

EEG Discrimination with Artificial Neural Networks

Sérgio Daniel Rodrigues¹, João Paulo Teixeira¹ and Pedro Miguel Rodrigues²

¹*Polytechnic Institute of Bragança, Bragança, Portugal*

²*University of Porto, Porto, Portugal*

Keywords: Electroencephalogram, Alzheimer's Disease, Artificial Neural Network.

Abstract: Neurodegenerative disorders associated with aging as Alzheimer's disease (*AD*) have been increasing significantly in the last decades. *AD* affects the cerebral cortex and causes specific changes in brain electrical activity. Therefore, the analysis of signals from the electroencephalogram (*EEG*) may reveal structural and functional deficiencies typically associated with *AD*. This study aimed to develop an Artificial Neural Network (*ANN*) to classify *EEG* signals between cognitively normal control subjects and patients with probable *AD*. The results showed that the *EEG* can be a very useful tool to obtain an accurate diagnosis of *AD*. The best results were performed using the Power Spectral Density (*PSD*) determined by Short Time Fourier Transform (*STFT*) with a *ANN* developed using *Levenberg – Marquardt* training algorithm, *Logarithmic Sigmoid* activation function and 9 nodes in the hidden layer (correlation coefficient training: 0.99964, test: 0.95758 and validation: 0.9653 and with a total of: 0.99245).

1 INTRODUCTION

Alzheimer's disease (*AD*) is a progressive degenerative neurological disorder of brain that leads to irreversible loss of neurons (Blennow et al., 2006). Lesions usually start in the hippocampus (which is an important structure in memory formation) and in the cerebral cortex (Blennow et al., 2006; Feldman and Woodward, 2005). In *AD* patients brain can be observed senile plaques formed by amyloid plaques and neurofibrillary tangles. Amyloid plaques are found outside the neurons, neurofibrillary plaques are found inside the neurons and result in the death of the cells. Gradually, the neurons degenerate and a generalized collapse of brain tissue occurs (Feldman and Woodward, 2005). The aging process increases the incidence of *AD* which is typically diagnosed in aging people (Moreira and Oliveira, 2005).

Researchers estimate that 1 in every 85 people may have *AD* in the next 35 years and this disease may affect about 100 million people by 2050 (Stahl, 2008). It is therefore necessary to reach a correct diagnosis of *AD* before significant loss of memory appears, because early diagnosis enables the treatment conditions (Blennow, 2005). A tool suitable for assisting *AD* diagnosis is the *EEG* (Hort et al., 2010).

One of the most frequently observed abnormalities in *EEG* activity of *AD* patients is an increase in power

at low frequency bands and a decreased power in the higher frequency bands (Baker et al., 2008; Rodrigues and Teixeira, 2011). The deceleration of *EEG* signal usually occurs in intermediate and severe stages of *AD* (Jeong, 2004).

In the present study we intend to develop an Artificial Neural Network (*ANN*) to discriminate *EEG* signals between cognitively normal control subjects (*Cs*) and *AD* patients.

2 MATERIALS AND METHODS

2.1 Selection of Patients and Controls/EEG Recording

We had the collaboration of thirty four subjects (14 Controls and 20 of *AD*). *EEGs* were recorded from the international system 10-20 of 19 electrodes. The frequency sample was 200 Hz. Then, *EEGs* were organized in 5 s artifact-free epochs (1000 samples) (Rodrigues and Teixeira, 2011). It should be noted that all recordings were digitally filtered with a band-pass filter between 1 Hz and 40 Hz.

