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## Self-Awareness and Self-Monitoring of Cognitive and Behavioral Deficits in Behavioral Variant Frontotemporal Dementia, Primary Progressive Aphasia and Probable Alzheimer's Disease

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## Abstract

Lack of insight is a core diagnostic criterion for behavioral variant frontotemporal dementia (bvFTD), and is believed to be intact in the early stages of primary progressive aphasia (PPA). In other neurological conditions, symptom-specific insight has been noted, with behavioral symptoms appearing especially vulnerable to reduced insight. Different components of insight, self-awareness and self-monitoring, are also often considered separate phenomena. The current study compared insight in patients with PPA, bvFTD, and probable Alzheimer's disease (PrAD) and a group of cognitively intact control subjects. Additionally, differences in insight for the domains primarily affected by the three types of dementia, namely, Behavior, Naming, and Memory, were assessed, and self-awareness and self-monitoring were compared. A total of 55 participants were enrolled. Participants were asked to complete self-estimate scales demonstrating their perceived ability immediately prior to, and immediately following a test in each domain of interest. Results indicated that PPA and normal control groups performed very similarly on control (Weight and Eyesight) and cognitive domains, whereas bvFTD and PrAD patients were unable to accurately assess Memory. All three diagnostic groups failed to accurately assess their behavioral symptoms, suggesting that this domain is vulnerable to loss of insight across diagnoses. Naming ability, in contrast, was either accurately assessed or underestimated in all groups. Finally, there were no notable differences between self-awareness and self-monitoring, potential explanations for this are examined.

## Keywords

Dementia; Primary Progressive Aphasia; Insight; Self-awareness; Self-Monitoring; Metacognition; Frontotemporal Dementia

## Introduction

The term "anosognosia" was originally coined to describe lack of insight into hemiplegia following right hemisphere stroke (Frederiks, 1985; McGlynn & Schacter, 1989; Schacter, 1990). Modern research on insight often breaks the concept down into two main components.

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Declarative knowledge of one's ability, or *self-awareness* is sometimes considered distinct from, although related to, *self-monitoring*, or the on-line tracking of ability (Toglia & Kirk, 2000). Furthermore, whereas insight was once considered relevant only to hemiplegia, it is now recognized as applicable to cognitive and behavioral domains affected by neurological disorders other than stroke, such as dementia (McGlynn & Schacter, 1989).

Alzheimer's disease (AD) is the most common form of dementia, and it initially affects the medial temporal lobes causing amnesia, which is sometimes associated with reduced insight (DeBettignies, Mahurin, & Pirozzolo, 1990; Migliorelli et al., 1995). Less common neurodegenerative diseases, such as those caused by frontotemporal lobar degeneration (FTLD), have different deficits when compared with the clinical syndrome of probable AD (PrAD.) Dementias caused by FTLD can largely be separated into two broad variants, the behavioral variant of frontotemporal dementia (bvFTD), and the language variant, often referred to as primary progressive aphasia (PPA; (McKhann et al., 2001; Neary et al., 1998). Initially, the two clinical presentations of FTLD have distinct profiles. BvFTD is marked by gradual change in personality and other behavioral dysfunction. In contrast, individuals with PPA have relatively isolated *cognitive*, specifically language, deficits, for at least the first two years of their disease (Mesulam, 1987, 2003). Over time, however, bvFTD patients develop language symptoms (Blair, Marczinski, Davis-Faroque, & Kertesz, 2007), and PPA patients develop behavioral symptoms (Kertesz, Davidson, McCabe, Takagi, & Munoz, 2003). Whereas loss of insight is a core diagnostic feature in bvFTD (Neary et al., 1998), insight is generally considered to be intact early in PPA, but deteriorates with progression of this disease (Marczinski, Davidson, & Kertesz, 2004). Neuroimaging studies using volumetric assessment techniques point to the involvement of the prefrontal cortex in bvFTD, including the ventromedial prefrontal, posterior orbitofrontal, and dorsolateral prefrontal cortices as well as the anterior cingulate cortex and insula (Rosen et al., 2002). Using similar techniques, the same group identified damage to the left perisylvian regions and temporal poles in PPA (Gorno-Tempini et al., 2004). As such, these two disorders present an attractive model with which to compare insight into both cognitive and behavioral symptoms in disorders where the neurological disease "respects" anatomical boundaries rather than vascular territories (Weintraub, 1993).

Anosognosia in PrAD has been extensively studied, although results tend to be mixed. Studies regarding cognitive anosognosia in PrAD have frequently, but not always, focused on memory. Findings include progressive loss of insight with extent of memory impairment, poorer insight into retrieval difficulties, and poorer insight in patients with evidence of frontal pathology (Ansell & Bucks, 2006; Dalla Barba, Parlato, Iavarone, & Boller, 1995; Gallo, Chen, Wiseman, & Schacter, in press; Reed, Jagust, & Coulter, 1993; Vogel, 2005). Several studies also report that patients with poor awareness into their condition overall tend to have higher levels of behavioral disturbance, such as disinhibition (Harwood, Sultzer, & Wheatley, 2000; Kashiwa et al., 2005; Starkstein, Jorge, Mizrahi, & Robinson, 2006). However, few studies report on insight into behavioral dysfunction itself despite its prevalence in PrAD. A notable exception to this is a recent report by Rankin and colleagues, discussed below, who found that patients with PrAD demonstrated relatively accurate perception of their personality change overall, although they tended to inaccurately report traits of submissiveness and extraversion (Rankin, Baldwin, Pace-Savitsky, Kramer, & Miller, 2005).

To date, research on insight in dementia related to FTLD has taken three distinct approaches. The first of these approaches is to assess overall level of insight. For example Marczinski et al. noted that loss of insight worsens over time in PPA, but remains fairly constant (and poor) in bvFTD (Marczinski et al., 2004).

The second approach has been to concentrate on insight into personality change or behavioral (or neuropsychiatric) symptoms. Rankin and colleagues (2005) used a novel technique of having patients and their caregivers complete assessments of the patient's personality at the time of assessment and at a hypothetical point in time several years prior. They found bvFTD patients to have less insight compared with normal controls or PrAD patients, and to minimize negative personality traits while exaggerating positive aspects of their current personality. In an interesting variation on this technique, Ruby and colleagues (2007) had patients with bvFTD complete a personality questionnaire on both themselves and their relatives. They found patients to be equally as accurate as normal controls when it came to characterizing their relatives. However, they considered their own personalities to be the same as ten years prior. This implies that they had poor insight into their own personality change, while demonstrating an intact ability to characterize others' traits. Eslinger and colleagues (2005) also assessed insight into behavioral change. Results indicated that their mixed FTD group (which combined behavioral and language variants) frequently underestimated symptoms, such as reduced empathy and flexibility, in comparison to their caregivers' complaints of these symptoms. Furthermore, the "social-behavioral" FTD subgroup (similar to the bvFTD group in the current study) showed far worse insight than the "aphasic" subgroup (akin to our PPA group; Eslinger et al., 2005).

