Semantic Error Patterns on the Boston Naming Test in Normal Aging, Amnestic Mild Cognitive Impairment, and Mild Alzheimer’s Disease: Is There Semantic Disruption?

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Naming difficulty is common in Alzheimer’s disease (AD), but the nature of this problem is not well established. The authors investigated the presence of semantic breakdown and the pattern of general and semantic errors in patients with mild AD, patients with amnestic mild cognitive impairment (aMCI), and normal controls by examining their spontaneous answers on the Boston Naming Test (BNT) and verifying whether they needed or were benefited by semantic and phonemic cues. The errors in spontaneous answers were classified in four mutually exclusive categories (semantic errors, visual paragonymia, phonological errors, and omission errors), and the semantic errors were further subclassified as coordinate, superordinate, and circumlocutory. Patients with aMCI performed normally on the BNT and needed fewer semantic and phonemic cues than patients with mild AD. After semantic cues, subjects with aMCI and control subjects gave more correct answers than patients with mild AD, but after phonemic cues, there was no difference between the three groups, suggesting that the low performance of patients with AD cannot be completely explained by semantic breakdown. Patterns of spontaneous naming errors and subtypes of semantic errors were similar in the three groups, with decreasing error frequency from coordinate to superordinate to circumlocutory subtypes.

Keywords: Alzheimer’s disease, mild cognitive impairment, naming test, semantic memory

Naming tests are simple neuropsychological tools that reveal several aspects concerning how the human mind stores knowledge. They involve visual perception, activation of linguistic and executive competencies that include semantic representations, lexical access decisions, and phonological retrieval. Naming complaints are very common in mentally healthy elderly people. Over the age of 70, individuals achieve significantly lower scores on these tests than those achieved by young adults (Albert, Heller, & Milberg, 1988; LaBarge, Edwards, & Knesevich, 1986; Zec, Markwell, Burkett, & Larsen, 2005). Some reports attribute this poor performance to difficulty in using semantic information for word retrieval, stating that lexical representation remains intact (Albert et al., 1988). Problems with naming and word finding are even more common in mild cognitive impairment (MCI) and particularly in Alzheimer’s disease (AD) (Adlam, Rozeat, Arnold, Watson, & Hodges, 2006; Dudas, Clague, Thompson, Graham, & Hodges, 2005). MCI is a clinical term applied to patients with objective cognitive problems, most commonly in episodic memory, without significant impairment of daily life activities. MCI can be classified according to the clinical presentation of symptoms as amnestic MCI (aMCI), multiple domain MCI, or single nonmemory domain MCI. It is assumed that there is a continuum in cognitive decline, and aMCI could be considered an intermediate stage between normal aging and AD, although not all patients will progress to dementia (Petersen, 2004; Winblad et al., 2004).

There are controversies regarding the nature of the naming deficit in AD over whether it should be considered a disruption of concepts and semantic knowledge or a difficulty in accessing the intact lexical-semantic field. A related methodological problem is that virtually all semantic memory tests involve other cognitive domains, which makes the exclusive assessment of the lexical-semantic system difficult, given the complexity of the cerebral organization of cognition. This difficulty could be overcome with procedures like the priming paradigm, which is an important way to evaluate the semantic field indirectly or implicitly by observing changes in the time and accuracy with which individuals perform simple word-nonword decisions (lexical decisions) or in overlearned language tasks, such as word reading (Milberg, McGlinchey-Berroth, Duncan, & Higgins, 1999).

Several authors believe that the main problem for patients with AD is a breakdown in semantic processing (Garrard, Lambon Ralph, Patterson, Pratt, & Hodges, 2005; Hodges, Salmon, & Butters, 1992; Lukatela, Edwards, & Knesevich, 1998), although other cognitive functions involved in the naming process, like working memory, attention, visuospatial skills, and lexical access, might also have an influence (Rogers, Ivanou, Patterson, & Hodges, 2006).