2.2 EEG Signal Processing

Fourier transform (FT) allows us to represent the signal in the frequency domain. It is the spectral EEG signal processing currently used (Rioul and Vetterli, 1992). FT constitutes a disadvantage of not being able to provide information both in time and frequency of signal characteristics. Indeed, in signal transformation from the time domain to the frequency domain there is a lost of information about the temporal location (Blennow,2005). Thus, by observing the FT of a signal is impossible to say where a particular event is located, since what is obtained is only the frequencies that compose the signal throughout the whole time interval considered. If a signal is stationary this drawback is irrelevant. However, if a signal contains many non-stationary or transient characteristics (characteristics which are usually the most important parts of a EEG signal) FT can not detect such processes (Blennow,2005). Seeking a solution to this problem it is common to use the Short-Time-Fourier-Transform ($STFT$). $STFT$ maps a signal using a bidimensional function defined in time and frequency and represents a form of compromise between representation both in time and frequency of this signal. Although the $STFT$ provides information on time and frequency, there is a disadvantage when choosing a particular size for the window that will go through the signal, because this window remains the same for all frequencies. It turns out that many signals have a window of variable length which allows a more precise location of a particular event both in time and frequency, because in most cases it is impossible to determine an optimal window size that can find events with enough resolution at very different frequencies(Blennow, 2005). So, $STFT$ also presents limitations. To overcome the problem, we used the $STFT$ because it uses a constant time window and therefore we can analyze the EEG signal in short time stationary periods. Thus, we calculated the power spectral density (PSD), functions that had the force of power variations as a frequency function. The PSD ($S_{x,w}[k]$) was calculated by the autocorrelation function ($R_{xx}(u)$) of $STFT$ of the signal, as can be seen in the follow equations (Rodrigues and Teixeira, 2011).

$$R_{xx}[u] = \begin{cases} \frac{1}{N} \cdot \sum_{n=0}^{N-m-1} x[n] \cdot x[n+u] & , u \geq 0 \\ R_{xx}^*[-u] & , u < 0 \end{cases} \quad (1)$$

$$\begin{aligned} S_{x,w}[k] &= \frac{1}{N} \cdot DFTS \{R_{xx}[u]\} \\ &= \frac{1}{N} \cdot \sum_{u=0}^{2N-1} R_{xx}[u] \cdot e^{-j \cdot \frac{2\pi k}{2N-1} \cdot u}, \end{aligned} \quad (2)$$

where, $k = 0, \dots, 2N - 1$.

The PSD was normalized to the scale 0 to 1 and it was designated PSD_n (Rodrigues and Teixeira, 2011), as can be observed in the next equation.

$$PSD_n[m, k] = \frac{S_{x,w}[n]}{\sum_{n=k_1}^{k_2} S_{x,w}[n]}, m = 0, \dots, N_t - 1 \quad (3)$$

where k_1 and k_2 represent discrete cutoff frequencies.

2.3 Feature Extraction

The relative power (RP) in EEG conventional frequencies bands was obtained by the sum of the components of the PSD_n in the conventional frequency bands: delta (δ , 1-4 Hz), theta (θ , 4-8 Hz), alpha (α , 8-13Hz), beta1 (β_1 , 13-19 Hz), beta2 (β_2 , 19-30 Hz) and gamma (γ , 30-40 Hz).

$$RP(\delta) = \sum_{f=1Hz}^{4Hz} PSD_n(f) \quad (4)$$

$$RP(\theta) = \sum_{f=4Hz}^{8Hz} PSD_n(f) \quad (5)$$

$$RP(\alpha) = \sum_{f=8Hz}^{13Hz} PSD_n(f) \quad (6)$$

$$RP(\beta_1) = \sum_{f=13Hz}^{19Hz} PSD_n(f) \quad (7)$$

$$RP(\beta_2) = \sum_{f=19Hz}^{30Hz} PSD_n(f) \quad (8)$$

$$RP(\gamma) = \sum_{f=30Hz}^{40Hz} PSD_n(f) \quad (9)$$

Four spectral ratios were used to resume the deceleration of the EEG as defined in (Rodrigues and Teixeira, 2011).

$$r_1 = \frac{RP(\alpha)}{RP(\theta)} \quad (10)$$

$$r_2 = \frac{RP(\alpha) + RP(\beta_1) + RP(\beta_2) + RP(\gamma)}{RP(\delta) + RP(\theta)} \quad (11)$$

$$r_3 = \frac{RP(\beta_1) + RP(\beta_2)}{RP(\delta)} \quad (12)$$

$$r_4 = \frac{RP(\beta_2)}{RP(\delta)} \quad (13)$$

In order to achieve a through analysis of the spectral characteristics of the EEG records of the patients diagnosed with probable Alzheimer's disease and Cs

in this study the following parameters were calculated using the PSD .