The final approach has been to assess insight into cognitive deficits seen in dementia caused by FTLD. In an additional component of their study, Eslinger and colleagues asked patients to complete visual analog scales to indicate how difficult various cognitive tasks (verbal fluency, word list learning and recall) would be. Following completion of the task, the patient filled out a second copy of the same scale. They found pre-test visual analog scale responses to correlate poorly with actual performance, even for controls, so they only analyzed post-test scales. Their bvFTD group behaved similarly to the normal controls when it came to post-test ratings of verbal fluency and word learning, but overestimated their verbal recall. However, the subset of subjects in their study with progressive non-fluent aphasia, a subtype of PPA, were poor at estimating verbal fluency, but accurate at estimating verbal learning and recall. Normal controls' post-test ratings were all correlated with actual performance, suggesting intact monitoring for these cognitive domains (Eslinger et al., 2005). O'Keeffe and colleagues used several methods to assess awareness in a group of patients with FTLD (11/14 had bvFTD, others had language subtypes), progressive supranuclear palsy, and cortico-basal ganglionic degeneration. They found FTLD patients to be particularly poor at monitoring their own errors on cognitive tasks, although all three patient groups fared worse than normal controls at the various aspects of insight into cognitive decline that they measured (O'Keeffe et al., 2007).

As yet, no studies with FTD patients have investigated insight into naming deficits, a common impairment in both bvFTD and PPA, as well as PrAD. A recent study of cognitive anosognosia noted intact insight into naming impairments in patients with PrAD (Barrett, Eslinger, Ballentine, & Heilman, 2005). The neuroanatomical basis of language, in the left hemisphere, might suggest that it is relatively protected from anosognosia, which is generally considered to be a right hemisphere phenomenon (Adair, Gilmore, Fennell, Gold, & Heilman, 1995; Cutting, 1978; Fordyce & Roueche, 1986), However, disturbed awareness for aphasia is seen in some patients with fluent aphasias, notably when jargonaphasia is present (Lebrun, 1987; Weinstein, Cole, Mitchell, & Lyerly, 1964).

Prior studies in bvFTD and PPA have used different techniques to assess *behavioral* and *cognitive* insight. Insight into changes in personality, or behavior, has been studied mostly by asking patients and their caregivers to complete identical questionnaires regarding some aspect of their personality or behavior. The discrepancy between the two scores is then used as an index for the patient's insight into their behavioral disorder or personality change. This *discrepancy technique* has been used to assess individual behavioral deficits (e.g., lack of

empathy) and personality change as a whole (Rankin et al., 2005; Ruby et al., 2007). Although this evidently provides a rich source of information on whichever trait or deficit is being studied, it does not provide any information regarding the relationship between insight and actual level of deficit in that domain.

The second technique, and the one used in the current study, involves *self-estimates* (Barrett et al., 2005; Eslinger et al., 2005; Souchay, Isingrini, Pillon, & Gil, 2003). This technique allows for the analysis of two metacognitive aspects of insight, self-awareness and selfmonitoring (which correspond to the 'metacognitive knowledge' and 'online awareness' notions posited by Toglia and Kirk's (2000) model of awareness following brain injury). Loss of insight into neurological deficits, or anosognosia, would logically involve a breakdown in both self-awareness and self-monitoring. To illustrate, individuals with anosognosia for hemiplegia deny that there is anything wrong with the affected limbs (i.e., they show poor selfawareness of hemiplegia), and, even when faced with the evidence, often confabulate reasons why their limbs are not functioning (Gerstmann, 1942). Discrepancies have been noted in levels of self-awareness and ability to self-monitor in patients with prefrontal damage (Stuss, 1991). Beer and colleagues (Beer, John, Scabini, & Knight, 2006) studied such patients, whose lesions were the result of either stroke or traumatic brain injury, and found patients with orbitofrontal damage to have deficient self-awareness when taking part in an interpersonal task, in comparison to patients with lateral prefrontal cortex damage and healthy controls. However, when the patients with orbitofrontal lesions watched their own performance on video, they did express some signs of embarrassment. The authors interpreted this as implying intact selfmonitoring, suggesting that these two processes are distinct. It is arguable however, that the fact that they are not reacting to their behavior in real time but rather on videotape, confounds Beer et al's argument that they were actually assessing self-monitoring. Thus far, studies that have used the self-estimate technique to investigate self-monitoring and self-awareness in dementia patients have been unsuccessful in showing this distinction (Barrett et al., 2005; Eslinger et al., 2005).

A self-estimate technique is used in the current study, with the aim of investigating selfawareness and self-monitoring of both cognitive and behavioral domains in patients with various forms of dementia. In targeting cognitive and behavioral domains commonly affected by bvFTD, PPA and PrAD, specifically memory, naming and behavior, the current study allows for direct comparison between awareness of cognitive and behavioral domains both within each form of dementia and among the three groups.

The design of the current study was multifactorial, 4 group (three diagnostic groups plus normal control comparison) X 5 domain (two cognitive, one behavioral, and two control) X 2 condition (self-awareness and self-monitoring). Comparisons between diagnostic groups were expected to show bvFTD patients to have the worse ability on both self-awareness or self-monitoring. PPA patients were expected to show a similar degree of self-awareness and self-monitoring to normal controls. Between domain comparisons were expected to demonstrate a particular tendency for patients to have poor insight into their behavioral change, and less for cognitive symptoms, especially naming. Finally, the between condition analyses were expected to demonstrate that patients are more accurate at the self-monitoring stage. To our knowledge, the current study is the first to directly compare awareness for two distinct groups of deficits seen in dementia, behavioral and cognitive, and is also the first to assess awareness of a naming deficit in PPA and bvFTD. The specific approach taken to assess self-monitoring and self-awareness, incorporating standardized scores of actual task performance, is also novel.

## **Methods**

## **Participants**

Patients with diagnoses of PPA (n = 14), bvFTD (n = 11), and probable PrAD (n = 15) as well as older normal controls (NC, n = 15) were recruited for this study. All participants were enrolled in the Clinical Core registry of the Northwestern Alzheimer's Disease Center. Patients and normal controls were invited to participate at the time of their scheduled annual research visit. Written consent was obtained from all subjects and the study was approved by the Institutional Review Board of Northwestern University.

Patients were diagnosed by consensus of a group of behavioral neurologists and neuropsychologists in a specialized dementia center, based on recent research diagnostic criteria (McKhann et al., 1984; Mesulam, 2003; Neary et al., 1998). Participants in the NC group were free of neurological or psychiatric disease. All patients were screened for their ability to complete the study using the Mini Mental Status Exam (MMSE(Folstein, Folstein, & McHugh, 1975). Patients were included only if their score was equal to, or greater than, 15/30, not beyond the range of moderate severity of dementia.