Even impairment in the ability to inhibit inappropriate or no-longer-relevant information might play a main role in naming errors when patients experience increased interference from a previous stimulus (Balota et al., 1991). In early AD, poor naming performance may result from changes in attentional control and/or lexical access processes. In this case, patients might present with...
difficulties in selecting the correct lexical-phonological response after activation of an intact semantic field (Chenery, Murdoch, & Ingram, 1996). Hajilou and Done (2007) suggested that one possible cause of object recognition impairment in AD could be a deficit in processing structural aspects of visually presented items. Some authors have cited these patients’ numerous semantic errors on visual confrontation naming as evidence for impaired semantic knowledge (Adlam et al., 2006; Barbarotto, Capitani, Jori, Lai-acona, & Molinari, 1998; Hodges et al., 1992), although these patients were qualitatively not so different from normal matched controls (Nicholas, Obler, Au, & Albert, 1996). Lukatela et al. (1998), after subclassifying the semantic naming errors, found that even in early AD the semantic system is damaged and these patients tend to commit subordinate errors (by naming the category instead of the object pictured). Poor performances on other lexical-semantic tasks, like category verbal fluency (Murphy, Rich, & Troyer, 2006) and semantic priming (Chertkow, Bub, & Siedenberg, 1989; Giffard, Desgranges, & Eustache, 2005), have also been cited as evidence for disruption of the semantic field.

In the present study, we evaluated the performance on the Boston Naming Test (BNT) of patients with aMCI, patients with mild AD, and normal controls in order to verify (1) the presence of semantic breakdown and (2) the pattern of general and semantic errors in these patients. With this purpose, we examined their spontaneous answers and investigated if they needed or were benefited by semantic and phonemic cues. We assumed that, if patients did not give a correct answer spontaneously or after a semantic cue, but significantly improved after a phonemic cue, this would mean that the semantic field is not necessarily damaged. If a phonemic cue does not improve naming performance, this indicates that semantic knowledge may be compromised. Thus, in order to study the error patterns in spontaneous answers, we classified them in four mutually exclusive categories (semantic errors, visual paragnosia, phonological errors, and omission errors) and the semantic errors were further subcategorized into three subclasses (coordinate, superordinate, and circumlocutory).

Method

We studied 48 subjects older than 50 years (16 with aMCI, 16 with mild AD treated at the Unit for Neuropsychology and Neurolinguistics [UNICAMP Clinic Hospital], and 16 controls). Routine laboratory examinations for dementia assessment (including B12 and folate dosage, serology for syphilis, and thyroid hormone measurement) and brain computed tomography were carried out in all patients. The local ethics committee approved this research. Diagnosis of aMCI in our clinic is carried out by trained neurologists using a standardized mental status battery. The diagnostic process consisted of a detailed interview with the patient and informant (usually a close relative of the patient). All patients underwent the Cambridge Mental Disorders of the Elderly Examination (CAMDEX), which is comprised of structured interviews with the patient and, separately, with an informant, evaluating the patient’s current medical condition, psychiatric status and family history. They also underwent the CAMDEX cognitive test battery (CAMCOG), which includes eight subscales: memory, orientation, language, attention, abstract thinking or similarities, calculation, and perception (Roth, Huppert, Tym, & Mountjoy, 1988). Diagnosis of MCI was made according to the criteria of the International Working Group on Mild Cognitive Impairment (Winblad et al., 2004): (i) the person is neither normal nor demented; (ii) there is evidence of cognitive deterioration shown by either objectively measured decline over time and/or subjective report of decline by self and/or informant in conjunction with objective cognitive deficits; and (iii) activities of daily living are preserved and complex instrumental functions are either intact or minimally impaired. We made a diagnosis of aMCI if the clinical history and cognitive performance pointed to an exclusive memory deficit and Clinical Dementia Rating (CDR; Morris, 1993) score of 0.5, with an obligatory and exclusive memory score of 0.5. This classification was performed by using a semistructured interview. All MCI subjects in this study met criteria for aMCI only.

For probable AD diagnosis, we used the criteria of the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and Alzheimer’s disease and Related Disorders Association (ADRDA) (McKhann et al., 1984), including only patients classified as CDR 1. Exclusion criteria were history of other neurological or psychiatric diseases, head injury with loss of consciousness, use of sedative drugs in the last 24 hours before the neuropsychological assessment, drug or alcohol addiction, and prior chronic exposure to neurotoxic substances. The control group consisted of subjects with CDR 0 without previous history of neurological or psychiatric disease or memory complaints.