The Mean Frequency (MF) is defined as the frequency comprising of 50% of the power (Rodrigues, 2011).

$$0.5 \sum_{1Hz}^{40Hz} PSD_n(f) = \sum_{1Hz}^{MF} PSD_n(f) \quad (14)$$

The PSD shows the highest peak at the α band. So we can calculate a parameter entitled Individual Alpha Frequency (IAF) in the extended α band between 4Hz and 15 Hz (Rodrigues, 2011).

$$0.5 \sum_{4Hz}^{15Hz} PSD_n(f) = \sum_{4Hz}^{IAF} PSD_n(f) \quad (15)$$

The Spectral Edge Frequency ($SEF95\%$) is calculated as the frequency that comprises 95% of the power spectrum (Rodrigues, 2011).

$$0.95 \sum_{1Hz}^{40Hz} PSD_n(f) = \sum_{1Hz}^{SEF95\%} PSD_n(f) \quad (16)$$

Finally, the Spectral Entropy (SE) is a measure of disorder that can be used as an irregularity estimate of the EEG as we can see on the equation below (Rodrigues, 2011):

$$SE = - \sum_{1Hz}^{40Hz} PSD_n(f) \cdot \text{Log}[PSD_n(f)] \quad (17)$$

3 METHODOLOGY

We developed an ANN in order to discriminate EEG signals between Cs and AD patients. It was created the input P matrix with EEG data of the subjects in study. This input matrix consists in one column for each segment of 5 s for all subjects (controls and patients). The lines of the matrix consist in the 14 features defined above (6 RP , 4 spectral ratios - r , MF , IAF , $SEF95\%$ and SE) for the 19 electrodes, in a total of $14 \times 19 = 266$ lines. In the future we intend to reduce this number of input nodes by a selection process. It was found that some electrodes presented probable noise, due to bad conduction during the acquisition process. These electrodes were identified by observation of the spectral components because they had similar energy in all bandwidth. To reduce that noise we proceeded to the average of electrodes without noise belonging to the same group of the subject involved, including the damaged electrode. For example, if the electrode 8 of a control subject presented noise we proceeded to the

substitution of the data of this electrode by the average of all electrodes of Cs without noise in any electrode, including the electrode 8. This method allowed to remove electrodes with wrong data. To identify noise moments we have used the *surf* function of *Matlab* software, which allowed to create a three-dimensional shaded surface from PSD components of signals of all five segments seconds presented in the electrode signal, which would allow the visualization of some useful information to create the P matrix.

Thus, in a first phase it was checked whether there were significant oscillations between electrodes positioned at the same position either Cs or AD patients and, if oscillations in subjects belonging to the same group were significant. It was found limited relevance for the identification by visualization, because differences were observed in electrodes of subjects belonging to the same group.

In a second phase we tried to find out if there were amplitude variations along the electrode segments and if the peaks were determinative. It was found that AD patient 4 presented small variations along the segments in frequency (view Figure 1), compared to other cases where there were no large amplitudes of the electrodes and that presented a greater distribution of the amplitudes per electrode. It has been assumed that AD patient 4 was in an advanced phase of the AD . As in patient 4, also the patient 10 and 38 and control subject 17 exhibited large variations with small amplitudes of energy in a restricted range of frequencies. It should be noted the fact that in the universe of 14 Cs the only one who showed higher accumulations of energy in a restricted range of frequencies of electrodes was the control subject 17.

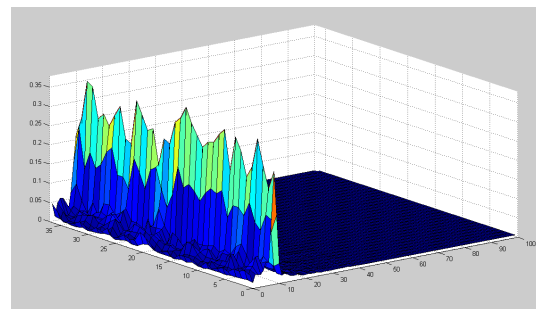


Figure 1: EEG signal Amplitude of successive segments (right axis) of one electrode along frequency (left axis).

