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Naming ability in patients with mild to moderate Alzheimer's disease: what changes occur with the evolution of the disease?

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OBJECTIVES: Naming deficit is a linguistic symptom that appears in the initial phase of Alzheimer's disease, but the types of naming errors and the ways in which this deficit changes over the course of the disease are unclear. We analyzed the performance of patients with Alzheimer's disease on naming tasks during the mild and moderate phases and verified how this linguistic skill deteriorates over the course of the disease.

METHODS: A reduced version of the Boston Naming Test was administered to 30 patients with mild Alzheimer's disease, 30 patients with moderate Alzheimer's disease and 30 healthy controls. Errors were classified as verbal semantic paraphasia, verbal phonemic paraphasia, no response (pure anomia), circumlocution, unrelated verbal paraphasia, visual errors or intrusion errors.

RESULTS: The patients with moderate Alzheimer's disease had significantly fewer correct answers than did both the control group and the group with mild Alzheimer's disease. With regard to the pattern of errors, verbal semantic paraphasia errors were the most frequent errors in all three groups. Additionally, as the disease severity increased, there was an increase in the number of no-response errors (pure anomia). The group with moderate Alzheimer's disease demonstrated a greater incidence of visual errors and unrelated verbal paraphasias compared with the other two groups and presented a more variable pattern of errors.

CONCLUSIONS: Performance on nominative tasks worsened as the disease progressed in terms of both the quantity and the type of errors encountered. This result reflects impairment at different levels of linguistic processing.

KEYWORDS: Alzheimer's disease; Language; Anomia.

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INTRODUCTION

Dementias represent a group of diseases that affect a growing number of people because of the aging of the world's population. Alzheimer's disease (AD) is the most common type of dementia (1), as it is responsible for 50% to 70% of the total number of dementia cases worldwide (2).

AD is a degenerative disease of the nervous system, and its most common characteristic is progressive and constant deterioration of cognitive function. Recently, new diagnostic criteria for AD have been advanced by the National Institute of Aging/Alzheimer's Association (3). The most common presentation of AD is the amnestic form. The first symptoms are progressive loss of recent memory and the ability to learn new facts, followed by impairment of other areas of cognition, and particularly language impairment.

Although memory impairment is the most evident initial symptom, changes in speech and language in amnestic AD have been studied more thoroughly in recent decades and have been investigated more attentively in clinical practice. Studies have indicated that the language deficit is progressive and affects all aspects of language (comprehension and production of oral discourse, reading and writing) during all stages of the disease (4,5).

Regarding language disorders, anomia is the most evident linguistic symptom, beginning in the initial phase of the disease (6). Several studies have been conducted on this topic, but the types of naming errors that occur and the ways in which the deficit changes over the course of the disease are still controversial (7–12). Whereas certain authors relate the deficit to the degradation of semantic memory (7), other studies relate the problem to failures in access to the

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phonological form of a word (8,9) or problems in other subsystems (10,11). However, most authors have not examined errors in different stages of the disease. One exception is Chenery's study (12), which compared subjects at different stages of AD and concluded that the nature of anomia in these patients changes over the course of the disease.

Studying the nature of naming ability is extremely important because anomia is correlated with a more rapid progression of the illness (13) and a greater likelihood of whole-brain atrophy (14). Moreover, recent studies have shown positive therapeutic effects for lexical semantic treatment in early AD (15). Therefore, providing a qualitative and quantitative description of naming deficits is essential.

Hence, the aim of this study was to analyze the performance of patients with AD on naming tasks during the mild and moderate phases of the disease to verify how this linguistic skill deteriorates over the course of the disease. The hypothesis of this study was that the anomia in AD changes both quantitatively and qualitatively and that the qualitative changes demonstrate compromise of different stages of the linguistic processing of naming over the disease course.

MATERIALS AND METHODS

Ethics

This cross-sectional, observational study was conducted at the outpatient clinic of the Behavioral Neurology Division in the Speech Pathology Department of São Paulo Federal University. The study was approved by the local research ethics committee (Protocol Number 0957/06). After the participants received complete information about the study, written informed consent was obtained from all enrolled subjects.

Subjects

The sample was composed of 90 individuals, who were split into an AD group (ADG) and a control group (CG). The general inclusion criteria were as follows: age $\geqslant$60 years; $\geqslant$4 years of education; no history of alcoholism or drug use; no use of psychotropic medications, except for atypical neuroleptics; and an absence of visual or auditory impairment that might affect the outcomes of cognitive tests.

