVLT-FC	131.20	.000	0.67	4.01	.047	0.03	0.00	.999	0.00	
LMI	9.71	.000	0.13	2.59	.110	0.02	3.64	.029	0.05	
LMII	5.29	.006	0.08	3.43	.066	0.03	6.83	.002	0.10	

for LMI indicated that, although patients and informants provided similar ratings, they both underestimated patient's actual performance. In addition, dementia patients performed worse than MCI patients. Lastly, comparison of prediction/performance means for LMII indicated that informants provided more favorable predictions than patients were actually able to perform. In addition, informants rated dementia patients less favorably than MCI patients, and dementia patients did, indeed, perform worse than MCI patients.

Sour	ce	Diagno	sis	Source x D	iagnosis
	р		p		p
CVLT-IR					
Pt x Inf	ns	MCI x Dem	0.012	Pt x Diag	ns
Pt x AP	0.044			Inf x Diag	ns
Inf x AP	ns			AP x Diag	0.000
CVLT-DR					
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns
Pt x AP	0.000			Inf x Diag	ns
Inf x AP	0.000			AP x Diag	0.018
CVLT-Rec					
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns
Pt x AP	0.000			Inf x Diag	ns
Inf x AP	0.000			AP x Diag	0.010
CVLT-FC					
Pt x Inf	0.015	MCI x Dem	0.047	Pt x Diag	ns
Pt x AP	0.000			Inf x Diag	ns
Inf x AP	0.000			AP x Diag	0.010
LMI					
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns
Pt x AP	0.000			Inf x Diag	ns
Inf x AP	ns			AP x Diag	0.001
LMII					
Pt x Inf	ns	MCI x Dem	ns	Pt x Diag	ns
Pt x AP	ns			Inf x Diag	0.002
Inf x AP	0.005			AP x Diag	0.005

Pairwise comparisons for the domain of memory for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

Note: Pt= Patient, Inf = Informant, AP = Actual Performance, Dem = Dementia, Diag = Diagnosis, ABC= Significant Multivariate Analyses, ABC= Interpretable Pairwise Comparisons

Executive Function

A two-way MANCOVA was conducted to determine the effect of information source (patient vs. informant vs. actual performance) and diagnosis group (MCI vs. dementia) on predictions of patient performance (or actual performance, when applicable) in the domain of executive function, including the WCST and Trails B, while controlling for age (see Table 30 and Figures 19-20). As presented in Table 31, the main effect of information source indicated a significant effect on the combined DV (Pillai's Trace=.29, F(4,268)=11.31, p=.000, $\eta^2=.14$). The main effect of diagnosis group also indicated a significant effect on the combined DV (Pillai's Trace=.08, F(2,133)=6.08, p=.003, $\eta^2=.08$). Lastly, the interaction effect between information source

Mean scores and standard deviations for the domain of executive function for patient and informant predictions of performance percentages and actual performance percentages for MCI and dementia patients

		Information Source							
Diagnosis	Patient					Informa	nt		
Group	n	Mean	SD		п	Mean	SD		
WCST									
MCI	24	41.61	24.08		22	37.27	24.53		
Dementia	25	58.78	23.15		21	24.29	11.65		
Total	49	50.37	24.92		43	30.93	20.21		
Trails B									
MCI	24	46.50	27.18		22	41.36	22.53		
Dementia	25	45.13	26.21		21	28.10	14.36		
Total	49	45.80	26.42		43	34.88	19.93		
	I	Performa	ance	-		Total			
	n	Mean	SD		п	Mean	SD		
WCST									
MCI	24	48.24	19.30		70	42.52	22.83		
Dementia	25	45.06	16.83		71	43.75	22.69		
Total	49	46.62	17.96		141	43.14	22.68		
Trails B									
MCI	24	37.38	26.88		70	41.76	25.62		
Dementia	25	13.15	20.17		71	28.83	24.84		
Total	49	25.02	26.45		141	35.25	25.96		

and diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.12, F(4,268)=4.09, p=.003, $\eta^2=.06$). The covariate did not significantly influence the combined DV (Pillai's Trace=.00, F(2,133)=.05, p=.952 multivariate $\eta^2=.00$).

Univariate ANOVA results are presented in Table 32. They indicated that both the WCST and Trails B predictions/performances were significantly effected by information source (F(2,134)=11.31, p=.000, partial $\eta^2=.14$; F(2,134)=9.30, p=.000, partial $\eta^2=.12$). Results indicated that Trails B predictions/performance was significantly effected by diagnosis group (F(1,134)=9.61, p=.002, partial $\eta^2=.07$) while WCST was not (F(1,134)=.02, p=.879, partial $\eta^2=.00$). Lastly, the interaction effect between information source and diagnosis group was significant for the WCST (F(2,134)=6.51, p=.002, partial $\eta^2=.09$), but not Trails B (F(2,134)=2.88, p=.060, $\eta^2=.04$).

Pairwise comparison data are presented in Table 33. Comparison of prediction/performance means for the WCST indicate that informants provided lower predictions of performance than patients provided and than patients were actually able to perform. In addition, MCI patients predicted that they would perform worse than

Multivariate analysis of covariance for the domain of executive function for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

			Hypothesis	Error		
Effect	Value	F	df	$d\!f$	Sig.	η^2
Age	0.00	0.05	2	133	0.952	0.00
Source	0.29	11.31	4	268	0.000	0.14
Diagnosis	0.08	6.08	2	133	0.003	0.08
Source * Diagnosis	0.12	4.09	4	268	0.003	0.06

Univariate analysis of covariance for the domain of executive function for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

	Main Effect of Source			Maii Di	n Effect iagnosis	of	Source x Diagnosis Interaction		
Measure	F	p	η^2	F	р	η^2	F	р	η^2
WCST	11.31	.000	0.14	0.02	.879	0.00	6.51	.002	0.09
Trails B	9.30	.000	0.12	9.61	.002	0.07	2.88	.060	0.04

Note: *ABC*= Significant Multivariate Analyses

Table 33

Pairwise comparisons for the domain of executive function for patient and informant prediction percentages and actual performance percentages for MCI and dementia patients

Sour	rce	Diagno	sis	Source x D	iagnosis
	p		р		р
WCST					
Pt x Inf	0.000	MCI x Dem	ns	Pt x Diag	0.037
Pt x AP	ns			Inf x Diag	0.027
Inf x AP	0.001			AP x Diag	ns
Trails B					
Pt x Inf	ns	MCI x Dem	0.002	Pt x Diag	ns
Pt x AP	0.000			Inf x Diag	0.042
Inf x AP	ns			AP x Diag	0.003

Note: Pt= Patient, Inf = Informant, AP = Actual Performance, Dem = Dementia, Diag = Diagnosis, *ABC*= Significant Multivariate Analyses, *ABC*= Interpretable Pairwise Comparisons

dementia patients, while informants predicted that dementia patients would perform worse than MCI patients. Lastly, dementia and MCI patients performed similarly. Comparison of prediction/performance means for Trails B indicated that, although patients and informants provided similar ratings, patients provided more favorable predictions than they were actually able to perform. In addition, dementia patients received less favorable ratings than MCI patients, regardless of the information source. Lastly, informants predicted that dementia patients would perform less favorably than MCI patients, and dementia patients did, in fact, perform worse than their MCI counterparts.

Hypothesis 3 – Emergent Awareness by Cognitive Domain General Cognitive Ability, Premorbid Function, and Effort

A two-way, mixed (doubly-multivariate) MANCOVA was conducted to determine the effect of diagnosis group (between subjects, MCI vs. dementia) and time (within-subjects, prediction vs. evaluation) on anosognosia ratios in the domain of general cognitive ability, premorbid function, and effort, including the 3MS, WTAR, and DC, while controlling for age (see Table 34 and Figures 21-23). As presented in Table 35, the between subjects main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.22, F(3,38)=3.60, p=.022, multivariate $\eta^2=.22$). The within-subjects main effect of time did not indicate a significant effect on the combined DV (Pillai's Trace=.05, F(3,38)=.64, p=.596, multivariate $\eta^2=.05$). The interaction effect between diagnosis group and time did not indicate a significant effect on the combined DV (Pillai's Trace=.16, F(3,38)=2.32, p=.091, multivariate $\eta^2=.16$). The covariate did not significantly influence the combined DV (Pillai's Trace=.03, F(3,38)=.03, p=.805, multivariate $\eta^2=.03$).

Univariate ANOVA results are presented in Table 36, and indicated that 3MS anosognosia ratios were significantly effected by diagnosis group (F(1,40)=10.90, p=.002, partial $\eta^2=.21$), while the WTAR and DC anosognosia ratios were not (F(1,40)=1.16,

94

p=.288, partial $\eta^2=.03$; F(1,40)=.093, p=.340, partial $\eta^2=.01$, respectively). Comparison of 3MS anosognosia ratio means indicated that anosognosia ratios for MCI patients were lower than those of dementia patients, indicating that MCI patients had the tendency to provide less favorable predictions of their performance relative to their actual performance in comparison to the dementia group.

Attention, Concentration, and Processing Speed

A two-way, mixed (doubly-multivariate) MANCOVA was conducted to determine the effect of diagnosis group (between subjects, MCI vs. dementia) and time

Table 34

MCI

Total

Dementia

21

22

43

-0.35

-0.21

-0.28

0.31

0.46

0.39

					Time				
Diagnosis		Predictio	on	•	Evaluati	on		Total	
Group	n	Mean	SD	n	Mean	SD	n	Mean	SD
3MS									
MCI	21	-0.24	0.19	21	-0.30	0.28	42	-0.27	0.19
Dementia	22	0.02	0.19	22	-0.12	0.23	44	-0.05	0.19
Total	43	-0.11	0.23	43	-0.21	0.27	86	-0.16	0.03
WTAR									
MCI	21	-0.30	0.35	21	-0.16	0.32	42	-0.23	0.08
Dementia	22	-0.10	0.38	22	-0.11	0.42	44	-0.10	0.08
Total	43	-0.20	0.37	43	-0.13	0.37	86	-0.17	0.05

21

22

43

-0.14

0.00

-0.07

0.29

0.34

0.32

42

44

86

0.22

0.03

0.13

0.08

0.08

0.05

Mean scores and standard deviations for the domain of general cognitive ability,



Figure 21. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for the Modified Mini Mental Status Examination (3MS)



Figure 22. Prediction and evaluation ratios (±SD) for the Wechsler Test of Adult Reading (WTAR)



Figure 23. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for the Dot Counting Test (DC)

Multivariate analysis of covariance analysis for the domain of general cognitive ability, premorbid function, and effort for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

				Hypothesis	Error		
Effect		Value	F	$d\!f$	$d\!f$	Sig.	η^2
Between	Intercept	0.04	0.53	3	38	0.665	0.04
	Age	0.03	0.33	3	38	0.805	0.03
	Diagnosis	0.22	3.60	3	38	0.022	0.22
Within	Time	0.05	0.64	3	38	0.596	0.05
	Time*Age	0.06	0.87	3	38	0.465	0.06
	Time*Diagnosis	0.16	2.32	3	38	0.091	0.16

	Main	Effect Time	of	Main Di	Effect Effect	of	Time x Diagnosis Interaction			
Measure	F(1,40)	р	η^2	F(1,40)	p	η^2	F(1,40)	p	η^2	
3MS	0.07	.791	0.00	10.90	.002	0.21	1.05	.313	0.03	
WTAR	1.04	.314	0.03	1.16	.288	0.03	7.18	.011	0.15	
DC	0.41	.526	0.01	0.93	.340	0.02	0.00	.979	0.00	

Univariate analysis of covariance analysis for the domain of general cognitive ability, premorbid function, and effort for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

(within-subjects, prediction vs. evaluation) on anosognosia ratios in the domain of attention, concentration, and processing speed, including Trails A, DSF, DSB, and the SDMT, while controlling for age (see Table 37 and Figures 24-27). As presented in Table 38, the between subjects main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.29, F(4,42)=4.23, p=.006, multivariate $\eta^2=.29$). The within-subjects main effect of time did not indicate a significant effect on the combined DV (Pillai's Trace=.05, F(4,42)=.56, p=.692, multivariate $\eta^2=.05$). The interaction effect between diagnosis group and time did not indicate a significant effect on the combined DV (Pillai's Trace=.06, F(4,42)=.062, p=.654, multivariate $\eta^2=.06$). The covariate did not significantly influence the combined DV (Pillai's Trace=.05, F(7,42)=.056, p=.693, multivariate $\eta^2=.05$).

Univariate ANOVA results are presented in Table 39, and indicated that Trails A, DSB, and SDMT anosognosia ratios were significantly effected by diagnosis group $(F(1,45)=9.58, p=.003, \text{ partial } \eta^2=.18; F(1,45)=5.84, p=.020, \text{ partial } \eta^2=.11;$ $F(1,45)=12.04, p=.001, \text{ partial } \eta^2=.21, \text{ respectively}), \text{ while DSF anosognosia ratios were}$

					Time						
Diagnosis		Predictio	on		Evaluatio	on		Total			
Group	п	Mean	SD	n	Mean	SD	n	Mean	SD		
Trails A											
MCI	24	-0.21	0.24	24	-0.17	0.19	48	-0.18	0.08		
Dementia	24	0.15	0.48	24	0.20	0.45	48	0.16	0.08		
Total	48	-0.03	0.42	48	0.02	0.39	96	-0.01	0.05		
DSF											
MCI	24	-0.25	0.44	24	-0.15	0.34	48	-0.22	0.07		
Dementia	24	-0.11	0.30	24	-0.11	0.36	48	-0.09	0.07		
Total	48	-0.18	0.38	48	-0.13	0.35	96	-0.16	0.05		
DSB											
MCI	24	-0.29	0.44	24	-0.16	0.46	48	-0.25	0.10		
Dementia	24	0.00	0.60	24	0.15	0.50	48	0.11	0.10		
Total	48	-0.14	0.54	48	-0.01	0.50	96	-0.07	0.07		
SDMT											
MCI	24	-0.25	0.41	24	-0.07	0.32	48	-0.16	0.08		
Dementia	24	0.20	0.50	24	0.30	0.39	48	0.25	0.08		
Total	48	-0.02	0.51	48	0.12	0.40	96	0.05	0.05		

Mean scores and standard deviations for the domain of attention, concentration, and processing speed for patient prediction anosognosia ratios for MCI and dementia patients



Figure 24. Prediction and evaluation ratios (±SD) for MCI and dementia patients for the Trail Making Test Part A (Trails A)



Figure 25. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for Digit Span Forward (DSF)



Figure 26. Prediction and evaluation ratios (±SD) for MCI and dementia patients for Digit Span Backward (DSB)



Figure 27. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for the Symbol Digit Modalities Test (SDMT)

Multivariate analysis of covariance analysis for the domain of attention, concentration, and processing speed for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

				Hypothesis	Error		
Effect		Value	F	$d\!f$	df	Sig.	η^2
Between	Intercept	0.03	0.36	4	42	0.838	0.03
	Age	0.05	0.56	4	42	0.693	0.05
	Diagnosis	0.29	4.23	4	42	0.006	0.29
Within	Time	0.05	0.56	4	42	0.692	0.05
	Time*Age	0.08	0.86	4	42	0.495	0.08
	Time*Diagnosis	0.06	0.62	4	42	0.654	0.06

	Main Effect of			Main	Effect	of	Time x Diagnosis		
	Time			Di	agnosis		Interaction		
Measure	F(1,45)	р	η^2	F(1,45)	р	η^2	<i>F</i> (1,45)	р	η^2
Trails A	0.04	.844	0.00	9.58	.003	0.18	0.03	.872	0.00
DSF	0.01	.915	0.00	1.74	.194	0.04	1.32	.257	0.03
DSB	1.69	.200	0.04	5.84	.020	0.11	0.19	.667	0.00
SDMT	0.47	.497	0.01	12.04	.001	0.21	1.06	.308	0.02

Univariate analysis of covariance analysis for the domain of attention, concentration, and processing speed for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

not (F(1,45)=1.74, p=.194, partial $\eta^2=.04$). Comparison of anosognosia ratio means for all three significant subtests (Trails A, DSB, and SDMT) indicated that anosognosia ratios for MCI patients were lower than those of dementia patients, indicating that MCI patients had the tendency to provide less favorable predictions of their performance relative to their actual performance in comparison to their dementia patient counterparts.

