

Thesis Paper

Predicting Epileptic Seizure from
Electroencephalography (EEG) using Hilbert Huang
Transformation and Neural Network

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Statement

We hereby proclaim that this thesis is based on the results we found by our hard work. Contents of the work found by other researcher(s) are motioned by references. This thesis has never been previously submitted for any degree neither in whole nor in part.

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Chapter 1: Abstract

Epilepsy is the most common chronic disease which involves 1% of the population in the world. It is a neurological disorder characterized by irregular brain tissue activity causing seizure. For many patients mental stress not knowing when seizure occurs is more than having the disease. In some cases physicians inspect lengthy EEG data visually for seizure prediction which involves a lot of time and sometimes tend towards faulty diagnosis. But *decision support systems* are used since 1960 by many physicians and in this paper we are trying to apply modern signal processing and machine learning techniques to improve the accuracy of these decision support systems. *Hilbert Huang Transform* (HHT) is a very new and powerful tool for analyzing data from non-stationary and nonlinear processing realm and capable of filtering data based on *empirical mode decomposition* (EMD). The EMD is based on the sequential extraction of energy associated with various intrinsic time scales of the signal; therefore total sum of the intrinsic mode functions (IMFs) matches the signal very well and ensures completeness. Artificial neural networks (ANNs) offer many potentially superior method of EEG signal analysis to the spectral analysis methods. In contrast to the conventional spectral analysis methods, ANNs not only model the signal, but also make a decision as to the class of signal [26–29]. Feed-forward neural networks are a basic type of ANNs capable of approximating generic classes of functions, including continuous and integrable ones. An important class of feed-forward neural networks is multilayer perceptron neural networks (MLPNNs). In this paper we proposed a model for predicting epileptic seizure using EMD for features extraction and MLPNN for classification.

Chapter 2: Introduction

2.1 Motivation for the thesis

Epileptic seizure is a group of disorders characterized by recurrent discharge from the cerebral cortex that result in irregular disturbance of brain function. According to the Epilepsia journal [1], epileptic seizure is defined as “*An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain*”. Each year, 2.4 million new cases are estimated to occur globally [2]. In most of the adult patients, it occurs in the mesial temporal structures such as hippocampus, amygdala, and Para hippocampal gyros [3]. Among them, approximately 30-40% suffers from medication-refractory epilepsy, and may require surgical measures for either curative or palliative therapy. For those individuals, the prospect of experiencing unpredictable seizures during daily activity can be harrowing. A significant amount of research investigations have been recently pursued that focus on techniques that predict seizures prior to their onset. This is of marked value to patients with refractory epilepsy, and may allow them to take preparatory steps to protect themselves from injury or to attempt to take medication preventively.

Electroencephalogram (EEG) is highly complex signal, containing a lot of information about the human brain function and neurological disorders and measurements of brain electrical activity with EEG have long been one of the most valuable sources of information for epilepsy research and diagnosis, yet this rich resource may still be underutilized. EEG carries a large amount of complex information that is valuable in detecting ongoing seizures. The communication in the brain cells take place through electrical impulses. It is measured by placing tiny electrodes on the scalp of the subject. The cortical nerve cell inhibitory and excitatory postsynaptic potentials generate the EEG signals. These postsynaptic potentials summate in the cortex and extend to the scalp surface where they are recorded as EEG.

The EEG records can easily display these electrical discharges as a rapid change in potential differences in real time. Thus, neurologists invariably use EEG records to investigate suspected seizure phenomena [1-7, 9-25]. Traditionally this job is done by an expert neurologist using a visual scanning of the EEG signals which is a time consuming process and may be inaccurate. These inaccuracies are particularly significant for long time duration EEG signals [4]. For this reason, an automated system for seizure prediction system can be very helpful and optimize reduce the volume of the data to be observed. The system work by transforming the mostly qualitative diagnostic criteria into a more objective quantitative signal feature classification problem and needs to be highly sensitive so that it can determine the false seizure and discard them [5]. The purpose of the system does not replace the neurologist, rather to relieve him off the burden of time consuming observation by providing alarms. For that reason, we are developing a system which can predict the upcoming seizure so that the patient can get some time to take necessary preparation so that he/she can avoid heavy work or take medicine to keep him/her functional.

2.2 Research Guideline

In our paper, we propose a feature extraction methodology for the classification of EEG signals involving four stages.

- a. The first stage of our work involves *down-sample* [41] the raw EEG data from 5000Hz to 200Hz so that we can reduce the data size to free some memory (our raw EEG data size is 56.2 GB) and speed up overall process and use a *bandpass filter* [3] to remove the unnecessary noise (like power line noise etc.) to extract the relevant features from the EEG signals.
- b. The second stage of the algorithm involves the Hilbert Huang Transformation and for that, we firstly calculate EMD which decomposes signal $x(t)$ into a number of

oscillatory components which is known as intrinsic mode function (IMF) through a shifting process of the EEG signals and giving a set of IMFs. We select first ten IMFs for further processing.