### Procedures

Self-estimate techniques similar to those used in prior studies (Barrett et al., 2005; Eslinger et al., 2005) were used to assess self-awareness and self-monitoring of symptoms within two cognitive domains (Memory and Naming) and one behavioral domain (Behavior). In addition, two control conditions were used to assure that subjects were capable of making ratings. The first was weight and the second eyesight. These control conditions were selected as they are self-related, but neither cognitive nor behavioral.

Prior to each test in the related domain, the participant was presented with a piece of regular, letter-sized, vertically-oriented, white paper with a simply-worded question at the top (e.g., "how good is your memory?") and a 18 cm line centered on the page with three labels, one at the bottom ("terrible"), one in the middle of the line ("average") and one at the top ("perfect"). The subject was then asked to draw an X on the line to mark where he or she believed their own ability to lie. The X could be placed anywhere on the line, making this a continuous scale. Figure 1 outlines the procedure for the naming domain. The distance between the bottom of the line and the subject's mark was taken as an index of the self-estimate.

The relevant test was then administered and the patient's level of ability was based on the test score. For Naming, the total number correct without cues on the Boston Naming Test [BNT (Kaplan, Goodglass, & Weintraub, 1976)] was used. For Memory, the total number correct on the delayed free recall of the Rey Auditory Verbal Learning Test [RAVLT(Rey, 1964)] was used. For Behavior, the patient was administered the Frontal Behavioural Inventory [FBI (Kertesz, Davidson, & Fox, 1997)] a 24-item questionnaire pertaining to common symptoms of dementias caused by FTLD. Although this questionnaire is meant to be completed by a caregiver or other informant, it was completed by both caregiver and patient, separately, in the current study. The caregiver FBI score served as the 'actual performance' for the calculations detailed below.

For the Weight condition, the participant was weighed and measured to calculate body mass index (BMI) which served as the standard against which to compare the subjects' self ratings. BMI was chosen over weight due to more accurate norms and less sex-related bias. For the self-ratings, participants were asked "How heavy are you compared to others your height and sex?". Finally, for Eyesight, the total number of words correctly read on a pocket visual acuity card, without eyeglasses, was used as the index of 'actual ability'. Following administration of the test, an identical page (i.e., with the same question and visual analog scale) was presented

Standardized scores were used to allow for meaningful comparisons between domains. Given that, for example, a score of 50% accuracy on the BNT is not comparable with a score of 50% accuracy on the delayed free recall of the RAVLT, tests scores were converted into T-scores using appropriate published norms (Bleecker, Bolla-Wilson, Agnew, & Meyers, 1988; Kertesz, Nadkarni, Davidson, & Thomas, 2000; Klein, Klein, Linton, & De Mets, 1991; Lethbridge-Cejku, Schiller, & Bernadel, 2004; Van Gorp, Satz, Kiersch, & Henry, 1986). By using this technique, an average memory score is comparable with an average naming score. There are obvious limitations to this approach; specifically, standardized data for each test are derived from different populations and so the scores are not entirely equivalent. However, using standardized scores allows for an improved, more ecologically valid comparison of scores among domains than those methods used in prior studies (Barrett et al., 2005; Eslinger et al., 2005).

Self-awareness scores (SAS) were calculated initially on the basis of the pre-test self-estimates. The self-estimate visual analog scale was converted to a percentage of best possible score, reflecting the number of centimeters from the bottom of the line (so an estimate of 'average' would be  $(9\text{cm}/18\text{cm}) \times 100 = 50\%$ , comparable to an 'average' T score of 50, a slightly above average estimate (e.g., 11cm/18cm) would be comparable to a T score of 61, etc.). The SAS was then calculated according to the following equation:

#### Self – Awareness Score (SAS)=(T – score – pre – test % estimate)

Self-monitoring scores (SMS) were also calculated, using a similar equation:

#### Self – Monitoring Score (SMS)=(T – score – post – test % estimate)

Actual scores were assessed as fraction of best possible score for the Naming, Memory and Eyesight domains, and as the participants' BMI for the Weight domain For the Behavioral domain, the caregiver FBI score was used to calculate the 'actual' T score (norms for depressed, non-demented older individuals (Kertesz et al., 2000)). In keeping with the other domains, patients completed the FBI via semi-structured interview between the pre and post self-estimates. Thus, they were encouraged to think about any of the behaviors queried on the FBI that they may have been exhibiting. Although this is not entirely consistent with reflecting on performance on a cognitive task, or ability to read an eyesight chart, by completing the FBI the patient was forced to consider these symptoms with regard to themselves.

Throughout the administration, the examiner answered questions or clarified the task if the patient was confused, but otherwise verbal communication was kept to a minimum.

#### **Data Analysis**

Demographic information, disease severity data, and T-scores for each of the tasks used in calculation of self-awareness and self-monitoring scores were compared between groups using ANOVA with Bonferroni-corrected post-hoc *t*-tests.

Group differences in the SAS and SMS were assessed using ANOVA. Initially, this was done using the 2 cognitive domains and the 2 control domains, with pairwise Dunnett's tests comparing each diagnostic group to the NC group. Since participants in the NC group were not expected to experience personality change, they were not asked to complete the FBI, or

the Behavior self estimates. Therefore, for the Behavior domain, the three diagnostic groups were compared using an ANOVA with Bonferroni corrected pair-wise comparisons.

Within each group, and within each domain, comparisons were made between SAS and SMS to ascertain if completing the associated task helped participants subsequently make more accurate judgments. Paired-sample *t*-tests were used to make these comparisons.

Within each group, domains were compared using a within group, repeated measures ANOVA with Bonferroni corrected post-hoc *t*-tests conducted for both the SAS and SMS conditions. These were completed first with all three diagnostic groups and the normal controls comparing the cognitive and control domains. Then an additional set of ANOVAs were completed without the NC group, who did not complete the Behavior task, comparing all five domains.

## Results

Demographic data, displayed in Table 1, indicated that bvFTD patients were significantly younger than PrAD patients or the NC group, that the NC group had significantly higher MMSE scores than all diagnostic groups, and that the PrAD group had higher CDR scores (i.e., were more severely demented, although still categorized as "mild", on average) than the PPA group. There were no statistically significant differences in education or disease duration.

Task performance data, displayed in Table 2, show no differences for the control domains (Weight and Eyesight). The NC group was significantly better at Naming than all diagnostic groups, and there were no differences within the diagnostic groups. Similarly, the NC group performed better on Memory than the diagnostic groups, and the PPA group performed better than the PrAD group. Within the diagnostic groups, the caregiver FBI for patients with PrAD and bvFTD was higher than that of the PPA group, but the PPA patients actually reported more (or more severe) symptoms on the FBI than did patients in the PrAD group.

The spread of SAS and SMS scores for each diagnostic group is depicted in figure 2.

## **Differences Between Diagnostic Groups**

#### Diagnostic groups and normal controls (Behavior omitted)

**Self-Awareness Score:** The effect of group was significant only for Memory (F (3, 54) = 6.95, p = .001). Both the bvFTD and PrAD patients significantly overestimated their memory ability in comparison with NC (p<.05).

