Abstract—The aim of this study was to analyse the electroencephalogram (EEG) background activity in Alzheimer’s disease (AD) with the Detrended Moving Average (DMA) method, a new approach to quantify correlation properties in non-stationary signals with underlying trends. EEGs were recorded from the 19 scalp loci of the international 10–20 system in 11 AD patients and 11 age-matched controls. Our results showed two scaling regions in all subjects’ channels, with a clear bend when their corresponding slopes ($\alpha_1$ and $\alpha_2$) were distinctly different. With the exception of electrode T4, the $\alpha_1$ values were lower in control subjects than in AD patients, with significant differences at T5, P3, P4 and O1 ($p < 0.01$, Student’s $t$-test). On the other hand, $\alpha_2$ values were higher in control subjects than in AD patients, with significant differences only at F4. Furthermore, we evaluated the ability of $\alpha_2$ to discriminate AD patients from control subjects at these electrodes using ROC plots. We obtained a maximum accuracy of 81.82% at O1 with $\alpha_2$ and at F4 with $\alpha_2$. These findings suggest that the scaling behaviour of the EEG is sensitive to AD and that the DMA method could help to increase our insight into brain dysfunction in AD.

I. INTRODUCTION

ALZHEIMER’S disease (AD) is a primary degenerative dementia of unknown aetiology that gradually destroys brain cells and represents the most prevalent form of dementia in western countries [1]. AD is characterized by progressive impairments in cognition and memory whose course lasts several years prior to the death of the patient [2]. Structural changes in AD are related to the accumulation of amyloid plaques between nerve cells in the brain and with the appearance of neurofibrillary tangles inside nerve cells, particularly in the hippocampus and the cerebral cortex [3].

The clinical diagnosis of AD is made primarily on the basis of medical history studies, psychiatric evaluation and different memory, reasoning and mental status tests. However, physicians only make a diagnosis of AD with an accuracy of about 90% and a definite diagnosis is only possible by necropsy [4]. Hence, new approaches are necessary to improve AD diagnosis.

The electroencephalogram (EEG) has been used as a tool for investigating dementias for several decades. This is mainly due to the fact that AD is a cortical dementia in which EEG abnormalities are more frequently shown. AD patients’ EEGs generally show a shift of the power spectrum to lower frequencies and a decrease of coherence among cortical areas [2]. However, in the early stages of the disease the EEG may exhibit normal frequencies and may be similar to that of elderly control subjects [5]. From another point of view, several studies have examined the non-linear dynamics of the EEG in AD (a detailed review can be found in [2]). In general, the EEG is less complex and more regular in AD patients than in controls [2], [6], [7]. Moreover, AD patients’ EEGs show reduced functional connections when compared to elderly controls [8]. EEG abnormalities in AD are thought to be associated with functional disconnections among cortical areas resulting from death of cortical neurons, axonal pathology, cholinergic deficits, etc. [2].

The complex nature of the electrical brain activity results in fluctuations in the EEG [9]. To understand EEG activity in a better way, it is necessary to characterize its fluctuations over different time scales. Recent studies show that EEG oscillations in the human brain show long-range temporal correlations [9]–[11] and that the scaling behaviour of the EEG is sensitive to AD [12].

In this preliminary study, we examined the scaling behaviour of the EEG in AD with the Detrended Moving Average (DMA) method, a new approach to quantify correlation properties in non-stationary signals with underlying trends [13]. We wanted to test the hypothesis that long-range temporal correlations in AD patients’ EEGs would be different from those of age-matched controls.
II. MATERIAL AND METHODS