Assessment of Naming Ability

The 60-item BNT (Kaplan, Goodglass, & Weintraub, 1983; translated and culturally adapted version for the Brazilian population by Dr. Cândida Camargo, Psychiatry Institute, Medicine School, University of São Paulo) was administered to all subjects where they were asked to name the presented pictures. We determined the total score by adding the number of correct spontaneous responses to the number of correct responses after a semantic cue, which consisted of a short explanation about the picture (e.g., for mask: it’s part of a carnival fantasy) or a superordinate category (e.g., for elephant: it’s a kind of animal). The semantic cue was only given if the patient had failed to recognize the picture (e.g., dog instead of tree) or if he or she said that they didn’t know what the picture was. We gave a phonemic rather than semantic cue if the spontaneous wrong answers were semantically related to the target word (e.g., dog instead of camel), or if the subject was unable to name the picture even after a semantic cue. A phonemic cue consisted of the first phonemes of the target word.

Error Classification

We modified the classification system described by Lukatela et al. (1998) and divided the spontaneous errors into four mutually exclusive types: omission (when the subject was unable to name the picture), visual paragnosia (when the subject answered with an unrelated word which may or may not have shared any common characteristics with the target word), phonologic (when the prominent reason for naming was the similarity with another unrelated word, generally the first phonemes) and semantic (when the answer was semantically related to the target word). At first glance, this classification could lead to some problems, mainly when the subjects’ answers contained more than one error, for example semantic and phonological (tatu instead of tamanduá—Brazilian
animals whose names start with the syllable ta and whose pictures share similarities). In cases like this, we considered the stronger semantic relationship between these animals and the error was classified as semantic.

Semantic errors were further classified into three mutually exclusive categories: circumlocutory (when responses described or indicated the function of the target word), coordinate (when responses were of the same category as the target word), and superordinate (responses that belonged to a broader category than that of the target word). Two independent researchers (MLFB, BPD) performed this classification, and the discordances were solved by consensus.

Additional Neuropsychological Evaluation

All subjects were submitted to tests of verbal fluency (VF) for the animals category (the score was the total number of different animal names given by the subject during one minute); Mini Mental Status Examination (MMSE; Folstein, Folstein, & McHugh, 1975; Brazilian version by Brucki, Nitrini, Caramelli, Bertolucci, & Okamoto, 2003); Rey auditory verbal learning test (RAVLT; Rey, 1964) to evaluate episodic memory delayed recall (RAVLT-A7); CAMCOG’s subscale of similarities between pairs of nouns: the patients were asked “In what way are they alike?” for the following pairs apple/banana, chair/table, shirt/dress and animal/vegetable. The score was calculated as the number of correct responses (zero to two for each pair; maximum score eight) (Roth et al., 1988); visual perception subtests of Luria’s Neuropsychological Investigation (LNI; maximum score 20; Christensen, 1979); the forward (FDS) and backward digit span (BDS) subtest of Wechsler Adult Intelligence Scale—Revised (WAIS–R; Wechsler, 1987); and the Cornell Scale for Depression in Dementia (CSDD; Alexopoulos, Abrams, Young, & Shamotan, 1988).

Data analysis was performed by means of Systat software, and we used analysis of variance (ANOVA) and a post hoc Tukey’s test for intergroup comparisons of demographic and cognitive scores and G Power 3 software to calculate the effect size. In accordance with Cohen (1988), we considered partial eta-squared ($\eta^2$) and $F$ values of 0.10 to represent a small effect, 0.25 a medium effect, and 0.4 a large effect size. With the aim of comparing the pattern of correctness after semantic and phonemic cues, we analyzed the percentage of correct answers for each participant using a separate one-way ANOVA. The same analysis was performed using the error type after spontaneous answers and the subtypes of the semantic errors. Multiple linear regressions for each group were carried out to compare the total BNT score as a dependent variable to other tests as independent variables: lexical-semantic (Similarities and VF), visual perception (LNI subtests), attention (FDS and BDS), episodic memory (RAVLT-A7), and MMSE. We also correlated BNT to age and education. In order to evaluate which cognitive problems might have possibly influenced spontaneous wrong answers for each group, we also compared the independent variables quoted above to semantic, omission, and visual paragnesia errors as separate dependent variables. We used the effect size metric $f^2$ for multiple regressions and, by convention, $f^2 = 0.01$, 0.15, and 0.35 for small, medium, and large effect sizes, respectively. $p$ values less than .05 were considered statistically significant.

