An investigation into the correlations found in the EEG data of Epileptic and Alzheimer's Disease patients

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Abstract

Alzheimer's Disease (AD) and epilepsy are both known pathological malfunctions of the brain. The former manifests itself in a patient's cognitive decline and in some cases the occurrence of seizures. The latter is observed as abnormal electrical activity in the brain, loss of conscious processing, disturbances in sensations and epileptic fits (otherwise known as convulsions or seizures). In this report we investigate the results of two echoencephalograms (EEG) performed on an epileptic and AD patient respectively by comparing the correlations of the data collected between the receptors during pre-ictal, ictal and post-ictal seizure for the epileptic patient and for an arbitrary time for the AD patient (whose results showed no significant disturbances). Our findings show that pre-ictal activity promotes stronger inter-hemispheric connections and synchrony which may be indicative of an approaching seizure, that the seizure focus is in the occipital cortices, that the recovery from seizure involves frontal lobe recruitment. Unfortunately, our analysis did not find any conclusive evidence to suggest that this AD patient in particular exhibited episodes of hyperexcitability often associated with epileptic brains.

Introduction

AD is associated with the increased cerebral levels of β-amyloid (Aβ) and consequent cognitive decline and increased incidence of seizures [1]. Epilepsy is defined as abnormal electrical activity in the brain which can cause convulsions, a loss of conscious processing and disturbances in sensations [2]. In 2009 data was obtained in transgenic mice expressing human APP (hAPP) in neurons which indicate that high levels of Aβ are sufficient to elicit epileptiform activity and seizures [1]. While neuro-degeneration and aging-related cofactors are often said to contribute to the development of seizures, this experiment in 2009 has opened a new pathway of scientific research into correlations between Epilepsy and AD. Analyisis of EEG data can give a greater insight into the brain functionality of patients of pathological malfunctions, and comparing can suggest possible links. Understanding this correlation better could open potential treatments and therapeutic avenues, as medications used on Epilepsy patients might be effective on AD patients and vice versa.
In this project, the goal is to establish features in patients with AD that are similar to that of epileptic patients. This will be achieved through the use of EEG data which measures electrical activity across the brain. EEG data will be taken from three spontaneous time periods of the seizure; ictal, pre-ictal and post-ictal. Characteristics in the EEG data from this will then be analysed against each other in order to decipher a conclusion.

Methods

An EEG is a recording of brain activity [3]. It is performed by placing a network of electrical sensors (channels) onto a patient's scalp, and then measurements are taken at a certain frequency for a particular interval of time. The individual channels act as electrical volt-meters, measuring the electrical activity in different regions of the brain over time and the number of them used for an EEG can vary depending on the doctor's or scientist's wishes. For the purposes of this report's investigation, the following data was provided:

- The EEG data collected for the Epileptic patient consists of 22 channels recording brain activity for 55 minutes at a sampling frequency of 500Hz.
- The EEG data collected for the Alzheimer's Patient consists of 64 channels recording brain activity for 3 minutes at a sampling rate of 250Hz.
- The samples were taken when both patients were sitting comfortably in a chair with their eyes open, discounting the possibility of the patients being asleep during the collection of data.

Figure 1: EEG data collected for 200 seconds from the Alzheimer's Disease patient. It shows no significant disturbances apart from during around 80 to 100 seconds. All this was incorporated into the selected time used for correlation.