- c. The third stage involves feature extraction which is done by calculating the *statistical* and *spectral* characteristics of the IMFs, which is the main contribution of this paper. For the calculation of statistical features, we have used autocorrelation, mean, variance and skewness of the IMFs and for spectral features, we have used spectral centroid, variation coefficient, spectral skew from the Hilbert transformed IMFs as this transformation can remove the DC offset from the spectral content of the signals which is one of the sources of non-stationary in the signals.
- d. The fourth stage involves the use of Artificial Neural Network (ANN) for the classification of EEG signals.

The paper is organized as follows: We will describe the related works on this aspect (**Chapter 3**) followed by the dataset used in this paper (**Chapter 4**) and after that we describe our proposed framework for classifying the EEG signals (**Chapter 5**). Later, we present our experimental results (**Chapter 6**) and conclude the paper (**Chapter 8**). A comprehensive work diagram is given bellow.

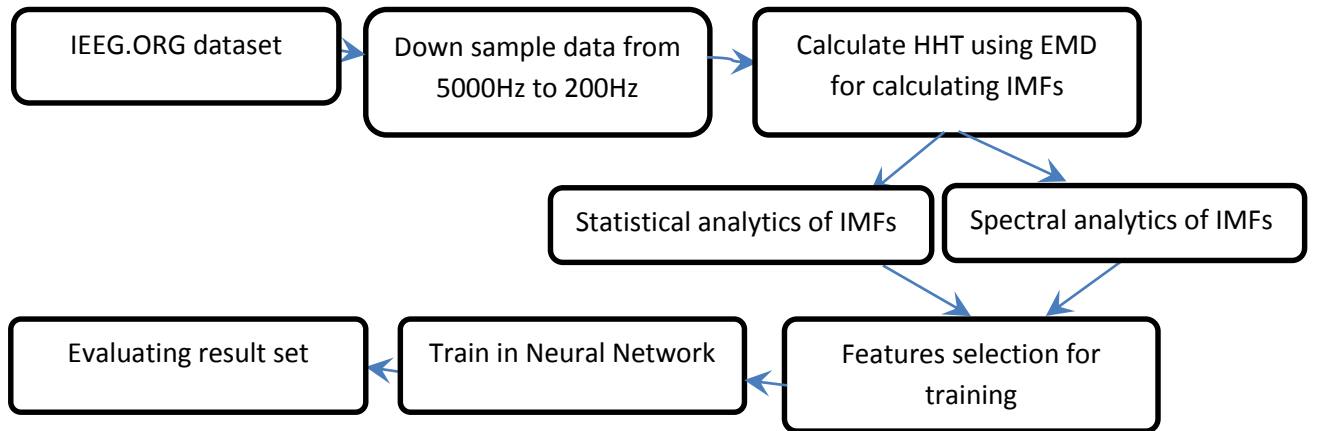


Fig.1: Flowchart of the proposed seizure prediction system.

Chapter 3: Related Works

3.1 What is epilepsy?

The epilepsy is a chronic neurological disorder in which clusters of nerve cells, or neurons, in the brain sometimes signal abnormally and cause seizures. Neurons normally generate electrical and chemical signals that act on other neurons, glands, and muscles to produce human thoughts, feelings, and actions. During a seizure, many neurons fire (signal) at the same time – as many as 500 times a second, much faster than normal. This surge of excessive electrical activity happening at the same time causes involuntary movements, sensations, emotions, and behaviors and the temporary disturbance of normal neuronal activity may cause a loss of awareness. Epilepsy can be considered a spectrum disorder because of its different causes, different seizure types, its ability to vary in severity and impact from person to person, and its range of co-existing conditions. Some people may have *convulsions* (sudden onset of repetitive general contraction of muscles) and lose consciousness. Others may simply stop what they are doing, have a brief lapse of awareness, and stare into space for a short period. Some people have seizures very infrequently, while other people may experience hundreds of seizures each day. There also are many different types of epilepsy, resulting from a variety of causes. Recent adoption of the term “the epilepsies” underscores the diversity of types and causes.

In general, a person is not considered to have epilepsy until he or she has had two or more unprovoked seizures separated by at least 24 hours. In contrast, a provoked seizure is one caused by a known precipitating factor such as a high fever, nervous system infections, acute traumatic brain injury, or fluctuations in blood sugar or electrolyte levels.

Anyone can develop epilepsy. About 2.3 million adults and more than 450,000 children and adolescents in the United States currently live with epilepsy. Each year, an estimated 150,000 people are diagnosed with epilepsy. Epilepsy affects both males and females of all races, ethnic

backgrounds, and ages. In the United States alone, the annual costs associated with the epilepsies are estimated to be \$15.5 billion in direct medical expenses and lost or reduced earnings and productivity. The majority of those diagnosed with epilepsy have seizures that can be controlled with drug therapies and surgery. However, as much as 30 to 40 percent of people with epilepsy continue to have seizures because available treatments do not completely control their seizures (called *intractable* or medication resistant epilepsy).

