The effectiveness of neurofeedback on cognitive functioning in patients with Alzheimer’s disease: Preliminary results

L’efficacité du neurofeedback sur le fonctionnement cognitif chez les patients atteints de la maladie d’Alzheimer : résultats préliminaires

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Alzheimer’s disease; Cognition; Memory; Neurofeedback; Performance

Summary
Objectives. — Alzheimer’s disease (AD) is the most common form of dementia. In quantified EEG (qEEG), the AD patients have a greater amount of theta activity compared with normal elderly individuals. Little is known about the effect of neurofeedback in patients with dementia. The objective of this study was to examine whether neurofeedback has a positive effect on cognitive performance in patients with AD.

Methods. — Ten patients with qEEG meeting criteria for AD received neurofeedback training. Participants were aged between 61 and 90 years. All patients underwent the CAMCOG test designed to assess cognitive functioning pre- and post-treatment.

Results. — The individual results, analyzed with a reliable change index (RCI), showed that patients who received neurofeedback treatment had stable cognitive functions. These patients showed improvement in memory after neurofeedback and other cognitive functions were stable. In addition, an improvement was observed in recall of information and recognition.

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Introduction

Dementia is a syndrome characterized by progressive deterioration of cognitive function, most commonly of memory, but other domains such as language, praxis, visual perception and most notably executive function are also often affected. As cognitive function worsens, there is increasing interference with the patients’ daily activities leading to loss of independence and eventually for some the need for nursing home care [23,24]. Dementia has an increasing incidence as people age. Dementia is a symptom of several clinical syndromes, of which Alzheimer’s disease (AD) is the most common form. Seventy percent of all patients with dementia have AD. The diagnosis of ‘probable’ or ‘possible’ AD is made based on clinical criteria established by the National Institute of Neurologic and Communicative Disorders and Stroke—Alzheimer’s disease and Related Disorders Association (NINCDS-ADRDA) [29]. Patients should have dysfunction of at least two or more areas of cognition (orientation to place and time, memory, language, praxis, attention, visual perception and problem solving skills), with progressive worsening of memory and other cognitive functions, no disturbance of consciousness and onset between ages 40 and 90, most often after the age of 65. Scales and inventories designed to screen for dementia contain orientation items, as these test functions that are sensitive to the most common dementing processes, such as both recent and remote memory, mental clarity, and some aspects of attention. Other areas of common interest are fund of knowledge and language skills [27]. AD is associated with functional and structural alterations in a distributed network of brain regions supporting memory and other cognitive domains [2]. Current therapies to treat AD are minimally effective and do not alter the disease process [26]. They may ease symptoms by providing temporary improvement and reducing the rate of cognitive decline [9]. Although the available non-pharmacological therapies for dementia can help with the management of symptoms, there is a need to develop more effective interventions [19], of which neurofeedback is a promising one.

Neurofeedback refers to a form of operant conditioning in which desirable brain activity is rewarded and undesirable brain activity is inhibited [10]. Neurofeedback training works directly with the brain. Each participant trains at his or her own pace. Neurofeedback can facilitate changes in brain wave patterns. These brain wave patterns, or electrical activity, are registered with an electroencephalograph (EEG). The EEG bands are named according to frequency and voltage. Slower frequencies tend to have higher amplitudes than faster frequencies. Neurofeedback training is aimed at changing the amplitude of a selected frequency. Neurofeedback training has been successfully applied in the treatment of different disorders in adults and children. It has shown reliable positive effect in the treatment of
Attention Deficit Hyperactivity Disorder (ADHD) [3,14,18,32] and epilepsy [12,32,42]. Secondly, it has shown positive preliminary results in the treatment of autism [8,25,32], depression [4,16,32] and anxiety [15,16,32,34].

In the normal aging process, the EEG changes in the pattern of brain electrical activity concern a decrease in frequency and amplitude (increased delta and/or theta) [5—7,37]. Patients with AD present a greater amount of theta activity compared to normal aging individuals. An excess of delta and a decrement of alpha and beta are also observed [5,38,40]. As previously stated, the aim of neurofeedback is to change the amplitude of a selected frequency. Therefore, it is expected that neurofeedback might have a positive effect on the treatment of AD, especially on the cognitive performance of patients with AD.