Sixty patients had AD (30 in the mild stage and 30 in the moderate stage) according to the clinical criteria proposed by the National Institute of Neurological and Communicative Disorders and Stroke – Alzheimer's Disease and Related Disorders Association (NINCDS-ADRDA) Work Group (16). The neurological assessment was performed by an AD expert. All of the patients diagnosed using the criteria were submitted to a complete neuropsychiatric evaluation followed by a neuropsychological evaluation. Cognitive screening tests, a neuropsychological battery and a functional assessment were used for patient selection and group classification.

The Mini-Mental State Examination (MMSE) was used as a screening tool (17). We used a Portuguese translation and Portuguese scoring of the MMSE (18). Only the individuals with an MMSE score greater than 12 who were undergoing treatment for AD with a therapeutic dose of acetylcholinesterase inhibitors (donepezil $\geqslant$5 mg, rivastigmine $\geqslant$9 mg or galantamine $\geqslant$8 mg) were selected. The subjects were also assigned a Clinical Dementia Rating (CDR) (19). The CDR scores were 0.5 or 1 for mild dementia and 2 for moderate dementia.

For neuropsychological evaluation, the patients were assessed using the protocol established by the Consortium to Establish a Registry for Alzheimer's Disease (CERAD) (20), which addresses attention, memory, evocation, recognition, language, praxias, gnosias, and abstract thinking using the following tests: verbal fluency, naming, word list memory, constructive praxis, word list evocation, word list recognition, apraxia evocation, and the trail test.

A functional scale questionnaire, or the Disability Assessment for Dementia (DAD) (21), which was adapted for the Brazilian population, was also performed (22).

The CG consisted of 30 healthy elderly volunteers with no neurological or psychiatric changes who were paired with the AD patients according to age and education. To evaluate general cognition, the MMSE was used as a screening tool, with cut-off scores adapted to the subjects' educational levels (18): elementary (1 to 4 years of education)=25; 5 to 8 years of education=26.5; 9 to 11 years of education=28; and highly educated (more than 12 years)=29.

In addition to the MMSE and a brief medical/neurological history, the family members or caregivers of the CG subjects completed the DAD to identify cognitive disorders that would preclude participation in this study. The results were analyzed according to the criteria proposed by Carthery et al. (22), with scores higher than 99.0 allowing inclusion.

The CG consisted of individuals who were accompanying patients, family members, friends, or students to the Senior Citizens' Open University; they were recruited after receiving authorization from the institution.

Procedure

All of the participants completed the revised version of the Boston Naming Test (RBNT). The instrument contains 20 picture cards from the original instrument, including the 15 pictures used by the CERAD Neuropsychological Battery (20), and five cards that represented low-frequency words.

The pictures used in the CERAD set depicted a tree, a bed, a whistle, a flower, a house, a boat, a toothbrush, a volcano, a mask, a camel, a harmonica, an ice gripper, a net, a funnel, and dominoes. The supplementary figures were a racket, a snail, an escalator, a harp, and a pyramid.

The test was administered according to the instructions in the original manual. After the presentation of each card, the individual was asked to name the object. Up to 20 seconds were allowed for the response. If the participant was unable to do so or if the first answer given was "I don't know" or "I don't remember," a semantic cue was given. If the participant still had no answer after 20 seconds, a phonemic cue was given (the initial sound of the word).

The responses obtained on the RBNT were classified according to whether they were correct or incorrect. Errors were categorized into the following groups:

- Verbal semantic paraphasia: substitution of another word semantically related to the target word (sheets x pillow);
- Verbal phonemic paraphasia: substitutions, omissions or additions of phonemes or syllables (funnel x tunnel);
- No response (pure anomia): absence of the name;
- Circumlocution: substitution of a word for a phrase (mask x it is used to go to a party);
- Unrelated verbal paraphasia: substitution of one word for another unrelated to it in form or content (camel x pen);

Table 1 – Descriptive analysis based on the mean measurements of naming abilities and the ANOVA results.

Naming abilities	Mean (SD)		ANOVA	F	*p*-value
	CG (30)	Mild AD (30)	Moderate AD (30)		
Correct answers	17.2 (2.2)	16.4 (2.3)	13.9 (3.9)	10.68	<0.001*
Verbal semantic paraphasia errors	1.7 (1.5)	1.6 (1.3)	2.1 (1.3)	−1.33	0.270
Verbal phonemic paraphasia errors	0 (0.2)	0.1 (0.3)	0.0 (0.0)	1.33	0.364
Unrelated verbal paraphasia errors	0.2 (0.5)	0.4 (0.6)	1.0 (1.1)	8.34	<0.001*
No-response (pure anomia) errors	0.3 (0.7)	0.9 (1.3)	1.9 (2.5)	7.15	0.001*
Circumlocution errors	0.2 (0.6)	0.3 (0.5)	0.3 (0.8)	0.22	0.799
Visual errors	0.3 (0.5)	0.4 (0.6)	0.7 (0.9)	3.19	0.046
Intrusion errors	0.0 (0.0)	0.0 (0.0)	0.1 (0.4)	1.00	0.372

- Visual: substitution of one word for another that represents a figure that is visually similar to the one presented on the card (pyramid x triangle);
- Intrusion: repetition of a word that was previously mentioned but that has no relation to the figure.