Language

A two-way, mixed (doubly-multivariate) MANCOVA was conducted to determine the effect of diagnosis group (between subjects, MCI vs. dementia) and time (within-subjects, prediction vs. evaluation) on anosognosia ratios in the domain of language, including FAS, Animals, and the BNT, while controlling for age (see Table 40 and Figures 28-30). As presented in Table 41, the between subjects main effect of diagnosis group did not indicate a significant effect on the combined DV (Pillai's Trace=.14, F(3,43)=2.33, p=.088, multivariate $\eta^2=.14$). The within-subjects main effect of time did not indicate a significant effect on the combined DV (Pillai's Trace=.06, F(3,43)=.86, p=.471, multivariate $\eta^2=.06$). The interaction effect between diagnosis

Diagnosis		Prediction			Evaluatio	on		Total		
Group	п	Mean	SD	п	n Mean SD		п	Mean	SD	
FAS										
MCI	23	-0.05	0.36	23	-0.21	0.36	46	-0.15	0.06	
Dementia	25	0.14	0.30	25	-0.05	0.29	50	0.06	0.06	
Total	48	0.05	0.34	48	-0.13	0.33	96	-0.04	0.04	
Animals										
MCI	23	-0.04	0.34	23	-0.18	0.39	46	-0.11	0.07	
Dementia	25	0.20	0.32	25	0.01	0.41	50	0.10	0.07	
Total	48	0.09	0.35	48	-0.08	0.41	96	0.00	0.05	
BNT										
MCI	23	-0.21	0.25	23	-0.19	0.23	46	-0.20	0.05	
Dementia	25	-0.09	0.22	25	-0.07	0.26	50	-0.08	0.05	
Total	48	-0.15	0.24	48	-0.13	0.25	96	-0.14	0.03	

Mean scores and standard deviations for the domain of language for patient prediction anosognosia ratios for MCI and dementia patients



Figure 28. Prediction and evaluation ratios (±SD) for MCI and dementia patients for FAS



Figure 29. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for Animals



Figure 30. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for the Boston Naming Test (BNT)

group and time did not indicate a significant effect on the combined DV (Pillai's

Trace=.02, F(3,43)=0.29, p=.833, multivariate $\eta^2=.02$). The covariate did not

significantly influence the combined DV (Pillai's Trace=.04, F(3,43)=.64, p=.592,

multivariate η^2 =.04). Univariate ANOVA results are presented in Table 42, though they are uninterpretable.

Visuoperception and Visuoconstruction

A two-way, mixed (doubly-multivariate) MANCOVA was conducted to determine the effect of diagnosis group (between subjects, MCI vs. dementia) and time (within-subjects, prediction vs. evaluation) on anosognosia ratios in the domain of visuoperception and

Table 41

Multivariate analysis of covariance analysis for the domain of language for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

				Hypothesis	Error		
Effect		Value	F	$d\!f$	$d\!f$	Sig.	η^2
Between	Intercept	0.04	0.54	3	43	0.659	0.04
	Age	0.04	0.64	3	43	0.592	0.04
	Diagnosis	0.14	2.33	3	43	0.088	0.14
Within	Time	0.06	0.86	3	43	0.471	0.06
	Time*Age	0.03	0.39	3	43	0.760	0.03
	Time*Diagnosis	0.02	0.29	3	43	0.833	0.02

Univariate analysis of covariance analysis for the domain of language for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

	Main Effect of			Main	Effect	of	Time x Diagnosis			
	Time			Di	agnosis		Interaction			
Measure	<i>F</i> (1,45)	р	η^2	F(1,45)	р	η^2	F(1,45)	р	η^2	
FAS	2.37	.131	0.05	5.46	.024	0.11	0.53	.471	0.01	
Animals	1.03	.315	0.02	4.08	.049	0.08	0.63	.433	0.01	
BNT	0.04	.841	0.00	3.30	.076	0.07	0.06	.801	0.00	

visuoconstruction, including the JLO and BD, while controlling for age (see Table 43 and Figures 31-32). As presented in Table 44, the between subjects main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.21,

F(2,43)=5.71, p=.006, multivariate $\eta^2=.21$). The within-subjects main effect of time did

not indicate a significant effect on the combined DV (Pillai's Trace=.07, F(2,43)=.1.50,

p=.235, multivariate $\eta^2=.07$). The interaction effect between diagnosis group and time did

not indicate a significant effect on the combined DV (Pillai's Trace=.06, F(2,43)=1.31,

p=.281, multivariate $\eta^2=.06$). The covariate did not significantly influence

Mean scores and standard deviations for the domain of visuoperception and visuoconstruction for patient prediction anosognosia ratios for MCI and dementia patients

		Time										
Diagnosis	Prediction				Evaluatio	on		Total				
Group	n Mean SD			п	Mean	SD	n	Mean	SD			
JLO												
MCI	24	-0.22	0.33	24	-0.10	0.22	48	-0.18	0.08			
Dementia	23	0.21	0.50	23	0.18	0.50	46	0.21	0.08			
Total	47	-0.01	0.47	47	0.03	0.41	94	0.02	0.06			
BD												
MCI	24	0.06	0.31	24	-0.20	0.38	48	-0.08	0.08			
Dementia	23	0.43	0.37	23	0.12	0.60	46	0.28	0.09			
Total	47	0.24	0.39	47	-0.04	0.52	94	0.10	0.06			



Figure 31. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for the Judgment of Line Orientation (JLO)



Figure 32. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for Block Design

Effect		Value	F	Hypothesis <i>df</i>	Error df	Sig.	η^2
Between	Intercept	0.02	0.37	2	43	0.694	0.02
	Age	0.02	0.35	2	43	0.709	0.02
	Diagnosis	0.21	5.71	2	43	0.006	0.21
Within	Time	0.07	1.50	2	43	0.235	0.07
	Time*Age	0.04	0.93	2	43	0.401	0.04
	Time*Diagnosis	0.06	1.31	2	43	0.281	0.06

Multivariate analysis of covariance analysis for the domain of visuoperception and visuoconstruction for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

Table 45

Univariate analysis of covariance analysis for the domain of visuoperception and visuoconstruction for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

	Main Effect of Time			Main Dia	Effect	of	Time x Diagnosis Interaction		
Measure	<i>F</i> (1,44)	p	η^2	<i>F</i> (1,44)	р	η^2	<i>F</i> (1,44)	р	η^2
JLO	2.21	.144	0.05	10.37	.002	0.19	2.01	.163	0.04
BD	1.17	.284	0.03	8.71	.005	0.17	0.43	.516	0.01

the combined DV (Pillai's Trace=.02, F(2,43)=.35, p=.709, multivariate η^2 =.02).

Univariate ANOVA results are presented in Table 45, and indicated that both JLO and BD anosognosia ratios were significantly effected by diagnosis group (F(1,44)=10.37, p=.002, partial $\eta^2=.19$; F(1,44)=8.71, p=.005, partial $\eta^2=.17$, respectively). Comparison of prediction means for both measures indicated that anosognosia ratios for MCI patients were lower than those of dementia patients, indicating that MCI patients had the tendency to provide less favorable predictions of their performance relative to their actual performance than their dementia group counterparts.

Memory

A two-way, mixed (doubly-multivariate) MANCOVA was conducted to determine the effect of diagnosis group (between subjects, MCI vs. dementia) and time (within-subjects, prediction vs. evaluation) on anosognosia ratios in the domain of memory, including the CVLT IR, CVLT DR, CVLT Rec, CVLT FC, LMI and LMII, while controlling for age (see Table 46 and Figures 33-38). As presented in Table 47, the between subjects main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.33, F(6,39)=3.23, p=.011, multivariate $\eta^2=.33$). The within-subjects main effect of time did not indicate a significant effect on the combined DV (Pillai's Trace=.17, F(6,39)=1.33, p=.596, multivariate $\eta^2=.17$). The interaction effect between diagnosis group and time did not indicate a significant effect on the combined DV (Pillai's Trace=.16, F(6,39)=1.19, p=.033, multivariate $\eta^2=.16$). The covariate did not significantly influence the combined DV (Pillai's Trace=.12, F(6,39)=.92, p=.494, multivariate $\eta^2=.12$).

Univariate ANOVA results are presented in Table 48, and indicated that CVLT IR, LMI and LMII anosognosia ratios were significantly effected by diagnosis group $(F(1,44)=10.16, p=.003, \text{ partial } \eta^2=.19; F(1,44)=12.20, p=.001, \text{ partial } \eta^2=.22;$ $F(1,44)=6.96, p=.011, \text{ partial } \eta^2=.14, \text{ respectively}), \text{ while CVLT DR, CVLT Rec, and}$ CVLT FC anosognosia ratios were not $(F(1,44)=3.61, p=.064, \text{ partial } \eta^2=.08;$ $(F(1,44)=.03, p=.874, \text{ partial } \eta^2=.00; F(1,44)=0.00, p=.961, \text{ partial } \eta^2=.00, \text{ respectively}).$ Comparison of prediction means indicated that anosognosia ratios for MCI patients were lower than those of dementia patients for all three significant measures (CVLT IR, LMI and LMII), indicating that MCI patients had the tendency to provide less favorable

Diagnosis	v	Predictio	n		Evaluatio	on		Total	
Group	п	Mean	SD	п	Mean	SD	п	Mean	SD
CVLT-IR									
MCI	23	-0.35	0.42	23	-0.30	0.39	46	-0.33	0.12
Dementia	24	0.23	0.64	24	0.16	0.65	48	0.20	0.11
Total	47	-0.05	0.61	47	-0.06	0.58	94	-0.06	0.08
CVLT-DR									
MCI	23	-0.42	0.47	23	-0.47	0.47	46	-0.42	0.11
Dementia	24	-0.03	0.58	24	-0.19	0.51	48	-0.13	0.10
Total	47	-0.23	0.56	47	-0.33	0.50	94	-0.28	0.07
CVLT-Rec									
MCI	23	-0.50	0.31	23	-0.39	0.30	46	-0.44	0.06
Dementia	24	-0.47	0.30	24	-0.38	0.32	48	-0.43	0.06
Total	47	-0.48	0.30	47	-0.39	0.31	94	-0.44	0.04
CVLT-FC									
MCI	23	-0.36	0.25	23	-0.21	0.26	46	-0.31	0.05
Dementia	24	-0.40	0.26	24	-0.26	0.18	48	-0.31	0.05
Total	47	-0.38	0.25	47	-0.23	0.22	94	-0.31	0.03
LMI									
MCI	23	0.00	0.40	23	-0.26	0.39	46	-0.15	0.08
Dementia	24	0.36	0.35	24	0.12	0.48	48	0.25	0.08
Total	47	0.18	0.42	47	-0.06	0.47	94	0.05	0.05
LMII									
MCI	23	-0.06	0.63	23	-0.02	0.62	46	-0.02	0.12
Dementia	24	0.49	0.48	24	0.40	0.51	48	0.42	0.11
Total	47	0.22	0.62	47	0.19	0.60	94	0.20	0.08

Mean scores and standard deviations for the domain of memory for patient prediction anosognosia ratios for MCI and dementia patients



Figure 33. Prediction and evaluation ratios (±SD) for MCI and dementia patients for the California Verbal Learning Test Immediate Recall (CVLT IR)



Figure 34. Prediction and evaluation ratios (±SD) for MCI and dementia patients for the California Verbal Learning Test Delayed Recall (CVLT DR)



Figure 35. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for the California Verbal Learning Test Recognition (CVLT Rec)



Figure 36. Prediction and evaluation ratios $(\pm SD)$ for MCI and dementia patients for the California Verbal Learning Test Forced Choice Recognition (CVLT FC)



Figure 37. Prediction and evaluation ratios (\pm SD) for MCI and dementia patients for Logical Memory I (LMI)



Figure 38. Prediction and evaluation ratios (±SD) for MCI and dementia patients for Logical Memory II (LMII)

				Hypothesis	Error		
Effect		Value	F	df	$d\!f$	Sig.	η^2
Between	Intercept	0.17	1.36	6	39	0.254	0.17
	Age	0.12	0.92	6	39	0.494	0.12
	Diagnosis	0.33	3.23	6	39	0.011	0.33
Within	Time	0.17	1.33	6	39	0.269	0.17
	Time*Age	0.19	1.56	6	39	0.185	0.19
	Time*Diagnosis	0.16	1.19	6	39	0.33	0.16

Multivariate analysis of covariance analysis for the domain of memory for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

Table 48

Univariate analysis of covariance analysis for the domain of memory for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

	Main Effect of			Main Effect of			Time x Diagnosis		
	r	Time		Die	agnosis		Interaction		
Measure	<i>F</i> (1,44)	p	η^2	F(1,44)	p	η^2	F(1,44)	p	η^2
CVLT-IR	3.93	.054	0.08	10.16	.003	0.19	5.06	.029	0.10
CVLT-DR	0.92	.342	0.02	3.61	.064	0.08	0.63	.432	0.01
CVLT-Rec	2.93	.094	0.06	0.03	.874	0.00	0.06	.803	0.00
CVLT-FC	0.00	.989	0.00	0.00	.961	0.00	0.06	.803	0.00
LMI	0.74	.393	0.02	12.20	.001	0.22	0.45	.505	0.01
LMII	0.00	.965	0.00	6.96	.011	0.14	1.22	.276	0.03

predictions of their performance relative to their actual performed in comparison to their dementia group counterparts.

