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THE EFFECTS OF PHONEMIC CUEING ON CONFRONTATION NAMING IN FRONTOTEMPORAL DEMENTIA AND ALZHEIMER'S DISEASE: EVIDENCE FROM THE POLISH VERSION OF THE BOSTON NAMING TEST

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SUMMARY

Background Confrontation naming of objects, as measured by the Boston Naming Test (BNT), is impaired in both fronto-temporal dementia (FTD) and Alzheimer's disease (AD). However, the profile of naming disturbances seems to be different in these two neurodegenerative conditions, which could provide clues for differential diagnosis. This study aimed to characterize the naming performance in FTD and AD as well as to test if the ability to benefit from phonemic cueing may be differentially impaired in FTD and AD.

Material/ Methods: We examined 28 patients who met the clinical criteria for FTD (16 men, 12 women) and 30 subjects with probable AD (22 women, 8 men). Naming was assessed twice in both groups, with the mean time between examinations ranging from 6 to 8 months. At each testing session, the patient's ability to name pictures from the Boston Naming Test (authorized Polish version) was assessed, with phonemic cueing whenever the patient failed to name the picture correctly

Results: The overall quantitative differences between the FTD and the AD groups on the BNT were not statistically significant. Nevertheless, in comparison to subjects with FTD, whose naming scores improved with phonemic cueing, the performance of patients with AD was characterized by perceptual/associative errors and did not benefit from phonemic cueing.

Conclusions: The preserved ability of patients with FTD to benefit from phonemic cueing, together with the qualitative analysis of performance on the BNT, might be useful in the differential diagnosis of FTD and AD.

Key words: neurodegenerative diseases, differential diagnosis, Pick complex, anomia, perceptual/associative errors

INTRODUCTION[1]

Alzheimer's disease (AD) is the most common form of dementia, remaining a major health care problem worldwide (Mendez & Cummings, 2003). Nonetheless, despite its high prevalence, it remains difficult to determine how many patients with a clinical diagnosis of AD actually have the pathological changes characteristic for this condition, that is, amyloid plaques and neurofibrillary tangles. In fact, a subset of these cases has been shown to have an underlying pathology of one of the diseases included by Kertesz in what he calls the "Pick complex" (Kertesz & Munoz 1998). For example, some clinical cases of AD have been subsequently diagnosed as fronto-temporal dementia (FTD) at autopsy. On the other hand, many patients with FTD, due to severe behavioral and personality changes, are misdiagnosed as being mentally ill. A great deal depends on the patient's age at onset and the clinic to which they initially report or are referred, especially in countries where physicians are less aware of the FTD syndromes. Importantly, however, the accurate diagnosis of dementia subtypes has not only psychological consequences for the patients and their caregivers, but also a major influence on the choice of pharmacological treatment (e.g. see Procter et al., 1999). Hence, the early differential diagnosis of AD and FTD remains one of the major aims of contemporary neuroscience.

Among the most characteristic features of FTD, as compared to AD, are early behavioral and/or language disturbances, depending on the localization of brain atrophy; only later do deficits of memory and other cognitive dysfunctions appear. In a typical patient with AD, the sequence of events is rather the reverse: first episodic memory deficits, then behavior and language abnormalities. Importantly, the language variant of FTD, often referred to as primary progressive aphasia (Mesulam, 2003), typically includes primary non-fluent aphasia (PNFA) (Grossman et al., 1998; Pąchalska 2008) and semantic dementia (SD) (Hodges et al., 1992; Snowden et al., 1989; Pąchalska 2008), two clinically different entities. When PNFA develops, patients present with impaired verbal output, whereas their comprehension is relatively well preserved. In the early phase, the predominant symptoms are then anomia and word-finding difficulties, often accompanied by mild agrammatism and apraxia of speech (Gorno-Tempini et al., 2004; Josephs et al., 2006; Pąchalska 2008). The patient's utterances are effortful and distorted by omissions (often verbs), improper use of prepositions, and paraphasias (Ash et al., 2009, 2010; Gunawardena at al., 2010; Hillis et al., 2004; Rohrer et al., 2010). Similar errors appear in reading and writing (Graham et al., 2004; Rohrer et al., 2010). In most cases the speech and language disturbances are isolated symptoms for about two years (Mesulam, 2003), although some patients may also develop early behavioral changes or extrapyramidal signs (Kertesz, 2008; Pąchalska 2008). In SD, on the other hand, disturbances of lexical-semantics predominate, whereas phonology and syntax, as well as day-to-