A. Subjects and EEG recording

Twenty-two subjects participated in this study. Eleven patients (5 men and 6 women; age = 72.5 ± 8.3 years, mean ± standard deviation, SD) fulfilling the criteria of probable AD were recruited from the Alzheimer’s Patients’ Relatives Association of Valladolid (AFAVA) and referred to the University Hospital of Valladolid (Spain), where the EEG was recorded. All of them had undergone a thorough clinical evaluation that included clinical history, physical and neurological examinations, brain scans and a Mini-Mental State Examination (MMSE), generally accepted as a quick and simple way to evaluate cognitive function [14]. The mean MMSE score for the patients was 13.1 ± 5.9. The control group consisted of 11 age-matched control subjects without past or present neurological disorders (7 men and 4 women; mean age = 72.8 ± 6.1 years). The local ethics committee approved the study and all control subjects and all caregivers of the patients gave their informed consent for participation in the current study.

EEGs were recorded from the 19 scalp loci of the international 10-20 system (electrodes F3, F4, F7, F8, Fp1, Fp2, T3, T4, T5, T6, C3, C4, P3, P4, O1, O2, Fz, Cz and Pz) using a Profile Study Room 2.3.411 EEG equipment (Oxford Instruments). More than five minutes of data were recorded from each subject. The sample frequency was 256 Hz, with a 12-bit A-to-D precision. Recordings were made under the eyes-closed condition in order to obtain as many artefact-free EEG data as possible. All EEGs were visually inspected by a specialist physician to check for eye movement and other artefacts. Afterwards, EEGs were organized in 5 second artefact-free epochs (1280 points) that were copied as ASCII files for off-line analysis on a personal computer. Furthermore, all recordings were digitally filtered with a band-pass filter with cut-off frequencies at 0.5 Hz and at 40 Hz in order to remove EMG activity prior to the computation of DMA.

B. Detrended Moving Average

Detrended Fluctuation Analysis (DFA) provides an estimation of scaling information and long-range correlations in time series, and is known for its robustness against non-stationarities [9], [15]. However, as the removal of trends in DFA is based on discontinuous polynomial fitting, oscillations in the fluctuation function and significant errors in crossover locations can be introduced. The Detrended Moving Average (DMA) method was introduced to estimate correlation properties of non-stationary signals without any assumption of the type of trends, the probability distribution or other characteristics of the underlying process [16].

Let the EEG time series be denoted by \( \{x(i)\} \), where \( i \) is the discrete time, ranging from \( i = 1 \) to \( N (N = 1280) \). To perform the DMA method, first we need to integrate the EEG time series to obtain:

\[
y(i) = \sum_{j=1}^{i} [x(j) - \bar{x}],
\]

where \( \bar{x} \) is the mean of the whole temporal series. Then, trends are detected in the data with a moving average.

There are two important categories of moving average: simple moving average and weighted moving average. In this study we consider the simple moving average, where equal weight is assigned to each data point in a window of size \( n \). The position to which the average of all weighted data points is assigned determines the relative contribution of the “past” and “future” points [16]. We consider the backward moving average, which for a window of size \( n \) is defined as

\[
y_{n}(i) = \frac{1}{n} \sum_{k=0}^{n-1} y(i-k),
\]

where \( y(i) \) is the aforementioned integrated signal. The average of the signal data points within the window refers to the last sample covered by the window. Thus, the operator in (2) is causal.

Once the moving average is obtained, we detrend the signal by subtracting it from the integrated profile:

\[
C_{n}(i) = y(i) - y_{n}(i).
\]

We then calculate the fluctuation for a window of size \( n \) as

\[
F(n) = \sqrt{\frac{1}{N-n+1} \sum_{i=1}^{N-n+1} [C_{n}(i)]^2}.
\]

A power law relation between the fluctuation function \( F(n) \) and the scale \( n \) (\( F(n) \propto n^{\alpha} \)) indicates a self-similar behaviour.

C. Statistical analysis

Student’s \( t \)-test was used to evaluate the statistical differences between the scaling exponents from AD patients and control subjects. Differences were considered statistically significant if the \( p \) value was lower than 0.01.

The ability to discriminate AD patients from control subjects at the electrodes where \( p < 0.01 \) was evaluated using Receiver Operating Characteristic (ROC) curves [17].