While many forms of epilepsy require lifelong treatment to control the seizures, for some people the seizures eventually go away. The odds of becoming seizure-free are not as good for adults or for children with severe epilepsy syndromes, but it is possible that seizures may decrease or even stop over time. This is more likely if the epilepsy starts in childhood, has been well-controlled by medication, or if the person has had surgery to remove the brain focus of the abnormal cell firing. Many people with epilepsy lead productive lives, but some will be severely impacted by their epilepsy. Medical and research advances in the past two decades have led to a better understanding of the epilepsies and seizures. More than 20 different medications and a variety of dietary treatments and surgical techniques (including two devices) are now available and may provide good control of seizures. Devices can modulate brain activity to decrease seizure frequency. Advance neuroimaging can identify brain abnormalities that give rise to seizures which can be cured by neurosurgery. Even dietary changes can effectively treat certain types of epilepsy. Research on the underlying causes of the epilepsies, including identification of genes for some forms of epilepsy, has led to a greatly improved understanding of these disorders that may lead to more effective treatments or even to new ways of preventing epilepsy in the future.

3.2 What causes the epilepsy?

The epilepsies have many possible causes, but for up to half of people with epilepsy a cause is not known. In other cases, the epilepsies are clearly linked to genetic factors, developmental brain abnormalities, infection, traumatic brain injury, stroke, brain tumors, or other identifiable problems. Anything that disturbs the normal pattern of neuronal activity – from illness to brain damage to abnormal brain development – can lead to seizures.

The epilepsies may develop because of an abnormality in brain wiring, an imbalance of nerve signaling in the brain (in which some cells either over-excite or over-inhibit other brain cells from sending messages), or some combination of these factors. In some pediatric conditions abnormal brain wiring causes other problems such as intellectual impairment.

In other persons, the brain's attempts to repair itself after a head injury, stroke, or other problem may inadvertently generate abnormal nerve connections that lead to epilepsy. Brain malformations and abnormalities in brain wiring that occur during brain development also may disturb neuronal activity and lead to epilepsy.

3.2.1 Genetics:

Genetic mutations may play a key role in the development of certain epilepsies. Many types of epilepsy affect multiple blood-related family members, pointing to a strong inherited genetic component. In other cases, gene mutations may occur spontaneously and contribute to development of epilepsy in people with no family history of the disorder (called “*de novo*” mutations). Overall, researchers estimate that hundreds of genes could play a role in the disorders.

Several types of epilepsy have been linked to mutations in genes that provide instructions for ion channels, the "gates" that control the flow of ions in and out of cells to help regulate

neuronal signaling. For example, most infants with *Dravet syndrome*, a type of epilepsy associated with seizures that begin before the age of one year, carry a mutation in the SCN1A gene that causes seizures by affecting sodium ion channels.

Genetic mutations also have been linked to disorders known as the *progressive myoclonic epilepsies*, which are characterized by ultra-quick muscle contractions (myoclonus) and seizures over time. For example, *Lafora disease*, a severe, progressive form of myoclonic epilepsy that begins in childhood, has been linked to a gene that helps to break down carbohydrates in brain cells. Mutations in genes that control neuronal migration – a critical step in brain development – can lead to areas of misplaced or abnormally formed neurons, called cortical dysplasia, in the brain that can cause these mis-wired neurons to misfire and lead to epilepsy. Other genetic mutations may not cause epilepsy, but may influence the disorder in other ways. For example, one study showed that many people with certain forms of epilepsy have an abnormally active version of a gene that results in resistance to anti-seizure drugs. Genes also may control a person's susceptibility to seizures, or *seizure threshold*, by affecting brain development.

3.2.2 Other Disorders which causes epilepsies:

Epilepsies may develop as a result of brain damage associated with many types of conditions that disrupt normal brain activity. Seizures may stop once these conditions are treated and resolved. However, the chances of becoming seizure-free after the primary disorder is treated are uncertain and vary depending on the type of disorder, the brain region that is affected, and how much brain damage occurred prior to treatment. Examples of conditions that can lead to epilepsy include:

- Brain tumors, including those associated with neurofibromatosis or tuberous sclerosis complex, two inherited conditions that cause benign tumors called hamartomas to grow in the brain
- Head trauma
- Alcoholism or alcohol withdrawal
- Alzheimer's disease
- Strokes, heart attacks, and other conditions that deprive the brain of oxygen (a significant portion of new-onset epilepsy in elderly people is due to stroke or other cerebrovascular disease)
- Abnormal blood vessel formation (arteriovenous malformations) or bleeding in the brain (hemorrhage)
- Inflammation of the brain
- Infections such as meningitis, HIV, and viral encephalitis

Cerebral palsy or other developmental neurological abnormalities may also be associated with epilepsy. About 20 percent of seizures in children can be attributed to developmental neurological conditions. Epilepsies often co-occur in people with abnormalities of brain development or other neurodevelopmental disorders. Seizures are more common, for example, among individuals with autism spectrum disorder or intellectual impairment. In one study, fully a third of children with autism spectrum disorder had treatment-resistant epilepsy.