The aim of the present pilot study is to answer the question whether neurofeedback has a positive effect on the decline of cognitive functioning in patients with AD. In patients with AD, it is hypothesized that the amount of delta and theta activity need to be decreased and the amount of alpha and beta activity need to be increased. Secondly, we hypothesize that the cognitive performance of patients with AD will stay stable, or preferably improve, after neurofeedback treatment.

Materials and methods

Participants

Participants for the neurofeedback pilot study were recruited through the outpatient memory clinic from the Catharina hospital in Eindhoven. In the present study, all participants are treated with cholinesterase inhibitors.

This study was carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki). Patients diagnosed with ‘probable’ AD were contacted if they met the inclusion criteria. The inclusion criteria comprised a positive advice for participation by the multidisciplinary team of the memory clinic. This team consisted of a geriatrician, neurologist, psychiatrist, psychologist and a nurse. Furthermore, patients had to have a score of 60 points or higher on a screening instrument for dementia, the Cambridge Cognitive examination (CAMCOG) [41]. This cut-off score was used as an indication that the patient was in an early stage of the disease. Patients were older than 60 years of age and living independently (or possibly assisted living). Additionally, they had to be able to visit the hospital twice a week for a period of fifteen weeks. With respect to the neuropsychological screening, a sufficient understanding of the Dutch language was required. Finally, their qEEG had to meet the typical pattern for people with AD. Patients with a medical history of neurological (epilepsy, stroke, tumor) or psychiatric disorders were excluded.

Procedure

Neuropsychological screening

There were two measurement periods: pre- and post-treatment. In the two measurement periods, the CAMCOG screening was administered in order to determine the cognitive function of the participants. Testing took place at the Catharina hospital by students of the Master in Medical Psychology course. When the participants were screened for dementia at the memory clinic, screening with the CAMCOG had already taken place. In the case the test having been performed longer than three months before inclusion, the CAMCOG was administered again.

qEEG

Pre-treatment, an EEG was performed at the ‘Neurofeedback Instituut Nederland’ (NIN). The EEG was recorded in two conditions: eyes open (EO, for ten minutes) and eyes closed (EC, for ten minutes). The EEG data was then transformed to a qEEG report. The qEEG data of the participant was compared to a normative database, Neuroguide [43]. Based on this qEEG, an individualized training protocol was determined (Table 1). Post-treatment, another EEG recording took place. The qEEG was recorded with the DeyMed True Scan, using True Scan software. The EEG data was processed to a qEEG with Neuroguide software. This software processes data with both ‘linked-ear’ and ‘Laplacian’ montages. The EEG signal was processed with a Fast Fourier Transformation (FFT) to the following frequencies: delta (1—4 Hz), theta (4—8 Hz), lower alpha (8—10 Hz), upper alpha (10—12 Hz), SMR (12—15 Hz), beta (15—18 Hz), high beta (18—25 Hz), gamma (30—35 Hz) and high gamma (35—40 Hz). For all these frequencies, z-scores (between 0 and 1) of the absolute and relative power were estimated for all 19-scalp locations. The normative database was normalized for age of the participants.

Neurofeedback

Within two weeks after the pre-treatment measurement, neurofeedback treatment started. The sessions took place twice a week for fifteen weeks, amounting to a total of thirty sessions. Data was acquired by placing electrodes on the scalp of the participant according to the International 10—20 system [22]. Recording with a single channel EEG required the placement of three separate leads on the head [10]. The active electrode was placed on the individual training location, depending on the training protocol (see Table 1). The reference electrode was placed on the earlobe contralateral to the location of the active electrode and the ground electrode was placed on the other earlobe. The scalp and earlobe locations were cleaned with an abrasive conductive gel (NuPrep Gel™) before the electrodes were placed. Each electrode cup was filled with special gel that conducts electricity (Ten20 conductive paste™). An impedance meter was used to determine if there was good contact between the electrode and the skin. Impedances were kept below 5 KΩ, which is a standard for assessment [20].