In a third and last stage we look for noise existence in AD patients and Cs and we confirmed noise in electrode 8 of the patient 5, in electrodes 8 and 9 of the control subject 7, in electrodes 8 and 16 of the control subject 13, in electrode 16 of patient 25 and in the electrodes 8,9 and 17 of the patient 50.

The matrix P was split into three randomly exclusive subgroups:

- Subset 1: Training set.
- Subset 2: Validation set.
- Subset 3: Test set.

The subset 1 presented the most part of the P matrix for training. It was about 80% of the P matrix. Therefore, this subset had the remaining segments for each subject in study. The subset 2 and 3 showed about 10% of the P matrix, respectively, and they had three segments of each subject randomly selected, from the 34 subjects in study. The size of each subset can be observed in table 1.

Table 1: Dimension of Training, Test and Validation Sets.

Total Dimension of matrix $P=266 \times 1066$		
Training Set	Validation Set	Test Set
266x862	266x102	266x102

In this work a feed-forward architecture of ANN was employed with 266 nodes input layer and 1 node in the output layer. The output node codes if the data features correspond to a control subject or in opposition corresponds to a patient. Different number of nodes in the hidden layer has experimented. For all ANN architectures, two learning algorithms were used, the Resilient Back-Propagation ($trainrp$) and the Levenberg - Marquardt ($trainlm$) and two Transfer Functions in the hidden layer were also used, Logarithmic Sigmoid ($logsig$) and the Tangential Sigmoid ($tansig$). Several nodes variations on the hidden layers were utilized and the $purelin$ was the transfer function of the output layer. The best ANN results were obtained with $trainlm$, $logsig$ and 9 nodes on the hidden layer, as can be seen by the autocorrelation coefficient (R) of the training, test and validation sets and overall P matrix presented in the table 2.

Once the output of the ANN is a linear function between 0 and 1 and we considered 0 for controls and 1 for AD patients a threshold value of 0.5 of output was considered. Therefore values >0.5 of the output are interpreted as AD patients and values < 0.5 are considered as Cs .

3.1 Train with Resilient Back-propagation Algorithm

Next will be showed the best results for regression and for the performance of ANN obtained with $trainrp$ algorithm (Riedmiller and Braun, 1993), $logsig$ transfer function and 14 nodes in the hidden layer (view Figure

2 and Figure 3). It must be noted that the result of the correlation coefficient in Test Set has the highest value for this ANN . The $tansig$ transfer function had noticed great results but not so greatest as $logsig$ transfer function.

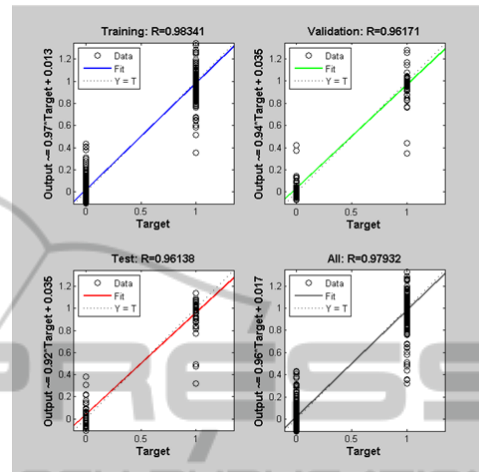


Figure 2: Regression Plot with $trainrp$ algorithm.

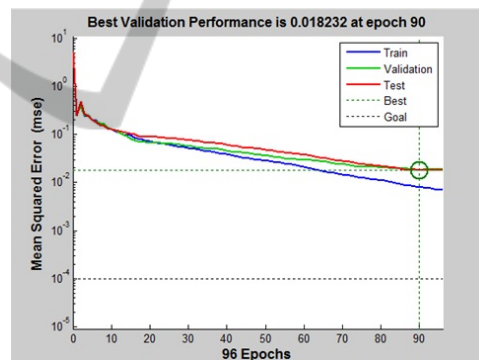


Figure 3: Performance Plot with $trainrp$ algorithm.