[1] The original research results presented in this article constitute an emended version of a portion of one of the authors' own monograph (Olszewski 2008).

day episodic memory, is usually unimpaired (Hodges & Patterson, 2007; Pąchalska, 2008). Of note, when the right temporal lobe is predominantly affected, progressive prosopagnosia or even voice recognition deficit are the first signs of the multimodal loss of semantic knowledge characteristic for SD (Gainotti et al., 2008; Hailstone et al., 2010; Josephs et al., 2008; see also Hodges, 2001). Importantly, thanks to the spared phonology, the speech of patients with SD is fluent, though with an impoverished vocabulary and little informative content. Word finding is a major problem, but, in contrast to PNFA, grammatical structures are relatively well preserved. The prevalence of behavioral disturbances in SD is high (Kertesz et al., 2010; Rosen et al., 2006; Snowden et al., 2001; Pąchalska 2011), contributing significantly to the social handicap.

Importantly, progressive aphasia is also common in AD and, similarly to FTD, may be among the first signs preceding the full blown of the dementia syndrome (Appell et al., 1982; Bayles & Tomoeda,1983; Blair et al., 2007; Cummings et al., 1985; Harciarek & Jodzio, 2005, Pąchalska 2008). Although the language impairment has long been considered a component of AD (e.g. even the original Alzheimer's patient was aphasic; see Alzheimer, 1907), aphasia as a primary deficit in AD has been emphasized only recently (e.g. Blair et al., 2007; Gorno-Tempini et al., 2004, 2008; Rabinovici et al., 2008). Initially, patients with AD generally present only with logopenia and anomia, making the early differential diagnosis relatively difficult (Gorno-Tempini et al., 2008; Rohrer et al., 2010). In contrast to PNFA, however, the speech of patients with AD is typically fluent until middle or late stages of the disease. As AD progresses, comprehension deficits, paraphasic errors, and semantic jargon appear, making the patient somewhat similar to individuals with transcortical sensory aphasia or Wernicke's aphasia. Global aphasia and mutism are typically not seen until the advanced stages of AD (Appell, et al., 1982; Cummings et al., 1985; Murdoch et al., 1987).

Nonetheless, although aphasia is frequently seen in both FTD and AD, the nature of language abnormalities, including naming deficits, may be somewhat different in these two neurodegenerative conditions. For example, there is evidence to suggest that, since subjects with AD frequently present with semantic impairment and visual agnosia (see Mendez & Cummings, 2003), perceptual/associative errors might be particularly common on a naming task in this group of patients. On the other hand, presumably most individuals with FTD should benefit from phonemic cues (Pąchalska 2008; 2011), since anomia in this group of patients is more likely to stem from the breakdown of the language/executive networks, rather than perceptual/associative problems. The purpose of our study, then, was to better characterize performance on naming testing in AD and FTD. Specifically, this research attempted to identify if FTD patients as a whole, in contrast to individuals with AD, would benefit from phonemic cueing when performing a confrontation naming task.