III. RESULTS

We performed the DMA method for channels F3, F4, F7, F8, Fp1, Fp2, T3, T4, T5, T6, C3, C4, P3, P4, O1, and O2. We studied the fluctuations using window sizes between 3 and
128 samples (from 0.01 s to 0.5 s). Furthermore, we plotted
the natural logarithm of $F(n)$ as a function of the natural
logarithm of $n$. If the plot displays a linear scaling region with
a certain scaling exponent, then there is a power-law
behaviour in the time series.

We found two scaling regions in the EEG with a clear bend
when the two slopes in the two regions are distinctly different.
These scaling properties were found in all channels for all
subjects. Fig. 1 and Fig. 2 show $F(n)$ vs. $n$ in a log-log plot for
one EEG epoch from a control subject at electrode T5 and for
one EEG epoch from an AD patient at electrode T5,
respectively. In both cases, two different slopes can be seen
and a bend in the transition between them can be observed.
We have denoted the scaling exponent of the first region as $\alpha_1$
and the exponent on the second one as $\alpha_2$.

To quantify the scaling exponents, we performed a linear fit
in region I for $1 < \ln n < 2.3$ and in region II for $3 < \ln n < 4.5$. The limits were chosen after a visual inspection of the
results. The $\alpha_1$ and $\alpha_2$ values for the AD patients and control
subjects and the $p$ values of the Student’s $t$-tests performed to
examine the differences between both groups are summarized
in Tables I and II, respectively. With the exception of electrode T4, the $\alpha_1$ values were lower in control subjects than
in AD patients, with significant differences at T5, P3, P4 and
O1 ($p < 0.01$). On the other hand, $\alpha_2$ values were higher in
control subjects than in AD patients, with significant
differences only at F4.

Finally, we evaluated the ability of the scaling exponents to
discriminate AD patients from control subjects at the
electrodes where significant differences were found using
ROC plots. Results are shown in Tables III and IV, where it can be seen that an accuracy of 81.82% was obtained at O1
with $\alpha_1$ and at F4 with $\alpha_2$.

**TABLE I**

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Control subjects (mean ± SD)</th>
<th>AD patients (mean ± SD)</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>F3</td>
<td>1.1250 ± 0.0422</td>
<td>1.1433 ± 0.0241</td>
<td>0.1808</td>
</tr>
<tr>
<td>F4</td>
<td>1.1268 ± 0.0443</td>
<td>1.1316 ± 0.0233</td>
<td>0.7536</td>
</tr>
<tr>
<td>F7</td>
<td>1.1155 ± 0.0414</td>
<td>1.1286 ± 0.0314</td>
<td>0.4109</td>
</tr>
<tr>
<td>F8</td>
<td>1.1185 ± 0.0301</td>
<td>1.1306 ± 0.0241</td>
<td>0.3112</td>
</tr>
<tr>
<td>Fp1</td>
<td>1.1294 ± 0.0323</td>
<td>1.1525 ± 0.0285</td>
<td>0.0903</td>
</tr>
<tr>
<td>Fp2</td>
<td>1.1306 ± 0.0417</td>
<td>1.1545 ± 0.0219</td>
<td>0.1072</td>
</tr>
<tr>
<td>T3</td>
<td>1.0606 ± 0.0697</td>
<td>1.0730 ± 0.0800</td>
<td>0.7007</td>
</tr>
<tr>
<td>T4</td>
<td>1.0748 ± 0.0656</td>
<td>1.0521 ± 0.1390</td>
<td>0.6300</td>
</tr>
<tr>
<td>T5*</td>
<td>1.0723 ± 0.0533</td>
<td>1.1304 ± 0.0382</td>
<td>0.0082</td>
</tr>
<tr>
<td>T6</td>
<td>1.0818 ± 0.0502</td>
<td>1.1286 ± 0.0403</td>
<td>0.0257</td>
</tr>
<tr>
<td>C3</td>
<td>1.0942 ± 0.0533</td>
<td>1.1191 ± 0.0420</td>
<td>0.2390</td>
</tr>
<tr>
<td>C4</td>
<td>1.0893 ± 0.0519</td>
<td>1.1082 ± 0.0542</td>
<td>0.4144</td>
</tr>
<tr>
<td>F3*</td>
<td>1.0862 ± 0.0462</td>
<td>1.1412 ± 0.0323</td>
<td>0.0042</td>
</tr>
<tr>
<td>F4*</td>
<td>1.0850 ± 0.0401</td>
<td>1.1345 ± 0.0327</td>
<td>0.0048</td>
</tr>
<tr>
<td>O1*</td>
<td>1.0566 ± 0.0481</td>
<td>1.1270 ± 0.0447</td>
<td>0.0028</td>
</tr>
<tr>
<td>O2</td>
<td>1.0712 ± 0.0508</td>
<td>1.1290 ± 0.0447</td>
<td>0.0102</td>
</tr>
</tbody>
</table>