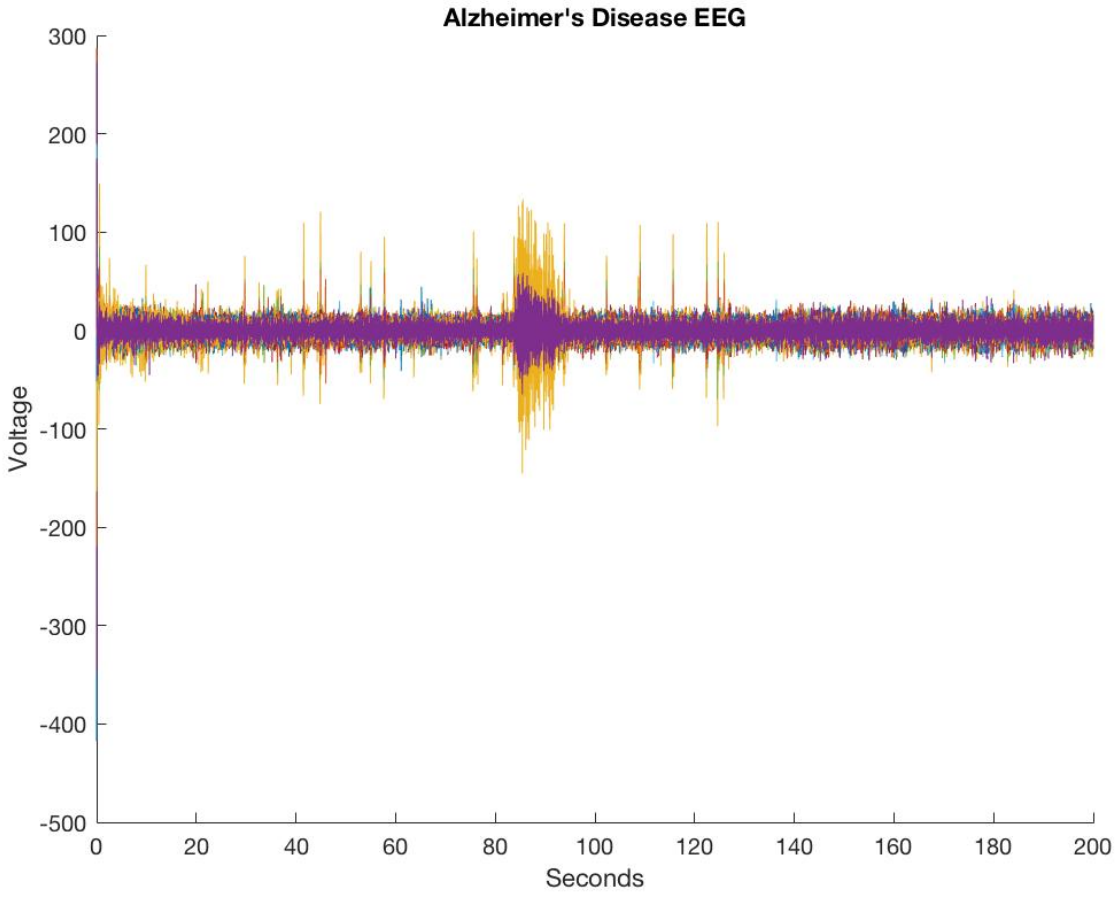
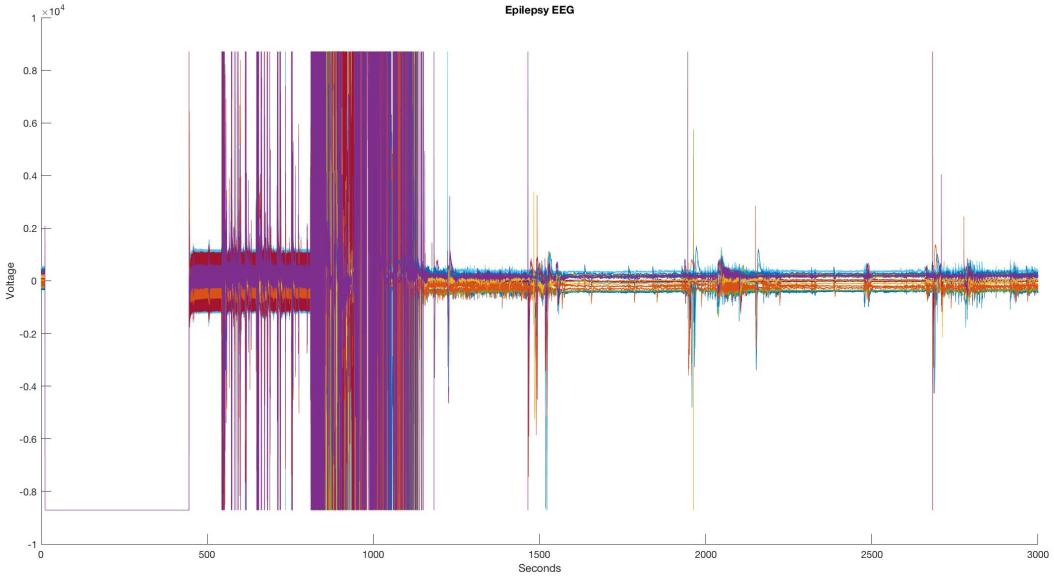


Figure 2: EEG data collected for 3000 seconds from the Epileptic patient. It shows clearly differing signals throughout the seizure, from pre-ictal, ictal and post-ictal.



In order to ascertain useful information from these data sets the following pre-processing steps are required.

Filtration

Like most electrical readings taken, there will be a certain degree of noise affecting the results. In the case of the EEG readings the noise originates from the electricity from the mains supply. By filtering out these signals the remaining results would be a more accurate representation of the signal measured from the brain. To do this a band-stop filter was applied to the epilepsy data for readings between 49-51 Hz, this is appropriate as the mains supply operates at a frequency of 50 Hz. The AD data did not need this filter, as the data given had already been filtered in this way. To do this a Butterworth filter was applied through MATLAB to the data.

When designing the filter, firstly, because the signal is not in a linear phase the filter needed is an IIR (infinite impulse response). Secondly, the Butterworth filter was chosen because this filter can be implemented without any rippling effect in the data, unlike the Chebyshev or Elliptic filters. Due to the narrow band filter the filter order was set to a high value of 20.

Having eliminated any noise from the epilepsy data, both sets of data can be visualised accurately. However, to have a greater understanding of the data, a further filter can be applied to each set to extract the signals for certain frequency bands. The band of frequencies were δ, θ, α, β and γ representing 1-4Hz, 4-8Hz, 8-16Hz, 16-32Hz and 32-50Hz respectively. To do this the data was passed through a band-pass filter selecting only the frequencies from within the half-power frequencies.
By filtering the signals into these selective bands the results for each can inform how the different brain components are effected during an epileptic seizure and in an AD patient. These bands also represent the different brain processes. The brain processes of each band can be identified as θ being memory, β being motor functions, α representing wakefulness and γ and δ representing sensory processing and sleep rhythm respectively. With this information the effects on the bodies functionality can be compared. In terms of the brain components θ is the temporal section of the brain, β is the feedback connections between cortex as well as the basal ganglia, α is the Thalamus, γ is the inhibitory neurons and δ is the hormones. By studying this, it can be made clear which elements of the brain are being activated and to what extent.

Data processing

In order to begin our investigation, we were provided with two sets of EEG data and thus it was necessary to ensure that the data we would be analysing was formatted consistently. The AD data, as supplied as a 64 x 1251 x 40 matrix, sampled at 250Hz, giving roughly 3 minutes and 20 seconds of data which had been divided into 40 sections. The matrix was first reshaped to 2-dimensional, 64 x 50040 matrix to make the data easier to handle and allow for the whole EEG time series to be more easily graphed. It was not necessary to reshape the epilepsy EEG data as it was supplied as a 2-dimensional matrix. As it was collected for research purposes, the AD EEG data consisted of 64 channels, whereas the epilepsy EEG data consisted of only 22 channels. Due to this, channels from each data set had to be compared like for like in order to find a subset of comparable channels from both sets of EEG data. The chosen channels and their respective locations are shown below.