MATERIAL AND METHODS

Participants

Twenty-eight individuals (16 men and 12 women) who met the criteria for a clinical diagnosis of FTD (Neary et al.,1998), as well as 30 patients (22 women and 8 men) with a clinical diagnosis of AD (McKhann et al., 1984), were enrolled in this study. All patients were examined twice: at baseline and again 6-8 months later. Importantly, the difference in timing between the baseline and the follow-up evaluation was not statistically significant between groups. The patients had been referred to the present authors for testing, primarily from the Department of Developmental Psychiatry, Psychoses, and Advanced Age at the Medical University of Gdansk, as well as several other medical facilities in Gdańsk, Poland. All the AD patients were from 64 to 86 years of age ($\bar{x}$ = 75.8; SD = 5.5) and were significantly older than the FTD patients, who ranged in age from 43 to 71 years ($\bar{x}$ = 57.1; SD = 7.6). The mean duration of illness was shorter, however, in the FTD group ($\bar{x}$ = 26.5 months; SD = 6.8) than in the AD group ($\bar{x}$ = 20.1 months; SD = 7.6). Nonetheless, the groups did not differ from each other in overall dementia severity, as measured by the Mini-Mental Status Examination (Folstein et al., 1975). The detailed clinical, language, and demographic characteristics have previously been published elsewhere (Olszewski 2008).

Measures and procedures

Naming was assessed with the Boston Naming Test (BNT) (Kaplan et al. 1983; Polish adaptation: Pachalska & MacQueen, 1998), which consists of 60 color pictures representing items from seven categories: objects of daily use, tools and machines, body parts, items of clothing, animals, fruits, plants. The test is designed not only to measure the degree of anomia, but also to analyze the specific nature of the naming impairment. A phonemic cue is provided whenever the patient fails to name the item correctly. A record is also made of the patient's erroneous answers and non-verbal behavior, which often provide important clues to the specificity of the naming disturbances.

In the first trial, without phonemic cueing, the examiner shows the examinee a series of drawings, pointing to each one in turn and asking, "What is this?" Both correct and incorrect answers are then recorded on the scoring sheet. Synonyms are acceptable (e.g. "pistol" for "revolver"), but not if the name given by the examinee is the name of a broader category of objects (e.g. "weapon" for "revolver" or "fruit" for "apple"). In the second trial, if the correct answer was not given on the first attempt, the examiner prompts with the first phoneme or syllable of the target word, and the reaction/answer is then noted on the scoring sheet. Polish norms were applied to evaluate naming disturbances (Pachalska 2008).

Statistical analyses

The longitudinal naming ability and the effects of phonemic cueing on naming in both groups were tested using 2×2×2 ANOVA with repeated measures, with

the within-subject factor being the naming condition (no cueing vs. phonemic cueing) and the testing time point (baseline vs. follow-up), and the between-subject factor being group (FTD and AD). The dependent variable was the total number of the subjects' correct responses for each picture. Post-hoc comparisons of between-group effects were in all cases conducted using Tukey's test, whereas pairwise comparisons were applied to test for within-group differences. In addition, the mean group differences in percentage improvement following phonemic cueing between baseline and follow-up were assessed using the t-student test for independent samples.

RESULTS

Statistical analysis revealed that, at baseline, both patients with AD and FTD showed an impaired ability to name objects before phonemic cueing, when compared to normative data. Also, these groups did not differ from each other, regardless whether it was the cued or non-cued condition. Importantly, however, as shown in Figure 1, no statistically significant improvement after cueing was seen in the AD group ($p = 0.87$), whereas in the FTD group scores on the BNT significantly increased following phonemic cueing ($p = 0.03$).