Results

As shown in Table 1, there was no significant difference between the three groups with regard to age, $F(2, 45) = 2.194, p = .123$, effect size: partial $\eta^2 = 0.08$, $f = 0.31$, or education, $F(2, 45) = 0.683, p = .51$, partial $\eta^2 = 0.02$, $f = 0.17$. With regard to the BNT total score, patients with AD performed worse than those with aMCI ($p < .001$) and controls ($p < .001$), while subjects with aMCI were similar to controls ($p = .646$) but performed worse than controls on BNT spontaneous answers (without cues; $p < .05$). AD patients also needed more semantic and phonemic cues than patients with aMCI ($p = .004$, and $p < .001$, respectively) and controls ($p < .001$ on both items), while there was no significant difference between aMCI patients and controls on these items ($p = .441$; see Table 2).

Table 1

Demographic and Additional Neuropsychological Data

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>AD (n = 16)</td>
</tr>
<tr>
<td>Age (years)</td>
<td>76.25 ± 7.75</td>
</tr>
<tr>
<td>Education (years)</td>
<td>5.31 ± 4.98</td>
</tr>
<tr>
<td>MMSE</td>
<td>22.56 ± 2.96<strong>b</strong>*</td>
</tr>
<tr>
<td>VF</td>
<td>10.12 ± 3.34<strong>b</strong>*</td>
</tr>
<tr>
<td>RAVLT-A7</td>
<td>1.00 ± 1.21<strong>b</strong>*</td>
</tr>
<tr>
<td>Similarities</td>
<td>4.87 ± 1.74<strong>b</strong>*</td>
</tr>
<tr>
<td>FDS</td>
<td>4.62 ± 1.08</td>
</tr>
<tr>
<td>BDS</td>
<td>3.12 ± 0.50a</td>
</tr>
<tr>
<td>Visuospatial LNI</td>
<td>17.31 ± 1.35<strong>b</strong>*</td>
</tr>
</tbody>
</table>

Note. AD = Alzheimer’s disease; MCI = mild cognitive impairment; MMSE = mini-mental status examination; VF = verbal fluency; RAVLT-A7 = delayed recall of Rey auditory verbal learning test; FDS = forward digit span; BDS = backward digit span; Visuospatial LNI = visual-spatial perception item of Luria’s Neuropsychological Investigation.

a Significantly different from controls.
b Significantly different from amnestic mild cognitive impairment (aMCI).

* $p < .01$. ** $p < .007$. *** $p < .001$. 

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We ran group comparisons of the mean percentages of correct answers between the three groups after semantic and phonemic cues to verify qualitatively whether there were different responses between the groups. As shown in Figure 1, patients with AD answered correctly after semantic cues 21.96% of the time, whereas patients with aMCI answered correctly at a rate of 38.98% and controls answered correctly 45.45% of the time. Analysis of variance for each participant after semantic cues was significant, $F(2, 45) = 7.171, p = .002$, partial $\eta^2 = 0.24, f = 0.56$.

A post hoc Tukey’s test showed that there was a difference between patients with AD and patients with aMCI ($p = .023$), as well as between patients with AD and controls ($p = .002$), but not between patients with aMCI and controls ($p = .674$). With regard to the mean percentage of correct answers by each group after phonemic cues, patients with AD answered correctly 37.02% of the time, whereas patients with aMCI answered correctly 39.86% of the time and controls answered correctly 45.45% of the time. Analysis of variance did not show any significant differences between the percentages for each group participant, $F(2, 45) = 0.926, p = .404$, partial $\eta^2 = 0.03, f = 0.20$. Another separate ANOVA was carried out to compare the percentage of each error type after spontaneous answers, and there was no significant difference between the three groups for omission errors, $F(2, 45) = 0.503, p = .608$, partial $\eta^2 = 0.02, f = 0.14$, visual paragnosis, $F(2, 45) = 2.728, p = .076$, partial $\eta^2 = 0.10, f = 0.34$, and semantic errors, $F(2, 45) = 2.284, p = .114$, partial $\eta^2 = 0.09, f = 0.31$. We excluded phonological errors from the analysis because the three groups made a small number of this type of error. These results are shown in Figure 2.