<u>Self-Monitoring Score:</u> Similarly, the groups significantly differed only for Memory (F (3, 54) = 7.25, p<.0005), with bvFTD and PrAD patients overestimating their ability (p<.005).

#### **Diagnostic groups only (Behavior included)**

<u>Self-Awareness Score</u>: Memory was, again, the only domain with a significant effect for the SAS (F (2,39) = 3.50, p = .04) although differences were significant with correction between the two groups (probably related to the correction).

**Self- Monitoring Score:** Memory differed across groups (F (2,39) = 3.32, p = .05), but again no two groups differed significantly (with correction) from each other. There was also a significant effect of diagnosis on the Behavior domain (F = 4.88 (2, 38), p = .011). The PrAD group significantly underestimated their behavioral change in comparison with the PPA group (p<.005), however the difference between the PPA and bvFTD groups did not reach significance.

#### **Comparisons of Self-Awareness Scores and Self-Monitoring Scores**

For the Weight condition, only PPA patients differed (t = 2.13, df = 13 p = .012), with a score suggesting more accurate performance on SMS than SAS. No patients differed significantly on Eyesight. On the cognitive domains, the NC group actually underestimated their ability more at the SMS of Naming (t = 3.50, df = 14, p < .005), and there were no significant differences in the Memory or Behavioral domains.

## **Comparison of Domains**

## Control and cognitive domains within group

Self-Awareness Score: The NC and PPA groups performed similarly on each domain, with no main effects. However, significant effects of domain were seen for the bvFTD group (Wilks'  $\Lambda = .29$ , F(1,8) = 6.50, p = .015, multivariate  $\eta^2 = .71$ ). Pairwise comparisons indicated that they were particularly poor at estimating their Memory, in comparison to Naming and Eyesight (p < .05). There was also a main effect of domain for the PrAD group (Wilks'  $\Lambda = .24$ , F(1,12) = 12.94, p < .001, multivariate  $\eta^2 = .76$ ). PrAD patients were also significantly worse at estimating Memory in comparison to Eyesight and Naming, and also in comparison to Weight (p < .05).

**Self-Monitoring Score:** There was no effect of domain for the NC group. The PPA group performed significantly differently across domains (Wilks'  $\Lambda = .46$ , F(1, 11) = 4.30, p = .03, multivariate  $\eta^2 = .54$ ), with comparisons indicating that Naming was significantly higher (in this case due to underestimation of ability) in comparison with Eyesight (p = .027). A significant effect of domain was also seen for the bvFTD group (Wilks'  $\Lambda = .18$ , F(1,8) = 12.18, p = .002, multivariate  $\eta^2 = .82$ ), who were significantly worse at estimating Memory than Naming (p = .003) or Eyesight (p = .004), and also underestimated their Naming to a greater degree than their Weight (p = .024). The PrAD group (Wilks'  $\Lambda = .70$ , F(1,12) = 9.48, p = .002, multivariate  $\eta^2 = .70$ ) overestimated their Memory in comparison to both control domains (Eyesight p = .003, Weight p = .001) as well as Naming (p = .003) and also underestimated their Naming in comparison with Eyesight (p = .047).

### Control, cognitive and behavioral domains within groups

**Self-Awareness Score:** Comparisons of ability on the cognitive and control domains with Behavior within each diagnostic group (i.e., excluding the NC group) for the SAS condition, revealed that there was a significant effect of domain for all three diagnostic groups. For the PPA group (Wilks'  $\Lambda = .24$ , F(1, 10) = 7.81, p = .004, multivariate  $\eta^2 = .76$ ) Behavior was lower than Memory and the two conditions (p < .05). For the BvFTD group (Wilks'  $\Lambda = .20$ , F(1,6) =5.86, p = .029, multivariate  $\eta^2 = .80$ ) Behavior was not different to any of the groups, so the main effect was driven by the differences in cognitive and control domains described above. The PrAD group (Wilks'  $\Lambda = .17$ , F(1,11) = 13.59, p < .0005, multivariate  $\eta^2 = .83$ ) performed similarly to the PPA patients, with Behavior lower than Memory and the two control conditions (Eyesight and Weight: p < .05).

**Self-Monitoring Score:** Comparisons of Behavior to other domains for the SMS condition indicated that all three diagnostic groups had overall significant effects of SMS domain. PPA patients (Wilks'  $\Lambda = .23$ , F(1,10) = 8.44, p = .003, multivariate  $\eta^2 = .77$ ) were less accurate at estimating Behavior than Weight, Eyesight or Naming. BvFTD patients (Wilks'  $\Lambda = .09$ , F (1,6) = 14.85, p = .003, multivariate  $\eta^2 = .90$ ) were less accurate at estimating Behavior than Eyesight or Naming. PrAD patients (Wilks'  $\Lambda = .10$ , F(1,11) = 25.80, p < .0005, multivariate  $\eta^2 = .90$ ) were less accurate at estimating Behavior than any of the other domains.

## Discussion

This study investigated differences in aspects of insight pertaining to deficits in cognitive and behavioral domains among groups with three distinct dementia diagnoses, namely, behavioral variant frontotemporal dementia, primary progressive aphasia, and probable Alzheimer's disease. Self-awareness and self-monitoring were both tested by assessing general awareness of limitations and the extent to which perceptions could be modified based on completion of a domain-specific task, respectively. Control tasks judging weight and eyesight were used to determine if any findings could be explained by a more general deficit in self-judgment. Results have implications for differences in insight among diagnostic groups, differences between self-awareness and self-monitoring as two components of insight, and differences in insight for different domains or symptoms. The main results were:

- 1. There were no group differences on either self-awareness or self-monitoring for the control domains, suggesting that the PPA, bvFTD and PrAD groups were equally accurate at non-cognitive, non-behavioral judgments as controls.
- 2. Differences between diagnostic groups for insight into certain non-control domains were evident, with bvFTD and PrAD patients being generally less accurate than controls or PPA patients.
- 3. There were very few differences between self-awareness and self-monitoring.
- **4.** Consistent with expectation, changes in behavior were more frequently associated with a reduction in insight than other symptoms. This was true across symptoms as a function of degree of behavioral change.
- **5.** Insight into behavioral symptoms did not differ significantly between bvFTD and PPA patients.
- 6. Memory loss was also susceptible to reduced insight, but in contrast naming ability was associated with good insight, even in bvFTD patients.