Executive Function

A two-way, mixed (doubly-multivariate) MANCOVA was conducted to determine the effect of diagnosis group (between subjects, MCI vs. dementia) and time (within-subjects, prediction vs. evaluation) on anosognosia ratios in the domain of executive function, including the WCST and Trails B, while controlling for age (see Table 49 and Figures 39-40). As presented in Table 50, the between subjects main effect of diagnosis group indicated a significant effect on the combined DV (Pillai's Trace=.21, F(2,45)=6.04, p=.005, multivariate $\eta^2=.21$). The within-subjects main effect of time did not indicate a significant effect on the combined DV (Pillai's Trace=.00, F(2,45)=.09, p=.916, multivariate $\eta^2=.00$). The interaction effect between diagnosis group and time did not indicate a significant effect on the combined DV (Pillai's Trace=.03, F(2,45)=.64, p=.534, multivariate $\eta^2=.03$). The covariate did not significantly influence the combined DV (Pillai's Trace=.02, F(2,45)=.56, p=.578, multivariate $\eta^2=.02$).

Univariate ANOVA results are presented in Table 51, and indicated that Trails B anosognosia ratios were significantly effected by diagnosis group (F(1,44)=10.70, p=.002, partial $\eta^2=.19$), while WCST anosognosia ratios were not (F(1,44)=3.18, p=.081, partial $\eta^2=.06$). Comparison of Trails B anosognosia ratio means indicated that anosognosia ratios for MCI patients were lower than those of dementia patients, indicating that MCI

	_	Time									
Diagnosis		Predictio	n		Evaluation	on		Total			
Group	n Mean SD			п	Mean	SD	п	Mean	SD		
WCST											
MCI	24	-0.13	0.39	24	-0.27	0.37	48	-0.19	0.06		
Dementia	25	0.12	0.26	25	-0.17	0.37	50	-0.04	0.06		
Total	49	-0.01	0.35	49	-0.22	0.37	98	-0.12	0.04		
Trails B											
MCI	24	0.19	0.59	24	0.21	0.59	48	0.18	0.11		
Dementia	25	0.69	0.45	25	0.65	0.47	50	0.70	0.11		
Total	49	0.45	0.57	49	0.44	0.57	98	0.44	0.07		

Mean scores and standard deviations for the domain of executive function for patient prediction anosognosia ratios for MCI and dementia patients



Figure 39. Prediction and evaluation ratios (±SD) for MCI and dementia patients for the Wisconsin Card Sorting Test (WCST)



Figure 40. Prediction and evaluation ratios (±SD) for MCI and dementia patients for the Trail Making Test Part B (Trails B)

				Hypothesis	Error		
Effect		Value	F	df	$d\!f$	Sig.	η^2
Between	Intercept	0.07	1.71	2	45	0.193	0.07
	Age	0.02	0.56	2	45	0.578	0.02
	Diagnosis	0.21	6.04	2	45	0.005	0.21
Within	Time	0.00	0.09	2	45	0.916	0.00
	Time*Age	0.01	0.29	2	45	0.751	0.01
	Time*Diagnosis	0.03	0.64	2	45	0.534	0.03

Multivariate analysis of covariance analysis for the domain of executive function for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

Table 51

Univariate analysis of covariance analysis for the domain of executive function for patient prediction and evaluation anosognosia ratios for MCI and dementia patients

	Main Effect of Time			Main Effect of Diagnosis			Time x Diagnosis Interaction		
Measure	F(1, 46)	p	η^2	F(1,46)	p	η^2	F(1, 46)	p	η^2
WCST	0.06	.806	0.00	3.18	.081	0.06	0.82	.370	0.02
Trails B	0.09	.767	0.00	10.70	.002	0.19	0.70	.406	0.02

patients had the tendency to provide less favorable predictions of their performance

relative to their actual performance than the dementia group.

Hypothesis 4

Logistic Regression – Enter Method

A logistic regression was conducted using the enter method to determine if

predictive anosognosia ratios for all 20 neuropsychological measures across six cognitive

domains, and age were predictors of diagnostic group membership (MCI vs. dementia,

see Table 52). A test of the full model with all predictors against a constant-only model

was statistically significant, χ^2 (21, *N*=42)=58.129, *p*=.000, indicating that the predictors, as a set, reliably distinguished between patients diagnosed with MCI and those diagnosed with dementia. Regression results indicated the overall model fit of the 21 predictors was impressive, such that it predicted group membership with 100% accuracy (-2 Log Likelihood=.000, Hostmer and Lemeshow χ^2 (7, *N*=42)=.000, *p*=1.00). Results also indicated that anosognosia ratios for the 3MS, Trails A, Animals, CVLT IR, BD, CVLT DR, CVLT Rec, LMI, JLO, and WTAR contributed to differential diagnosis between MCI and dementia groups (*Exp*(*B*)=4.882E+070; *Exp*(*B*)=9.501E+039; 3.612E+038; *Exp*(*B*)=3.906E+11; *Exp*(*B*)=2.297E+31; *Exp*(*B*)=160739907; *Exp*(*B*)=76.999; 20.562; *Exp*(*B*)=9.843E+27; *Exp*(*B*)=5.239E+18, respectively).

Logistic Regression – Forward Method

A forward logistic regression was conducted to determine the most predictive set of anosognosia ratios for diagnostic group membership, using the same set of predictors as the first logistic regression (20 neuropsychological measures across six domains, and age). The anosognosia ratios for the 3MS and LMII were the only two predictors to enter the model (see Table 53). Regression results indicated the overall model of fit for the two predictors was questionable (-2 Log Likelihood=32.545, Hostmer and Lemeshow χ^2 (8, N=42)=18.421, p=.018) but was statistically reliable in distinguishing between diagnostic groups, χ^2 (1, N=42)=7.264, p=.007. Regression results also indicated that the model impressively predicted group membership, with 85% accuracy for the MCI group and 81.8% accuracy for the dementia group, for an overall accuracy of 83.3%. These results indicate that anosognosia ratios for the 3MS and LMII, in isolation of the rest of the anosognosia ratios, were significant predictors of diagnostic group membership and

reliably distinguished between MCI and dementia groups.

Measure	В	SE	Exp(B)	p			
Age	-1.02	4618.03	0.36	1.000			
General Cognitive Ability, Premorbid Function, and Effort							
3MS	1.63	1459.22	5.09	.999			
WTAR	0.64	885.24	1.89	.999			
DC	-1.13	1598.60	0.32	.999			
Attention, Concentration, and Processing Speed							
Trails A	0.92	865.57	2.51	.999			
DSF	-0.11	2274.33	0.89	1.000			
DSB	-0.20	1707.89	0.82	1.000			
SDMT	-0.21	293.19	0.81	.999			
Language							
FAS	-0.91	981.04	0.40	.999			
Animals	0.89	405.97	2.43	.998			
BNT	-0.01	1182.65	0.99	1.000			
Visuoperceptual and Visuoconstructional							
JLO	0.25	487.19	1.29	1.000			
BD	0.72	1155.55	2.06	1.000			
Memory							
CVLT IR	0.27	1086.87	1.31	1.000			
CVLT DR	0.19	533.32	1.21	1.000			
CVLT Rec	0.04	896.89	1.04	1.000			
CVLT FC	-0.91	735.45	0.40	.999			
LMI	0.03	918.30	1.03	1.000			
LMII	-0.08	813.39	0.92	1.000			
Executive Function							
WCST	-0.09	378.08	0.91	1.000			
Trails B	-0.05	1044.84	0.95	1.000			

Summary of simultaneous logistic regression analysis predicting diagnostic group membership

Summary of forward logistic regression analysis predicting diagnostic group membership

Measure	В	SE	Exp(B)	р
3MS	.082	.028	1.085	.003
LMII	.020	.009	1.021	.021

CHAPTER SIX

DISCUSSION

The current study sought to standardize the assessment of anosognosia using a clinically relevant, easily replicable protocol that could readily be incorporated into existing routine assessments of patients with cognitive complaints related to mild cognitive impairment and dementia diagnoses. In addition, investigation into the relationships between patient predicted performance, informant predicted performance, and actual performance were performed in order to assess the validity of current anosognosia assessment procedures using informant input as the benchmark for assessment of presence and severity of anosognosia (Evans et al., 2005; Flashman & McAllister, 2002; Hart, Seignourel, & Sherer, 2009; Sherer et al., 1998). The study also sought to investigate the relationship between informant and patient performance as they relate to actual performance, a methodology seldom used in current anosognosia literature (Fleming, Strong, & Ashton, 1996; Barrett et al., 2005). Emergent awareness, which is currently unexamined in anosognosia literature, was assessed by investigating the difference between predictions of performance and evaluations of performance before and after each neuropsychological measure, respectively, to examine changes in awareness after having completed a measure. Lastly, the ability to predict diagnosis based on patient predictions of performance relative to actual performance was assessed.

Hypothesis 1 – Patient Versus Informant Ratings

It was hypothesized that a significant difference would be present between patient and informant predictions of patient performance, such that patients would estimate their performance more favorably than would their respective informants. Analysis compared patient and informant prediction percentages, irrespective of actual performance, for each domain of neuropsychological function.

General Cognitive Ability, Premorbid Function, and Effort

Patient and informant ratings were significantly different for the domain of general cognitive ability, premorbid ability, and effort, with patients providing more favorable predictions than informants for a measure of general cognition (3MS). Interestingly, patients had the tendency to provide less favorable predictions on a measure of effort asking patients to quickly count a series of cards with dots on them (DC). It is possible that the difference seen between these two ratings is due to an effect of order, because the 3MS is given first in the neuropsychological battery and the DC is administered last, thus possibly reflecting a tendency to provide less favorable ratings throughout the course of the evaluation. Patients and informants provided similar predictions of patient ability to read a list of irregular words (WTAR). Thus, patients provided differentially favorable predictions of their general cognitive ability, but less favorable predictions of their ability to perform simple speeded counting tasks.

Attention, Concentration, and Processing Speed

Patients and informants provided significantly different ratings for the domain of attention, concentration and processing speed. Patients demonstrated the tendency to rate themselves more favorably on a measure of speeded visual attention (Trails A) than their informant counterparts. Patients and informants provided similar predictions of ability of verbal attention and working memory (DSF and DSB, respectively) and psychomotor processing speed (SDMT). Thus, patients provided differentially favorable predictions of their performance on speeded visual attention tasks (Trails A), but not for tasks reflective of verbal attention and concentration or psychomotor processing speed.

Language

Both groups provided similar ratings for the language domain. There were no differences found in patient and informant ratings on phonemic and semantic fluency ability (FAS and Animals, respectively) or confrontational naming (BNT). Thus, patients did not provide differentially favorable predictions of their language skills.

Visuoperception and Visuoconstruction

Both groups provided similar ratings for the visuospatial domain. No differences were found in patient and informant predictions of ability for visuoperceptual and speeded visuoconstructional ability (JLO and BD, respectively). Thus, patients did not provide differentially favorable predictions of their visuospatial skills.

Memory

Patient and informant ratings were found to be significantly different for the memory domain. Patients more favorably rated their ability to recognize previously presented discrete information in forced choice format (CVLT FC). Interestingly, patients were found to rate themselves less favorably on delayed contextual memory for stories (LMII) than their informant counterparts. No differences were found between patient and

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informant predictions for immediate or delayed recall of discrete information (CVLT IR, CVLT DR, respectively), yes/no recognition ability for previously presented discrete information (CVLT Rec), or immediate recall of contextual information (LMI). Thus, patients had the tendency to provide differentially more favorable predictions of their forced choice recognition ability, but differentially less favorable predictions of their delayed contextual memory. In addition, they did not provide differentially favorable predictions of ability on other facets of memory, including discrete immediate, delayed, and recognition memory, or immediate contextual memory.

Executive Function

Lastly, the groups were found to provide significantly different ratings for the domain of executive function. Patients provided differentially more favorable predictions of their performance in novel, ambiguous problem solving (WCST) as well as in speeded mental set shifting (Trails B). Thus, on measures of executive function, patients had the tendency to provide differentially favorable predictions of their performance than their informant counterparts.

Conclusions

In sum, the hypothesis that patients would provide significantly more favorable predictions of their neuropsychological performance ability received mixed support across cognitive domains, with support in the areas of general cognitive ability, speeded visual attention, forced choice recognition memory, and executive function as a whole.

The reverse pattern of patient and informant ratings between the 3MS and Trails A is interesting, given that patients provided more favorable responses than informants when asked about their general cognitive ability, while the reverse was seen when asked to serially connect 25 numbers on a page, a measure of speeded visual processing. It is likely that the manner in which the 3MS measures "general cognitive ability" is conceptually different than what is implied when the lay public thinks about general cognitive skills, and thus the way in which the question was worded may not have translated well to the general public. The reverse pattern of ratings seen in Trails A also provides evidence for the idea that patients do not consistently provide more favorable predictions of their performance than their informant counterparts. This was also seen on a measure of delayed memory for contextual information. The fact that patients failed to consistently rate themselves more favorably across all measures, as well as across all domains of function, falls in line with research previously conducted, however the domains within which anosognosia was apparent differed. Barrett and colleagues (2005) found that anosognosia was present primarily for visuospatial function in Alzheimer's dementia patients. It is possible that the heterogeneous diagnostic sample employed in the current study diluted the impact of anosognosia seen in dementia, such that the pattern of anosognosia seen in their study is specific to Alzheimer's dementia and not dementia as a whole. The current study found that patients and informants provided commensurate predictions within the domains of language and visuospatial skills.

Previous anosognosia research traditionally measures anosognosia for memory as a single construct, but neuropsychological tests assess different aspects of memory, which would make it difficult for patients to provide reliable predictions and evaluations

of their performance on memory measures (Barrett et al., 2005; Carr, Gray, Baty, & J. C. Morris, 2000; Derouesné et al., 1999). The current study conducted multistep ratings of different aspects of memory, parallel with the method of measuring memory skills used in traditional neuropsychological assessment. As such, patients were asked for prediction and evaluation ratings for immediate and delayed portions memory, as well as yes/no recognition memory and forced choice memory for previously presented information. Interestingly, differences in patterns of responding were seen across memory measures, with patients rating themselves more favorably on forced choice recognition paradigms than informants, and less favorably on delayed recall for contextual information. No differences were found for immediate, delayed, and yes/no recognition memory for discrete information or on immediate memory for contextual information. In addition, patients rated themselves less favorably on delayed recall for contextual information than informants. Thus, "memory" is clearly not a unitary construct, and more fine-grained analysis of anosognosia within this domain is warranted.