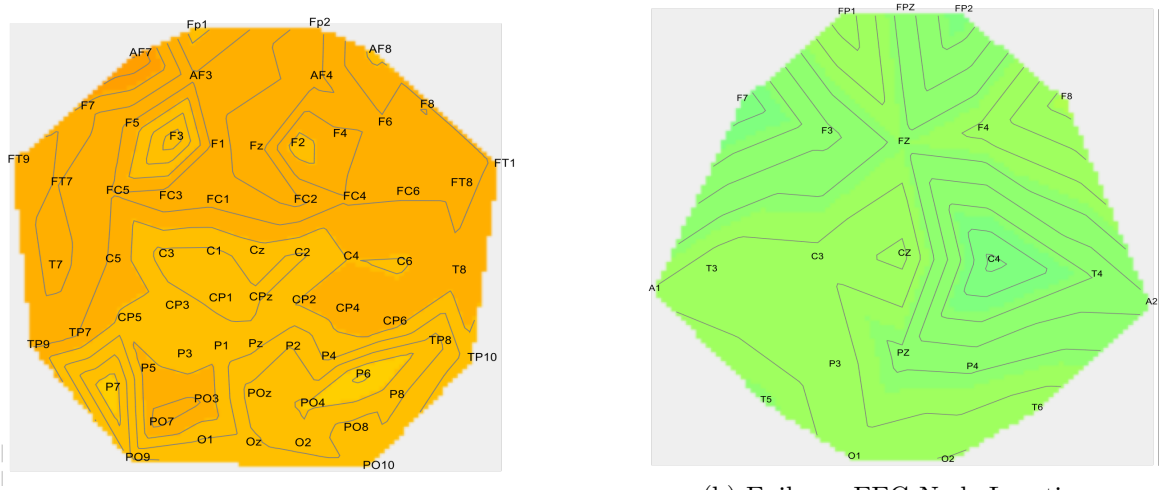
Table 1: Corresponding EEG channels chosen for analysis

Channel	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19
Epilepsy Data	C3	C4	Cz	F3	F4	F7	F8	Fz	FP1	FP2	O1	O2	P3	P4	Pz	T3	T4	T5	T6
AD Data	C3	C4	Cz	F3	F4	F7	F8	Fz	FP1	FP2	O1	O2	P3	P4	Pz	T7	T8	P7	P8

Following this, it was decided that the best approach would be to compare a sample of the AD EEG data against 3 samples from the Epilepsy EEG data (pre-ictal, ictal, post-ictal). Three 90 second

samples were extracted from the epilepsy EEG data shown in Figure 1, the pre-ictal sample ranging from 450 - 540 seconds, ictal ranging from 900 - 990 seconds and post-ictal ranging from 2500 - 2590 seconds. The epilepsy EEG data was sampled at twice the frequency of the AD EEG data, therefore in order to have a comparable amount of data, it was necessary to extract a 180 second sample from the AD EEG data (Figure 2). The 180 second sample was chosen ranging from 10 - 190 seconds as the data series we had been supplied for the AD patient was significantly shorter than that of the epileptic patient.

Figure 3: Channel Locations



(a) Alzheimer's Disease EEG Node Locations

(b) Epilepsy EEG Node Locations

Creating the brain networks

Once the filtering and data processing is complete the data is visualized in the form of 'brain networks' that show the strength of correlations between the receptors/nodes during pre-ictal, ictal and post-ictal periods for each of the frequencies alpha, beta, delta, gamma and theta. The reason for doing this is that one can then see which parts of the brain behave in a similar manner during each period of seizure. The correlations are computed using Matlab's inbuilt linear correlation coefficient function to form a matrix of correlations for each of the pre-/post-/ictal periods for each of the frequencies mentioned above. Then, using these matrices, network graphs are generated and the nodes are placed into the appropriate positions to form a brain structure. The programming of the networks include normalizing the correlation matrices to each other so that the network graphs can be compared, giving those of with a maximum linear correlation an edge weighting of 5 and those with no correlation that of 0. Finally, to reduce over-clustering a threshold is included such that all the edges between the nodes that have edge weights of less than 4 are set to 0.1, so that only the strong correlations are visible. Of course, this threshold can be adjusted if lower correlations wish to be investigated.

To clarify the process an example is provided: Let $\mathbf{A}$ represent the matrix of ictal data collected during a seizure. Then this matrix consists of 19 columns each representing one of the selected nodes (see Table 1) and 45001 rows containing the data collected from the EEG at a certain frequency. The correlation coefficient is a measure of linear dependence between two random variables and here we are applying this to the rows of matrix $\mathbf{A}$, so as to see how correlated the node signals are. The correlation coefficient is determined by this formula:

$$\rho(C_j, C_k) = \frac{1}{N-1} \sum_{i}^{N} \left(\frac{(C_j)_i - (\mu_j)_i}{\sigma_{C_j}} \right) \left(\frac{(C_k)_i - (\mu_k)_i}{\sigma_{C_k}} \right)$$

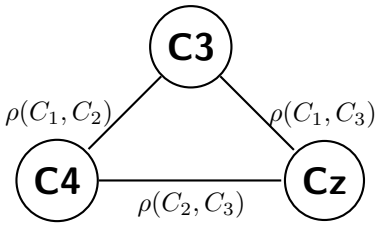
where C_j, C_k are the columns for $j, k = 1...12$. The matrix of the correlations $\mathbf{R}$ which is then formed

looks as follows:

$$\mathbf{R} = \begin{bmatrix} \rho(C_1,C_1) & \rho(C_1,C_2) & \rho(C_1,C3) & \dots \\ \rho(C_2,C_1) & \rho(C_2,C_2) & \rho(C_2,C_3) & \dots \\ \rho(C_3,C_1) & \rho(C_3,C_2) & \rho(C_3,C_3) & \dots \\ \vdots & \vdots & \vdots & \ddots \end{bmatrix} = \begin{bmatrix} 1 & \rho(C_1,C_2) & \rho(C_1,C3) & \dots \\ \rho(C_2,C_1) & 1 & \rho(C_2,C_3) & \dots \\ \rho(C_3,C_1) & \rho(C_3,C_2) & 1 & \dots \\ \vdots & \vdots & \vdots & \ddots \end{bmatrix}$$