3.3 Definitions of the Epilepsy classifications:

The classification of etiologies of the epilepsies is divided into four main categories, which are given bellow.

a. Idiopathic epilepsy:

Defined as epilepsy of predominately genetic or presumed genetic origin and in which there is no gross neuro-anatomic or neuro-pathologic abnormality. Included here are epilepsies of presumed multiagency or complex inheritance, but for which currently the genetic basis has not been elucidated.

b. Symptomatic epilepsy:

Defined as epilepsy of an acquired or genetic cause; associated with gross anatomic or pathologic abnormalities, and/or clinical features, indicative of underlying disease or condition. We thus include in this category developmental and congenital disorders where these are associated with cerebral pathologic changes, whether genetic or acquired (or indeed cryptogenic) in origin. Also included are single gene and other genetic disorders in which epilepsy is only one feature of a broader phenotype with other cerebral or systemic effects.

c. Provoked epilepsy:

Defined as epilepsy in which a specific systemic or environmental factor is the predominant cause of the seizures and in which there are no gross causative neuro-anatomic or neuro-pathologic changes. Some “provoked epilepsies” will have a genetic basis and some an acquired basis, but in many no inherent cause can be identified. The reflex epilepsies are included in this category (which are usually genetic) as well as the epilepsies with a marked seizure precipitant.

d. Cryptogenic epilepsy:

Defined as epilepsy of presumed symptomatic nature in which the cause has not been identified. The number of such cases is diminishing, but currently this is still an important category, accounting for at least 40% of adult-onset cases of epilepsy.

3.4 Treatment for epileptic seizures:

Once epilepsy is diagnosed, it is important to begin treatment as soon as possible. For about 70 percent of those diagnosed with epilepsy, seizures can be controlled with modern medicines and surgical techniques. Some drugs are more effective for specific types of seizures. An individual with seizures, particularly those that are not easily controlled, may want to see a neurologist specifically trained to treat epilepsy. In some children, special diets may help to control seizures when medications are either not effective or cause serious side effects.

While epilepsy cannot be cured, for some people the seizures can be controlled with medication, diet, devices, and/or surgery. Most seizures do not cause brain damage, but ongoing uncontrolled seizures may cause brain damage. It is not uncommon for people with epilepsy, especially children, to develop behavioral and emotional problems in conjunction with seizures. Issues may also arise as a result of the stigma attached to having epilepsy, which can lead to embarrassment and frustration or bullying, teasing, or avoidance in school and other social settings. For many people with epilepsy, the risk of seizures restricts their independence (some states refuse drivers licenses to people with epilepsy) and recreational activities.

3.5 Can Epileptic Seizures Be Predicted?

Recent multi-center clinical studies showed evidence of premonitory symptoms in 6.2% of 500 epileptic patients [30]. Another interview-based study found that 50% of 562 patients felt “auras” before seizures [31]. These clinical observations give an incentive to search for premonitory changes on Electroencephalographic (EEG) recordings from the brain. The epileptic brain consecutively transitions through different states of activity: from normal interictal (far from seizures) to preictal (minutes or sometimes hours before the seizure) then ictal (seizure) and postictal, before returning to the interictal state [32]. Despite the current lack

of a complete neurological understanding of the preictal brain state, which is patient and condition specific, researchers increasingly hypothesize that brainwave synchronization patterns might differentiate interictal, preictal and ictal states [33]. The specific seizure prediction task thus becomes a classification problem where one aims at discriminating between interictal and preictal patterns of brain activity [32]. Ictal and postictal states are discarded from the classification because the task is not to detect undergoing seizures, but to warn the patient or clinician about future ones [32].

3.6 Reviews on Epilepsy prediction:

Many prediction works is done so far using various signal processing methods and each of them different from another. As a whole, we can divide the prediction task into two part; firstly processing the EEG data for feature extraction, then use those features to predict seizure analyzing patients EEG recording. For EEG signal classification, parameters extracted from the EEG signals using various signal processing methods are very useful for diagnostics seizure related cases. Here we describe some selected works which performs well at prediction.

3.6.1 Fourier transform

Spectral parameters based on the Fourier transform are useful for analyzing the EEG signals and have shown good results on their classification [6], [7]. However, it is important to note that the Fourier domain does not exhibit any time-domain characteristics in the signal giving the features which are suboptimal for feature extraction from some signal processing scenarios [8].

3.6.2 Short time Fourier transforms

Several time-frequency domain based methods have been developed for detection of epileptic seizure from EEG signals. These methods include the short time Fourier transform (STFT) [9] where Characteristics of the spectrogram and scalogram are studied in order to get more insight in how these transforms might be useful to derive suitable features for myoclonic seizure detection.