We also used one-way ANOVA to compare the percentages of the semantic subtype of errors among the three groups, and there were no significant differences observed for circulocutory, $F(2, 45) = 0.620, p = .542$, partial $\eta^2 = 0.02, f = 0.14$; coordinate, $F(2, 45) = 0.260, p = .772$, partial $\eta^2 = 0.01, f = 0.10$; or superordinate errors, $F(2, 45) = 0.032, p = .968$, partial $\eta^2 = 0.001, f = 0.03$. These results are shown in Figure 3.

The only variables that contributed significantly to the BNT variance on multiple regression analysis were Similarities ($t(10) = 2.878, p = .035$) in the aMCI group ($R^2 = 0.58$, $f^2 = 1.38$) and Similarities ($t(10) = 3.429, p = .019$) and MMSE ($t(10) = 3.553, p = .016$) in the control group ($R^2 = 0.933$, $f^2 = 13.92$). There were no significant relationships between any variable and the BNT in the mild AD group. In the AD group, the only variable that contributed significantly to spontaneous errors was the RAVLT delayed recall on omission errors ($t(7) = 2.322, p = .049, R^2 = 0.378$, $f^2 = 0.60$). In the aMCI group, there was a significant relationship between omission errors ($R^2 = 0.496$, $f^2 = 0.98$) and Similarities ($t(7) = -2.949, p = .018$) and between visual paragnosis ($R^2 = 0.421$, $f^2 = 0.72$) and Similarities ($t(7) = -2.983, p = .018$).

![Figure 1](image1.png)  
**Figure 1.** Percentage of correct answers by each group after semantic and phonemic cues.

![Figure 2](image2.png)  
**Figure 2.** Percentage of subtypes of errors from total naming errors among patients with Alzheimer’s disease (AD), patients with amnestic mild cognitive impairment (aMCI), and control subjects.
Discussion

Our results showed that patients with aMCI demonstrated a normal performance on the BNT and needed fewer semantic and phonemic cues than patients with mild AD. After semantic cues, aMCI and control subjects correctly named more pictures than patients with mild AD, but after phonemic cues there was no significant difference between the three groups. This finding suggests that patients with AD may have some degree of preserved knowledge about the pictured object, but they need some help to retrieve the phonological information about the presented item. We have found that cues, like primes, could facilitate picture naming by spreading activation of semantic relations, which indicates that semantic knowledge may not be the main cognitive domain that is disrupted. Semantic errors in object naming can also arise from impairment of any level in the naming process, including input, semantic, and output levels, as shown by Hillis and Caramazza’s (1995) study of aphasics patients. Picture naming deficits in AD may also be, at least in part, due to a decline in inhibitory control over phonological output processes related to phonological implementation of conceptual information (Faust, Balota, & Multhaup, 2004). In addition, some studies point to preserved semantic priming as evidence that patients with AD do not suffer from a degradation or loss of semantic knowledge, but rather from a loss of retrieval or other attentionally mediated processes (Albert et al., 1988; Balota & Duchek, 1991; Ober & Shena, 1988). In fact, the attention performance of our patients with mild AD, as assessed by the backward digit span task, was lower than that of the control group, which could suggest that lexical access and attention may have played a major role in their naming deficits.

To verify the performance of our patients in other lexical-semantic tasks, we applied the VF test for category animals and the CAMCOG’s item of Similarities. Both tests showed that patients with mild AD performed significantly worse than patients with aMCI and controls. There were no significant relations between the BNT and other tests in the AD group. In the aMCI and control groups, Similarities performance was related to the BNT overall score, which suggests that lexical-semantic field integrity is important for this naming test.

