

Review

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## Clinical psychoacoustics in Alzheimer's disease central auditory processing disorders and speech deterioration

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### Abstract

**Background:** Difficulty in speech understanding in the presence of background noise or competing auditory signals is typically present in central auditory processing disorders. These disorders may be diagnosed in Alzheimer's disease as a result of degeneration in the central auditory system. In addition perception and processing of speech may be affected.

**Material and Methods:** A MEDLINE research was conducted in order to answer the question whether there is a central auditory processing disorder involved in Alzheimer's disease. A second question to be investigated was what, if any is the connection, between central auditory processing disorders and speech deterioration?

Articles were retrieved from the Medline to find relevance of Alzheimer's disease with central auditory processing disorders, they summed up to 34. Twelve papers were studied that contained testing for CAPD through psychoacoustic investigation. An additional search using the keywords 'speech production' and 'AD' produced a result of 33 articles, of them 14 are thoroughly discussed in this review as they have references concerning CAPD. The rest do not contain any relevant information on the central auditory system.

**Results:** Psychoacoustic tests reveal significantly lower scores in patients with Alzheimer's disease compared with normal subjects. Tests concerning sound localization and perception of tones as well as phoneme discrimination and tonal memory reveal deficits in Alzheimer's disease. Central auditory processing disorders may exist several years before the onset of clinical diagnosis of Alzheimer's disease. Segmental characteristics of speech are normal. Deficits exist concerning the supra-segmental components of speech.

**Conclusions:** Central auditory processing disorders have been found in many cases when patients with Alzheimer's disease are tested. They may present as an early manifestation of Alzheimer's disease, preceding the disease by a minimum of 5 and a maximum of 10 years. During these years changes in the central auditory system, starting in the temporal lobe, may produce deficits in speech processing and production as hearing and speech are highly connected human functions. Another theory may be that spread of degeneration of the central nervous system has as a consequence, speech deterioration. Further research and central auditory processing disorders testing in the elderly population are needed to validate one theory over the other.

## Background

Degeneration of the central auditory system in Alzheimer's disease (AD) is well documented [1]. The structural changes involve the ventral nucleus of the medial geniculate body (MGB) and the central nucleus of the inferior colliculus [2]. The MGB is one of the most important relay stations for central auditory function, its projections are arranged tonotopically as a function of frequency. Central auditory processing disorders (CAPD) have the predominant symptom of difficulty in speech understanding in the presence of background noise or competing auditory signals.

The aim of the current study was to investigate the correlation of AD with auditory deficits in general and more specific CAPD. The question was whether presbycusis, high frequency hearing loss related to the aging process, is responsible for the abnormal results in word recognition and psychoacoustic tests or if there is a CAPD involved. Speech in patients diagnosed with the above disease has been shown to have impairments involving perception and production and affecting semantic, morphological and phonological elements.

## Material and Methods

Articles were retrieved from the MEDLINE to find relevance of Alzheimer's disease with central auditory processing disorders, they summed up to 34. Twelve papers were studied that contained testing for CAPD through psychoacoustic investigation. An additional search using the keywords 'speech production' and 'AD' produced a result of 33 articles, of them 14 are thoroughly discussed in this review as they contain information on the central auditory system. The rest of them do not include any information on the central auditory pathway.

## Results

### CAPD

Clinical psychoacoustic investigations on patients with AD involve the dichotic digits test, the dichotic sentence test and the synthetic sentence identification with ipsilateral competing messages (SSI-ICM) [3]. These psychoacoustic tests reveal significantly lower scores in the AD group when compared with normal subjects [4,5]. Testing of the peripheral auditory function is a prerequisite in order to rule out presbycusis or peripheral hearing loss. This is important in order to correctly interpret the results of the psychoacoustic investigations as being the evidence of CAPD. Strouse, Hall et al [6] tested 10 subjects diagnosed with mild to moderate AD based on a neuropsychological test battery and neuroimaging techniques and compared them with 10 normal subjects matched for age, gender and average threshold of hearing. The AD group scored significantly lower than the control group on four of the five psychoacoustic tests. Higher perform-

ance in the right versus the left ear could be recognized in the AD group. The investigators recommended screening for CAPD in the AD population, since this could influence psychiatric assessment of the cognitive deficit and audiologic management in this population. Gates, Karzon et al [7] tested 42 patients with AD using psychoacoustic tests, of them 20 were in the mild category and compared them with 40 normal subjects. They concluded that there was evidence of central auditory dysfunction in subjects with even mild cases of AD, whereas peripheral auditory function was not different from that of the control group.