Next, an array of nodes is created using the channels from Table 1, $N = C3, C4, Cz, ...$ Each of these nodes behave as an index to a column and row in the matrix $\mathbf{R_{ij}}$, where each element now acts as weighted edge between the nodes. Since the matrix is symmetric one can transform the matrix into an upper or lower triangular matrix to increase computation speed. So for example, if we only consider the first three channels $C3, C4$ and Cz respectively the network formed would be as follows:

$$\begin{array}{c} \\ C_3 \\ C_4 \\ C_z \end{array} \begin{array}{c} \begin{array}{ccc} C_3 & C_4 & C_z \end{array} \\ \begin{pmatrix} 1 & \rho(C_1,C_2) & \rho(C_1,C3) \\ 0 & 1 & \rho(C_2,C_3) \\ 0 & 0 & 1 \end{pmatrix} \end{array} \Rightarrow$$



Since there are 19 channels in total, the generated networks will be very clustered and difficult to infer information from. To overcome this issue, the threshold mentioned afore is added. Furthermore, because the correlation coefficients are bounded between -1 and 1, their absolute value is taken and all coefficients are multiplied by a factor of 5 to make the weighting more visible in the network diagrams. Finally, in order to be able to compare the networks at different frequencies and at different stages of seizure, the weighted edges need to be normalised. This is achieved by finding the maximum correlation coefficient value of all networks and then dividing each weighted edge by that value.

Results

The following plots show the network graphs generated for the frequencies alpha, beta, delta, gamma and theta during the Epilepsy patient's pre-ictal and ictal and post-ictal seizure stages. The edges are weighted and this is shown on the graphs both through increased line width and colour coding, whereby dark red indicates a very large correlation between the nodes and dark blue indicates the opposite.

Figure 4: Brain Activity for Delta frequency 1 - 4Hz

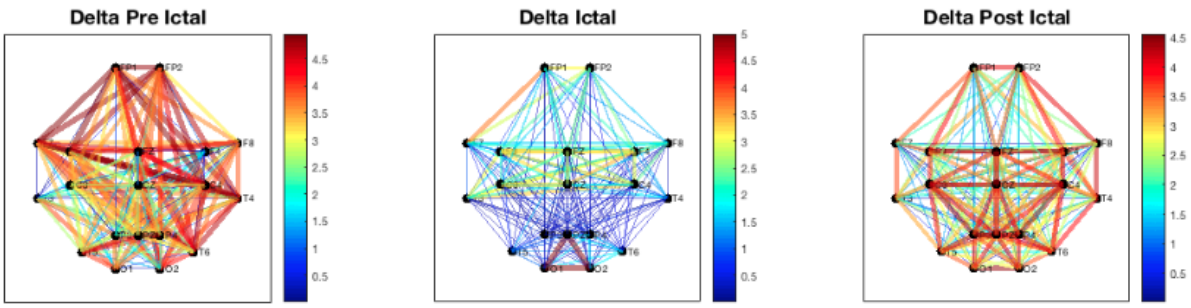
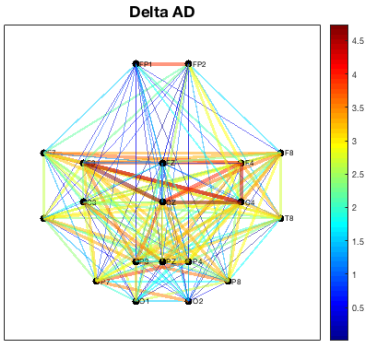


Figure 5: Alzheimer's Disease correlation network for Delta frequencies



The delta frequency range is the lowest range of frequencies found within the human brain, however they are the frequencies with the highest amplitude. In healthy adults, delta wave activity usually begins to appear in Stage 3 of Non-rapid eye movement (NREM) sleep and by Stage 4 of NREM sleep almost all spectral activity is dominated by delta waves [4]. Delta waves are often associated with suspended external awareness, and so are characteristic of deep sleep or in more extreme cases, a coma. It is generally accepted that delta waves are absent from a healthy adult's waking EEG, however coginative impairment is, in some instances, also a known cause of waking EEG delta wave detection [5]. Figure 4 shows the inter-nodal correlations, and as can be seen, the pre- and post-ictal networks show significant correlation across large areas of the brain. Pre-ictal analysis shows that there are strong correlations in the anterior and medial regions of the brain, whilst post-ictal correlations seem more focussed around the medial to anterior regions. This does not necessarily imply that there is a lot of pre- and post-ictal delta wave activity, rather, this may indicative of there being little to no delta wave activity across large portions of the brain which would contribute to many edges within the network being heavily correlated, despite the magnitude of the values assigned to each edge being small, due to the nature of the linear correlation used to calculate the correlations assigned to each weighted edge.