The group differences found in this study were partially consistent with predictions, in that bvFTD patients showed less awareness than PPA patients and controls in some domains. However, the finding that the PrAD patients were the poorest at estimating their behavioral change was unexpected. Research on insight in PrAD patients indicates that a sizeable proportion (20-60 %) suffer from some degree of reduced awareness (Bozzola, Gorelick, & Freels, 1992; Migliorelli et al., 1995), with research often concentrating on awareness of memory and other cognitive deficits (Feher, Larrabee, Sudilovsky, & Crook, 1994; Howorth & Saper, 2003; Reed et al., 1993). Other studies have associated loss of insight in PrAD with behavioral changes such as apathy (Ott et al., 1996; Starkstein, Petracca, Chemerinski, & Kremer, 2001), irritability, anxiety (Vasterling, Seltzer, & Watrous, 1997) and disinhibition (Starkstein, Garau, & Cao, 2004; Starkstein et al., 2006). Depression, logically, appears to be associated with intact insight (Vasterling et al., 1997). Although there is evidence from these studies that PrAD patients have poor insight into the majority of behavioral symptoms, a recent study indicated that PrAD patients are less susceptible to reduced insight into personality deficits than bvFTD patients (Rankin et al., 2005). A possible explanation for the difference between Rankin and colleagues' results and our own is that they used a different technique (the discrepancy between patients' and caregivers reports on personality questionnaires) and concentrated on traits as opposed to specific behavioral deficits (Rankin et al., 2005).

The finding that PPA patients performed similarly to normal controls on all cognitive and control domains confirms the assumption that these patients, especially in the earlier stages of the disease, have intact insight into their symptoms. However, the bvFTD and PPA groups did not differ significantly on the behavioral domain, although PPA patients had significantly lower

(caregiver) scores on the FBI in comparison with bvFTD patients. It seems, then, that PPA patients have reduced insight into those behavioral changes that do occur (both at the self-monitoring and self-assessment stages).

No significant differences were found within any diagnostic group between the self-awareness score and the self-monitoring score in the cognitive or behavioral domains. In fact, even when the normal control group's self-awareness scores were inaccurate, as in the case of naming, they went on to further underestimate their ability at the self-monitoring stage. It may be that the methodology was ineffective at detecting any differences between self-awareness and selfmonitoring. The theoretical underpinnings of these two constructs remains logical, both from a conceptual point of view and in terms of the neural networks that they would utilize. Selfawareness of different domains may be expected to use some shared regions, such as those involved in the recall of episodic memories, or in self reflection, independent of the domain being tested. They may, in addition, involve domain specific networks. Self-monitoring, however, may use less distributed networks which activate online. Evidence from studies with normal controls using event-related fMRI (Hester, Foxe, Molholm, Shpaner, & Garavan, 2005) and event-related potentials (Nieuwenhuis, Ridderinkhof, Blom, Band, & Kok, 2001; O'Connell et al., 2007) point to an increasingly well-defined self-monitoring network involving the anterior cingulate cortex and regions of the parietal and frontal cortex. This distinction may be more made more apparent by adapting the method used in the current study, making errors more readily apparent to the participant, or giving them feedback of their success or failure (e.g., "You recalled 3 of the 10 words correctly"), may provide more ecologically valid and clinically useful information.

All three patient groups showed the least insight (self-awareness or self-monitoring) for behavior. This was consistent with expectation, given the evidence for a close association between poor insight and behavioral disturbances seen in PrAD (Kashiwa et al., 2005; Migliorelli et al., 1995; Starkstein et al., 2004) and findings within dementias caused by FTLD of reduced insight into personality change (Rankin et al., 2005). In the current study, we assessed insight into personality change overall, as opposed to specific deficits. Earlier studies in PrAD have pointed to distinctions regarding insight for various behavioral symptoms. Intact awareness for depression has been demonstrated in PrAD (Barrett et al., 2005; Vasterling et al., 1997), and PPA patients often show depressive symptomatology (Medina & Weintraub, 2007). In contrast, anosognosia has been associated with disinhibition (Kashiwa et al., 2005; Starkstein et al., 2004) and apathy (Migliorelli et al., 1995), symptoms common in bvFTD (Banks & Weintraub, In Press; Rosen et al., 2005). Future studies might be more specific in their examination of awareness of personality or behavioral changes, since interesting dissociations may exist within the range of behavioral and mood disturbances seen in the various dementias.

Memory was another domain that appeared vulnerable to loss of insight in PrAD and bvFTD groups, although not in PPA or NC groups. Research on loss of insight into memory changes has suggested that poor insight is more strongly associated with effortful retrieval of verbal information (Gallo et al., in press), similar to the delayed recall test used in the current study. Tests of recognition memory, or other aspects of memory that involve less effort than uncued retrieval, may have been associated with better awareness in the bvFTD group. This is a potential focus for future research.

Perhaps the most surprising finding was the tendency of all diagnostic groups to underestimate naming ability to a similar degree as they overestimated the maintenance of their usual behavior. The apparent protected status of language from anosognosia is intriguing. The notable exception is patients with Wernicke's aphasia who show signs of jargonaphasia and seem to show no insight into their production of meaningless speech. There have been

surprisingly few studies of anosognosia in aphasia (Lebrun, 1987; Weinstein et al., 1964) and none to date in PPA. It may be that patients with more fluent, Wernicke's-like subtypes of PPA are more likely to have reduced insight into their language deficits. An alternative explanation for participants' acute awareness of language problems is the frequency with which they are encountered in every day life, even by normally aging individuals. Memory lapses, on the other hand, may be more easily forgotten or unnoticed.

This dichotomy between preserved insight for naming and reduced insight for behavioral changes may well have neuroanatomical underpinnings. Personality change in dementia is generally associated with right hemisphere and/or frontal damage (Miller et al., 2001; Rosen et al., 2005), the same areas commonly involved in anosognosia (Mendez & Shapira, 2005), whereas anomia is related to left perisylvian damage.

These findings have various clinical implications. For bvFTD patients, intact insight for language provides a potential avenue both to assist patients in comprehending that they have undergone some form of disease process, and also to provide a focus for therapy. Therapy with Speech Language Pathologists has proven to be beneficial in PPA (Thompson & Johnson, 2006), but no trials have yet been completed in bvFTD. Their lack of insight into behavioral changes or reduced ability on tests of memory confirms the futility in trying to convince these patients of the extent of such deficits. Both lack of insight (Seltzer, Vasterling, Yoder, & Thompson, 1997) and behavioral disturbance (Rymer et al., 2002) have been associated with higher levels of caregiver burden in Alzheimer's disease. Caregiver education is important in mediating this burden in all dementia caregivers, especially when patients exhibit disruptive behaviors (Ostwald, Hepburn, Caron, Burns, & Mantell, 1999). Caregivers may find it reassuring that not only the behavioral and cognitive changes, but also the lack of insight, are a direct result of the disease process, and the patient is not behaving in a difficult, hurtful or indifferent manner on purpose. For PPA patients, the lack of insight into behavioral change is perhaps the most clinically pertinent finding, since these symptoms tend to emerge as the disease progresses. This has important implications in terms of planning as much as possible early in the disease course, when the affected individual is still able to take an active role in decision making about their future care. From a societal prospective, the finding that patients with bvFTD have such limited insight specifically into their behavioral changes is important given the tendency of some of these patients to engage in antisocial behaviors (Mendez, Chen, Shapira, & Miller, 2005; Passant, Elfgren, Englund, & Gustafson, 2005). It may be that adaptations to social and legal policies need to be made for patients with bvFTD.