Additionally, the fact that patients rated themselves less favorably on measures of delayed recall for contextual information relative to their informant counterparts speaks to the real world frustration that many family members and caregivers of MCI and dementia patients face. Informants predict, and likely expect, that patients remember more contextual information, or presented in story format as opposed to discrete lists, than patients predict they will. Thus, it is likely that these family members and caregivers are providing information to patients with this idea in mind, which likely leads to a disconnect between patient and caregiver expectations of patient ability. In addition, the current analysis also revealed that patients tend to overestimate their ability to solve

problems, as discussed below, and likely lack the insight to make appropriate compensations for their memory ability.

Lastly, patients rated themselves consistently more favorably on measures of executive function. This may be related to the fact that these types of measures, by definition, are novel and require the patient to perform tasks that are not typically asked of them. Thus, patients have little experience with these types of tasks to provide a basis for prediction of their ability. This is often clinically evident during the neuropsychological evaluation process, such that patients are often surprised and/or perplexed by the difficulties they encounter when attempting these tasks.

Hypothesis 2 – Accuracy of Ratings

Research to date has yet to compare the accuracy of patient predictions of their performance or informant predictions of patient performance relative to actual performance ability. It was hypothesized that there would be significant differences between information sources (patient prediction, informant prediction, and actual performance), with informant predictions more accurate than patient predictions. Accuracy of patient and informant predictions were assessed relative to patient's actual performance on each measure. In addition, it was hypothesized that there would be significant differences between informant predictions based on diagnosis groups, such that the accuracy of informant ratings would be better for MCI patients than dementia patients. Lastly, domain specific awareness was hypothesized to be accurate for memory ability, but poor for visuospatial ability for both patient and informant predictions.

actual performance), as well as actual performance for MCI and dementia patients for each domain of neuropsychological function.

General Cognitive Ability, Premorbid Function, and Effort

Significant differences were found between information sources and diagnosis group for the domains of general cognitive ability, premorbid function, and effort. An interaction effect was found for a measure of general cognitive ability (3MS), with significant overall differences observed for both source of information and diagnosis. For both MCI and dementia groups, informants significantly underestimated patient general cognitive ability relative to actual performance, and they predicted dementia patient performance significantly lower than MCI patient performance. Lastly, MCI patients performed significantly better than their dementia counterparts. Thus, patients were actually better predictors of their performance than informants for patient general cognitive ability, regardless of their diagnosis. It is possible that this finding speaks to the limited ecological validity of neuropsychological assessment measures in their sensitivity to everyday functional capacity. While patients may, indeed, have areas of preserved cognitive function, if their ability to compensate for general areas of weakness is impaired, the likelihood of informants to be able to accurately predict their actual ability is limited.

For a measure of premorbid function (WTAR), an interaction effect was seen between source of information and diagnosis, with significant overall differences also seen between information sources. No significant overall differences were seen for diagnosis group. Patients and informants both underestimated patient actual performance,

with informants predicting significantly lower performance for dementia patients than MCI patients. There was no difference in performance between MCI and dementia patients on this measure. Thus, MCI and dementia patients had similar levels of estimated premorbid function, but both were inaccurate predictors of their performance.

On a measure of effort (DC), significant overall differences were seen for source of information and diagnosis group. No interaction effect was observed. Patients and informants provided significantly different predictions, with patient predictions significantly lower than informant predictions. Both patients and informants predictions were significantly lower than actual performance. Informants rated MCI patient performance more favorably than dementia patient performance, and MCI patients performed better than dementia patients. Thus, both patients and informants were inaccurate predictors of performance. As discussed previously, it is possible that this finding is reflective of emergent awareness across the course of the entire evaluation, since the DC is given last in the battery. Thus, patients may be more cautious in their ratings on this measure by virtue of having performed several other measures prior.

Attention, Concentration, and Processing Speed

Significant differences were found between source of information and diagnosis group for the domain of attention, concentration, and processing speed. On a measure of visual attention, scanning, and processing speed (Trails A), there was a significant interaction between source of information and diagnosis, with significant overall differences for both source and diagnosis. Patient and informant predictions were significantly different, with patients providing more favorable responses. Significant

differences were found between informant predictions and actual performance, with informants underestimating patient performance. Informants predicted significantly lower performance for dementia than MCI patients, with dementia patients actually performing worse on this task than MCI patients. Thus, patients were actually better predictors of their ability on Trails A than informants, regardless of their diagnosis.

On a measure of simple verbal attention (DSF), significant differences were seen for information source. No significant interaction effect or overall difference for diagnosis group was observed. Patient and informant ratings were similar to one another, and both were significantly lower than actual performance. Informants rated MCI patient performance more favorably than dementia patients, and MCI patients actually performed better than dementia patients. Thus, both patients and informants were poor predictors of patient performance on this measure of simple attention.

On a measure of verbal attention and working memory (DSB), no significant interaction effects were found between information source and diagnosis, and nor overall differences for source of information or group were found. Thus, patients and informants were accurate predictors of actual performance, and MCI and dementia patients performed similarly on this working memory task.

On a measure of processing speed (SDMT), a significant interaction effect was seen between information source and diagnosis group, and significant overall differences were seen between groups. Both patients and informants provided similar predictions, and both were accurate predictors of actual performance. MCI patients performed significantly better than their dementia counterparts. Thus, both patients and informants were accurate predictors of patient performance on this processing speed task.

Language

Significant differences were found between sources of information as well as between diagnosis group. On a measure of phonemic verbal fluency (FAS), informants predicted less favorable performance for dementia patients than MCI patients. On a measure of semantic verbal fluency (Animals), dementia patients received less favorable ratings than their dementia counterparts across all sources of information, including actual performance. In addition, informants rated dementia patients significantly less favorably than MCI patients. Lastly, on a measure of confrontational naming (BNT), informants predicted that patients would perform significantly worse than they actually did perform, regardless of diagnosis. Dementia patients, overall, received lower predictions than MCI patients regardless of the source of information. Lastly, dementia provided predictions that were significantly lower than the MCI patients, and dementia patients did, in fact, perform worse than their MCI counterparts. Thus, patients were more accurate predictors of their actual semantic fluency ability than informants, while the reverse was true for confrontational naming ability.

Visuoperception and Visuoconstruction

Significant differences were found between sources of information and diagnosis group for the visuoperception and visuoconstruction domain. On a measure of visuoperception (JLO), significant interaction was found between information source and diagnosis group, with significant overall differences seen between diagnosis groups. Both informant and patient predictions were similar to actual performance. Informants predicted significantly lower performance for dementia patients than MCI patients, and

dementia patients performed significantly worse than their MCI counterparts. Thus, both patients and informants were accurate predictors of actual performance for both MCI and dementia patients.

On a measure of visuoconstruction (BD), a significant interaction was found between source of information and diagnosis, and significant overall differences were seen between information sources and between diagnosis groups. Patients and informants provided similar predictions, and both informant and patient predictions significantly overestimated patient actual performance. Informants provided significantly lower performance for dementia patients than MCI patients, and dementia patients actually performed significantly worse than their MCI counterparts. Thus, both patients and informants were poor predictors of visuoconstructional ability.

Memory

Significant differences were found between source of information and diagnosis group for the memory domain. On a measure of immediate memory for discrete information (CVLT IR), a significant interaction effect was found between source of information and diagnosis group, with significant overall differences found between sources of information as well as between diagnosis groups. Patient predictions were significantly lower than their actual performance, regardless of their diagnosis. MCI patients performed significantly better than their dementia group counterparts. Thus, patients were poor predictors of their immediate memory ability for discrete information regardless of their diagnosis, while informant ratings were accurate for both groups.

On a measure of delayed recall for discrete information (CVLT DR), a significant interaction effect was found between source of information and diagnosis group, with significant overall differences found between sources of information but not between diagnosis groups. Patients and informant predictions were similar and both significantly lower than patient actual performance. MCI patients performed significantly better than dementia patients. Thus, both patients and informants were poor predictors of patient performance, regardless of diagnosis.

On a measure of recognition (CVLT Rec), a significant difference was found between sources of information. No significant interaction effect or overall differences between diagnosis groups were observed. Both patients and informants significantly underestimated patient ability relative to their actual performance. Additionally, MCI patients performed better than dementia patients. Thus, both patients and informants were poor predictors of patient ability, regardless of diagnosis.

On a measure of forced choice recognition (CVLT FC), significant differences were found for both source of information and diagnosis. No significant interaction effect was observed. Patient and informant predictions were significantly different. Though both informants and patients significantly underestimated patient performance for both MCI and dementia patients, the extent to which informants underestimated performance was significantly lower than that of patients'. MCI patients performed significantly better than dementia patients. Thus, both patients and informants were poor predictors of patient's forced choice recognition ability.

On a measure of immediate memory for contextual information (LMI), a significant interaction effect between source of information and diagnosis group was seen,

with significant overall differences seen between source of information. There was no significant overall difference observed between diagnosis groups. Patient and informant predictions were similar regardless of diagnosis, with both significantly overestimating patient actual performance. MCI patients performed significantly better than dementia patients. Thus, patients and informants were both poor predictors of patient actual ability for immediate memory for contextual information.

On a measure of delayed memory for contextual information (LMII), a significant interaction was seen between source of information and diagnosis group, and a significant overall difference was seen between information sources. No significant overall difference was observed in between diagnosis groups. Though patient and informant ratings were similar, informants significantly overestimated patient performance relative to actual performance, and predicted significantly better performance in MCI patients than in dementia patients. MCI patients performed significantly better than dementia patients. Thus, patients were more accurate predictors of their performance on measures of delayed contextual memory than informants, with informants predicting better performance than the patients actually performed.

Executive Function

Significant differences were found between source of information and diagnosis group for the executive function domain. On a measure of novel, ambiguous problem solving (WCST), a significant interaction effect was found between sources of information and diagnosis group, with significant overall differences between sources of information. No significant overall differences were seen between diagnosis groups.

Patient and informant predictions were significantly different, with patient predictions similar to actual performance and informant predictions significantly lower than actual performance. Dementia patients provided more favorable responses than their MCI counterparts, whereas informant predictions were more favorable for MCI patients than dementia patients. MCI patients actually performed similarly to dementia patients. Thus, patients were better predictors of their actual performance than informants were, with informants underestimating patient performance on novel, ambiguous problem solving.

On a measure of speeded mental set shifting (Trails B), a significant overall effect was found for source of information, and a significant overall effect was also found for diagnosis group. No interaction effect was observed. Informants and patients provided similar predictions, but patient predictions were significantly higher than actual performance. Patient predictions were similar regardless of diagnosis group, but informants had the tendency to provide more favorable estimations for MCI patients than dementia patients. MCI patients performed better than dementia patients. Thus, patients were more accurate predictors of performance than informants for tasks requiring speeded mental set shifting.

Conclusions

In sum, the hypothesis that informants were more accurate predictors of patient performance received little support. Out of 20 cognitive measures, informants were only more accurate predictors of patient performance on two, immediate memory for discrete information and confrontational naming. On five measures, informant and patient predictions were both commensurate with actual performance, including measures of

auditory working memory, psychomotor processing speed, phonemic verbal fluency, and a measure of visuoperception.

On seven measures, informant and patient predictions were equally poor estimations of patient ability, with both sources of information underestimating actual patient performance. These measures included a measure of premorbid ability, a measure of effort, a measure of simple auditory attention, a measure of visuoconstruction, and three measures of memory (delayed recall for discrete information, yes/no recognition ability, and forced choice recognition ability). On a measure of immediate memory for contextual information, informant and patient predictions were equally poor estimations of patient ability, with both sources overestimating actual performance.

Importantly, on six measures, informants were less accurate predictors of patient ability than patients were. These measures included measures of general cognitive ability, visual attention, semantic verbal fluency, and delayed recall of contextual information, novel problem solving, and speeded mental set shifting. Of note, informants provided more favorable predictions than did patients on both immediate and delayed memory for contextual information.

This set of findings provides consistent, potentially alarming evidence for the notion that informant reports of patient ability are not universally accurate, and in fact are more often inaccurate in relation to actual patient performance. The underlying explanation for this discrepancy is likely multifactorial and dependent upon a number of factors, including the relationship between the informant and patient (i.e. child vs. spouse vs. caregiver), the amount and nature of time spent with the patient (i.e. living together vs. visiting regularly vs. visiting on holidays), the cognitive status of the informant, and the

context within with the initial referral was placed that may create bias with respect to the informant's perception of the patient's ability. Thus, the current trend in anosognosia literature across neurological diagnoses to use informant ratings as the "benchmark" for assessing anosognosia is problematic (Evans, Sherer, Nick, Nakase-Richardson, & Yablon, 2005; Flashman & McAllister, 2002; Hart, Seignourel, & Sherer, 2009; Sherer et al., 1998). In fact, the current study showed that patients are often better reporters of their own ability than informants are, regardless of the severity of their diagnosis (MCI vs dementia). Thus, a patient could potentially be evaluated as having anosognosia when, in fact, their informant is providing unjustifiably low reports of their function, while their own reported functioning is actually commensurate with their actual function. It is important to note that the current study is investigating the accuracy of reports of neuropsychological function, while informants may be more focused on functional ability. This is likely an important factor to consider when formulating anosognosia measures, such that informant predictions are relative to real world functionality, not neuropsychological performance. Lastly, this analysis provides evidence for the superior sensitivity of discrepancy between patient predictions and actual performance than that of the discrepancy between informant and patient predictions in determining anosognosia of cognitive ability.

It is certainly possible, and logically conceivable, that informant ratings are negatively biased just by the nature of their relationship to the patient. Clinically, informants are typically spouses or other family members that are intimately connected to the patient, and often also have the responsibility of "filling in the gaps" with regard to patients' everyday functioning, both of which can naturally negatively skew their view of

patient ability. This idea is supported by the fact that informant ratings are consistently lower for dementia patients than MCI patients, even on tasks where their actual performances are commensurate with one another.

Hypothesis 3 – Emergent Awareness by Cognitive Domain

Emergent awareness has not been systematically studied in formal neuropsychological assessment paradigms. It was hypothesized that emergent awareness would vary across cognitive domains, with more emergent awareness seen for tasks allowing for physical manipulation of objects and/or verbal feedback from the examiner (i.e. BD and WCST, respectively). It was additionally hypothesized that this emergent awareness would be greater in MCI patients than dementia patients. Data was analyzed using anosognosia ratios of patient predicted performance to actual performance taken prior to administration of each measure, and anosognosia ratios of patient evaluations of performance to actual performance taken subsequent to administration of each measure.