3.6.3 Wavelet transform

Discrete wavelet transforms (DWT) analysis and approximate entropy (ApEn) of EEG signals is another popular method for seizure detection and it is performed in two stages. In the first stage, EEG signals are decomposed by DWT to calculate approximation and detail coefficients. In the second stage, Approximation Entropy (ApEn) values of the approximation and detail coefficients are calculated. Significant differences have been found between the ApEn values of the epileptic and the normal EEG allowing us to detect seizures with 100 % classification accuracy using artificial neural network [10]. In [11], Discrete Wavelet Transform (DWT) with the Multi-Resolution Analysis (MRA) is applied to decompose EEG signal at resolution levels of the components of the EEG signal and the Parseval's theorem are employed to extract the percentage distribution of energy features of the EEG signal at different resolution levels.

3.6.4 Multi-wavelet transform

Multi-wavelets, which contain several scaling and wavelet functions, offer orthogonally, symmetry and short support simultaneously, which is not possible for scalar wavelet.

Multi-wavelet is considered as the generalization of scalar wavelet. However, some important differences exist between these two types of wavelet. Specifically, scalar wavelets only have a single scaling and wavelet function, whereas multi-wavelets have two or more scaling and wavelet functions and with these functions, they classifies seizure quite well[12].

Although good results are obtained using these methods, the STFT does not yield a multi-resolution analysis of the signals. This is because of the fact that the STFT uses the filters of the same bandwidth for signal decomposition at all frequencies. This limitation is typically resolved using the wavelet analysis in which a multi-resolution time-frequency analysis is facilitated by forming band pass filters with varying bandwidths [25].

3.6.5 Discrete wavelet transforms

The sub-band frequencies of the sub-band signals obtained from the DWT have been used as a feature for classification of normal and seizure EEG signals [13]. The line length feature of the sub-band decomposed signals obtained by using DWT has been used for classification of healthy and epileptic seizure EEG signals [14]. The approximate entropy in conjunction with autoregressive model parameters extracted from the Fourier transforms of the EEG signals is employed in linear and nonlinear classifiers [15].

3.7 Recent methodologies for seizure prediction:

3.7.1 Empirical mode decomposition (EMD)

More recently, new techniques for the analysis of non-stationary and nonlinear signals have been proposed which are mainly based on empirical mode decomposition (EMD) [16]. The EMD is a time-frequency based method which decomposes signals into a number of intrinsic mode functions (IMF) which are oscillatory components. This characteristic of EMD has motivated the researchers to use it for the analysis of EEG signals.

In [17], The EMD decomposes an EEG signal into a finite set of band-limited signals termed intrinsic mode functions (IMFs). The mean frequency (MF) for each IMF has been computed using Fourier-Bessel expansion. The MF measure of the IMFs has been used as a feature in

order to identify the difference between ictal and seizure-free intracranial EEG signals. It has been shown that the MF feature of the IMFs has provided statistically significant difference between ictal and seizure-free EEG signals. In [18], the weighted frequency has been used as a feature of IMFs for the classification between healthy and epileptic seizure EEG signals.

3.7.2 Multivariate EMD

After applying Hilbert-Huang Transform in the EEG data, it facilitated the extraction of the EEG intrinsic modes as well as the eventual EEG frequency/energy content analysis. The analysis of the frequency and energy content of every extracted mode has been performed via Hilbert Transform, which was achieved through the tracking of the instantaneous frequencies and amplitudes. Hilbert weighted frequency has been used to help discriminate between healthy and seizure EEG patterns [18, 19].

3.7.3 Higher Order Statistics in EMD

In [20], higher order statistics such as variance, skewness, and kurtosis are utilized for classifying the EEG signals in the EMD for detecting seizure and epilepsy. The appropriateness of these moments in distinguishing the EEG signals is investigated through an extensive analysis in the EMD domain. Besides the strengths of feature extraction methods related to instantaneous frequencies (IF), it is important to note that the extraction of IF is more meaningful when the IMFs extracted from the EEG signals are mono-component.

3.7.4 Hilbert Huang Transform with Bayesian Classifier

In [22], after preprocessing of intracranial EEG data for removing noise and obtain data segments for sliding window analysis, feature extraction was done using Hilbert-Huang transform. Feature selection using correlation based feature selection algorithm, binary

classification by Bayesian networks, and a simple post-processing algorithm to remove spurious detections, thus they got better performance for big number of EEG recordings.

3.7.5 Phase space representation of IMFs

In this paper [23], we have proposed the new features based on the phase space representation (PSR) for classification of epileptic seizure and seizure-free EEG signals. The EEG signals are firstly decomposed using empirical mode decomposition (EMD) and phase space has been reconstructed for obtained intrinsic mode functions (IMFs). For the purpose of classification of epileptic seizure and seizure-free EEG signals, two-dimensional (2D) and three-dimensional (3D) PSRs have been used. New features based on the 2D and 3D PSRs of IMFs have been proposed for classification of epileptic seizure and seizure-free EEG signals. These measured parameters show significant difference between epileptic seizure and seizure-free EEG signals. The combination of these measured parameters for different IMFs has been utilized to form the feature set for classification of epileptic seizure EEG signals.