This study has several limitations. The number of patients in each diagnostic group was relatively small. This reduced power also prevented exploration of relevant concepts within diagnostic groups, such as the impact of duration of illness on reduced insight. The choice not to complete the behavioral task on the Normal Control group, while logical, resulted in unanswered questions relating to awareness of personality change by individuals experiencing normal aging. Behavior was assessed as a unitary domain, when in fact it encompasses a wide range of symptoms some of which may be more likely associated with reduced insight than others. Future studies may find distinctions between insight into depression and disinhibition, for example. Finally, the neuropsychological measures and control tasks chosen to compare different domains were not similar. The tasks that were selected had strong psychometric properties and reliable normative data, but were quite distinct from each other in terms of format. Despite this, another potential problem may be the ceiling effect seen in BNT norms. Future studies might incorporate experimental measures that have more similarities (e.g., remembering 10 words, naming 10 objects, answering 10 questions about behavior) in order to reduce the confound of different domain specific tasks on the validity of results.

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## References

- Adair JC, Gilmore RL, Fennell EB, Gold M, Heilman KM. Anosognosia during intracarotid barbiturate anesthesia: unawareness or amnesia for weakness. Neurology 1995;45(2):241–243. [PubMed: 7854519]
- Ansell EL, Bucks RS. Mnemonic anosognosia in Alzheimer's disease: a test of Agnew and Morris (1998). Neuropsychologia 2006;44(7):1095–1102. [PubMed: 16324727]
- Banks S, Weintraub S. Neuropsychiatric Symptoms in Behavioral Variant Frontotemporal Dementia and Primary Progressive Aphasia. Journal of Geriatric Psychiatry and Neurology. In Press
- Barrett AM, Eslinger PJ, Ballentine NH, Heilman KM. Unawareness of cognitive deficit (cognitive anosognosia) in probable AD and control subjects. Neurology 2005;64(4):693–699. [PubMed: 15728294]
- Beer JS, John OP, Scabini D, Knight RT. Orbitofrontal cortex and social behavior: integrating selfmonitoring and emotion-cognition interactions. Journal of Cognitive Neuroscience 2006;18(6):871– 879. [PubMed: 16839295]
- Blair M, Marczinski CA, Davis-Faroque N, Kertesz A. A longitudinal study of language decline in Alzheimer's disease and frontotemporal dementia. Journal of the International Neuropsychological Society 2007;13(2):237–245. [PubMed: 17286881]
- Bleecker ML, Bolla-Wilson K, Agnew J, Meyers DA. Age-related sex differences in verbal memory. Journal of Clinical Psychology 1988;44(3):403–411. [PubMed: 3384968]
- Bozzola FG, Gorelick PB, Freels S. Personality changes in Alzheimer's disease. Arch Neurol 1992;49 (3):297–300. [PubMed: 1536633]
- Cutting J. Study of anosognosia. Journal of Neurology, Neurosurgery and Psychiatry 1978;41(6):548–555.
- Dalla Barba G, Parlato V, Iavarone A, Boller F. Anosognosia, intrusions and 'frontal' functions in Alzheimer's disease and depression. Neuropsychologia 1995;33(2):247–259. [PubMed: 7746367]
- DeBettignies BH, Mahurin RK, Pirozzolo FJ. Insight for impairment in independent living skills in Alzheimer's disease and multi-infarct dementia. Journal of Clinical and Experimental Neuropsychology 1990;12(2):355–363. [PubMed: 2341561]
- Eslinger PJ, Dennis K, Moore P, Antani S, Hauck R, Grossman M. Metacognitive deficits in frontotemporal dementia. Journal of Neurology, Neurosurgery and Psychiatry 2005;76(12):1630– 1635.
- Feher EP, Larrabee GJ, Sudilovsky A, Crook TH 3rd. Memory self-report in Alzheimer's disease and in age-associated memory impairment. Journal of Geriatric Psychiatry and Neurology 1994;7(1):58– 65. [PubMed: 8192832]
- Folstein MF, Folstein SE, McHugh PR. Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. Journal of Psychiatry Research 1975;12(3):189–198.
- Fordyce DJ, Roueche JR. Changes in perspectives of disability among patients, staff and relatives during rehabilitation of brain injury. Rehabilitation Psychology 1986;312:217–229.
- Frederiks, J. The Neurology of Aging and Dementia. In: Vinken, P.; Bruyn, J.; Klawans, H., editors. Handbook of Clinical Neurology. New York: Elsevier; 1985.
- Gallo DA, Chen JM, Wiseman AL, Schacter DL. Retrieval monitoring and anosognosia in Alzheimer's disease. Neuropsychology. in press
- Gerstmann J. Problems of imperception of disease and impaired body territories with organic lesions. Archives of Neurology and Psychiatry 1942;48:890–913.
- Gorno-Tempini ML, Dronkers NF, Rankin KP, Ogar JM, Phengrasamy L, Rosen HJ, et al. Cognition and anatomy in three variants of primary progressive aphasia. Annals of Neurology 2004;55(3):335– 346. [PubMed: 14991811]