**TABLE II**

<table>
<thead>
<tr>
<th>Electrode</th>
<th>Control subjects (mean ± SD)</th>
<th>AD patients (mean ± SD)</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>F3</td>
<td>0.8876 ± 0.0479</td>
<td>0.8079 ± 0.1125</td>
<td>0.0429</td>
</tr>
<tr>
<td>F4*</td>
<td>0.8974 ± 0.0478</td>
<td>0.7955 ± 0.1032</td>
<td>0.0075</td>
</tr>
<tr>
<td>F7</td>
<td>0.8741 ± 0.0433</td>
<td>0.8187 ± 0.0943</td>
<td>0.0923</td>
</tr>
<tr>
<td>F8</td>
<td>0.8855 ± 0.0384</td>
<td>0.8263 ± 0.1002</td>
<td>0.0823</td>
</tr>
<tr>
<td>Fp1</td>
<td>0.8947 ± 0.0175</td>
<td>0.8650 ± 0.0990</td>
<td>0.3386</td>
</tr>
<tr>
<td>Fp2</td>
<td>0.8983 ± 0.0257</td>
<td>0.8621 ± 0.0750</td>
<td>0.1458</td>
</tr>
<tr>
<td>T3</td>
<td>0.8593 ± 0.0567</td>
<td>0.7696 ± 0.0933</td>
<td>0.0131</td>
</tr>
<tr>
<td>T4</td>
<td>0.8661 ± 0.0706</td>
<td>0.7715 ± 0.1010</td>
<td>0.0192</td>
</tr>
<tr>
<td>T5</td>
<td>0.8007 ± 0.0593</td>
<td>0.7359 ± 0.1014</td>
<td>0.0823</td>
</tr>
<tr>
<td>T6</td>
<td>0.8312 ± 0.0690</td>
<td>0.7508 ± 0.1088</td>
<td>0.0517</td>
</tr>
<tr>
<td>C3</td>
<td>0.8551 ± 0.0683</td>
<td>0.7993 ± 0.1092</td>
<td>0.1662</td>
</tr>
<tr>
<td>C4</td>
<td>0.8588 ± 0.0459</td>
<td>0.7774 ± 0.1328</td>
<td>0.0691</td>
</tr>
<tr>
<td>P3</td>
<td>0.7992 ± 0.0665</td>
<td>0.7515 ± 0.1094</td>
<td>0.2305</td>
</tr>
<tr>
<td>P4</td>
<td>0.7937 ± 0.1126</td>
<td>0.7437 ± 0.1203</td>
<td>0.3272</td>
</tr>
<tr>
<td>O1</td>
<td>0.7798 ± 0.0899</td>
<td>0.7374 ± 0.1144</td>
<td>0.3451</td>
</tr>
<tr>
<td>O2</td>
<td>0.8071 ± 0.1084</td>
<td>0.7516 ± 0.1006</td>
<td>0.2281</td>
</tr>
</tbody>
</table>

---

Fig. 1. $F(n)$ vs. $n$ in a log-log plot for one EEG epoch from electrode T5 of a control subject. The scaling exponents $\alpha_1$ and $\alpha_2$ are depicted with a solid line and their numerical values included.