The neurofeedback treatment started with a single channel EEG record of one minute with ‘eyes open’. Subsequently, a twenty-minute training session was conducted with three breaks after five minutes. During the training sessions, the participant sat in front of a computer screen and watched a movie. He or she received visual and auditory feedback. If the training went according to the protocol, the movie was shown in a higher contrast and the participant heard a beep. The thresholds were adjusted manually to maintain a reward frequency around 70 percent and an inhibited threshold around 10 percent. After ending the four
training sessions, a final EEG registration of one minute with 'eyes open' was administered.

Outcome measurements

The primary outcome measurement of this study is the results of the CAMCOG. The Cambridge Cognitive Examination (CAMCOG), a neuropsychological screening, is the objective test portion of an instrument developed for the early diagnoses and monitoring of dementia in the elderly, the Cambridge Mental Disorders of the Elderly Examination-Revised (CAMDEX) [27]. It was mainly developed to contribute to the early diagnosis of dementia in people older than 65 years [41]. This study used the Dutch translation [11].

The CAMCOG's 67 items are grouped into eight subscales:

- orientation (ten items dealing with time and place);
- language (seven comprehension items, six naming items, category fluency, and four word definitions);
- memory (recall and recognition of six pictured objects, name and address recall and ten 'information type' items);
- attention (counting from 20 to 1 and serial sevens [five subtractions of seven]);
- praxis (copying geometric figures and following commands);
- calculations;
- abstract thinking (similarities between pairs of items);
- perception (e.g. recognition of objects depicted from unusual angles and stereognosis).

Impairments on the scales Total CAMCOG, Orientation and Memory are indicative for AD [13,27]. Therefore, this study measured cognitive impairment using these three scales and their subscales. Seven items do not contribute to the total score but are included to permit calculation of an MMSE total score (five items) or to acquire additional qualitative information (two items). The scores range from 0 (severe cognitive impairment) to 105 (no cognitive impairment). In the present study, when a CAMCOG score is mentioned, this score is not the 'real' CAMCOG score, but a proportion of that score: the obtained score on a subscale divided by the maximum score of the same subscale.

The CAMCOG divides the educational level into three classes: low, average and high. People with a low educational level are able to score lower on the CAMCOG than people with an average or high level of education before their scores are interpreted as impaired. People with a low level of education usually have a lower IQ, hence it is more difficult for them to answer the questions and perform the tasks than more educated people. In the present study, the level of education was not included.

Statistical analyses

Analyses were performed using SPSS 19.0 for Windows. $P$ values of $<0.05$ were considered significant.

Individual performance on the CAMCOG was analysed with a reliable change index (RCI). The RCI for the CAMCOG and the various subscales were computed according to the formula of Jacobson and Truax [21]:

$$RCI = \frac{X_2 - X_1}{S_{diff}}$$

$$S_{diff} = \sqrt{2 \times (SE)^2}$$

$$SE = s_1 \times \sqrt{1 - r_{xy}}$$

The outcomes of the previous mentioned analysis were used to calculate the RCI's of the subscales.

The group performance on the CAMCOG and the subscales of the CAMCOG were analysed with a Mixed Linear Model analysis, using the procedure *lme* in the R statistical Software [36] with Cognitive Function as dependent variable, Measurement (pre-treatment, post-treatment) as fixed factor and participant ID as random factor. Age is included as a covariate. The simple effects were analysed with a paired-samples t-tests in order to investigate changes in scores (decrease, increase or stabilization) for each group separately.

Results

A total of ten patients (three females and seven males) with AD participated in this neurofeedback study. They were aged $71.5 \pm 6.74$ years, had a mean CAMCOG score of $0.80 \pm 0.10$
and a mean of 153.40 ± 52.59 days between pre- and post-treatment measurements.