The correct answers, the answers given after a semantic cue, and the answers given after a phonemic cue were tallied on the test application form. The errors were also counted and analyzed. Error classification was derived from the most common nomenclature used in the literature (23).

Statistical analysis

The answers provided by the groups of patients with mild or moderate AD were compared with each other and with the results obtained from the CG. The differences between the means of continuous data were tested using parametric tests and corresponding non-parametric tests. When similar results were achieved with the two tests, the parametric results were used; when there was a discrepancy, however, the non-parametric results were used. Non-parametric tests are often robust enough to handle a degree of deviation from normality, with the advantage of providing useful confidence intervals (CIs), given that our preference is to use parametric tests whenever possible.

Student's t test (t) was used for paired samples, and its corresponding non-parametric test, or the Wilcoxon signed rank test (Z), was used for two dependent samples. For three or more independent samples, we used one-factor one-way analysis of variance (ANOVA) (F) and the corresponding non-parametric Friedman's test. When the ANOVA showed that differences were statistically significant, Bonferroni's multiple comparison test was conducted to identify the specific differences. Spearman's correlation coefficient (ρ) was also used to evaluate the relationship between continuous variables.

A probability (P) value less than 0.05 was considered statistically significant, and all of the tests were two tailed. The 95% CIs were calculated for the differences between averages and for odds ratios (ORs). All of the calculations were performed using Statistical Package for the Social Sciences (SPSS) 11.5.1 statistical software for Windows.

RESULTS

General characteristics

The mean age was 74.2 years (SD ± 6.0) in the mild ADG, 74.4 years (SD ± 5.2) in the moderate ADG, and 73.1 years (SD ± 6.5) in the CG. On average, the mild ADG had 7.6 (SD ± 5.1) years of education, the moderate ADG had 6.9 (SD ± 4.7) years, and the CG had 6.6 (SD ± 5.3) years. No significant differences were found between the ADGs and the CG with respect to age or years of schooling; thus, it was possible to compare the performance of the groups.

The mean MMSE score was 25.4 (SD ± 1.6) in the mild ADG, 19.8 (SD ± 2.9) in the moderate ADG, and 28.6 (SD ± 1.4) in the CG. With the groups paired by age and education, the mean MMSE score notably decreased as the disease progressed. The group with mild AD had lower scores than the CG did according to the score expected for each education level, and the moderate ADG had lower scores than the mild ADG did.

Evaluation of naming ability - intergroup analysis

An analysis of the performance of the three groups on the RBNT is shown in Table 1. We observed that the number of errors increased with progression of the disease. The nature of the errors also differed among the three groups studied (Table 1). According to the ANOVA, there were significant differences in the mean numbers of correct answers, unrelated verbal paraphasias, and no-response errors (pure anomia) among the three groups of subjects. To determine where these differences occurred, we performed Bonferroni's multiple comparison test (Table 2). We noted that patients with moderate AD had fewer correct answers and made more unrelated verbal paraphasias than did the subjects with mild AD and the subjects in the CG. With regard to no-response errors (pure anomia), a significant difference was found only between the group of patients with moderate AD and the control subjects.

Types of errors - intragroup analysis

To evaluate which types of errors were most prevalent in each group of subjects, we performed Wilcoxon's signed rank test; the results are presented in Table 3. We noted that in the CG, there were significantly more verbal semantic paraphasias than all other types of errors analyzed. In the mild ADG, there was a significantly greater number of verbal semantic paraphasias than all other types of errors, except for no-response errors (pure anomia), which occurred at a similar rate. Additionally, significantly more no-response errors (pure anomia) were observed compared with intrusion and verbal phonemic paraphasias. However, the pattern of errors in the group of patients with moderate AD was more varied, including a greater frequency of visual errors and unrelated verbal paraphasias than in the other groups.

DISCUSSION

The analysis of the performance of all of the groups on the RBNT (Table 1) showed that the number of errors increased

Table 2 – Results of Bonferroni's multiple comparison test for the mean numbers of correct answers, unrelated verbal paraphasias, and no-response errors (pure anomia) for the different groups of subjects.