At follow-up, the results before cueing were, again, similarly impaired in both the FTD and the AD group (see Figure 2). After cueing, however, the performance of patients with AD remained unchanged ($p = 0.91$), whereas there was a signif-

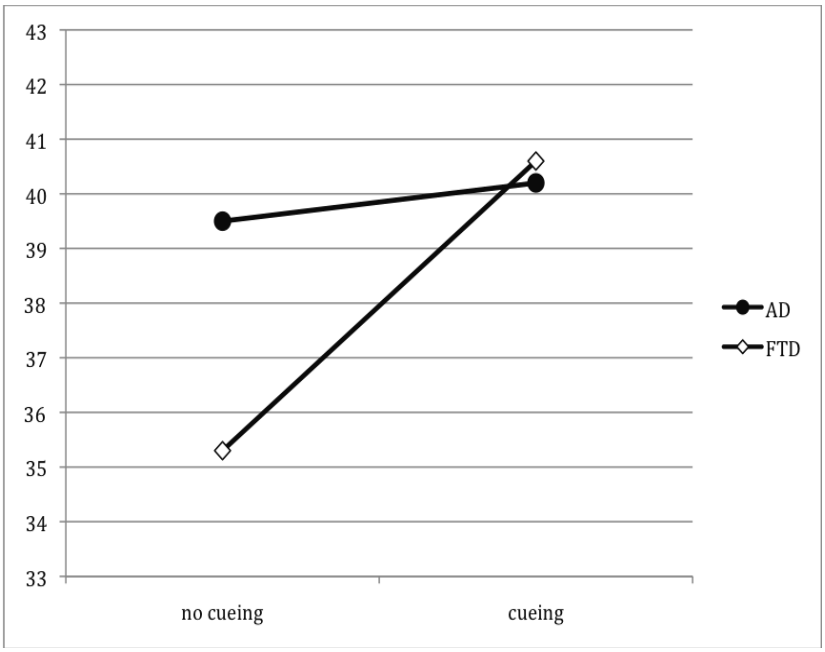

Figure 1. Baseline difference in naming scores obtained before and after phonemic cuing by patients with FTD and AD

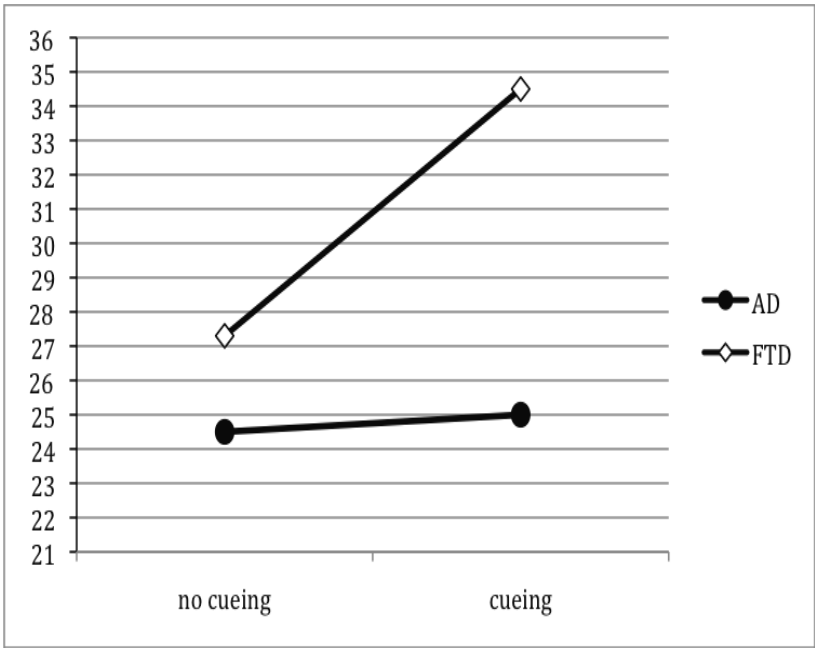

Figure 2. Follow-up difference in naming scores obtained before and after phonemic cuing by patients with FTD and AD

icant improvement in scores in the FTD group ($p < 0.001$). Also, in contrast to baseline testing, during follow-up the differences between the cued mean scores of the FTD and AD groups reached the level of statistical significance, with FTD subjects outperforming the AD group ($p = 0.02$).

Nonetheless, the analysis also revealed that, as the diseases progressed, the performance of both dementia groups declined significantly over time ($p < 0.001$), regardless of the testing condition (cued vs. not cued).