In a study conducted by Kurylo, Corkin et al [8] tests of sound localization and perception of tones as well as phoneme discrimination and tonal memory were administered to 19 mild to moderate AD patients, 21 elderly control subjects and 14 young control subjects. The AD group differed from the elderly control subjects on sound localization and synthetic speech discrimination. The investigators concluded that specific auditory deficits exist in AD patients but increased impairment of functions mediated by primary auditory centers and auditory association cortices could not be revealed. This could probably be attributed to an auditory deficit affecting the structures of the brain stem.

In an experiment of Mohr, Cox et al [9] AD patients showed significantly worse performance in dichotic testing compared to controls. The scores were lower as the dichotic list length was increasing and as a function of stimulus set content, using semantically, phonetically and unmatched dichotic items. The investigators concluded that AD patients were unable to attend selectively to only one ear and as a consequence could not increase right or left ear advantages, so AD may be associated with an impairment of selective allocation of attention. This is especially important in interpreting the results of the psychoacoustic tests. Is it an attention deficit or a central auditory one? Auditory processing has a preattentive (automatic) and attentive (controlled) component [10]. In a dichotic test situation when a person is asked to attend to auditory information coming from one ear, he or she fails to recall the semantic content of information arriving in the other ear but the acoustic changes are being detected. In a work of Wood, Hiscock and Widrig [11] three different experiments with 60 normal right-handed adults revealed that selective attention to one ear increased the accuracy of localization but not detection at the attended ear. The lag effect, the preferential process of auditory information on either stimulus in a dichotic test when the presentation is delayed by 60–90 ms, remained invariant as attention was manipulated. The investigators concluded that attention instructions do not influence the relative access of left and right ear stimuli but involve alteration at a later stage of information processing.

The Framingham Heart Study is a population-based cohort consisting originally of 5,209 men and women aged 30–62 with the first examination starting at 1948. This study has collaborated with two non-vascular projects, the hearing and the dementia cohort [12]. Dementia cohort with hearing testing consists of 789 subjects, limited down to 740 when excluding those with stroke or dementia at the time of hearing testing. 40 of them developed a diagnosis of AD and 15 of them had CAPD. Seven (46.7%) of the 15 subjects with CAPD developed AD during the follow up period compared with 33 of the 725 (4.6%) of the subjects with no evidence of CAPD. Peripheral auditory function is not responsible for the very low scores in SSI-ICM observed in the CAPD and AD subjects. Time from testing to diagnosis was shorter for the CAPD subjects (6.5+1.4 years) as compared with the non-CAPD subjects (8.8+2.5 years). Adults with the poorest SSI-ICM scores had the shortest times from hearing testing to diagnosis. Five of the seven subjects who were later diagnosed with AD had near normal score of the SSI-ICM in one ear and very poor levels in the opposite ear. The investigators concluded that although the SSI-ICM test involves executive control, the predominately unilateral findings and the long term interval between auditory testing and AD diagnosis argue against the executive dysfunction hypothesis as the mechanism of the cohort findings.

#### **speech perception and production**

Language deficits in perception and production are reserved at an average level till the late stage of AD. The typical time from diagnosis to late stage is 10 years. The impairment of speech comprehension is subtle and requires careful testing. Its cause has been attributed to reduced working memory capacity [13], loss of core linguistic knowledge [14] and to loss of comprehension abilities and in particular 'the ability to activate and manipulate phonological and semantic information from auditory input' [15]. This last theoretical position points out that auditory comprehension is one of the abilities impaired in AD patients. More specifically, MacDonald et al [15] concluded that AD patients exhibit a deficit in being able to convert acoustic input into a rich syntactic and semantic representation.