Thus, our patients with mild AD demonstrated a poor performance overall on the BNT (spontaneous and semantic cued naming) and other lexical-semantic tasks, but their semantic field tended to be at least partly preserved, since they scored normally after phonemic cues. A possible explanation for this finding is provided by Butterworth, Howard, and McLoughlin (1984) who, in a study of aphasics subjects, proposed that a semantic deficit with incomplete activation of semantic knowledge is likely to produce either a semantic error or a correct response (if the information available is sufficient to retrieve the correct phonological form). In a similar way, Moreau, David, Charnallet, and Pellat, (2001), by evaluating 15 patients with AD, offered a conciliatory theory that a loss of semantic knowledge for some items (as proposed by Hodges et al., 1992) may coexist with a deficit of lexical retrieval for other items (Nebes, 1992; Nicholas et al., 1996). Chenery et al. (1996) found that in early AD, the main problem is attentional, but that later in the progression of the disease, naming deficits reflect increased compromise of core semantic structures and processes. It could be very difficult to demonstrate that poor performance on semantic tasks is caused by storage disorders, since disruption in other cognitive processes (mainly attention) may theoretically explain the observed outcomes as well, as discussed by Storms, Dirix, Saerens, Verstraeten, and De Deyn (2003). Attentional problems alone, however, cannot explain the semantic errors of all AD cases. In our sample, for example, subjects with aMCI, which might be representative of very early AD, scored lower than patients with AD on digit span tests (although the difference was not statistically significant). Furthermore, there is substantial clinical heterogeneity (both cognitive and behavioral) among patients with AD, even in the early phase of the disease (Cummings, 2000). Thus, the primary initial disturbance can be attentional-executive, as well as visuospatial-apraxic or aphasic (semantic anomia), depending on which brain region is predominantly degenerated.

With regard to spontaneous naming errors, there was a continuum between the three groups, with patients with AD committing the most errors, controls committing the fewest errors, and subjects with aMCI showing an intermediate performance. Nevertheless, when we analyzed the percentage of naming errors, the three groups were similar regarding the pattern of errors: each group committed semantic errors most frequently, followed by visual paragnosias and omissions. Phonological spontaneous errors were very uncommon. Analysis of relationships between spontaneous errors and other cognitive tests showed a significant correlation only between RAVLT delayed recall and omission errors in the AD group. A plausible explanation for this finding could be that naming partly depends on active retrieval (lexical-semantic selection) from long-term declarative memory, as in the RAVLT delayed recall task. In the aMCI group, Similarities was negatively related to omission and visual paragnosia errors (that is, committing fewer omission and visual paragnosia errors implied a better performance on Similarities), suggesting that these errors might have been influenced by semantic field integrity.

Analysis of the semantic subtype of errors showed that the three groups had a similar pattern of errors: they differed quantitatively, but not qualitatively. They made the most coordinate errors, followed by superordinate and circumlocutory errors. Why did this pattern of errors exist even among controls? Why did our patients with AD not make more superordinate than coordinate errors when compared with aMCI and controls? A plausible explanation for this pattern of errors even among controls is that naming of basic level entities (e.g., house, chair, hammer, dog), as well as of unique or subordinate entities (e.g., White House, rocking chair, sledgehammer, collie) requires finergrained discrimination and access to more information than the naming of higher level categories (e.g., animal, fruit, tool), as suggested by Martin and Chao (2001). The predominance of coordinate errors
made by our patients with AD (whose mean MMSE score was 22.5 ± 2.9) is in disagreement with the higher frequency of superordinate errors found by Lukatela et al. (1998) in their AD group with similar MMSE scores (23.9 ± 3.2), although we used a slightly different classification. In spite of this discordance, our findings support the theory of Lukatela et al. (1998) and earlier proposals (Chertkow & Bub, 1990; Hodges, Salmon, & Butters, 1991) that, in AD, differentiation of within-category exemplars is impaired, whereas knowledge of broader semantic categories is preserved.