- Harwood DG, Sultzer DL, Wheatley MV. Impaired insight in Alzheimer disease: association with cognitive deficits, psychiatric symptoms, and behavioral disturbances. Neuropsychiatry, Neuropsychology and Behavioral Neurology 2000;13(2):83–88.
- Hester R, Foxe JJ, Molholm S, Shpaner M, Garavan H. Neural mechanisms involved in error processing: a comparison of errors made with and without awareness. Neuroimage 2005;27(3):602–608. [PubMed: 16024258]
- Howorth P, Saper J. The dimensions of insight in people with dementia. Aging and Mental Health 2003;7 (2):113–122. [PubMed: 12745389]
- Kaplan, E.; Goodglass, H.; Weintraub, S. Boston Naming Test. Experimental Edition. Boston: Aphasia Research Center, Boston University; 1976.
- Kashiwa Y, Kitabayashi Y, Narumoto J, Nakamura K, Ueda H, Fukui K. Anosognosia in Alzheimer's disease: association with patient characteristics, psychiatric symptoms and cognitive deficits. Psychiatry and Clinical Neurosciences 2005;59(6):697–704. [PubMed: 16401246]
- Kertesz A, Davidson W, Fox H. Frontal behavioral inventory: diagnostic criteria for frontal lobe dementia. Canadian Journal of Neurological Sciences 1997;24(1):29–36. [PubMed: 9043744]
- Kertesz A, Davidson W, McCabe P, Takagi K, Munoz D. Primary progressive aphasia: diagnosis, varieties, evolution. Journal of the International Neuropsychological Society 2003;9(5):710–719. [PubMed: 12901777]
- Kertesz A, Nadkarni N, Davidson W, Thomas AW. The Frontal Behavioral Inventory in the differential diagnosis of frontotemporal dementia. Journal of the International Neuropsychological Society 2000;6(4):460–468. [PubMed: 10902415]
- Klein R, Klein BE, Linton KL, De Mets DL. The Beaver Dam Eye Study: visual acuity. Ophthalmology 1991;98(8):1310–1315. [PubMed: 1923372]
- Lebrun Y. Anosognosia in aphasics. Cortex 1987;23(2):251-263. [PubMed: 2440639]
- Lethbridge-Cejku M, Schiller JS, Bernadel L. Summary health statistics for U.S. adults: National Health Interview Survey, 2002. Vital Health Statistics 2004;10(222):1–151.
- Marczinski CA, Davidson W, Kertesz A. A longitudinal study of behavior in frontotemporal dementia and primary progressive aphasia. Cognitive and Behavioral Neurology 2004;17(4):185–190. [PubMed: 15622012]
- McGlynn SM, Schacter DL. Unawareness of deficits in neuropsychological syndromes. Journal of Clinical and Experimental Neuropsychology 1989;11(2):143–205. [PubMed: 2647781]
- McKhann G, Albert MS, Grossman M, Miller B, Dickson D, Trojanowski JQ. Clinical and pathological diagnosis of frontotemporal dementia: report of the Work Group on Frontotemporal Dementia and Pick's Disease. Archives of Neurology 2001;58(11):1803–1809. [PubMed: 11708987]
- McKhann G, Drachman D, Folstein M, Katzman R, Price D, Stadlan EM. Clinical diagnosis of Alzheimer's disease: report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. Neurology 1984;34(7):939–944. [PubMed: 6610841]
- Medina JE, Weintraub S. Depression in Primary Progressive Aphasia. Journal of Geriatric Psychiatry and Neurology 2007;20:153–160. [PubMed: 17712098]
- Mendez MF, Chen AK, Shapira JS, Miller BL. Acquired Sociopathy and Frontotemporal Dementia. Dementia and Other Geriatric Cognitive Disorders 2005;20(2–3):99–104.
- Mendez MF, Shapira JS. Loss of insight and functional neuroimaging in frontotemporal dementia. Journal of Neuropsychiatry and Clinical Neurosciences 2005;17(3):413–416. [PubMed: 16179666]
- Mesulam MM. Primary progressive aphasia--differentiation from Alzheimer's disease. Annals of Neurology 1987;22(4):533–534. [PubMed: 3324947]
- Mesulam MM. Primary progressive aphasia--a language-based dementia. New England Journal of Medicine 2003;349(16):1535–1542. [PubMed: 14561797]
- Migliorelli R, Teson A, Sabe L, Petracca G, Petracchi M, Leiguarda R, et al. Anosognosia in Alzheimer's disease: a study of associated factors. Journal of Neuropsychiatry and Clinical Neurosciences 1995;7 (3):338–344. [PubMed: 7580194]
- Miller BL, Seeley WW, Mychack P, Rosen HJ, Mena I, Boone K. Neuroanatomy of the self: evidence from patients with frontotemporal dementia. Neurology 2001;57(5):817–821. [PubMed: 11552010]

- Neary D, Snowden JS, Gustafson L, Passant U, Stuss D, Black S, et al. Frontotemporal lobar degeneration: a consensus on clinical diagnostic criteria. Neurology 1998;51(6):1546–1554. [PubMed: 9855500]
- Nieuwenhuis S, Ridderinkhof KR, Blom J, Band GP, Kok A. Error-related brain potentials are differentially related to awareness of response errors: evidence from an antisaccade task. Psychophysiology 2001;38(5):752–760. [PubMed: 11577898]
- O'Connell RG, Dockree PM, Bellgrove MA, Kelly SP, Hester R, Garavan H, et al. The role of cingulate cortex in the detection of errors with and without awareness: a high-density electrical mapping study. Eur J Neurosci 2007;25(8):2571–2579. [PubMed: 17445253]
- O'Keeffe FM, Murray B, Coen RF, Dockree PM, Bellgrove MA, Garavan H, et al. Loss of insight in frontotemporal dementia, corticobasal degeneration and progressive supranuclear palsy. Brain 2007;130(Pt 3):753–764. [PubMed: 17347257]
- Ostwald SK, Hepburn KW, Caron W, Burns T, Mantell R. Reducing caregiver burden: a randomized psychoeducational intervention for caregivers of persons with dementia. Gerontologist 1999;39(3): 299–309. [PubMed: 10396888]
- Ott BR, Lafleche G, Whelihan WM, Buongiorno GW, Albert MS, Fogel BS. Impaired awareness of deficits in Alzheimer disease. Alzheimer Disease and Associated Disorders 1996;10(2):68–76. [PubMed: 8727167]
- Passant U, Elfgren C, Englund E, Gustafson L. Psychiatric symptoms and their psychosocial consequences in frontotemporal dementia. Alzheimer Disease and Associated Disorders 2005;19 (Suppl 1):S15–18. [PubMed: 16317252]
- Rankin KP, Baldwin E, Pace-Savitsky C, Kramer JH, Miller BL. Self awareness and personality change in dementia. Journal of Neurology, Neurosurgery and Psychiatry 2005;76(5):632–639.
- Reed BR, Jagust WJ, Coulter L. Anosognosia in Alzheimer's disease: relationships to depression, cognitive function, and cerebral perfusion. Journal of Clinical and Experimental Neuropsychology 1993;15(2):231–244. [PubMed: 8491848]
- Rey, A. L'examen clinique en psychologie. Paris: Presses Universitaires de France; 1964.
- Rosen HJ, Allison SC, Schauer GF, Gorno-Tempini ML, Weiner MW, Miller BL. Neuroanatomical correlates of behavioural disorders in dementia. Brain 2005;128(Pt 11):2612–2625. [PubMed: 16195246]
- Rosen HJ, Gorno-Tempini ML, Goldman WP, Perry RJ, Schuff N, Weiner M, et al. Patterns of brain atrophy in frontotemporal dementia and semantic dementia. Neurology 2002;58(2):198–208. [PubMed: 11805245]
- Ruby P, Schmidt C, Hogge M, D'Argembeau A, Collette F, Salmon E. Social mind representation: where does it fail in frontotemporal dementia? Journal of Cognitive Neuroscience 2007;19(4):671–683. [PubMed: 17381257]
- Rymer S, Salloway S, Norton L, Malloy P, Correia S, Monast D. Impaired awareness, behavior disturbance, and caregiver burden in Alzheimer disease. Alzheimer Disease and Associated Disorders 2002;16(4):248–253. [PubMed: 12468899]
- Schacter DL. Toward a cognitive neuropsychology of awareness: implicit knowledge and anosognosia. Journal of Clinical and Experimental Neuropsychology 1990;12(1):155–178. [PubMed: 2406281]
- Seltzer B, Vasterling JJ, Yoder JA, Thompson KA. Awareness of deficit in Alzheimer's disease: relation to caregiver burden. Gerontologist 1997;37(1):20–24. [PubMed: 9046701]
- Souchay C, Isingrini M, Pillon B, Gil R. Metamemory accuracy in Alzheimer's disease and frontotemporal lobe dementia. Neurocase 2003;9(6):482–492. [PubMed: 16210230]
- Starkstein SE, Garau ML, Cao A. Prevalence and clinical correlates of disinhibition in dementia. Cognitive and Behavioral Neurology 2004;17(3):139–147. [PubMed: 15536301]
- Starkstein SE, Jorge R, Mizrahi R, Robinson RG. A diagnostic formulation for anosognosia in Alzheimer's disease. Journal of Neurology, Neurosurgery and Psychiatry 2006;77(6):719–725.
- Starkstein SE, Petracca G, Chemerinski E, Kremer J. Syndromic validity of apathy in Alzheimer's disease. American Journal of Psychiatry 2001;158(6):872–877. [PubMed: 11384893]
- Stuss, D. Disturbance of self awareness after frontal systems damage. In: Prigatano, GP.; Schacter, DL., editors. Awareness of Deifcit after Brain Injury. New York: Oxford University Press; 1991. p. 63-83.
- Thompson, CK.; Johnson, N. Language intervention in dementia. In: ADK; W-BKA, editors. Geriatric Neuropsychology. New York: Guilford; 2006. p. 315-332.