**Test-retest reliability**

When looking at the manual of the CAMDEX-R [11] the test-retest reliability ($r_{xy}$) after one year for the total score was 0.97 ($n = 387$) and varied between the subscales from 0.49 to 0.87. In the TAU group, the $r_{xy}$ for the total CAMCOG score was 0.84 and the scores from the various subscales varied from 0.56 to 0.78 (Table 2).

**Individual performance (RCI)**

RCI’s for each neurofeedback participant were computed. The RCI for the Total CAMCOG score of the first participant (see Table 3 for the values) is:

$$RCI = \frac{(X_2 - X_1)}{S_{diff}}$$

$$S_{diff} = \sqrt{n*SE^2}$$

$$SE = S_1 \times \sqrt{1 - r_{xy}}$$

The RCI’s for the other subscales and participants were calculated similarly. Positive values indicate an increase in score, negative values a decrease. A zero score means no change. Scores above 1.96 or below -1.96 indicate a reliable change ($P < .05$). In other words; it is unlikely that the change is due to a measurement error.

Table 4 shows an overview of the various pre- and post-treatment measurements for each participant and subscale.

Table 5 displays the various RCI’s for each participant. One participant had a significant increase in the subscales Total CAMCOG Score and Learning Memory. Another had a significant increase in the subscales Orientation in Place and Past Memory, and a decrement in the subscale Recent Memory. A third participant also had a decrement in the subscale Recent Memory. Overall the neurofeedback participants did not show an increase in scores. However, they also did not show a decrement. Simply put, the participants who had neurofeedback treatment had stable scores on the various subscales.

**Group performance (LME)**

For the following subscales, scores on the pre-treatment measurement were significantly higher than scores on the post-treatment measurement: Total CAMCOG ($F(1,131) = 24.648; \ P = .000$), Total Orientation ($F(1,131) = 10.187; \ P = .002$), Orientation in Place ($F(1,131) = 17.926; \ P = .000$), Total Memory ($F(1,131) = 19.243; \ P = .000$), Past Memory ($F(1,131) = 4.083; \ P = .045$), Recent Memory ($F(1,131) = 28.035; \ P = .000$) and Learning Memory ($F(1,131) = 7.258; \ P = .008$). No difference between the measurements was found for the subscale Orientation in Time ($P = .363$). Table 6 displays an overview of the mean scores of the pre- and post-treatment measurements for each participant.

To analyze the effect of the total days between the pre- and post-treatment measurement, the factor Total days was included as continuous factor instead of the fixed factor Measurement. The Group ($F(1,131) = 7.786; \ P = .006$), Measurement ($F(1,131) = 27.933; \ P = .000$) and Interaction effects ($F(1,131) = 4.983; \ P = .027$) of the total CAMOG score remained. Age (when included as covariate) did not have an effect or interaction effect on the total CAMCOG score.

In summary, Total days and Age did not have an impact on the effects that were found.

Paired-sample t-tests were conducted in order to investigate changes in scores (decrease, increase or stabilization) for each group separately. A higher mean score on Memory Learning after the treatment ($t(9) = —2.613; \ P = .028$) was observed. There was no improvement on the other subscales, but also no decline (resp, $P = .207, .468, .343, .726, .052, .269$ and .223).

**Discussion**

The preliminary results of this study suggest that neurofeedback treatment may have beneficial effects on the cognitive functioning of AD patients. The aim was to explore whether neurofeedback is a potential intervention in decreasing the cognitive decline in patients with AD. Based on the previously discussed literature [5–7,37,38,40], it is hypothesized that the cognitive performance of patients with AD will stay stable, or preferably improve, after neurofeedback treatment. An improvement in learning memory was observed, as well as an increase in recognition and recall of information. Other cognitive functions were stable.