The varied findings and controversies concerning coordinate versus superordinate errors as well as lexical retrieval deficit versus semantic knowledge loss found by several authors are probably related to variations in the dementia stage and, in the early stages, to the heterogeneous distribution of regional degeneration. In different patients with AD, this could affect predominantly stricto sensu language areas for lexical access (naming) and/or higher level cortical association areas related to semantic (conceptual) organization.

An additional plausible explanation for these varied findings in early AD is Milberg et al.’s (1999) Gain/Decay hypothesis, which represents a further development based upon Collins and Loftus’s (1975) model of dynamic spreading activation and Hasselmo’s (1994) theory of AD pathology as characterized by changes in synaptic density and deregulations of connectivity, which occur early in the course of the disease. According to this hypothesis, knowledge is stored in a semantic network made up of a series of representational (conceptual) units which vary in how “active” they are and when activated beyond some threshold, will produce a wave of activation that spreads to other units within the network. The central assumption is that a reduction in the time constant of spreading activation in AD produces dynamic changes that allow semantic representations to be either more available or less available than normal, depending on the time frame in which this information has to be accessed. In AD, there may be a change in the modulation of activation, rather than the loss of activation proposed by models that claim a degradation of semantic knowledge associated with brain atrophy (cf. Farah & Tippett, 1996; Martin & Fedio, 1983).

Knowledge degradation attributable to neural atrophy and loss of representational units cannot be a plausible explanation for the semantic deficits found in early AD, since (1) many other degenerative conditions (Parkinson’s disease, Huntington’s disease, alcoholism) are not associated with such an extensive impairment of semantic memory as seen in this disease (as argued by Milberg et al., 1999), and (2) there is increasing evidence that the earliest pathological change in AD is an intraneuronal accumulation of AB oligomers (not fibrils) leading to mitochondrial abnormalities, a decreased rate of glucose utilization, oxidative damage, and synaptic dysfunction, which can impair cognition long before the appearance of neuritic plaques, neurofibrillary tangles, and brain atrophy (Dodart et al., 1999; Kelly & Ferreira, 2006; Reiman et al., 1996; Selkoe, 2002).

Early synaptic changes plus a reduction in the number of longer axons and dendrites by the disease process (tangles and plaques) would have the effect of reducing the total resistance and capacitance of the dendritic membrane, thus reducing the time constants of both excitatory and inhibitory postsynaptic potentials arriving at affected neural cell bodies while increasing the gain and the decay rate of activation within the neural network (Milberg et al., 1999).

Another relevant aspect of our findings is that our subjects answered correctly after a phonemic cue, even if they had spontaneously made a semantic error. In such cases, for example, making a semantic-coordinate error on spontaneous naming might imply semantic integrity at this and higher levels and maybe a disruption at a more basic level. Semantic disruption would be expected to occur from the more detailed nodes of the semantic network to the more generic levels of semantic hierarchical organization as aging leads to the progression of aMCI and AD. Chery et al. (1996) showed that the naming responses of subjects severely affected by the disease reflect increased compromise of core semantic structures and processes, which is not necessarily true in the early phases. It is possible that if we had included patients with moderate and severe AD, they would not have answered properly even after phonemic cues. Should this be the case, we could have found a different pattern of semantic errors, maybe with a higher prevalence of the superordinate subtype.

In conclusion, we have found that subjects with aMCI performed similarly to controls with regard to the BNT total score (spontaneous plus cued naming), while there was a significantly decreased performance from normal aging to aMCI to AD on BNT spontaneous naming (without cues). The poor performance of patients with AD cannot be completely explained by semantic breakdown, since they performed as well as aMCI and control subjects after phonemic cues, and this relative sparing of semantic knowledge could be attributable to the early disease phase of our patients. We also found that the overall pattern of spontaneous naming errors and the subtypes of semantic errors were similar in the three groups, with decreasing frequency of errors from coordinate to superordinate to circumlocutory subtypes. These naming difficulties are most likely explained by a combination of loss of semantic knowledge, impaired lexical access, and higher taxing of cognitive resources for finer-grained discrimination between basic level lexical-semantic fields. Additional studies with larger sample sizes and a more comprehensive battery of tests to assess the cognitive architecture of the semantic system, including lexical access and appropriate control tasks, are needed for more reliable conclusions.

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