- Toglia J, Kirk U. Understanding awareness deficits following brain injury. NeuroRehabilitation 2000;15 (1):57–70. [PubMed: 11455082]
- Van Gorp WG, Satz P, Kiersch ME, Henry R. Normative data for the Boston Naming Test for a group of normal older adults. Journal of Clinical and Experimental Neuropsychology 1986;8:702–705. [PubMed: 3782449]
- Vasterling JJ, Seltzer B, Watrous WE. Longitudinal assessment of deficit unawareness in Alzheimer's disease. Neuropsychiatry, Neuropsychology and Behavioral Neurology 1997;10(3):197–202.
- Vogel C. Cognitive and functional neuroimaging correlate for anosognosia in mild cognitive impairment and Alzheimer's disease. International Journal of Geriatric Psychiatry 2005;20(3):238–246. [PubMed: 15717342]
- Weinstein EA, Cole M, Mitchell MS, Lyerly OG. Anosognosia and Aphasia. Archives of Neurology 1964;10:376–386. [PubMed: 14107687]
- Weintraub, S.; Mesulam, M-M. Handbook of Neuropsychology. Vol. 8. 1993. Four neuropsychological profiles in dementia; p. 253-282.

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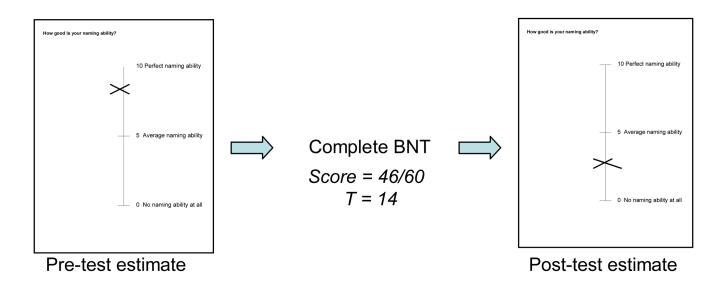
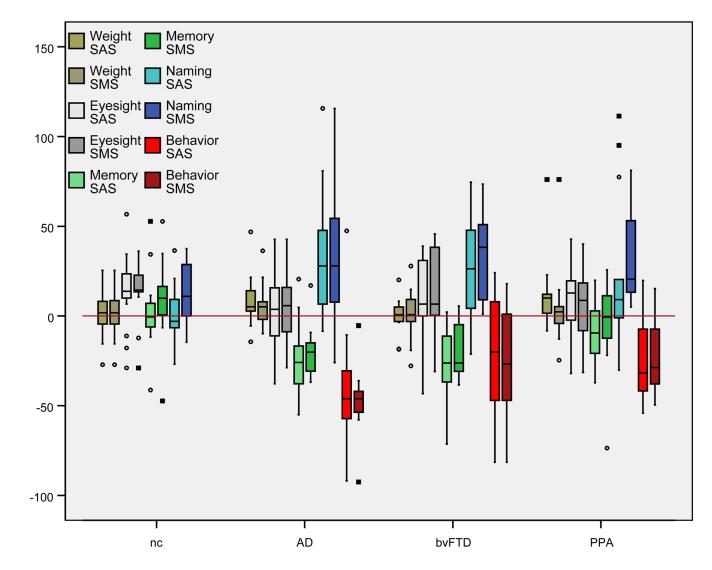
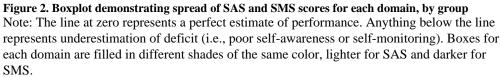


Figure 1. An example of the procedure taken from a 62 year-old male with a diagnosis of PrAD Note: When asked to complete the visual analog scale initially, he considers his naming ability to be quite good, indicating this with a mark that is 83% of the way up the line. His actual BNT score is poor (T=14). This difference between pretest estimate and actual performance suggests self-awareness was poor. However, he realized that he should have known some of the words he missed, and this is reflected on the post-estimate, where his mark is just 33% from the bottom of the line. Thus, in this case, self-monitoring was judged more intact than self-awareness.





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Table 1

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		NC	PrAD	bvFTD	PPA	Group differences (p<.05)
	Mean	15	15	11*	14	
Age	Mean	72.60	77.47	63.82	69.79	BvFTD < NC, PrAD
	s.d.	6.03	8.81	6.27	7.87	
Education	Mean	15.93	13.07	15.91	16.00	no differences
	s.d.	2.63	3.81	2.21	2.96	
Duration of disease	Mean	n/a	4.60	5.55	4.21	no differences
	s.d.		2.75	3.30	1.57	
MMSE	Mean	29.33	21.93	24.45	23.00	NC > PrAD, bvFTD, PPA
	s.d.	1.05	4.28	4.23	6.65	
CDR	Mean	n/a	1.11	1.05	0.50	PrAD > PPA
	s.d.		0.40	0.55	0.39	

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Banks and Weintraub

		NC	PrAD	bvFTD	PPA	Group differences (p<.05)
WEIGHT	Mean	51.49	54.17	61.93	51.14	no differences
BMI	s.d.	7.82	13.18	20.31	20.41	
EYESIGHT	Mean	50.00	52.47	56.84	51.76	no differences
Visual Acuity	s.d.	10.00	10.10	12.95	7.65	
BEHAVIOR, FBI (caregiver)	Mean	;	61.84	69.34	48.40	PPA < PrAD, BvFTD
	s.d.	;	12.34	14.13	9.86	
FBI (patient)	Mean	;	38.25	41.99	43.62	PPA > PrAD
	.b.s	;	3.04	8.46	4.36	
NAMING	Mean	58.93	27.14	31.73	29.96	
BNT	s.d.	3.61	26.97	26.97	24.69	NC > PrAD, bvFTD, PPA
MEMORY	Mean	61.86	21.10	29.55	37.21	NC > PrAD, bvFTD, PPA
RAVLT Delayed Recall	s.d.	17.76	4.03	16.07	18.75	PPA > PrAD