Figure 6: Brain Activity for Theta frequency 4 - 8Hz

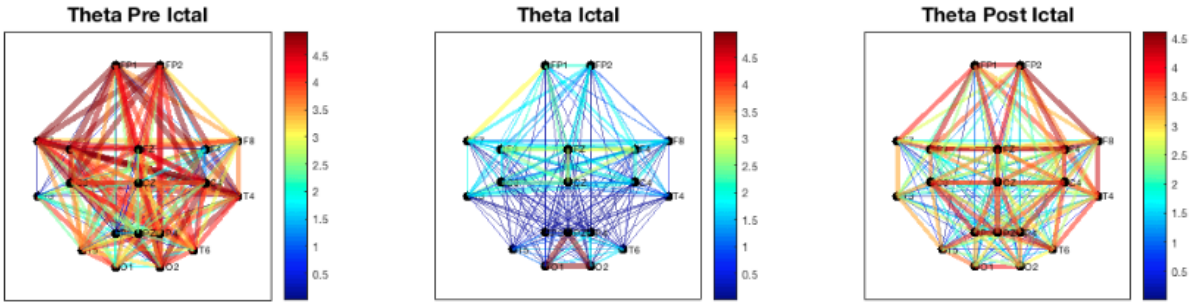
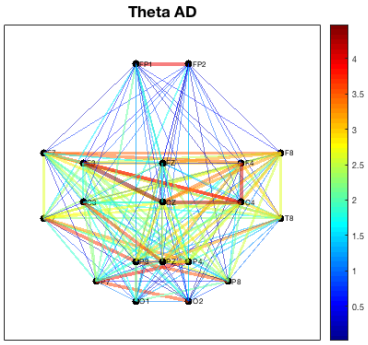


Figure 7: Alzheimer's Disease correlation network for Theta frequencies



The theta band with a frequency of 4-8 Hz is associated with memory and emotional regulation. Theta activity in the brain is seen to occur in arousal and deep meditation but most often in drowsiness where one is in the state of waking or falling asleep[6]. Theta activity also occurs in daydreaming where tasks become so automatic that one can mentally disengage from them. Where alpha waves dominates in EEG of adult humans, theta dominates in the EEG of non human mammals[7]. The results display heavy correlation during the pre-ictal period with little correlation occurring in ictal. The cause of the greater activity in theta waves during pre-ictal could be a result of the patient feeling tired of drowsy until ictal of the seizure where theta waves deplete due to more active thoughts leading to dispersions of other brain frequency bands.

Figure 8: Brain Activity for Alpha frequency 8 - 16Hz

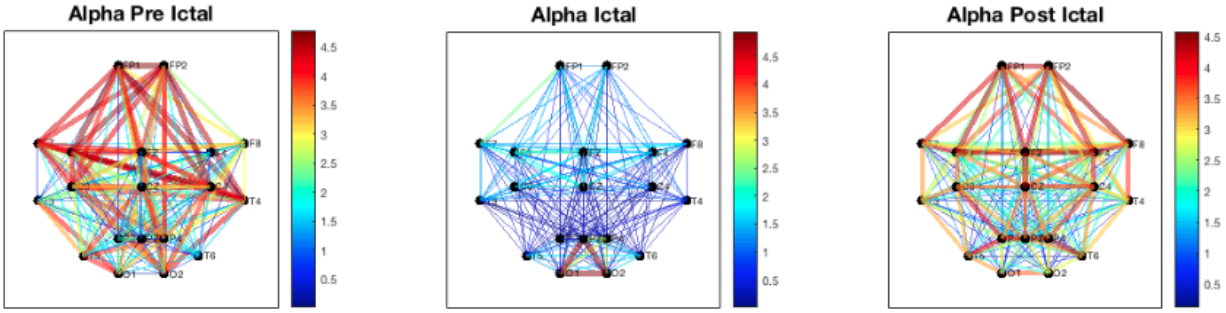
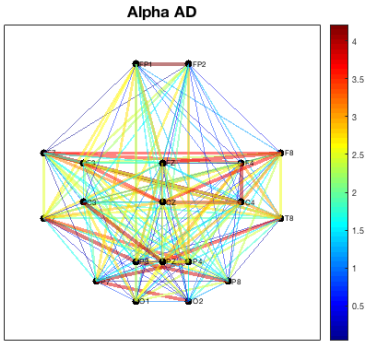


Figure 9: Alzheimer's Disease correlation network for Alpha frequencies



In the literature surrounding brain activity and brain waves collected from EEG data, the alpha frequency is said to be associated with wakefulness and to predominantly originate from the occipital lobe [8]. The occipital lobe is located at the back of the brain and is responsible for visual processing [9]. The receptors on the EEG that pick up signals from this region are primarily O1 and O2.

Furthermore, the amplitude of the alpha frequency is said to be enhanced by internal tasks, such as mental calculations and working memory [10]. The results show large correlations between the data collected for receptors/nodes during the pre-ictal period, and more of the correlations seem to occur with the right hemisphere of the occipital lobe. During seizure (ictal) the main correlations are focused to the occipital region of the brain, from which one could perhaps conclude that the occipital lobe, and thereby visual processing is largely effected. Post seizure, the correlations seem to occur primarily in the upper half of the brain (the frontal lobe). Interestingly the correlations during the post-ictal and ictal seizure seem almost symmetric, while during pre-ictal seizure they do not. Together the results appear to show a dispersion of the alpha waves, then a concentration during seizure and then a dispersion again. In relation to the AD correlation network at alpha frequency (Figure 9), it is difficult to identify any general similarities to the networks of the Epilepsy patient at the same frequency. The correlations between the nodes for the AD patient appear scattered and chaotic, and are, be it inconclusive, most similar to that of the Epilepsy patient during pre-ictal seizure.

Figure 10: Brain Activity for Beta frequency 16 - 32Hz

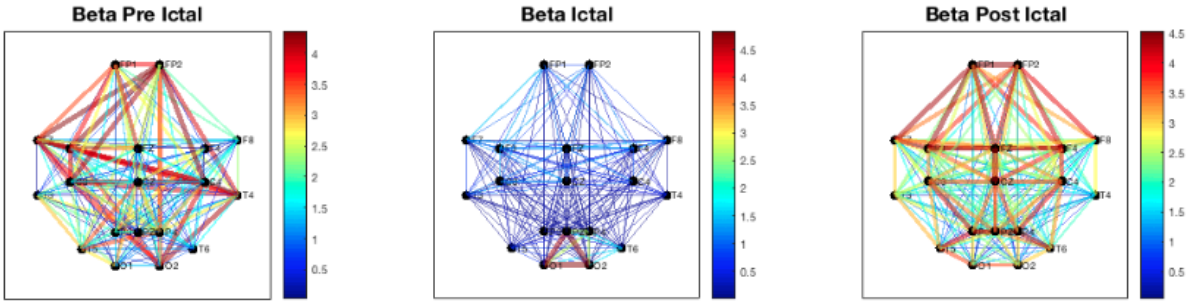
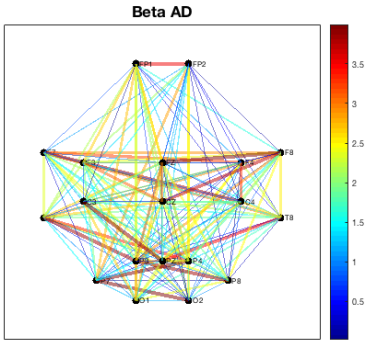