Imamura, Takatsuki et al [16] examined 150 patients with mild to moderate AD. Word comprehension and sequential commands testing revealed greater deficits in early onset patients than in late onset ones. The existence of greater comprehension impairments in the early onset AD patients has been confirmed by other studies [17-19].

Patients with Alzheimer's disease have difficulties in processing the emotional qualities of words as well as in recognizing prosody [20,21]. There appears to exist for the

patients a great difficulty in expressing emotions by speech, as well as in properly understanding emotional responses in their environment [22]. Speech output is also affected, as patients with Alzheimer's disease have difficulties in properly vesting their speech with emotions.

Emotions are processed in the right hemisphere. Although it is the motor areas of the brain in the left hemisphere that control speech programming, the right hemisphere seems to play a major role in fine-tuning the entire procedure [23]. The production of speech remains intact in neurological patients with right hemisphere damage; however, their prosody is seriously affected [24]. The opposite is true for neurological patients with left hemisphere damage; even though production of speech is deteriorated, these patients maintain the ability to properly convey their emotions by using prosody alone [25].

In a study conducted at the Aristotle University of Thessaloniki, patients with mild to moderate Alzheimer's disease were subjected to various neurolinguistic tests in order to have their speech investigated [26]. The tests assessed the patients' abilities of naming and word retrieval, their overall production of grammatical and syntactic features, as well as the non-verbal features of speech.

From those findings of the study that concern the phonological and non-verbal elements of speech, the patients show a statistically significant deficiency in controlling certain phonological features as well as the phonological programming of speech, as compared to the control group. At an early stage of the disease there is no statistically significant difference concerning the phonological and non-verbal features of speech that are devoid of semantic importance and are being controlled by the right hemisphere. On the contrary, there appears to exist a statistically significant relation concerning the phonological features of speech that have semantic importance and are being controlled by the left hemisphere. The brain area responsible for controlling the semantic features of speech is Wernicke's area, which is situated on the temporal lobe, the first lobe to be affected by Alzheimer's disease. The findings show that although Alzheimer's disease gradually affects both hemispheres of the brain, the left hemisphere is affected first.

#### **Conclusions**

Evidence exists that CAPD may be found in patients with Alzheimer's disease. Studies conducted reveal the presence of CAPD at an early or moderate stage of AD. The Framingham Heart Study included a Dementia cohort with hearing testing of 789 subjects. The study revealed that CAPD may present as an early manifestation of AD, preceding the disease by a minimum of 5 and a maximum of 10 years. During these years changes in the central audi-

tory system, starting from the temporal lobe, may result in deficits in speech production as hearing and speech are highly connected human functions. The alternative view is that further degeneration of the central nervous system has as a consequence, speech deterioration. Further research and CAPD testing in the elderly population may contribute in validating one theory over the other.