General Cognitive Ability, Premorbid Function, and Effort

No significant differences were found between time of rating for anosognosia ratios for measures of cognitive ability, premorbid function, and effort, though significant differences were found between diagnostic groups. On a measure of general cognitive ability, MCI patients had the tendency to provide less favorable predictions and evaluations of their performance relative to their actual performance in comparison to the dementia group. Thus, MCI patients significantly underestimated their ability in comparison to their dementia counterparts, which may be indicative of a general increased sensitivity to their general cognitive function than dementia patients.

Attention, Concentration, and Processing Speed

No significant differences were found between time of evaluation for anosognosia ratings for anosognosia ratios for measures of attention, concentration, and processing speed, but significant differences were found between diagnostic groups. On measures of speeded visual attention, working memory, and processing speed, MCI patients rated themselves less favorably both pre and post testing than dementia patients, which may also be indicative of a general increased sensitivity to their functioning in these areas relative to dementia patients.

Language

No significant differences were found between time of evaluation and diagnosis group for anosognosia ratios for measures of language ability. Thus, there were no differences between pre and post ratings for MCI and dementia patients relative to actual performance on measures of language.

Visuoperception and Visuoconstruction

No significant differences were found between time of evaluation for anosognosia ratios for measures of visuoperception and visuoconstruction, but there was a significant difference between diagnostic groups. On both measures, MCI patients had the tendency to underestimate their performance relative to their actual performance, regardless of the time in which the rating was collected.

Memory

No significant differences were found between time of evaluation for anosognosia ratios for measures of memory, but there was a significant effect between diagnostic groups. On a measure of immediate recall of discrete information, as well as immediate and delayed memory for contextual information, MCI patients had the tendency to provide less favorable ratings of their performance, regardless of the time in which the rating was solicited, compared to their dementia counterparts. This may also be indicative of an increased sensitivity to their cognitive function in this area.

Executive Function

No significant differences were found between time of evaluation for anosognosia ratios for measures of executive function, but significant effects were found between diagnostic groups. On a measure of speeded mental set shifting, MCI patients had the tendency to provide less favorable ratings of their performance, regardless of time, than their dementia counterparts.

Conclusions

In sum, there was no evidence of emergent awareness across time (pre and post assessment) in any cognitive domain assessed for either MCI patients or dementia patients. This is inconsistent with previous research (Barrett et al., 2005) and the nature of the lack of appreciable differences is unclear. The fact that previous research typically uses Likert scales, requiring patients to provide ordinal responses, and the current study employed visual scales, where patients provided their responses along an unmarked line, led to difficulty quantifying meaningful change before and after assessment. In other words, Likert scales provide a structural context in which to provide responses, with clearly delineated markers between ratings that may lend themselves easier to making appreciably distinct ratings pre and post assessment. The nature of the currently employed scales was to release the patient from that very structure imposed by Likert scales, but may have left the determination of "appreciable changes" between prediction and evaluation ratings to the discretion of the respondent. Thus, while a one-inch difference in ratings for one patient may mean the same decrement in performance that a three-inch difference in ratings may mean to a different patient.

A second influencing factor may be the confrontational nature of the prediction and evaluation paradigm used in the current study. Since patients provided their prediction and evaluation responses on the same sheet of paper, they are confronted with their predicted performance ratings at the time that they are asked to provide their evaluation of performance ratings. Previous studies lack the confrontational nature in their pre and post assessment, which may also contribute to the disparity in results seen across studies (Evans, Sherer, Nick, Nakase-Richardson, & Yablon, 2005; Flashman & McAllister, 2002; Hart, Seignourel, & Sherer, 2009; Sherer et al., 1998).

There was, however, significant effects of diagnostic group across all domains except for language, with MCI patients consistently providing less favorable ratings of their performance relative to their actual performance, regardless of the time in which the

ratings were collected. This provides evidence for the fact that MCI patients are anosognostic with regard to their cognitive function in the negative direction, such that they consistently underpredict and underevaluate their actual performance. However, the hypothesis that emergent awareness would differ across cognitive domains received no support.

Hypothesis 4 – Diagnostic Utility of Anosognosia Assessment

It was hypothesized that measures of anosognosia across the entire evaluation would be reliably predictive of group membership, and thus diagnostically informative. It was also hypothesized that anosognosia measures of memory would be the best predicting measures of diagnostic group membership.

Logistic Regression – Enter Method

In order to investigate the possibility of predicting diagnosis group based on emergent awareness over the course of the evaluation, a logistic regression was performed using the enter method and force entering all 21 anosognosia prediction ratios for neurocognitive measures and age for both MCI and dementia patients. Statistical analyses revealed that MCI and dementia diagnoses could be reliably predicted based on anosognosia ratios with 100% accuracy for both groups.

Logistic Regression – Forward Method

In order to investigate the possibility of predicting diagnosis group based on predictive anosognosia ratios for specific measures, a second logistic regression was

performed using for forward method, such that only predictors that significantly accounted for variance in anosognosia ratios entered the model. The 21 anosognosia prediction ratios for neurocognitive measures and age were entered into the analysis. Two iterations were completed, with the anosognosia prediction ratios for general cognitive function (3MS) and delayed contextual memory (LM II) remaining in the model. MCI group membership was predicted with 85% accuracy, and dementia group membership was predicted with 81.8% accuracy based on the model. Thus, group membership can be reliably predicted using only predictive anosognosia ratios for general cognitive function measures and delayed contextual memory. With respect to predictive anosognosia ratios for general cognitive ability, MCI patients had the tendency to provide less favorable estimations of their performance relative to their actual performance, while dementia patients provided more accurate predictions relative to their actual performance ability. For delayed contextual memory, MCI patients had the tendency to provide slightly less favorable predictions relative to their actual ability, while dementia patients had the tendency to provide largely more favorable predictions of their performance relative to their actual performance.

Conclusions

No prior published study to date has utilized regression models to predict group membership based on measures of anosognosia. The current regression analyses provide both strong and convincing evidence for the clinical relevance of anosognosia assessment in diagnosis of MCI and dementia. The fact that, by using the 21 prediction anosognosia ratios, it was possible to predict group membership in the current sample with 100%

accuracy speaks to the clinical sensitivity of discrepancies between patient predicted and actual performance and the importance of including awareness assessment in the clinical standard of care for neuropsychological work-ups secondary to complaints of dementiarelated symptomatology.

In addition, the predictive power of two single measures of anosognosia, such that MCI and dementia diagnosis was predicted with such high rates of accuracy in the current sample is equally compelling. It is conceptually fitting that awareness of ability on a measure of general cognitive ability, in conjunction with that of a measure of long term contextual memory, would accurately predict diagnostic group membership. Since general cognitive status is highly related to patient functional ability, and functional ability is the single differential diagnostic criterion between MCI and dementia diagnosis, it is parsimonious that this predictive anosognosia rating would explain a large amount of the variance between diagnostic groups. Similarly, since contextual memory measures are often viewed as increasingly complex in relation to discrete memory measures, and that memory complaints also constitute a diagnostic criterion in both MCI and dementia evaluations, it is also logical that this measure of predictive anosognosia would also explain a large amount of variance between diagnostic groups.

General

The current study provides evidence for the notion that anosognosia is multifaceted construct, and not an "all or none" phenomenon, such that patients display anosognosia for specific areas of cognitive function while maintaining preserved awareness in other domains of cognitive function. It also provides novel evidence to

show that informant predictions of patient ability are not uniformly accurate, and that patient predictions of their own ability may be more accurate measures of their actual ability. Importantly, the current study provides evidence for the fact that, while there is much debate in the literature regarding the presence of anosognosia in MCI populations, that there were numerous areas measures of awareness across multiple cognitive domains that failed to show differences between patient and informant ratings, meaning to say that MCI and dementia patients showed similar levels of awareness and similar levels of anosognosia across different areas of cognitive function. Lastly, the current study speaks to the clinical utility and unique value of anosognosia assessment in MCI and dementia evaluation, as well as the predictive power of anosognosia ratings in isolation.

While the current study provides novel and informative results, there are a number of limiting factors that could potentially confound its results. First, the relatively small sample size used in the study (n=49) could potentially inadvertently accentuate any significant differences between groups. Also, diagnostic categories were collapsed across different types of MCI and dementia, which lead to heterogeneous populations within each group. By increasing the overall study sample, thus bolstering the number of contributing diagnoses, would be helpful in providing finer distinctions within each overarching diagnostic group, and allow for more detailed analysis investigating differences between various types of MCI and dementia with well-documented differences in neuropathology. Second, the current study did not employ counterbalancing techniques to prevent the impact of order of test administration on the results. While unlikely, it is conceivably possible that the nature of the results are related to the order in which neuropsychological tests were administered, independent of the

nature of the tests themselves. Third, given the nature of the populations studied, the age range of the sample was relatively restricted in comparison to the lifespan. As such, it is possible that the current results are reflective of simple cohort effects between elderly adults, who are also more likely to be diagnosed with dementia, and their younger counterparts, who are more likely to be diagnosed with MCI. Thus, it is possible that the nature of the upbringing of these different generations of patients may lend themselves to simple cohort differences related to awareness, introspection, education, and employment.

The horizon for future research is plentiful, with novel evidence providing new pathways to pursue. First, future research could conduct a similar study and employ a healthy control group, which would provide for additional comparisons. It would also be helpful to include clinician predictions of patient performance using the current paradigm, which is also used in current literature as a benchmark for evaluating patient awareness. It would be interesting to investigate the cognitive status of the informant as well, as they relate to more similar or more discordant ratings of patient ability. Second, given that the current study found informants to be poor predictors of patient performance, future research could also investigate differences in accuracy across different types of informants, such as spouses, children, and caregivers. Given the various relationships informants may have with the patient, it is possible that the bias associated with patientinformant interactions is qualitatively different depending on who the informant is. Lastly, it would be interesting to systematically investigate the relationship between levels of anosognosia in MCI patients and their associated diagnostic trajectory to see if anosognosia evident early on is predictive of conversion to dementia.

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APPENDIX A

LLUMC FREQUENTLY ASKED QUESTIONS SHEET

Loma Linda University Medical Center Department of Neuropsychology

FAQ Sheet

Welcome, I am Dr. Travis Fogel, Director of the Department of Neuropsychology. You have been referred by your physician for neuropsychological assessment. If you are like most individuals, you have many questions about our services. I have prepared the following Frequently Asked Questions (FAQ) and am hopeful that this will answer many of your questions.

What is your address?

Department of Neuropsychology Outpatient Rehabilitation Center 11406 Loma Linda Drive, South Entrance Loma Linda, CA 92354

How can I reach you?

For appointments, please contact our scheduler, Melissa Abraham at (909) 558-4000, ext. 66142. If you need to reach me directly, you can also call my direct office line at (909) 558-4000, ext. 66105. You may also reach me by email at: tfogel@llu.edu.

Where are your offices located?

Our offices are located in the Outpatient Rehabilitation Center (ORC) or Ambulatory Care Services. This is a single-story peach-colored building about one mile east of the main hospital. Below are directions from the 10 Freeway. Our reception area is at the South Entrance of the building (middle of the building facing away from Barton Road).

DIRECTIONS FROM LOS ANGELES:

10 Freeway East

Take Mountain View Ave. exit

Turn RIGHT onto Mountain View Ave. (stay on this for about 2 miles)

Turn RIGHT onto Barton Rd. at light (as you turn, a gas station will be on your right) Turn LEFT onto Loma Linda Dr. (this will be the first traffic light after taking a right on Barton)

Turn RIGHT into the Outpatient Rehabilitation Center (a peach-colored single-story Spanish style building immediately before some condo-type homes -- about 400 yards after turning onto the road)

Enter SOUTH ENTRANCE (it will be facing away from Barton Road toward a small wall with condos on the other side)

Check in with the receptionist and I will be paged.

DIRECTIONS FROM PALM SPRINGS:

10 Freeway West

Take Mountain View Ave. exit

Turn LEFT onto Mountain View Ave. (stay on this for about 2 miles)

Turn RIGHT onto Barton Rd. at light (as you turn, a gas station will be on your right) Turn LEFT onto Loma Linda Dr. (this will be the first traffic light after taking a right on Barton)

Turn RIGHT into the Outpatient Rehabilitation Center (a peach-colored single-story Spanish style building immediately before some condo-type homes -- about 400 yards after turning onto the road)

Enter SOUTH ENTRANCE (it will be facing away from Barton Road toward a small wall with condos on the other side)

Check in with the receptionist and I will be paged.

Why was I referred to see a neuropsychologist?

Your physician has referred you for a neuropsychological evaluation. This evaluation may be of help in:

- \Box finding possible problems with your brain functioning,
- \Box forming a diagnosis,
- □ defining your thinking skill strengths and weaknesses,
- □ guiding treatment for your personal, educational or vocational needs,
- \Box making relevant recommendations to your health care provider(s), and/or,
- □ documenting possible changes in your functioning over time.

What is a neuropsychologist?

A neuropsychologist is a licensed psychologist specializing in the area of brain-behavior relationships. Although a neuropsychologist has a doctoral degree in psychology, he or she does not just focus on emotional or psychological problems. The neuropsychologist has additional training in the specialty field of clinical neuropsychology. That means a neuropsychologist is educated in brain anatomy, brain function, and brain injury or disease. The neuropsychologist also has specialized training in administering and interpreting the specific kinds of tests included in your neuropsychological evaluation. As a part of the required education, a neuropsychologist also has years of practical experience working with people who have had problems involving the brain.

What will happen during my first appointment and what should I bring?

The first appointment will consist of a 60-90 minute clinical interview. Some very brief preliminary testing also may be conducted at the very end of the interview in order to help develop the subsequent test battery. You are more than welcome to bring someone with you to this appointment. Such a person can provide valuable supplementary information about you (for example, their observations regarding your condition or clarification of dates).

It is also very helpful to prepare a list of your medications, current medical providers, and chronology of events. This chronology might include the date of onset of your condition, dates of any hospitalizations, and dates of any significant changes in your symptoms. It is also helpful to obtain copies of any medical records you might possess. I can make a copy of these records at the office. You do not need to bring any MRI films with you. Please be on time to your appointment. Unlike physicians who may have many overlapping appointments during their clinic hours, we block off the scheduled time for you alone. That is, the time slot is reserved for you and you alone. As such, we are unable to extend the appointment beyond the allotted time if you arrive late.

What will happen after the first appointment?

After the first appointment, if it is deemed appropriate, you will be scheduled for neuropsychological testing, to occur at a later date.

How long does the testing last?