At baseline, the average improvement after phonemic cueing in the AD group was no more than 3%, as compared to a significant improvement of nearly 31% in the FTD group ($p = 0.03$). By the second examination, the difference became even more prominent: 2.5% in the AD group vs. 49.3% in the FTD group ($p = 0.002$).

Interestingly, qualitative differences were also noted in the course of the testing in respect to the nature of the naming errors committed by patients from each of the two groups. The AD patients more often mistook the displayed item for something else (sometimes something from the same semantic category), or failed to recognize it at all (perceptual/associative errors), especially at follow-up. By comparison, patients with FTD could typically not recall what the item was called (anomic errors), regardless of the time of the assessment.

DISCUSSION

By investigating the effects of phonemic cuing and by analyzing the type of errors on the authorized Polish version of the BNT, this study aimed to better

characterize the nature of naming problems in patients with AD and FTD. As expected, when compared to norms, the performance on the BNT in both the FTD and the AD groups was markedly impaired. Moreover, the analysis showed that phonemic cueing improves naming only in patients with FTD, supporting the hypothesis that the word-finding difficulties in FTD may be primarily the result of the breakdown of the language/executive networks (for review see Mendez & Cummings, 2003). Nevertheless, unless phonemic cueing was provided on the second trial, the two groups of patients did not differ from each other when the overall quantitative score on the BNT was analyzed. This result seems to be in contrast to the established notion that language impairments, including anomia, are typically more prominent in the course of FTD than AD. It is worth mentioning, however, that in the present study the FTD group may have predominantly consisted of patients with the behavioral variant of FTD (bvFTD), although the available data did not allow for testing this assumption.

Based on the longitudinal assessment, the performance on the BNT of both the FTD and the AD group was not only markedly impaired at the two testing points, but also declined significantly over time. This observation, therefore, confirms previous research indicating the high prevalence of increasing naming problems in both FTD and AD. In a relatively recent study, Blair and coworkers (2007) showed that, indeed, the majority of early FTD and AD cases could be classified as patients with anomic aphasia. Significantly, the same results also indicated a steeper decline in the FTD group when compared to less aphasic patients with AD. This could have been due, at least partly, to a shorter duration of illness in the AD group, especially since at the same time the language impairment in both FTD and AD was found to be associated with the severity of dementia.

Anomia is one of the characteristic features of both FTD and AD (Appell et al., 1982; Blair et al., 2007; Gustafson 1987; see also Harciarek & Kertesz, 2009). Importantly, however, as also shown in the present study, different cognitive processes may lead to poor confrontation naming performance in these two neurodegenerative diseases. In AD perceptual/associative errors are frequently observed, while in FTD naming errors seem most likely to reflect the breakdown of the language/executive networks. Since the qualitative analysis of the patients' errors can provide data on the nature of anomia (e.g. word-finding difficulty vs. optic aphasia/visual agnosia), naming tests such as the BNT might be useful in the assessment of naming when differentiating between AD and FTD.

To better understand the mechanism that underlies the differences in naming between subjects with FTD and individuals with AD, it may be helpful at this point to refer to Luria's neuropsychological studies on naming (Luria 1973). In his concept, the following four factors underlie the process of naming:

1) Exact visual perception of an object. Any inaccuracies, or the absence of the visual optical base, make it difficult or even impossible to give the proper name of the object. Such a state has been associated with damage to temporal-occipital structures.