Figure 11: Alzheimer's Disease correlation network for Beta frequencies



The beta band has a frequency between 12-30 Hz and is usually separated into further sub-bands however this was not measured for the purpose of the model used in this project. The beta band is associated with sensorimotor behaviour and affects activities such as active, task-oriented, busy or anxious thinking and active concentration[6]. Beta waves over the motor cortex are suppressed prior and during voluntary muscle movements but after there are bursts of beta waves. The brain however can respond the same way when one imagines or observes movement without any muscular activity[7]. The results show more beta activity pre-ictal and post-ictal compared to ictal as seen in the other frequency bands. During pre-ictal there seems to be larger correlation in the left hemisphere of the brain whereas ictal and post-ictal are seen to be more symmetric. During the ictal period the main correlation is at the back of the head in the occipital region whereas post-ictal most of the correlation occurs in the frontal lobe of the brain.

Figure 12: Brain Activity for Gamma frequency 32 - 50Hz

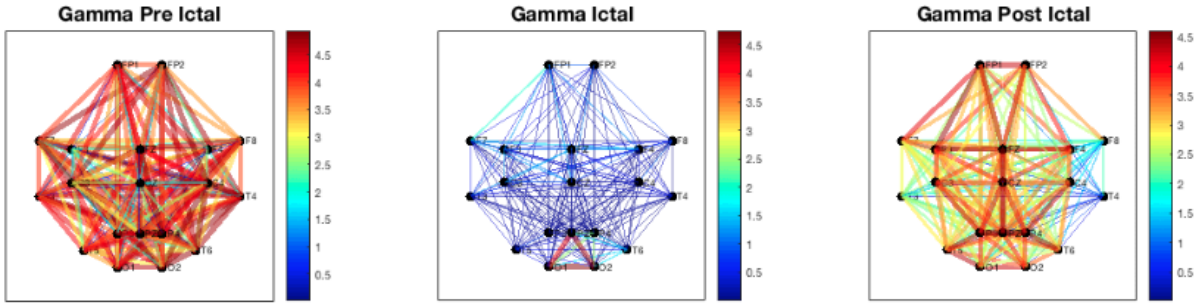
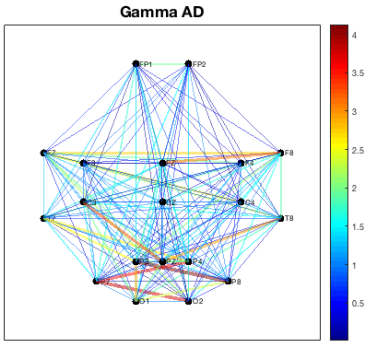


Figure 13: Alzheimer's Disease correlation network for Alpha frequencies



The gamma frequencies of 32-50 Hz, as previously mentioned, when considering brain functionality, is predominantly associated with sensory processing. It is commonly known that sensory inputs can be particularly influential in ictogenesis [11], and this is seen by the strong links in the gamma pre-ictal graph. In comparison to the other pre-ictal graphs, the brain is transmitting signals of stronger links across the brain, putting emphasis on the influx of sensory processing leading up to a seizure. In the gamma ictal graph this abundance of brain signals being transmitted suddenly reduces from the most active frequency band to one of the least animated. This gamma band represents the inhibitory neurons in the brain structure, so by revealing such a lack of transmission, it is clear that during the seizure these inhibitory neurons are not functioning as expected. These inhibitory neurons, when activated, hyperpolarize the postsynaptic neurons, making it harder for the neurons to reach the threshold to fire an action potential causing the intended inhibition [12]. This inhibition is clearly not occurring during the ictal stage, allowing constant unbounded excitation. The correlation network of the AD data shows that the channels have neither strong nor weak correlations. This is most like the ictal stage in the gamma band however there is an absence of these strong correlations from front to back seen after the epileptic seizure.

Correlation Probability Analysis

Figure 14: Probability of Correlation levels throughout the epilepsy seizure within the Alpha bands

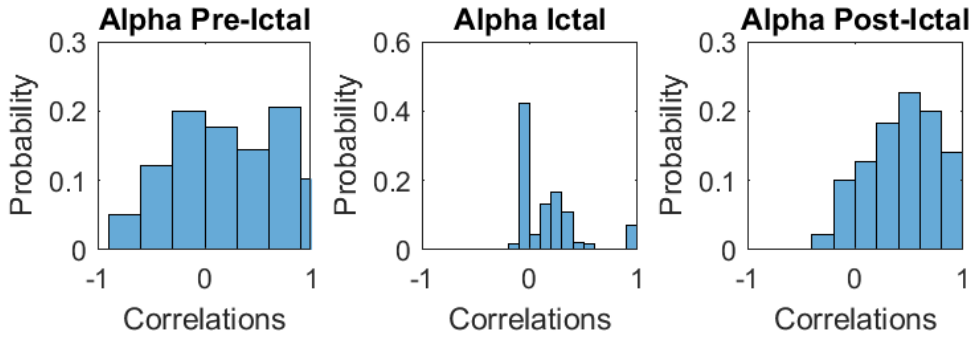


Figure 15: Probability of Correlation levels throughout the epilepsy seizure within the Beta bands