The length of this testing can vary greatly and depends on the nature of the referral question and many other factors. Sometimes the evaluation will last only a couple of hours. More frequently, the evaluation will last all day (6-8 hours). As examples, the test battery for persons referred to see me as part of their pre-surgical epilepsy evaluation requires about 6-8 hours. Persons referred for dementia evaluations may only require 1-3 hours. For longer test batteries, we may divide testing into a couple of different days.

What will happen during testing?

Testing involves taking paper-and-pencil measures or answering questions of a wide range of mental abilities including your memory, attention and concentration, processing speed, language skills, visuospatial skills, cognitive flexibility, planning, and organization. Questionnaires may also be given to assess your coping skills. I also may give you questionnaires to provide persons who know you well in order to obtain their impressions about certain aspects of your neuropsychological functioning.

Is there anything I need to do to prepare for the day of testing?

Get plenty of rest. Otherwise the testing may simply reflect how tired you were rather than your optimal performance. Bring your glasses and hearing aids if you typically use them. Take your medications as you normally would. Dress comfortably. Bring a jacket or sweater as temperatures can vary. Feel free to also bring drinks or snacks. We will take rest breaks periodically. This is not an endurance contest; I want you to be at your best so that I can obtain accurate and meaningful results. If testing will last all day, we will break for about one hour for lunch. There are restaurants within walking distance. Lastly, if someone drives you to your appointment, there is no need for them to stay. Third party observers are not allowed to be present in the room during testing, and they will be asked to wait outside. It is perfectly fine for persons to drop you off and leave. However, I would recommend that you have some way to reach them in case we end early.

Who will administer the battery?

Typically I administer the test battery. I also have three doctoral neuropsychology interns whom may perform portions of the battery. These are individuals whom I have selected to train with me for one year as part of their doctoral training through Loma Linda University's Department of Psychology.

What happens after the testing is completed?

After you complete testing, in some ways, my work just begins. The test data will be scored and interpreted. A formal neuropsychological evaluation report will then be prepared. Included in this report will be a summary of your history of illness, pertinent background, test performance, interpretation of findings, and recommendations. This report will then be sent to the physician who referred you. Typically you and I will then meet for a formal feedback session to review the results and my recommendations. This feedback session lasts about an hour. You are welcome to bring whomever you would like to this feedback session.

I look forward to working with you and believe you will find the evaluation experience rewarding.

Travis G. Fogel, Ph.D., ABPP-CN Neuropsychologist, PSY 17746 Director, Department of Neuropsychology and Rehabilitation Psychology Assistant Professor, Physical Medicine and Rehabilitation Loma Linda University Medical Center OFFICE: (909) 558-4000, ext. 66105 FAX: (909) 558-6418 EMAIL: tfogel@llu.edu

Loma Linda University Medical Center Department of Neuropsychology

Here is a checklist of things to bring with you to your first appointment.

- \Box List of medications.
- □ Copies of available medical records (I can make copies of your copies during the interview).
- □ List of current and past medical providers (name, address, phone number, fax, general purpose for seeing them)
- \Box Description of medical condition.
- \Box List/description of symptoms.
- □ Chronology of major events (e.g., date of injury or diagnosis, hospital admissions, change in symptoms, etc.).
- Date of Appointment: _____

Time of Appointment: _____

Appointment with: Dr. Travis Fogel_____

Referred By: _____

APPENDIX B

DEMOGRAPHIC AND PATIENT HISTORY VARIABLES COLLECTED

Marital Status:				Ethnicity:			Language:	
Years Married:				Education:			D.Hand:	
Informant Present: DNo DYes – If yes, Relationship to Patient? Years Known?								
Subjective complaints Yes No	If yes, d	lescribe.			Collateral complaints Yes	If yes, descript from whom?	tion of complaints	and
			1			Onset:		
Current Living Arrangement			 □ Alone □ Alone w/Nursing □ w/Spouse □ w/Children □ w/Roommate □ Assisted Living □ Skilled Nursing Facility □ Other (Please List) 					
Premorbid Occupation								
PERSONAL MEDICAL HISTORY								
TBI/LOCIf yes, how many? Severity?								
□Yes □No								
Stroke	Stroke If yes, how many? Residual effects?							
□ Yes □ No								
Medical Dia	ignoses	If ye	s, please list:					
☐ Yes ☐ No								
Surgery If y			If yes, please list type and date:					
			If was plance list type and data					
$\Box V_{es} \Box N_{c}$		s nye	in yes, piease nsi type and date:					
ADD /LD Diagnosis If		If ve	If yes, please list type and date:					
\Box Yes \Box No		11 9 0	o, prouse not	Spe and date.				
Psychiatric Treatment If $\Box \mathbf{V}_{ee} \Box \mathbf{N}_{e}$		nt If ye	If yes, please list type, date, and duration:					
			.1. 0. 1	· • □ • • □ • •				
\Box Vac \Box No \Box		Cur	Urrently Smoking? Li Yes Li No					
Li res Li No li yes, please list quantity and duration:								
Alcohol Use Curr			Currently Drinking? Yes No					
□Yes □No If		If ye	If yes, please list quantity and duration:					
Illicit Drug	Ilee	0						
\Box Yes \Box No			If yes, please list quantity and duration:					
FAMILY MEDICAL HISTORY								
Medical Disorders			If yes, please list type:					
∐Yes ∐No				-				
Neurologic 1	Neurologic Disorders		If yes, please list type:					
∐Yes ∐N	10							
Notes								
APPENDIX C

INFORMANT PACKET – PATIENT HISTORY FORM

For how long have you known the patient? _____ Relationship: _____ Patient Occupation/Former Occupation: _____ Patient Education (Years) _____

Please answer the following questions to the best of your ability. If you don't know the answer, just check "Don't know". The questionnaire is long, but it serves several important purposes; it stimulates you to think about your own observations in greater depth and detail and helps you to include information you might not have thought important. And it will be read.

1. Was onset of problem with memory, language, or daily function sudden _____ or gradual

Don't

__?

2. Has there been a steady progression _____, abrupt decline _____, or no

progression _____?

Is there a problem with:

	1	No	Yes	Know
ME	MORY			
3.	Remembering people's names			
4.	Recognizing familiar faces			
5.	Finding way indoors			
6.	Finding way on familiar streets			
7.	Remembering a short list of items			
8.	More confusion late in the day			
EX	PRESSION			
9.	Finding the right word			
10.	Understanding words			
DA	ILY FUNCTIONING			
11.	Trouble with household tasks			
12.	Trouble handling money			
13.	Doesn't grasp situations or explanations			
14.	Difficulty at work (check if NA)			
				Don't

		No	Yes	Know
15.	Trouble dressing or caring for self			
16.	Trouble feeding self			
17.	Trouble controlling bladder and bowels			
PE	RSONALITY			
18.	More irritable			
19.	Less interested			
20.	Less sensitive to others			
21.	Loss of social graces or manners			
22.	Loss of initiative			
23.	Physical violence			
24.	Developed odd habits or interests			
<u>SL</u>	EEPING AND EATING			
25.	Excessive daytime sleepiness			
26.	Vivid dreams; dreams seem real			
27.	Violent movement/talking in sleep			
28.	Overeating/Consuming sweets			
29.	Appetite loss			
30.	Eating nonfood substances			
TH	INKING			
31.	More suspicious			
32.	Delusions or false beliefs			
33.	Hallucinations (sight, sound, odor)			
34.	Illusions; mistakes one thing for another			
35.	Thinks others are doubles or imposters			
36.	Talks of suicide or attempts suicide			
37.	Aware of having a problem			
				Don't
<u>OT</u>	HER PROBLEMS	No	Yes	Know
38.	Poor hearing			
39.	Poor eyesight			

40.	. High cholesterol						
41.	Stroke(s)						
42.	High blood pressure						
43.	Heart attack						
44.	Abnormal heart beat						
45.	Unexplained falls						
46.	Parkinson disease (shaking, shuffling gait)						
47.	Fainting spells						
48.	Head injury with loss of consciousness						
49.	Seizure or epilepsy						
50.	Brain tumor						
51.	Diabetes						
52.	High or low thyroid function						
53.	Treated for mental/emotional problems						
	a. Diagnosis						
	b. Hospitalized? No Yes						
54.	Down Syndrome No Self Fami	ly Member _					
55.	Other medical problems						
56.	Drugs: medication for memory?						
	a		_				
	b						
	c		_				
	d		_				
				Don't			
57		No	Yes	Know			
51.	Drugs: medication for calming?						
	Drugs: medication for calming?						
	ab						
	<pre>Drugs: medication for calming? a. b. c.</pre>						

	d		_	
	e. Side Effects (specify)			
58.	Illegal street drugs?			
	a. Other drug abuse/dependence (prescription, e	etc)?		
59.	Alcohol Use			
	a. Current number of ounces per week?			
60.	Alcohol Abuse			
61.	Toxic chemical exposure			
	a. Type			
62.	Syphilis			
63.	Other infection (HIV, hepatitis, etc)			
	a. Specify			
64.	Cancer (other than skin)			
	a. Type			
	b. Treatment: None Radiation	Che	motherapy	
	Surgery Other			
65.	Cataract surgery or other eye surgery			
66.	Surgery with general anesthesia			
	a		_	
	b		_	
	c		_	
	d		_	
67.	CAT scan or MRI (Head)			
				Don't
		No	Yes	Know
	a. Allergies			
	b. Type			
68.	Anyone in family with similar problem			
	a. Relationship to patient			

69.	. Ever had psychiatric neurological exam					
	a. Diagnosis					
70.	Name and address of doctors seen for same or similar purpose					

INFORMANT PREDICTION OF PATIENT PERFORMANCE

In general, how concerned are you about the patient's overall cognitive skills/thinking ability? Please circle your answer on the following scale.

	1	2	3	4	5	6	7	8	9	10
Not Con	cerned			About	Averag	ge			Very	Concerned

Based on your knowledge of the patient, please answer the following questions. Please compare the patient to his/her same aged peers using the following scale:

1	2	3	4	5	6	7	8	9	10
Relatively	Poorly		1	About A	verage			Rel	atively Well

- 1. Overall, how do you think the patient's general cognitive (thinking) ability compares with his/her same aged peers? _____
- 2. How well do you think the patient could initially memorize a list of 9 words if we read the list out loud to him/her several times? _____
- 3. How well do you think the patient would remember those words after about 10 minutes? _____
- 4. If we were to read a list of more words to the patient, some of which were on the original list and some of which were new words, how well do you think the patient would be able to recognize the words from the original list? _____
- 5. If we were then to give the patient two words at a time, one of which was on the original list and one of which wasn't, how accurately do you think the patient would be able to pick out the word from the list? _____
- 6. If we were to ask the patient to initially memorize two short stories, how well do you think he/she would do compared to his/her same aged peers? _____
- 7. How well do you think the patient would remember those stories after 25 minutes?
- 8. If we were to give the patient a set of blocks and ask him/her to arrange them so that they matched a picture, how well do you think he/she would do compared to his/her same aged peers? _____
- 9. How well do you think the patient could visually judge the angle of two lines if given a reference like a protractor (assume he/she cannot place the protractor directly over the lines given)? _____
- 10. If we were to show the patient a series of pictures of objects and asked him/her to name them, how well do you think he/she would do? _____
- 11. If we were to give the patient a letter of the alphabet and ask him/her to come up with as many words as he/she could within one minute, how well do you think he/she would do? _____

- 12. If we were to ask the patient to name as many animals as he/she could in one minute, how well do you think he/she would do? _____
- 13. If we were to read the patient a string of numbers and asked him/her to repeat them back to us in the same order that they were read, how well do you think he/she would do? _____
- 14. If we were to read the patient a string of numbers and asked him/her to repeat them back to us in the reverse order that they were read (backwards), how well do you think he/she would do? _____
- 15. If we were to give the patient a key that showed a list of shapes that corresponded to numbers and asked him/her to substitute the appropriate numbers for a row of shapes as quickly as he/she could, how well do you think he/she would do? _____
- 16. If we were to give the patient a sheet with 25 randomly placed numbers and asked him/her to connect them in order as fast as he/she could, how well do you think he/she would do? _____
- 17. If we were to give the patient a sheet with 25 randomly placed numbers AND letters and asked him/her to connect them by alternating between connecting numbers and letters in order (i.e. 1-A-2-B-3-C, etc) as fast as he/she could, how well do you think he/she would do?
- 18. If we were to give the patient a test that assessed his/her ability to problem solve in new ways without really knowing where to start, how well do you think he/she would do? _____
- 19. If we were to give the patient a list of uncommon words to pronounce out loud, how well do you think he/she would do? _____
- 20. In general, how depressed do you think the patient is?
- 21. If we were to show the patient a series of cards, each with dots on them, how quickly do you think he/she would be able to count the dots on the card and tell us his/her answer? _____

LEVEL OF FUNCTION SCALE

	Independent, as good as ever	Independent, not as good as past	Needs prompting or reminding to perform	Needs hands-on help or step-by- step	Can't do, depends on others to do
Work responsibilities	0	1	task 2	directions 3	4
Hobbies	0	1	2	3	4
Household chores	0	1	2	3	4
Shopping for needs	0	1	2	3	4
Driving	0	1	2	3	4
Appointments	0	1	2	3	4
Finding one's things	0	1	2	3	4
Dressing	0	1	2	3	4
Washing and Grooming	0	1	2	3	4
Eating	0	1	2	3	4
Toileting	0	1	2	3	4
Other:	0	1	2	3	4
Other:	0	1	2	3	4

Please circle the patient's current level of function at these tasks of everyday life.

Is there anything else you'd like to mention?

APPENDIX D

INFORMANT PACKET – PATIENT PSYCHIATRIC HISTORY

For how long have you known the patient? _____ Relationship:

How would you rate your knowledge of the patient?

- □ Very familiar/provides daily care
- □ Somewhat familiar/often cares for
- \Box Not very familiar, has minimal interaction with

If you do not live with the patient, how many hours per week do you see him/her?

Please indicate whether the patient has displayed any of the behaviors listed below within the past four weeks. If you answer yes to any of the following questions, please continue to the page noted to answer follow up questions related to that item.

A. <u>Delusions</u>: Does the patient have beliefs that you know are not true? For example, insisting that people are trying to harm him/her. Has he/she said that family members are not who they say they are, or that the house is not their home? (Please do not include suspicious activity, please only indicate true beliefs on the part of the patient.)

- \square No (If no, please proceed to B)
- □ Yes (If yes, please see additional related questions)
- □ Not applicable

B. <u>Hallucinations</u>: Does the patient have hallucinations such as false visions or voices? Does he/she see, hear, or experience things that are not present? By this, we do not mean just mistaken beliefs such as stating that someone who has died is still alive; rather we are asking if the patient actually has abnormal experiences of sounds or visions.