Fig. 2. $F(n)$ vs. $n$ in a log-log plot for one EEG epoch from electrode T5 of an AD patient. The scaling exponents $\alpha_1$ and $\alpha_2$ are depicted with a solid line and their numerical values included.
that patients and control subjects at T5, T6 and O1 in the second and we only found significant differences between AD and discriminates AD patients from control subjects at these would weaken or even block the interactions. temporal correlations of EEG should originate from the strong accuracies ranging from 77.27% to 81.82%. These findings electrodes was evaluated using ROC plots. We obtained that subtracting a continuous function, the moving average. Additionally, DMA is more accurate since the moving average

**IV. DISCUSSION AND CONCLUSIONS**

We analysed the EEG background activity of 11 AD patients and 11 control subjects with the DMA method with backward moving average, a new approach to quantify correlation properties in non-stationary signals with underlying trends [13]. We found two different scaling regions in the EEG that depend on the window size \( n \). The first region corresponds to small time scales (less than 0.04 s) and could be characterised by a scaling exponent \( \alpha_1 \). The second one corresponds to time scales from 0.08 s to 0.43 s and could be described with an exponent \( \alpha_2 \).

We have found that \( \alpha_1 \) was significantly lower in the control subjects’ EEG at electrodes T5, P3, P4 and O1 and that \( \alpha_2 \) was significantly higher in that group at F4. Furthermore, the ability of both scaling exponents to discriminate AD patients from control subjects at these electrodes was evaluated using ROC plots. We obtained accuracies ranging from 77.27% to 81.82%. These findings suggest that the scaling behaviour of the EEG is sensitive to AD. From the view of many body systems, long-range temporal correlations of EEG should originate from the strong interactions of the neural cells. It is logical to assume that AD would weaken or even block the interactions.

We have previously analysed the same database with DFA and we only found significant differences between AD patients and control subjects at T5, T6 and O1 in the second scaling region used in this study [12]. It has been suggested that DMA is an improvement over DFA [18], as the former does not require dividing the series into non-overlapping windows. Instead, the DMA method determines the series by subtracting a continuous function, the moving average. Additionally, DMA is more accurate since the moving average is a better low-pass filter when compared to the polynomial filter used for DFA [18]. Thus, EEG analysis with the DMA method might be more appropriate than with DFA. However, it should be considered that, while the main significant differences between both groups were found at the first scaling region with DMA, DFA showed significant differences only at the second scaling region. It could be argued that, although the DMA method might be more appropriate than DFA, both methods could provide complementary information. Hence, we should investigate whether the combined use of DFA and DMA could improve the results obtained independently with each technique.

Some limitations of this preliminary study must be mentioned. Firstly, the sample size was small. To prove the usefulness of these techniques as an AD diagnostic tool, this approach should be extended on a much larger patient population. Moreover, the backward moving average is affected by a rather slow reaction to changes in the signal, due to a delay of half the window size compared to the signal [16]. Thus, other moving average schemes should be evaluated to check their possible usefulness in the analysis of EEG background activity in AD. Furthermore, as AD diagnosis is only definite after necropsy, the sample may not fully represent this disease. Finally, the scaling properties of the EEG should be studied in depression or other dementias to verify if the reported changes are specific to AD.

In summary, our results indicate that the DMA method could be useful in AD diagnosis. In future studies we should investigate whether the combination of information obtained with DMA at several electrodes, or even the combination of results obtained with different techniques, could provide a better classification between AD patients and control patients.

**REFERENCES**