Comparison among the groups	Difference in means	95% CI (difference)	*p*-value
Correct responses			
Controls x Mild AD	0.83	−1.00 to 2.67	0.810
Controls x Moderate AD	3.33	1.50 to 5.17	<0.001*
Mild AD x Moderate AD	2.50	0.67 to 4.33	0.004*
Unrelated verbal paraphasias			
Controls x Mild AD	−0.17	−0.67 to 0.34	1.000
Controls x Moderate AD	−0.80	−1.30 to −0.30	0.001*
Mild AD x Moderate AD	−0.63	−1.14 to −0.13	0.009*
No response (pure anomia)			
Controls x Mild AD	−0.63	−1.67 to 0.41	0.422
Controls x Moderate AD	−1.60	−2.64 to −0.56	0.001*
Mild AD x Moderate AD	−0.97	−2.01 to 0.07	0.077

CI = confidence interval

Table 3 – Comparisons between the types of errors according to the groups of subjects.

Comparison	Controls		Mild AD		Moderate AD	
	Z	*p*-value	Z	*p*-value	Z	*p*-value
Verbal phonemic paraphasia - Verbal semantic paraphasia	−4.1	<0.001*	−4.3	<0.001*	−4.6	<0.001*
Unrelated verbal paraphasia - Verbal semantic paraphasia	−3.9	<0.001*	−3.9	<0.001*	−3.3	0.001
No response (pure anomia) - Verbal semantic paraphasia	−3.8	<0.001*	−1.9	0.062	−1.1	0.258
Circumlocution - Verbal semantic paraphasia	−3.7	<0.001*	−3.8	<0.001*	−4.4	<0.001*
Visual error - Verbal semantic paraphasia	−3.8	<0.001*	−3.9	<0.001*	−3.9	<0.001*
Intrusion - Verbal semantic paraphasia	−4.1	<0.001*	−4.3	<0.001*	−4.6	<0.001*
Unrelated verbal paraphasia - Verbal phonemic paraphasia	−1.9	0.059	−2.2	0.029	−3.8	<0.001*
No response (pure anomia) - Verbal phonemic paraphasia	−2.3	0.020	−3.1	0.002*	−3.8	<0.001*
Circumlocution - Verbal phonemic paraphasia	−1.9	0.063	−2.5	0.011	−2.3	0.024
Visual error - Verbal phonemic paraphasia	−2.8	0.005	−2.3	0.020	−3.6	<0.001*
Intrusion - Verbal phonemic paraphasia	−1.0	0.317	−1.4	0.157	−1.0	0.317
No response (pure anomia) - Unrelated verbal paraphasia	−0.5	0.589	−2.2	0.027	−1.7	0.092
Circumlocution - Unrelated verbal paraphasia	−0.3	0.773	−0.2	0.816	−2.5	0.011
Visual error - Unrelated verbal paraphasia	−1.1	0.257	0.0	1.000	−1.0	0.340
Intrusion - Unrelated verbal paraphasia	−2.1	0.034	−2.8	0.005	−3.7	<0.001*
Circumlocution - No response (pure anomia)	−0.3	0.729	−2.2	0.027	−2.8	0.005
Visual error - No response (pure anomia)	0.0	1.000	−2.1	0.033	−2.4	0.017
Intrusion - No response (pure anomia)	−2.3	0.024	−3.3	0.001*	−3.6	<0.001*
Visual error - Circumlocution	−0.8	0.414	−0.3	0.796	−1.6	0.107
Intrusion - Circumlocution	−2.1	0.038	−2.9	0.004	−1.5	0.143

$p < 0.002$ was considered to indicate statistical significance according to Bonferroni's correction; Z=Wilcoxon signed rank test; P=probability

with progression of the disease. The literature agrees that individuals with AD perform worse on visual confrontation naming tasks compared with healthy subjects (4,6,7), even though healthy older people also have word-finding deficits (24). Studies have also indicated that it is possible to identify naming failures early, even in the mild phase of AD, and that this impairment worsens in proportion to the degree of the illness (25,26).

In addition to the increased number of errors, the most important finding of our study is that the patterns of naming errors observed in AD patients changed as the disease evolved. It was possible to measure the quantitative and qualitative changes using a revised version of the Boston Naming Test. Although verbal semantic paraphasias and no-response errors (pure anomia) were observed in all groups, the number of no-response errors (pure anomia) increased as the disease progressed, and there were significantly more unrelated verbal paraphasias among the moderate AD patients. We also observed that patients in the moderate stage showed a more variable pattern of errors, including visual errors (Tables 2 and 3).