3.7.6 Ngram-Derived Pattern Recognition

In this paper [24], the presented approach specifically applies Ngram-based pattern recognition. After data pre-processing with similarity metrics, including the Hamming distance and Needleman-Wunsch algorithm for identifying unique patterns within epochs of time where pattern counts within each epoch are used as measures to determine seizure detection and prediction markers.

According to the paper [25] Scale-free dynamics of the intracerebral EEG are quantified through robust estimates of the scaling exponents—the first cumulants—derived from a wavelet leader and bootstrap based multifractal analysis. The cumulants are investigated for the discriminability between preictal and interictal epochs. Patient-specific seizure prediction

method uses a set of three measures, namely the *persistence*, *distance* and *inclusion*, referred to as *state similarity measures*, which quantify the similarity between the states underlying iEEG epochs and a reference state underlying the immediate preictal (90s) iEEG epoch. The state similarity measures are derived from the two dimensional thermodynamic profiles of the iEEG epochs obtained from time-series of wavelet energy and entropy calculated using the wavelet transform modulus maxima method (WTMM). Here the prediction method use cumulants individually and in combination with state similarity measures as feature sets to predict seizures, with no change to the parameters used in training and testing procedures of the original algorithm.

Comprehensive studies on recent methods with dataset names are given bellow to summarize the whole related work under an umbrella.

Reference	Method	Used Dataset	Objective	Year	Accuracy/ Specificity
Alam et al. [20]	EMD statistics + ANN	University of Bonn, 173.61Hz sampling rate	Epileptic Seizure	2013	100%
Bajaj et al. [10]	EMD + SVM	University of Bonn, 173.61Hz sampling rate	Seizure	2012	99.50%
Oweis et al. [18]	Hilbert Huang transform + Clustering	University of Bonn, 173.61Hz sampling rate	Seizure	2011	94%
Nilufer et al. [21]	Hilbert Huang transform + Bayesian Classifiers	University of Freiburg EEG database, 256Hz sampling rate	Epileptic Seizure	2014	96.55%
Rajeev et al. [22]	EMD + 2D,3D Phase Space Representation	University of Bonn, 173.61Hz sampling rate	Seizure	2014	98.67%

Reference	Method	Used Dataset	Objective	Year	Accuracy/ Specificity
Eftekhari et al. [23]	Ngram-Derived pattern recognition algorithm	University of Freiburg EEG database, 256Hz sampling rate	Epileptic Seizure	2014	75.16%
Gadhoumi et al. [24]	wavelet leader based scaling + cumulant estimation	Montreal neurological Institute, 2000Hz sampling rate	Epileptic Seizure	2015	80.5%

Figure: 2.0 Comparison among methods with dataset name of recent approaches to predict seizure

Chapter 4: Dataset

To understand data we have to understand the classification of data and which data we have chosen. There is emerging evidence that the temporal dynamics of brain activity can be classified into 4 states: Interictal (between seizures, or baseline), Preictal (prior to seizure), Ictal (seizure), and Post-ictal (after seizures). Seizure forecasting requires the ability to reliably identify a preictal state that can be differentiated from the interictal, ictal, and postictal state. The primary challenge in seizure forecasting is differentiating between the preictal and interictal states. So we need three kind of data one is interictal EEG clip second one is preictal EEG clip and finally test EEG clip. In this paper we have used raw EEG data obtained from ieeg.org through an international competition at [kaggle.com](https://www.kaggle.com). In our datasets from different patients with epilepsy undergoing EEG monitoring to identify a region of brain that can be resected to prevent future seizures. These datasets have varying numbers of electrodes and are sampled at 5000 Hz, with recorded voltages referenced to an electrode outside the brain. Each preictal clips contains ten minutes of EEG recording covering an hour prior to seizure and other clips have the recording of interictal activity. Total size of the data is 56.2 GB. Each file is in .mat format which consists of recording time, sampling frequency and channel information along with matrix of EEG clip. The dimension of the matrix is 3 million columns and 24 rows and each row represents the EEG clips from different channel and each column represents the amplitude of the signal on that particular time. Our challenge is to use this data and distinguish between ictal and preictal data.