To the authors’ knowledge, only two studies applied neurofeedback to the elderly with the aim of improving cognitive activity. Becerra et al. [5] assessed the effectiveness...
<table>
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<tr>
<th>ID</th>
<th>Total CAMCOG Score</th>
<th>Total Orientation Score</th>
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<th>Total Memory Score</th>
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of neurofeedback in healthy elderly people with abnormally high theta activity. Positive changes were observed in cognition including attention, executive functions and memory. However, the improvement of memory was also observed in the control group. The control group in the previous mentioned study was treated with a sham neurofeedback treatment. Becerra et al. [5] stated that the improvement in memory processes observed in both groups might be due to a placebo effect. Angelakis et al. [1] reinforced alpha power, which correlated positively with cognitive performance. Their results suggest that neurofeedback improves memory. 

Becerra et al. [5] found an improvement in memory for the treatment as well as the control group. Angelakis et al. [1] found an improvement in memory processes. The present study found stable cognitive functions and an increase in learning memory after neurofeedback treatment, which is not in agreement with the previously discussed studies. This could be due to the fact that both Becerra et al. [5] and Angelakis et al. [1] included normally elderly with only subjective complaints of memory loss but no objective evidence of memory dysfunction. This study, as well as other studies [33,39], suggests that a certain level of neuronal plasticity persists, even in AD.

A strong point of this study is that each included patient used cholinesterase inhibitors that might suggest a positive (combination) effect of neurofeedback and cholinesterase inhibitors on the cognitive performance of patients with AD, especially on the recognition and recall of information. In order to explore the effect of neurofeedback alone, patients would have to stop taking their medication. As long as neurofeedback is not acknowledged as a treatment for AD, the termination of the medication cannot be ethically justified. Another strength of this study is that the training protocol is individualized. This is important, since research has shown that there is considerable heterogeneity in the EEG patterns that are associated with diagnostic categories and symptoms, like AD. The use of one standard protocol may increase the risk on an ineffective or adverse treatment [17].

It should be noted that there is a methodological issue that could, besides the operand training, explain the stabilized cognitive performance. Patients were obliged to visit the hospital twice a week, which could be a form of social stimulation to undertake more activities. Patients with AD frequently show an increase in apathy, which is a recurring symptom of AD [28]. It is possible that increased activity results in improved cognitive functions since apathy and cognitive performance are related [30]. Further research could address this issue by implementing a device that monitors the physical activity of a patient (e.g. pedometer). That way, insight could be obtained into the physical activity of participants in both groups.

A limitation is the pilot design of this study, including only 10 patients. Another issue is the level of education, which has not been taken into account. People with higher IQ, education or occupational attainment have lower risks of developing dementia, AD or VD, and are considered to have greater cognitive reserves. The cognitive reserve hypothesis postulates that among those who have greater initial cognitive reserves (in contrast to those with fewer reserves) greater brain pathology occurs before the clinical symptoms of disease become manifest [31,35]. Thus, if there are differences in educational level, the group with the highest educational level will show a faster decline in cognition and function after diagnosis.

In this pilot study, we included patients with a definite diagnosis of AD and a ‘probable’ diagnosis of AD. In terms of confirmed AD, cognitive functions may be very altered and no longer modifiable, thus the stability of cognitive function

Table 5 RCI scores for 10 neurofeedback participants.

<table>
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<tr>
<th>ID</th>
<th>Total CAMCOG Score</th>
<th>Total Orientation Score</th>
<th>Orientation in time</th>
<th>Orientation in place</th>
<th>Total Memory Score</th>
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x = RCI ≤ or ≥ than 1.96. 0 = no change between pre- and post-treatment measurements. Positive values: increase in score between pre- and post-treatment measurements; negative values: decrease in score between pre- and post-treatment measurements.

Table 6 Pre- and post-treatment measurements (mean ± SD).

<table>
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<th>Measure</th>
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<th>Measurement</th>
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with neurofeedback could be explained by a floor effect. Patients with "probable AD", perhaps at the beginning of the pathology, could show greater possibility for change in cognitive functions.

Conclusion

The preliminary results of this pilot study might indicate that neurofeedback, in combination with treatment with cholinesterase inhibitors, may be a potential treatment by which the progressive deterioration in patients with AD can be stabilized. Future research can investigate if neurofeedback leads to changes in the behavior and qEEG of patients with AD.

Disclosure of interest

The authors declare that they have no competing interest.

References