Several studies have shown naming difficulties in AD patients, but research on the changing nature of the errors over the course of the disease is less common. In this context, Chenery et al. (12) found similar results. The authors specifically used the Boston Naming Test (27) to assess subjects with AD, who were divided into three groups according to dementia severity: mild (n=8), moderate (n=7) and moderately severe (n=8). The results showed that in the milder stages, anomia is related to the inability of a patient to access the phonological label for a particular word. Over the course of the disease, the responses of severely affected subjects reflect the increased compromise of core semantic structures and processes. However, the authors discussed the need for studies with larger samples and further empirical validation.

Verbal semantic paraphasias and no-response errors (pure anomia) may be more strongly related to failures in the semantic system. Certain authors have hypothesized that AD patients perform worse than healthy individuals do in visual confrontation naming tasks because of a semantic

breakdown caused by store degradation (7). Performance impairment results as neurodegeneration spreads to the association cortices that presumably store semantic representations (28). However, semantic errors do not systematically reflect only a deficit of semantic knowledge in AD patients; instead, they reveal a deficit in retrieval of the phonological form and a deficit in accessing semantic knowledge in the visual modality (8). Verbal semantic paraphasias in early phases of AD could reflect a breakdown in access to phonological representations of object names as a consequence of reduced inhibitory control over other highly active alternatives (9), whereas the increase in no-response errors (pure anomia) in moderate AD is possibly related to a loss of semantic information.

In our study, we observed that errors of a visual nature also gradually increased with the course of the disease; in particular, these errors were more prevalent in the group with moderate AD. Because the naming task was carried out using visual confrontation, it is important to remember that naming alterations could exist because of a failure in the visual object recognition system or a failure to access the semantic system because of the visual processing requirement. However, our results showed that even receiving phonemic and semantic cues did not interfere with the results. Thus, in this case, naming problems stemmed from deterioration of the detailed conceptual knowledge of an item and should not be attributed only to a specific deficit of semantic processing. Nevertheless, as the illness progresses, perception problems, including visual agnosia, also contribute to patients' naming difficulties (10,11) in visual confrontation naming tasks because visual perception otherwise helps patients to identify an item.

In addition to a greater number of visual errors in the group with moderate AD compared with the subjects with mild AD and the CG, the results indicated an increase in unrelated verbal paraphasias and a more varied pattern of errors in general. These results seem to reflect the impairment of several linguistic subsystems, with alterations at different levels of linguistic processing, in addition to the increase in cognitive modifications in general (such as visual perception), resulting from the involvement of various cerebral areas and consequently leading to greater linguistic-cognitive compromise.

The fact that the temporal lobe becomes more involved as AD advances may explain the deficit in word retrieval (29). However, it is necessary to consider more recent studies that have suggested the involvement of more anterior and ventral frontal portions, and especially the inferior frontal gyrus (30). Left lateral temporal atrophy and other distinct neuroanatomical signs of AD are consistent with the hypothesis that a large-scale neural network supports naming (31). We should expect, however, that more linguistic and cognitive systems will be affected as the disease progresses and as cortical areas are damaged. Therefore, semantic processing involves extensive areas of brain function, and no-response errors (pure anomia) may be attributable to lesions on multiple areas of the brain that are involved in diverse processes. Longitudinal studies have shown that the most severe naming deficits increase from mild to moderate dementia. The possible causes are lexical-semantic and visual-perceptive dysfunctions (13), and the appearance of visuospatial and constructional impairment is most likely due to increasing pathological involvement of the posterior association cortex. The ability to visually discriminate nameable objects in AD also strongly predicts patients' performance in both picture naming and semantic association (32).

In sum, in analyzing the errors found in this study, we noted a quantitative difference in the errors made by the three groups. Similarly, in addition to the increase in the number of errors as the disease progressed, we noted that the types of errors (which also differed qualitatively) might differentiate healthy subjects from patients with various stages of AD. This result seems to reflect impairment at different levels of linguistic processing and suggests that the underlying basis of the anomia changes as the disease progresses. Identifying the types of naming errors in AD is essential for the appropriate implementation of therapeutic strategies for these patients. In addition, qualitative analysis of naming errors in AD is an important tool to help with differentially diagnosing AD and other forms of dementia.

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AUTHOR CONTRIBUTIONS

Silagi ML collected, analyzed and interpreted the data and drafted the submitted material. Bertolucci PH performed critical revision of the paper. Ortiz KZ supervised the collection, analysis and interpretation of the data and performed critical revision of the paper.

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