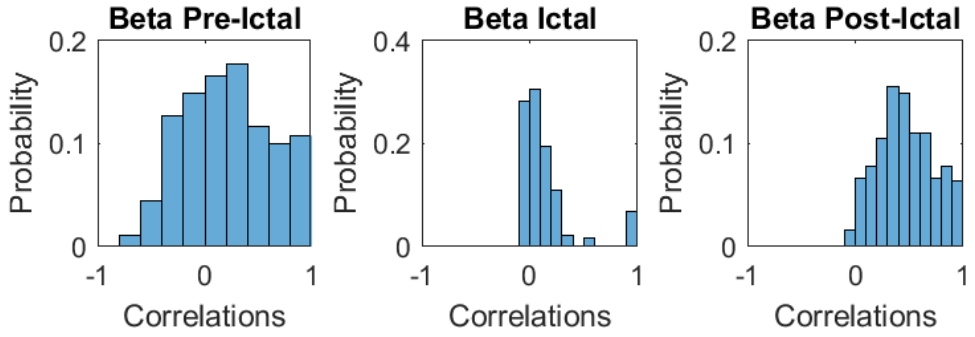


Figure 16: Probability of Correlation levels throughout the epilepsy seizure within the Gamma bands

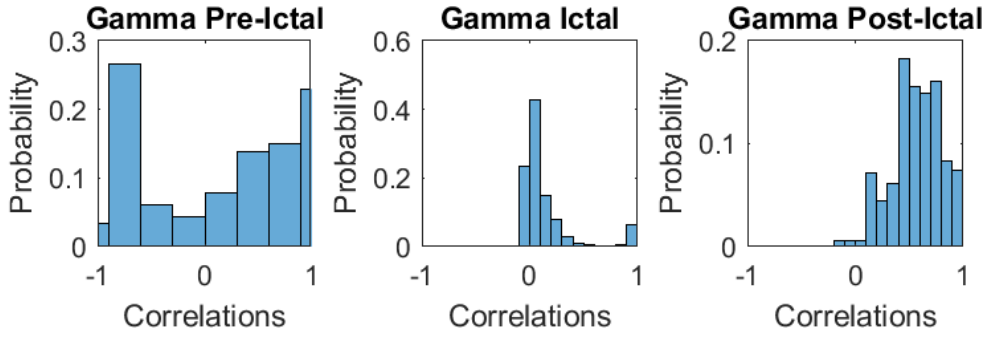


Figure 17: Probability of Correlation levels throughout the epilepsy seizure within the Delta bands

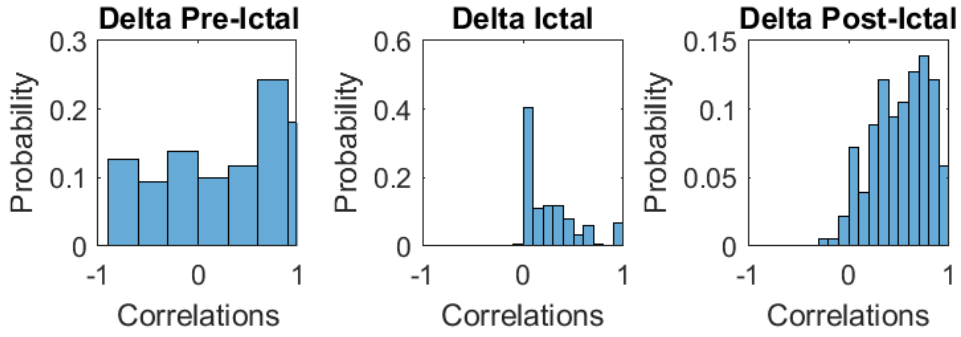
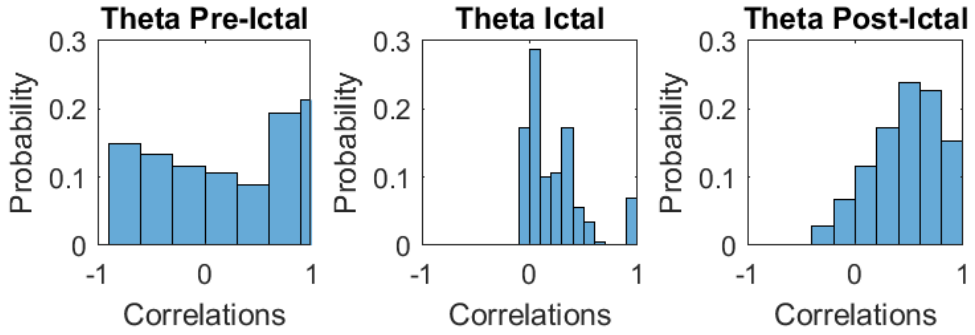


Figure 18: Probability of Correlation levels throughout the epilepsy seizure within the Theta bands



Having produced these histograms, it is clear to see there are obvious similarities between the bands throughout the ictal and post-ictal stages. In the pre-ictal time frame the probability of correlation levels vary across the whole spectrum from -1 to 1. The pattern of this probability distribution is completely dependant on the frequency band with little to no similarities between them. From the Beta band revealing an almost Gaussian distribution to the Delta band which shows a relatively level uniform distribution, not one of these pre-ictal histograms are the same. This reveals that before the seizure the brain is functioning with selected desired other sections. However, as mentioned, at the ictal and post-ictal periods there are obvious contingent features. Throughout each of the ictal histograms there are two evident similarities. Firstly, there are almost no negative correlations between nodes. This is a stark difference from the pre-ictal stage, as the correlations were previously more widely spread. Another observation is that the nodes seem to be largely uncorrelated, with dramatic spikes in the probability of a given correlation of around zero. This suggests that during the seizure the signals from nodes are not dependant on the signals from the rest of the nodes. The post-ictal histograms again present the fact that there is a negligible portion of negative correlations between nodes. In addition, in most of the graphs, a negatively skewed Gaussian distribution can be observed. With peaks of between 0.4 and 0.9, after a seizure, on average the nodes are highly correlated. This contrasts with the ictal stage of the seizure as instead of each node acting independently, now the signals from each part of the brain are fluctuating simultaneously in a similar fashion.