2) Preservation of the acoustical structure of the name of this object. Disturbances in this aspect are associated with damage to the left temporal cortex.
3) Finding the essential designation of the target object, while simultaneously inhibiting all other collateral acoustical alternatives. The naming of an object is intertwined with a network of diverse connections, such as the verbal designation of various features of the object, designations pertaining to nearby semantic categories, designations that are proximate in respect to their sound structure or morphology. In normal verbal behavior this condition is met without the problems that appear in the case of any brain pathology in the specified structures. This is obviously a far more complex factor than the two preceding ones. For example, if a patient cannot recall the name of the given object, this is described as amnestic aphasia, which is associated with damage to parietal-occipital structures.
4) Normal dynamics of cortical processes, which involves shifting from one word to another. This becomes especially difficult with damage to the left inferior premotor cortex, or the left fronto-temporal cortex.

In order to explicate the research results reported in the present study, it may also be also interesting to discuss two basic mechanisms of naming disturbances. Luria (1973) described disturbances of linguistic memory as a phenomenon specific to the given modality. When there are pathomorphological changes in the temporal region of the dominant hemisphere, the memory deficits are closely associated with auditory processes and speech. In other words, a patient with temporal damage does not remember the name of a given object and searches for it. Luria also claimed (1973) that when there are analogous lesions in the parietal or parietal-occipital region, the patient will have difficulties with simultaneous synthesis of processed information, and so the disturbances of mnestic processes will be a consequence of these gnostic disturbances. The patient with damage to the parietal or parietal-occipital regions does not recognize the object and accordingly does not have access to the name of that object. Disturbances in the mechanism of recalling the names of objects (which Luria called amnestic aphasia), with this localization, are the result of the pathologically changed neurodynamics of these brain structures, leading to the probable appearance of diverse utterances containing systems of word traces (similar in respect to phonetics, morphology, and semantics). Verbal paraphasias then appear in the patient's utterances.

All these phenomena might have had an impact on the results obtained in this study, including the ineffectiveness of phonemic cueing in persons with AD. For example, frequent perceptual/associative errors in the AD group (however, not explicitly reported in the Results section, cf. Olszewski, 2008) indicate that the first step in the word-finding process as described by Luria, namely exact visual perception of the object, might be primarily impaired in AD (see also Farah, 2004). Also, the positive effect of phonemic cueing in FTD suggests that in this group the acoustical word structure and the selection of the correct word from the lexicon may typically be well preserved. Thus, when the item is presented to the patient, it is correctly recognized, but the name cannot be spontaneously retrieved.

Nevertheless, when the phonemic cue is provided, the preserved phonemic structure of the word enables access to the appropriate name. It should be borne in mind that if the acoustical structure had been affected, phonemic cueing would have led to phonological errors. Alternatively, if the selection process had been deficient, the patient, when provided with a phonemic cue, would have given another phonologically correct word that did not designate the object shown.

This study has several limitations that need to be considered. First, the available data did not allow for classifying patients with FTD into its three major syndromes, that is bvFTD, NFPA, and SD. Thus, since in SD patients present with a progressive and multimodal loss of semantic knowledge, it seems indisputable that the severely impaired performance on naming tasks seen in these patients will not improve following phonemic cueing. Therefore, the bias in this study could have likely resulted from the overrepresentation of patients with bvFTD in the FTD group. Second, since in this study the groups did not differ on the MMSE, it is likely patients with FTD were at more advanced stages of the disease than the subjects with AD. Along the same line, the participants in this study were not matched on aphasia type and the severity of language impairment. Thus the conclusions about the effects of phonemic cueing and the error type on confrontation naming seem to be limited. Also, since in this study semantic cueing was not included in the testing procedure, the precise nature of anomia in FTD and AD is still debatable. Lastly, it would be interesting to see if there are any differences in the effects of cueing on the specific semantic category of the given picture (e.g. living vs. non-living, fruit vs. animals). Thus, future studies are warranted to investigate in more depth the mechanisms that underlie naming abnormalities in these two neurodegenerative diseases.

CONCLUSIONS

Our research points to the need for qualitative analysis of naming errors measured by the Boston Naming Test in differentiating AD from FTD. Perceptual/associative errors in confrontation naming tasks seem to be typical for AD. In FTD phonological cueing is more effective than in AD.

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