Chapter 5: Proposed system

5.1 Decimation:

As our data sampled at a very high frequency rate about 5000 Hz for that the data is too big for computation and will slow out our overall process. There are many process of decimation but we will use decimation by an integer factor as we want to down sample our signal into 200Hz by the factor of 25. Down sampling can be done by two stages

1. Apply low-pass filter to the signal
2. Resample the filtered data by selecting every M^{th} point

Let our signal is $x(t)$ and we convert it to sequence of n samples which is $x[n]$, in decimation we start with a discrete-time signal $x[n]$ and convert it into another discrete-time signal $y[n]$. Thus, the formal definition of M -fold decimation, or down-sampling, is defined by Equation 1.0[1]. In decimation, the sampling rate is reduced from F_s to F_s/M by discarding $M - 1$ samples for every M samples in the original sequence.

$$y[n] = v[nM] = \sum_{k=-\infty}^{\infty} h[k]x[nM - k] \quad \dots \dots 1.0$$

The block diagram notation of the decimation process is depicted in Figure 3.0

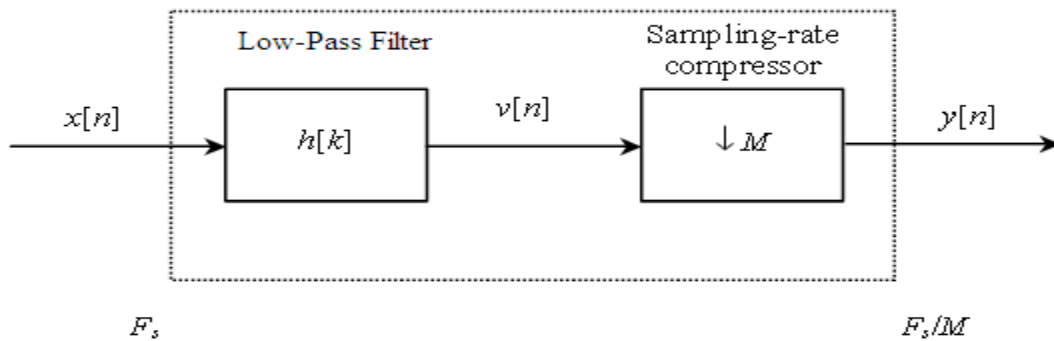


Figure 3.0: Block diagram notation of decimation, by a factor of M .

Figure 3.1 shows the first 0.2 seconds of raw EEG data sampled at 5000 Hz and figure 1.2 shows first 0.2 seconds of down sampled 200Hz of EEG