Figure 19: Probability of Correlation levels in the AD patient within the Alpha, Beta and Gamma bands

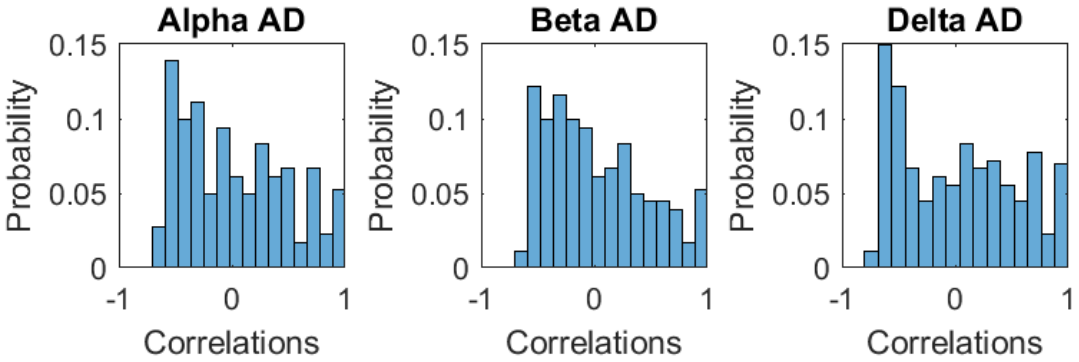
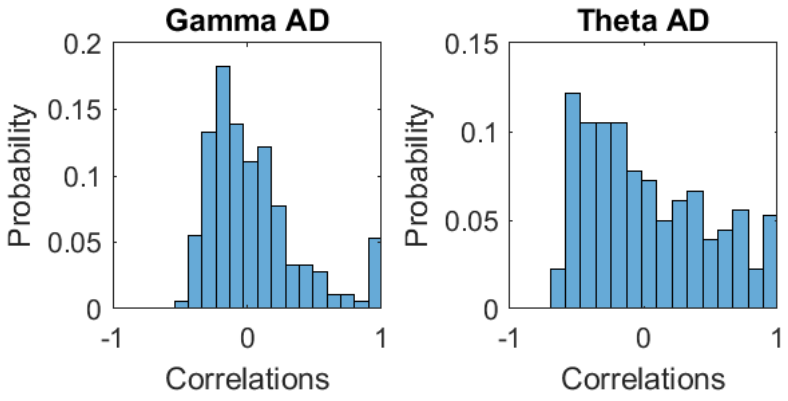


Figure 20: Probability of Correlation levels in the AD patient within the Gamma and Theta bands



Within the AD histograms, again, there are clear similarities that are maintained throughout each of the bands. Each band roughly shows an initial negative spike followed by a shallower slope down. These histograms draw resemblances with each of the different periods in a epileptic seizure. Firstly, these span almost the entirety of the possible values of correlation from around -0.8 up to 1. This is similar to the pre-ictal stage where, although unlike in the AD graphs there are reoccurring features, the irregular patterns of the correlation probability covering the whole spectrum.
Another comparison to make is that, just as in the ictal stages, there is this initial spike of probability, whereas unlike the ictal stage of the seizure there is a gradient up to the spike then a much less steep gradient following. This resembles the post-ictal stage with this Gaussian distribution. In the AD histograms the distribution is strongly positively skewed, as oppose to being negatively skewed after the seizure. This curve represents an average correlation within the mid-range negatives however with such a shallow slope following, a lot of the probabilities is still within the positive correlation making it just as likely for the two nodes to be positively correlated.

Conclusion

Generally, the network analysis shows that the most correlated brain activity occurs prior to seizure. This seems to suggest that highly synchronous brain activity, found across all frequency bands is indicative of an impending seizure. However, to the limitations in the data provided, it was not possible to test this conclusion in its entirety. In order to test this, data collected significantly prior to the initiation of a seizure would be required to determine whether synchrony and correlations increases as a seizure episode approaches. The most synchronous frequency band prior to seizure seems to be the gamma band and this is consistent, to some extent with the notion of the large correlations being indicative of an impending seizure as the gamma band is associated with sensory processing. During seizure it is evident that across all frequency bands, the synchrony is focussed in the occipital region of the brain, indicating a disturbance to visual processing during the seizure. From the EEG plot it is clear to see the period over which the seizure takes place as it is characterised by the large spike in the EEG data. It would be expected that this large spike would correspond to significant correlations and widespread synchrony across the brain network, however the network diagrams suggest that during the seizure, brain activity is erratic as there are a large range of correlations across the network, as can be seen by the wide variety of colours, particularly in the ictal representations for the Delta and Theta frequency ranges (Figures 4 and 6). Unfortunately, we were only able to match 19 corresponding data channels from the two sets of EEG data and thus it was not possible to find correlations across the whole brain network. As a result of this, some correlations may not have been taken into consideration and this could contribute to the lack of convincing evidence for a link between the brain activity during an epileptic seizure and that of a patient with AD. Furthermore, the correlations calculated for the given networks were calculated using Matlab's inbuilt linear correlation coefficient function. Since this function only considers linear correlations, any other non-linear correlations would not be considered by the function meaning that there may be relationships in the AD and Epileptic patients' data which are not considered in the analysis. Overall, we were mostly unsuccessful in finding a direct link

between hyperexcitability associated with epileptic seizures and Alzheimer's Disease, however we can not dismiss the theory that there are links between the two conditions due to the limitations associated with the data provided. Given more time and more patient data to test, we may have been able to find more conclusive results.

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