- \Box No (If no, please proceed to C)
- □ Yes (If yes, please see additional related questions)
- □ Not applicable

C. <u>Agitation/Aggression:</u> Does the patient have periods when he/she refuses to cooperate or won't let people help him/her? Is he/she hard to handle?

- \Box No (If no, please proceed to D)
- □ Yes (If yes, please see additional related questions)
- □ Not applicable

D. <u>Depression/Dysphoria</u>: Does the patient seem sad or depressed? Does he/she say that he/she feels sad or depressed?

- \Box No (If no, please proceed to E)
- ☐ Yes (If yes, please see additional related questions)
- □ Not applicable

E. <u>Anxiety:</u> Is the patient very nervous, worried, or frightened for no reason? Does he/she seem very tense or fidgety? Is he/she afraid to be apart from you?

- \Box No (If no, please proceed to F)
- □ Yes (If yes, please see additional related questions)
- \Box Not applicable

F. <u>Elation/Euphoria</u>: Does the patient seem too cheerful or too happy for no reason? We don't mean the normal happiness that comes from seeing friends, receiving presents, or spending time with family members. We are asking if he/she has a persistent and abnormally good mood or finds humor where others do not.

- \Box No (If no, please proceed to G)
- □ Yes (If yes, please see additional related questions)
- □ Not applicable

G. <u>Apathy/Indifference</u>: Does the patient sit quietly without paying attention to things going on around him/her? Has he/she lost interest in the world around him/her? Has he/she lost interest in doing things or lack motivation for participating in activities? Is it difficult to involve him/her in conversation or in doing chores?

- \Box No (If no, please proceed to H)
- □ Yes (If yes, please see additional related questions)
- \Box Not applicable

H. <u>Disinhibition</u>: Does the patient seem to act impulsively without thinking? Does he/she do or say things that are not usually done or said in public? Does he/she say things that are embarrassing to you or others?

- \Box No (If no, please proceed to I)
- □ Yes (If yes, please see additional related questions)
- \Box Not applicable

I. <u>Irritability/Lability</u>: Does that patient get irritated and easily disturbed? Are his/her moods very changeable? Is he/she abnormally impatient? We do not mean frustration over memory loss or inability to perform usual tasks; we are interested to know if the patient has abnormal irritability, impatience, or rapid emotional changes different from his/her usual self.

- \Box No (If no, please proceed to J)
- □ Yes (If yes, please see additional related questions)
- \Box Not applicable

J. <u>Aberrant Motor Behavior</u>: Does the patient pace, do things over and over such as opening closets or drawers, or repeatedly pick at things, or wind string or threads?

- \Box No (If no, please proceed to K)
- □ Yes (If yes, please see additional related questions)
- \Box Not applicable

K. <u>Sleep</u>: Does the patient have difficulty sleeping (aside from getting up once or twice to go to the restroom and falling back asleep immediately)? Is he/she up at night? Does he/she wander at night, get dressed, or disturb your sleep?

- \Box No (If no, please proceed to L)
- □ Yes (If yes, please see additional related questions)
- \Box Not applicable

L. <u>Appetite and Eating Disorders:</u> Has the patient had any changes in appetite, weight, or eating habits? Has there been any change in type of food he/she prefers? (Please mark Not Applicable if the patient is incapacitated and has to be fed.)

- \Box No (If no, please stop here)
- □ Yes (If yes, please see additional related questions)
- \Box Not applicable

CONTINUED FROM QUESTION A: DELUSIONS

 Does the patient believe that he/she is in danger – that others are planning to hurt him/her?

 \Box No \Box Yes \Box Not Applicable

- Does the patient believe that others are stealing from him/her?
 □No □Yes □Not Applicable
- 3. Does the patient believe that his/her spouse is having an affair? □No □Yes □Not Applicable
- 4. Does the patient believe that unwelcome guests are living in his/her house?
 □No □Yes □Not Applicable
- 5. Does the patient believe that his/her spouse or others are not who they claim to be?
 □No □Yes □Not Applicable
- 6. Does the patient believe that his/her home is not his/her home?□No □Yes □Not Applicable
- 7. Does the patient believe that family members plan to abandon him/her?□No □Yes □Not Applicable
- 8. Does the patient believe that television or magazine figures are actually present in the home/room?
 □No □Yes □Not Applicable
 If yes, does he/she try to talk or interact with them? □No □Yes
- 9. Does the patient believe any other unusual things that have not been covered here?

If so, please describe (use the back of this page if necessary):

- 10. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - \Box Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 11. How severe do you consider the patient's symptoms?
 - □ Mild delusions are present but seem harmless, and do not upset the patient that much
 - □ Moderate delusions are stressful and upsetting to the patient and cause unusual or strange behavior
 - □ Marked delusions are very stressful and upsetting to the patient and cause a major amount of unusual or strange behavior
- 12. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - □ Mildly
 - □ Moderately
 - □ Severely
 - □ Very severely or extremely

CONTINUED FROM QUESTION B: HALLUCINATIONS

- Does the patient describe hearing voices or act as if he/she hears voices?
 □No □Yes □Not Applicable
- Does the patient talk to people who are not there?
 □No □Yes □Not Applicable
- 3. Does the patient describe seeing things not seen by others, or behave as he/she is seeing things not seen by others (i.e. people, animals, lights, etc.)?
 □No □Yes □Not Applicable
- 4. Does the patient report smelling odors not smelled by others?
 □No □Yes □Not Applicable
- 5. Does the patient describe feeling things on his/her skin, or otherwise appear to be feeling things crawling or touching him/her?
 □No □Yes □Not Applicable
- 6. Does the patient describe tastes that are without known cause?
 □No □Yes □Not Applicable

- 7. Does the patient describe any other unusual sensory experiences?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 8. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - \Box Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 9. How severe do you consider the patient's symptoms?
 - □ Mild hallucinations are present but seem harmless, and do not upset the patient that much
 - □ Moderate hallucinations are distressing and disruptive
 - □ Marked hallucinations are very disruptive and are a major source of behavioral disturbance (medications may be required to control them)
- 10. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - □ Mildly
 - □ Moderately
 - □ Severely
 - \Box Very severely or extremely

CONTINUED FROM QUESTION C: AGITATION/AGGRESSION

- Does the patient get upset when people are trying to care for him/her to resist activities such as bathing or changing clothes?
 □No □Yes □Not Applicable
- Is the patient stubborn, having to have things his/her way?
 □No □Yes □Not Applicable
- 3. Is the patient uncooperative, resistive to help from others? □No □Yes □Not Applicable
- 4. Does the patient have any other behaviors that make him/her hard to handle?
 □No □Yes □Not Applicable
- 5. Does the patient shout or curse angrily?□No □Yes □Not Applicable
- 6. Does the patient slam doors, kick furniture, or throw things?

- 7. Does the patient attempt to hurt or hit others?□No □Yes □Not Applicable
- 8. Does the patient have any other aggressive or agitated behavior?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 9. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - □ Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 10. How severe do you consider the patient's symptoms?
 - □ Mild behavior is disruptive but can be managed with redirection or assurance
 - □ Moderate behaviors are disruptive and difficult to redirect or control
 - □ Marked agitation is very disruptive and a major source of difficulty; there may be a threat of personal harm. Medications are often required.
- 11. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - □ Mildly
 - □ Moderately
 - □ Severely
 - \Box Very severely or extremely

CONTINUED FROM QUESTION D: DEPRESSION/DYSPHORIA

1. Does the patient have periods of tearfulness or sobbing that seem to indicate sadness?

 \Box No \Box Yes \Box Not Applicable

- Does the patient say or act as if he/she is sad or in low spirits?
 □No □Yes □Not Applicable
- 3. Does the patient put him/herself down or say that he/she feels like a failure? □No □Yes □Not Applicable
- 4. Does the patient say that he/she is a bad person or deserves to be punished?
 □No □Yes □Not Applicable
- 5. Does the patient seem very discouraged or say that he/she has no future?

- 6. Does the patient say he/she is a burden to the family or that the family would be better off without him/her?
 □No □Yes □Not Applicable
- Does the patient express a wish for death or talk about killing him/herself?
 □No □Yes □Not Applicable
- 8. Does the patient show any other signs of depression or sadness?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 9. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - □ Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 10. How severe do you consider the patient's symptoms?
 - □ Mild depression is distressing, but usually responds to redirection or reassurance
 - □ Moderate depression is distressing, depressive symptoms are spontaneously voiced by the patient and difficult to alleviate
 - □ Marked depression is very distressing and a major source of suffering for the patient
- 11. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - \Box Minimally
 - □ Mildly
 - □ Moderately
 - □ Severely
 - \Box Very severely or extremely

CONTINUED FROM QUESTION E: ANXIETY

- Does the patient say that he/she is worried about planned events?
 □No □Yes □Not Applicable
- 2. Does the patient have periods of feeling shaky, unable to relax, or feeling excessively tense?
 □No □Yes □Not Applicable
- 3. Does the patient have periods of or complain of shortness of breath, gaping, or sighing for no reason other than nervousness?

- 4. Does the patient complain of butterflies in his/her stomach, or of racing or pounding of the heart in association with nervousness?
 □No □Yes □Not Applicable
 If yes, is this associated with a medical condition?
 □No □Yes
- 5. Does the patient avoid certain places or situations that make him/her more nervous such as riding in the car, meeting with friends, or being in crowds?
 □No □Yes □Not Applicable
- 6. Does the patient become nervous or upset when separated from you or his/her caregiver?
 □No □Yes □Not Applicable
 If yes, does he/she cling to you to keep from being separated?
 □No □Yes
- 7. Does the patient show any other signs of anxiety?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 8. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - □ Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 9. How severe do you consider the patient's symptoms?
 - □ Mild anxiety is stressful, but usually responds to redirection or reassurance
 - □ Moderate anxiety is stressful, anxiety symptoms are spontaneously voiced by the patient and difficult to alleviate
 - □ Marked anxiety is very distressing and a major source of suffering
- 10. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - □ Mildly
 - □ Moderately
 - □ Severely
 - □ Very severely or extremely

CONTINUED FROM QUESTION F: ELATION/EUPHORIA

- Does the patient appear to feel good or to be too happy, different from his/her usual self?
 □No □Yes □Not Applicable
- 2. Does the patient find humor and laugh at things that others do not find funny? □No □Yes □Not Applicable
- Does the patient seem to have a childish sense of humor with a tendency to giggle or laugh inappropriately (such as when something unfortunate happens to others)?
 □No □Yes □Not Applicable
- 4. Does the patient tell jokes or make remarks that have little humor? □No □Yes □Not Applicable
- 5. Does the patient play childish pranks such as pinching or playing "keep away" for the fun of it?
 □No □Yes □Not Applicable
- 6. Does the patient "talk big" or claim to have more abilities or wealth than is true?
 □No □Yes □Not Applicable
 If yes, does he/she cling to you to keep from being separated?
 □No □Yes
- 7. Does the patient show any other signs of feeling to good or being too happy?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 8. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - □ Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 9. How severe do you consider the patient's symptoms?
 - \Box Mild the patient is too happy at times
 - □ Moderate the patient is too happy at times, and this sometimes causes strange behavior
 - □ Marked the patient is almost always too happy and finds nearly everything to be funny
- 10. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - \square Mildly

- □ Moderately
- \Box Severely
- \Box Very severely or extremely

CONTINUED FROM QUESTION G: Apathy/Indifference

- Does the patient seem less spontaneous and less active than usual?
 □No □Yes □Not Applicable
- 2. Is the patient less likely to initiate a conversation?□No □Yes □Not Applicable
- 3. Is the patient less affectionate or lacking in emotions when compared to his/her usual self?
 □No □Yes □Not Applicable
- 4. Does the patient contribute less to household chores?□No □Yes □Not Applicable
- 5. Does the patient seem less interested in the activities and plans of others? □No □Yes □Not Applicable
- 6. Has the patient lost interest in friends and family members?□No □Yes □Not Applicable
- 7. Is the patient less enthusiastic about his/her usual interests?
 □No □Yes □Not Applicable
- 8. Does the patient show any other signs that he/she doesn't care about doing new things?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 9. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - \Box Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 10. How severe do you consider the patient's symptoms?
 - Mild apathy is notable but produces little interference with daily routines; only mildly different from the patient's usual behavior; the patient responds to suggestion to engage in activities

- □ Moderate apathy is very evident; may be overcome with coaxing and encouragement; responds spontaneously only to powerful events such as visits from close relatives or family members
- □ Marked apathy is very evident and usually fails to respond to any encouragement or external events
- 11. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - \square Mildly
 - □ Moderately
 - □ Severely
 - \Box Very severely or extremely

CONTINUED FROM QUESTION H: DISINHIBITION

- 1. Does the patient act impulsively without appearing to consider the consequences? □No □Yes □Not Applicable
- 2. Does the patient talk to total strangers as if he/she knew them? □No □Yes □Not Applicable
- 3. Does the patient say things to people that are insensitive or hurt their feelings? □No □Yes □Not Applicable
- 4. Does the patient say crude things or make sexual remarks that they would not usually have said?
 □No □Yes □Not Applicable
- 5. Does the patient talk openly about very personal or private matters not usually discussed in public?
 □No □Yes □Not Applicable
- 6. Does the patient take liberties or touch or hug others in a way that is out of character for him/her?
 □No □Yes □Not Applicable
- 7. Does the patient show any other signs of loss of control of his/her impulse?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 8. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - \Box Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day

- 9. How severe do you consider the patient's symptoms?
 - □ Mild disinhibitiion is notable, but usually responds to redirection and guidance
 - □ Moderate disinhibition is very evident and difficult to overcome by the caregiver
 - □ Marked disinhibition usually fails to respond to any intervention by the caregiver and is a source of embarrassment or social distress
- 10. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - \square Mildly
 - □ Moderately
 - \Box Severely
 - □ Very severely or extremely

CONTINUED FROM QUESTION I: IRRITABILITY/LABILITY

1. Does the patient have a bad temper, "flying off the handle" easily over little things?

□No	\Box Yes	□Not	Applicable
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- Does the patient rapidly change moods from one to another, being fine one minute and angry the next?
 □No □Yes □Not Applicable
- 3. Does the patient have sudden flashes of anger? □No □Yes □Not Applicable
- 4. Is the patient impatient, having trouble coping with delays or waiting for planned activities?
 □No □Yes □Not Applicable
- 5. Is the patient cranky and irritable? □No □Yes □Not Applicable
- 6. Is the patient argumentative and difficult to get along with? □No □Yes □Not Applicable
- 7. Does the patient show any other signs of irritability?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 8. How frequently does the patient experience these symptoms? □ Occasionally less than once per week