3.2 Train with Levenberg-Marquardt Back-propagation Algorithm

Next will be showed the best results for regression and for the performance of ANN obtained with $trainlm$ algorithm (Marquardt, 1963; Hagan and Menhaj, 1994), $logsig$ transfer function and 9 nodes in the hidden layer of entrance in the neural network (view Figure 4 and Figure 5). This ANN was the performance with the best correlation coefficient of the P matrix and of the Training and Validation Sets. Similar to the $trainrp$ algorithm, the $tansig$ activation function had noticed equally great results but not so greatest as $logsig$ activation function.

Table 2: Classification results.

Training Algorithm	Transfer Function	Nodes of the Hidden Layer	Training Set (R)	Test Set (R)	Validation Set (R)	P Matrix (R)
TrainRP	Logsig	14	0.98341	0.96138	0.96171	0.97932
TrainLM	Logsig	9	0.99964	0.95758	0.9653	0.99245
TrainRP	Tansig	11	0.97885	0.93211	0.95087	0.97173
TrainLM	Tansig	9	0.99807	0.91164	0.95555	0.98622

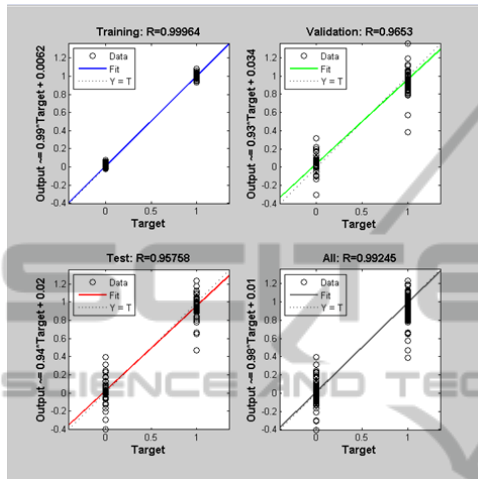


Figure 4: Regression Plot with *trainlm* algorithm.

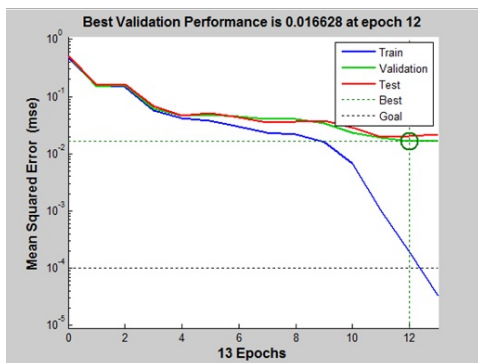


Figure 5: Performance Plot with *trainlm* algorithm.

4 CONCLUSIONS AND FUTURE WORK

AD keeps on defying medical research, even despite the progress achieved in better knowledge of the complex processes of deterioration of memory levels, this disease still remains with no prospect of a cure. Therefore, it is very important to have an early diagnose of disease in order to avoid their evolution. In this paper we developed a model based on ANN to discriminate between Cs and AD patients. A set of 14 features

for each electrode was used in the input of the ANN. These features are based in the PSD determined with the STFT. The features consists in the Relative Power of the PSD in the bandwidths of delta, theta, alpha, beta1, beta2 and gamma, four spectral ratios, the Mean Frequency, the Individual Alpha Frequency, the Spectral Edge Frequency and the Spectral Entropy. The output consisted in a binary to classify Cs or AD patients. Several ANN were experimented with different number of nodes in the hidden layer and activations functions. The Resilient Back-Propagation and the Levenberg-Marquardt training algorithms were also experimented. The best results give a correlation coefficient of 0.961 in the test set. Once the data of this set were never seen during the training process we may consider it as a very competitive result compared with similar previous works. Namely, the obtained classification results can be compared with the studies presented in (Vialatte et al., 2005a; Vialatte et al., 2005b; Vialatte et al., 2008; Rodrigues et al., 2011; Rodrigues and Teixeira, 2011; Rodrigues, 2011; Melissant et al., 2005).