## References

- Ohn TG, Braak H: **Auditory brainstem nuclei in Alzheimer's disease.** *Neurosci Lett* 1989, **2**:60-3.
- Sinha UK, Hollen KM, Rodriguez R., Miller CA: **Auditory system degeneration in Alzheimer's disease.** *Neurology* 1993, **43**:779-85.
- Iliadou V, Iakovidis S: **Contribution of psychoacoustics and neuroaudiology in revealing correlation of mental disorders with central auditory processing disorders.** *Annals of General Hospital Psychiatry* 2003, **2**:5.
- Strouse AL, Hall JW 3rd, Burger MC: **Central auditory processing in Alzheimer's disease.** *Ear Hear* 1995, **16**:230-8.
- Grady CL, Grimes AM, Patronas N, Sunderland T, Foster NL, Rapoport SI: **Divided attention, as measured by dichotic speech performance, in dementia of the Alzheimer type.** *Arch Neurol* 1989, **46**:317-20.
- Gates GA, Karzon RK, Garcia P, Peterlein J, Storandt M, Morris JC, Miller JP: **Auditory dysfunction in aging and senile dementia of the Alzheimer's type.** *Arch Neurol* 1995, **52**:626-34.
- Kurylo DD, Corkin S, Allard T, Zatorre RJ, Growdon JH: **Auditory function in Alzheimer's disease.** *Neurology* 1993, **43**:1893-9.
- Mohr E, Cox C, Williams J, Chase TN, Fedio P: **Impairment of central auditory function in Alzheimer's disease.** *J Clin Exp Neuropsychol* 1990, **12**:235-46.
- Shiffrin RM, Schneider W: **Controlled and automatic human information processing II. Perceptual learning, automatic attending, and a general theory.** *Psychology Review* 1977, **84**:127-190.
- Wood S, Hiscock M, Widrig M: **Selective attention fails to alter the dichotic listening lag effect: evidence that the lag effect is preattentive.** *Brain and Language* 2000, **71**:373-390.
- Gates GA, Beiser A, Rees TS, D'Agostino R, Wolf PA: **Central auditory dysfunction may precede the onset of clinical dementia in people with probable Alzheimer's disease.** *J Am Geriatr Soc* 2002, **50**:482-488.
- Hopper T, Bayles KA, Kim E: **Retained Neuropsychological Abilities of individuals with Alzheimer's disease.** *Seminars in Speech and Language* 2001, **22**:261-273.
- Rochon E, Waters G, Caplan D: **Sentence comprehension in patients with Alzheimer's disease.** *Brain and Language* 1994, **46**:329-349.
- Grober E, Bang S: **Sentence comprehension in Alzheimer's disease.** *Developmental Neuropsychology* 1995, **11**:95-107.
- MacDonald MC, Almor A, Henderson VW, Kempler D, Andersen ES: **Assessing working memory and language comprehension in Alzheimer's disease.** *Brain Lang* 2001, **78**:17-42.
- Imamura T, Takatsuki Y, Fujimori M, Hirono N, Ikejiri Y, Shimomura T, Hashimoto M, Yamashita H, Mori E: **Age at onset and language disturbances in Alzheimer's disease.** *Neuropsychologia* 1998, **36**:945-949.
- Binetti G, Magni E, Padovani A, Cappa SF, Bianchetti A, Trabucchi M: **Neuropsychological heterogeneity in mild Alzheimer's disease.** *Dementia* 1993, **4**:321-326.
- Brandt J, Mellits ED, Rovner B, Gordon B, Selnes OA, Folstein MF: **Relation of age at onset and duration of illness to cognitive functioning in Alzheimer's disease.** *Neuropsychiatry, Neuropsychology and Behavioural Neurology* 1989, **2**:93-101.
- Sevush S, Leve N, Brickman A: **Age at disease onset and pattern of cognitive impairment in probable Alzheimer's disease.** *Journal of Neuropsychiatry and Clinical Neurosciences* 1993, **5**:66-72.
- Weintraub S, Mesulam M, Kramer L: **Disturbances in prosody.** *Archives of Neurology* 1981, **38**:742-744.
- Testa JA, Beatty WW, Gleason AC, Orbelo DM, Ross ED: **Impaired affective prosody in AD.** *Neurology* 2001, **57**:1474-1481.
- Schlanger BB, Schlanger P, Gerstmann LJ: **The perception of emotionally toned sentences by right hemisphere damaged and aphasic subjects.** *Brain and Language* 1976, **3**:396-403.
- De Bleser R, Poeck K: **Analysis of prosody in the spontaneous speech of patients with CV-recurring utterances.** *Cortex* 1985, **21**:405-416.
- Tucker DM, Watson RT, Heilman KM: **Affective discrimination and evocation in patients with right parietal disease.** *Neurology* 1977, **17**:947-950.
- Ross ED: **The aprosodias: functional-anatomic organization of the affective components of language in the right hemisphere.** *Annals of Neurology* 1981, **38**:561-589.
- Kaprinis SG: **A Psycholinguistic Investigation of Patients with Alzheimer's Disease.** *PhD Thesis, 1st Psychiatric Department, Aristotle University of Thessaloniki, Thessaloniki* 2003.

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