- \Box Often about once per week
- \Box Frequently several times per week, but less than every day
- \Box Very Frequently once or more per day
- 9. How severe do you consider the patient's symptoms?
 - □ Mild irritability or lability is notable but usually responds to redirection and reassurance
 - □ Moderate irritability and lability are very evident and difficult to overcome by the caregiver
 - □ Marked irritability and lability are very evident, they usually fail to respond to any intervention by the caregiver, and they are a major source of distress
- 10. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - \square Mildly
 - □ Moderately
 - \Box Severely
 - \Box Very severely or extremely

CONTINUED FROM QUESTION J: ABBERANT MOTOR BEHAVIOIR

- 1. Does the patient pace around the house without purpose? □No □Yes □Not Applicable
- 2. Does the patient rummage around opening and unpacking drawers or closets? □No □Yes □Not Applicable
- 3. Does the patient repeatedly put on and take off clothing? □No □Yes □Not Applicable
- 4. Does the patient have repetitive activities or "habits" that he/she performs over and over?
 □No □Yes □Not Applicable
- 5. Does the patient engage in repetitive activities such as handling buttons, picking, wrapping string, etc?
 □No □Yes □Not Applicable
- 6. Does the patient fidget excessively, seem unable to sit still, or bounce his/her feet or tap his/her fingers a lot?
 □No □Yes □Not Applicable
- 7. Does the patient do any other activities over and over?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):

- 8. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - \Box Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 9. How severe do you consider the patient's symptoms?
 - □ Mild abnormal motor activity is notable but produces little interference with daily routines
 - □ Moderate abnormal motor activity is very evident; can be overcome by the caregiver
 - □ Marked abnormal motor activity is very evident, it usually fails to respond to any intervention by the caregiver and is a major source of distress
- 10. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - □ Mildly
 - □ Moderately
 - □ Severely
 - \Box Very severely or extremely

CONTINUED FROM QUESTION K: SLEEP

- 1. Does the patient have difficulty falling asleep? □No □Yes □Not Applicable
- Does the patient get up during the night (do not count if the patient gets up once or twice per night only to go to the bathroom and falls back asleep immediately)?
 □No □Yes □Not Applicable
- 3. Does the patient wander, pace, or get involved in inappropriate activities at night? □No □Yes □Not Applicable
- 4. Does the patient awaken you during the night?□No □Yes □Not Applicable
- 5. Does the patient wake up at night, dress, and plan to go out, thinking that it is morning and time to start the day?
 □No □Yes □Not Applicable
- 6. Does the patient wake up too early in the morning (earlier than was his/her habit)?□Yes □No □Not Applicable
- 7. Does the patient sleep excessively during the day?

- 8. Does the patient have any other night-time behaviors that bother you that haven't been asked about here?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 9. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - □ Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day

10. How severe do you consider the patient's symptoms?

- □ Mild night-time behaviors occur but they are not particularly disruptive
- □ Moderate night-time behaviors occur and disturb the patient and the sleep of the caregiver; more than one type of night-time behavior may be present
- Marked night-time behaviors occur; several types of behavior may be present; the patient is very distressed during the night and the caregiver's sleep is markedly disturbed
- 11. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - □ Minimally
 - □ Mildly
 - □ Moderately
 - □ Severely
 - \Box Very severely or extremely

CONTINUED FROM QUESTION L: APPETITE AND EATING DISORDERS

- Does the patient have poor appetite (loss of appetite)?
 □No □Yes □Not Applicable
- 2. Does the patient have an abnormally good appetite (increase in appetite)? □No □Yes □Not Applicable
- 3. Has the patient lost weight?□No □Yes □Not Applicable
- 4. Has the patient gained weight?□No □Yes □Not Applicable
- 5. Does the patient have unusual eating behavior, such as putting too much food in his/her mouth at once?
 □No □Yes □Not Applicable

- 6. Has the patient had a change in the kind of food he/she likes, such as wanting too many sweets or other specific types of food?
 □No □Yes □Not Applicable
- 7. Has the patient developed eating behaviors, such as eating exactly the same types of food each day, or eating the food in exactly the same order?
 □No □Yes □Not Applicable
- 8. Have there been any other changes in appetite or eating that haven't been asked about here?
 □No □Yes □Not Applicable
 If so, please describe (use the back of this page if necessary):
- 9. How frequently does the patient experience these symptoms?
 - \Box Occasionally less than once per week
 - \Box Often about once per week
 - □ Frequently several times per week, but less than every day
 - \Box Very Frequently once or more per day
- 10. How severe do you consider the patient's symptoms?
 - □ Mild appetite changes are present but usually responds well to redirection and reassurance
 - □ Moderate appetite changes are very evident and difficult to overcome by the caregiver
 - □ Marked appetite changes are very evident, they usually fail to respond to any intervention by the caregiver, and they are a major source of distress
- 11. How emotionally distressing do you find this behavior?
 - \Box Not at all
 - \Box Minimally
 - □ Mildly
 - □ Moderately
 - \Box Severely
 - \Box Very severely or extremely

APPENDIX E

NEUROPSYCHOLOGICAL BATTERY ORDER OF

ADMINISTRATION

NOTE: Use BLUE pen for predictions, and GREEN pen for evaluations

"Throughout the testing process today, I am going to ask you some questions that require you to estimate how well you would do on a certain task compared to people your same age and education level that haven't had complaints about their thinking ability. Please draw a line on this scale indicating where you think your performance would fall, with the very top of the line being extremely well (point) and the very bottom of the line being extremely poorly (point). Remember that you are rating yourself in comparison to the

average person your same age and education."

- 1. Concern (PRE)(1)
- 2. Read SCRIPT Above
- 3. WCST Prediction (2)
- 4. 3MS Prediction (3)
- 5. 3MS
- 6. 3MS Evaluation (3)
- 7. Trails A Prediction (4)
- 8. Trails A
- 9. Trails A Evaluation (4)
- 10. Trails B Prediction (5)
- 11. Trails B
- 12. Trails B Evaluation (5)
- 13. FAS Prediction (6)
- 14. FAS
- 15. FAS Evaluation (6)
- 16. Animals Prediction (7)
- 17. Animals
- 18. Animals Evaluation (7)
- 19. CVLT Immediate Recall (IR) Prediction (8)
- 20. CVLT Immediate Free Recall
- 21. CVLT Short Delay Free Recall
- 22. CVLT Immediate Recall (IR) Evaluation (8)
- 23. Block Design (BD) Prediction (9)
- 24. Block Design
- 25. Block Design (BD) Evaluation (9)
- 26. CVLT Delayed Recall (DR) Prediction (10)
- 27. CVLT Long Delay Free Recall
- 28. CVLT Long Delay Cued Recall
- 29. CVLT Delayed Recall (DR) Evaluation (10)

- 30. CVLT Recognition Prediction (11)
- 31. CVLT Long Delay Recognition
- 32. CVLT Recognition Evaluation (11)
- 33. SDMT Prediction (12)
- 34. SDMT Written
- 35. SDMT Evaluation (12)
- 36. GDS Prediction (13)
- 37. GDS
- 38. GDS Evaluation (13)
- 39. CVLT Forced Choice Prediction (14)
- 40. CVLT Forced Choice
- 41. CVLT Forced Choice Evaluation (14)
- 42. Logical Memory I (LMI) Prediction (15)
- 43. Logical Memory I
- 44. Logical Memory I (LMI) Evaluation (15)
- 45. Logical Memory II (LMII) Pre-Prediction (16)
- 46. Digit Span Forward (DS-F) Prediction (17)
- 47. Digit Span Forward
- 48. Digit Span Forward (DS-F) Evaluation (17)
- 49. Digit Span Backward (DS-B) Prediction (18)
- 50. Digit Span Backward
- 51. Digit Span Backward (DS-B) Evaluation (18)
- 52. Judgment of Line Orientation (JoLO) Prediction (19)
- 53. Judgment of Line Orientation (JoLO)
- 54. Judgment of Line Orientation (JoLO) Evaluation (19)
- 55. Boston Naming (BNT) Prediction (20)
- 56. Boston Naming Test
- 57. Boston Naming (BNT) Evaluation (20)
- 58. WTAR Prediction (21)
- 59. WTAR
- 60. WTAR Evaluation (21)
- 61. WCST
- 62. WCST Evaluation (2)
- 63. Logical Memory II (LMII) Prediction (22)
- 64. Logical Memory II
- 65. Logical Memory II (LMII) Evaluation (22)
- 66. Dot Counting (DC) Prediction (23)
- 67. Dot Counting
- 68. Dot Counting (DC) Evaluation (23)
- 69. General Evaluation (24)
- 70. Concern (POST) (25)

APPENDIX E

PREDICITON AND EVALUATIONS RATING SCRIPT

		Prediction	Evaluation
1	CONCERN	Evaluation 1 (B): In general, how concerned is your family about your current thinking skills or cognitive ability? Evaluation 2 (G): In general, how concerned are you about your current thinking skills or cognitive ability?	Evaluation 1 (B): Now that we've completed all these different tasks, how concerned is your family about your current thinking skills or cognitive ability? Evaluation 2 (G): In general, how concerned are you about your current thinking skills or cognitive ability?
2	WCST	If I were to give you a test that assessed your ability to solve problems in new ways without really knowing where to start, how well do you think you'd do?	Earlier you said you'd do this well if I tested your ability to solve problems in new ways. Now that we've done the task, how well do you think you actually did?
3	3MS	Overall, how do you think your general cognitive ability compares with your same age and educated peers?	Now that we've done a short set of tasks that look at your overall cognitive ability, how well do you think you actually did in comparison to your same age and educated peers?
4	Trails A	If I were to give you a sheet with 25 randomly distributed, numbered circles and ask you to connect them in order as fast as you could, how well do you think you'd do?	Earlier you said you'd do this well if I asked you to connect 25 randomly distributed, numbered circles. Now that we've done the task, how well do you think you actually did?
5	Trails B	If I were to give you a sheet with 25 randomly distributed circles, half of them numbered and half of them lettered, and asked you to connect them alternating between numbers and letters in order as fast as you could, how well do you think you'd do?	Earlier you said you'd do this well if I asked you to connect 25 randomly distributed circles alternating between numbers and letters in order. Now that we've done the task, how well do you think you actually did?
6	FAS	If I were to give you a letter of the alphabet and you were given one minute to come up with as many words as you could that start with that letter, how well do you think you'd do?	Earlier you said you would do this well if given one minute to say as many words as you could that started with a given letter. Now that we've actually done the task, how well do you think you did?
7	Animals	If I were to give you a category (like things you could find in a kitchen) and ask you to name as many items that fall within that category as you could in one minute, how well do you think	Earlier you said you would do this well if given a category and asked to name as many items as you could in one minute. Now that we've actually done the task, how well do you think you actually did?

		you'd do?	
8	CVLT IR	If I were to read you a list of words to memorize and asked you to repeat as many words as you could, how well do you think you'd do?	Earlier you said you'd do this well (point to estimate) if asked to memorize a list of words I read to you. Now that we've done the task, how well do you think you actually did?
9	BD	If I were to give you a set of blocks and ask you to arrange the blocks so that they look like a given picture I show you, how well do you think you'd do?	Earlier you said you'd do this well if asked to recreate a picture out of blocks. Now that we've done the task, how well do you think you actually did?
10	CVLT DR	Earlier I read you a list of words and asked you to repeat back as many words as you could. If I were to ask you to repeat back as many of those words as you can now, how well do you think you'd do?	Earlier you said you'd do this well (point to estimate) if asked to repeat as many words as you could remember from a list I read to you some time ago. Now that we've done the task, how well do you think you actually did?
11	CVLT Recognition	Earlier I read a list of words to you several times and asked you to repeat back as many of them as you could remember. How well do you think you'd be able to recognize those words from a list including words that were and were not from that original list?	Earlier you said you'd do this well (point to estimate) if asked to recognize the words from the list that I read to you earlier from a list including words that were and were not from that original list. Now that we've actually done the task, how well do you think you actually did?
12	SDMT	If I were to give you a key that showed a list of shapes that corresponded to numbers, how quickly do you think you could substitute the appropriate numbers for a row of shapes?	You said you'd do this well if asked to substitute the appropriate numbers for a row of shapes if given a key. Now that we've done the task, how well do you think you actually did?
13	GDS	In general, how often do you think you've experienced depression over the past two weeks in comparison to other people your same age?	You just completed a questionnaire that assesses your level of depression. How do you think your answers compare to other people your same age?
14	CVLT FC	Earlier I read a list of words to you several times and asked you to repeat back as many of them as you could remember. If I were to give you words two at a time, one of which was on the list and one of which was not, how accurately do you think you'd be able to pick out the word from the list?	Earlier you said you'd do this well (point to estimate) if asked to pick out the correct word if given two choices. Now that we've actually done the task, how well do you think you actually did?

15	LMI	If I were to read you a story to memorize and asked you to repeat it back to me, how well do you think you'd do?	Earlier you said you'd do this well (point to estimate) if asked to memorize a story I read to you. Now that we've done the task, how well do you think you actually did?
16	DS-F	If I were to read you a string of numbers and asked you to repeat them back to me in the same order, how well do you think you'd do?	Earlier you said you'd do this well if asked to repeat a string of numbers in the same order that I read them to you in. Now that we've done the task, how well do you think you actually did?
17	DS-B	If I were to read you a string of numbers and asked you to repeat them back to me in reverse order, how well do you think you'd do?	Earlier you said you'd do this well if asked to repeat a string of numbers in reverse order. Now that we've done the task, how well do you think you actually did?
18	JoLO	How well do you think you could pick out the line in the group above (point to sample) that matches the line shown below (point to sample)?	Earlier you said you'd do this well if asked to pick out the lines in the group that corresponded to the lines shown below. Now that we've actually completed the task, how well do you think you did?
19	BNT	If I were to assess your word finding ability by showing you a series of pictures of objects and asking you to name them, how well do you think you'd do?	Earlier you said that you'd do this well if asked to name items when shown a picture of them. Now that we've done the task, how well do you think you actually did?
20	WTAR	If I were to give you a list of uncommon words to pronounce out loud, how well do you think you'd do?	Earlier you said you'd do this well if I asked you to pronounce a list of uncommon words out loud. Now that we've done the task, how well do you think you actually did?
21	LMII	I read you two stories earlier, do you remember? If I were to ask you to tell me as much about those stories as you can remember now, how well do you think you'd do?	Earlier you said you'd do this well (point to estimate) if asked to repeat back as much of those stories as you could. Now that we've done the task, how well do you think you actually did?
22	.]		In general, how well do you think you did on the tasks you engaged in today?
	GENERAI		If you were to have engaged in these same tasks before you (or your family) began noticing changes in your cognitive ability, how well do you think you would have done?

APPENDIX G

SAMPLE RATING SCALES