For a future work we intend to reduce the number of features used in the input of the ANN and identify electrodes that can be removed due to low discriminative power. Experiment the use of the PSD determined using the Wavelet Transform as presented by Rodrigues in (Rodrigues, 2011; Rodrigues and Teixeira, 2011; Rodrigues et al., 2011).

REFERENCES

- Baker, M., Akrofi, K., Schiffer, R., and Michael, W. O. B. (2008). Eeg patterns in mild cognitive impairment (mci) patients. *Open Neuroimaging J.*, 2:52–55.
- Blennow, K. (2005). *Amsterdam: European College of Neuropsychopharmacology.* ECNP.
- Blennow, K., Leon, M., and Zetterberg, H. (2006). Alzheimer’s disease. *The Lancet*, 368:387–403.
- Feldman, H. and Woodward, M. (2005). The staging and assessment of moderate to severe alzheimer disease. *Neurology*, 65:10–17.
- Hagan, M. T. and Menhaj, M. (1994). Training feedforward networks with the marquardt algorithm. *IEEE Transactions on Neural Networks*, 5:989–993.

- Hort, J., O'Brien, J. T., Gainotti, G., Pirtila, T., Popescu, B. O., Rektorova, I., Sorbi, S., and Scheltens, P. (2010). Efn guidelines for the diagnosis and management of alzheimer's disease. *European Journal of Neurology*, 17:1236–1248.
- Jeong, J. (2004). Eeg dynamics in patients with alzheimer's disease. *Clin. Neurophysiol*, 115:1490–1505.
- Marquardt, D. (1963). An algorithm for least-squares estimation of nonlinear parameters. *SIAM Journal on Applied Mathematics*, 11:431–441.
- Melissant, C., Ypma, A., Frietman, E., and Stam, C. (2005). A method for detection of alzheimer's disease using ica-enhanced eeg measurements. *Artif Intell Med*, 33:209–222.
- Moreira, P. and Oliveira, C. (2005). *A Doença de Alzheimer e outras Demências em Portugal*, chapter Fisiopatologia da doença de Alzheimer e de outras demências., pages 41–60. Lisboa: Lidel Edições Técnicas.
- Riedmiller, M. and Braun, H. (1993). A direct adaptive method for faster backpropagation learning: The rprop algorithm. *Proceedings of the IEEE International Conference on Neural Networks*, 1:586–591.
- Rioul, O. and Vetterli, M. (1992). Wavelets and signal processing. *IEEE Signal Processing Magazine*, 8:14–38.
- Rodrigues, P. (2011). Diagnóstico da doença de alzheimer com base no electroencefalograma. Master's thesis, Instituto Politécnico de Bragança - Escola Superior de Tecnologia e Gestão.
- Rodrigues, P. and Teixeira, J. (2011). Artificial neural networks in the discrimination of alzheimer's disease. *Communications in Computer and Information Science*, 221:272–281.
- Rodrigues, P., Teixeira, J., Hornero, R., Poza, J., and Careres, A. (2011). Classification of alzheimer's electroencefalograms using artificial neural networks and logistic regression. *Japan - Portugal Nano-Biomedical Engineering Symposium 2011*, 1 (ISBN-4-904157-20-6):33–34.
- Stahl, S. (2008). *Stahl's Essential Psychopharmacology. Neuroscientific Basis and Practical Applications*. Cambridge University Press, third edition.
- Vialatte, F., Cichocki, A., Dreyfus, G., Musha, T., Rutkowski, T., and Gervais, R. (2005a). Blind early detection of alzheimer's disease by blind source separation and bump modelling of eeg signals. *Lectues Notes in Computer Science*, 3596:683–692.
- Vialatte, F., Cichocki, A., Dreyfus, G., Musha, T., Shishkin, S., and Gervais, R. (2005b). Early detection of alzheimer's disease by blind source separation, time frequency representation, and bump modeling of eeg signals. *Lecture Notes in Computer Science*, 3696:683–692.
- Vialatte, F., Maurice, M., and Cichocki, A. (2008). Why sparse bump models? *Neuroimage*, 41:159.