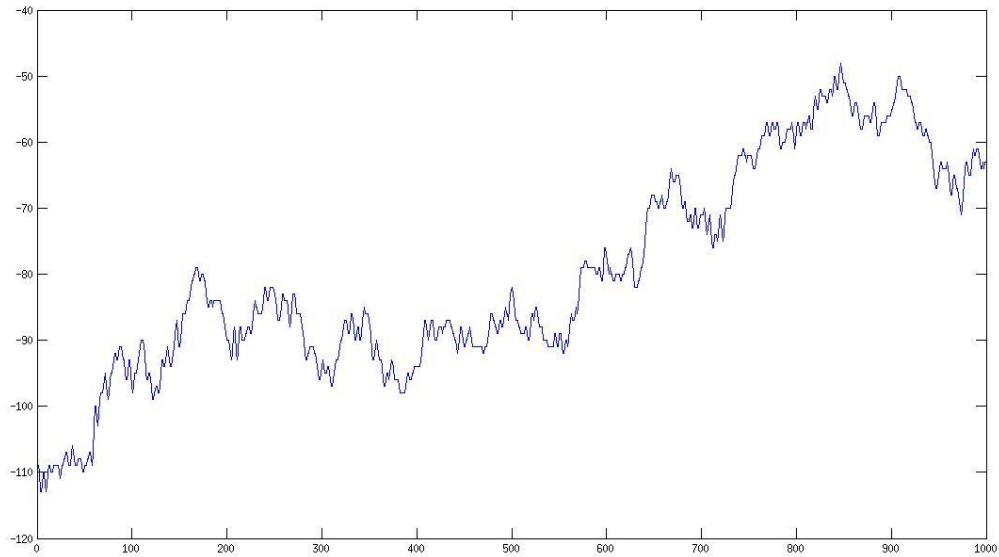


Figure 3.1: first 0.2 seconds of raw EEG data

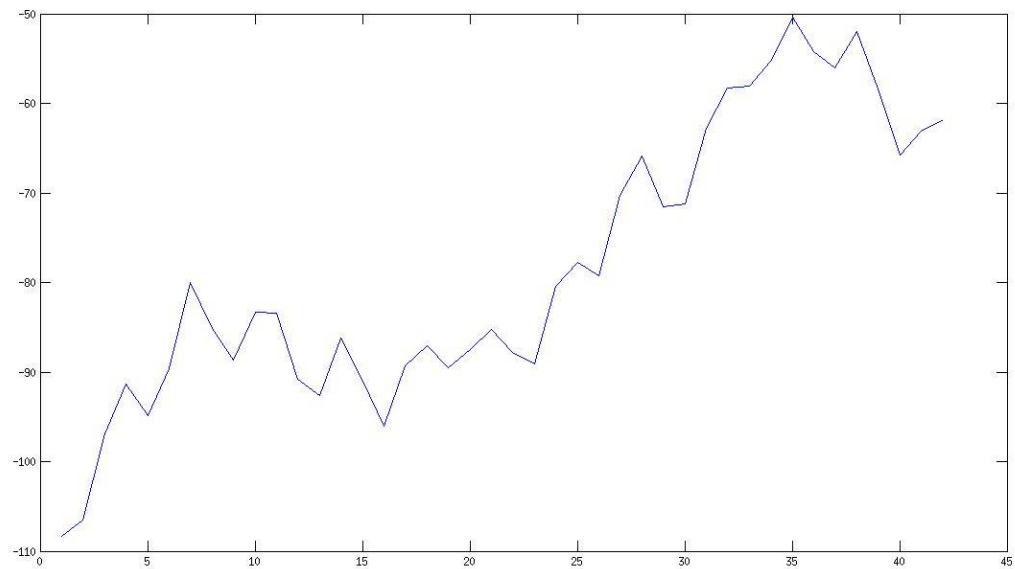


Figure 3.2: first 0.2 seconds of down sampled EEG data

5. 2 Band-Pass Filter:

The real EEG data Lies between [0.1 – 60Hz] [3] and frequencies greater than that are considered power line noise. To get better feature out of these signals we have to apply band-pass filter to the signal at [0.1 – 60Hz]. We can design our band pass by two ways one by cascading which involves apply two filters to the signal first is low-pass and second one is high-pass filter. Second technique is to multiply low-pass and high-pass filter by convolution and apply the impulse response to the signal. Figure 3.3 threes shows cascading method and figure 3.4 shows single stage method.

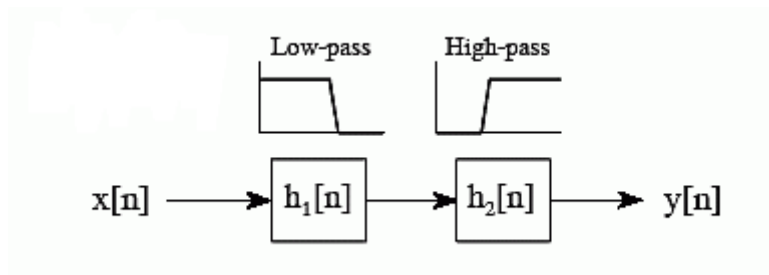


Figure 3.3: Low-Pass filter using cascading stage

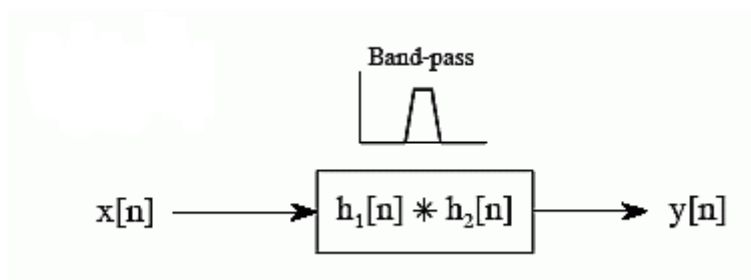


Figure 3.4: Low-Pass filter using single stage

Figure 3.5 shows EEG signal after applying Band-Pass filter

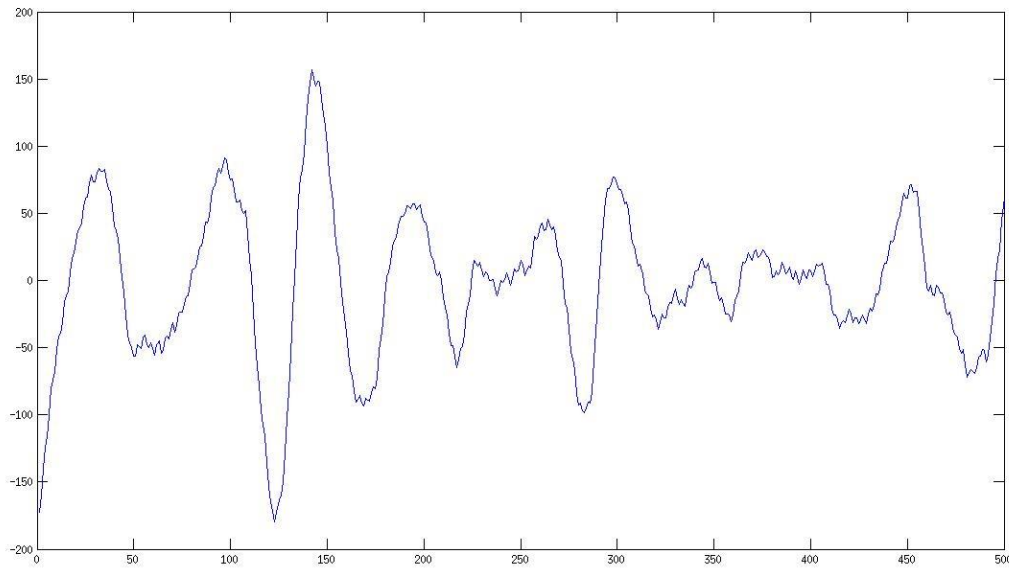


Figure 3.5 EEG data after applying Band-Pass filter

5.3 Hilbert-Huang Transformation:

The Hilbert–Huang transform (HHT) is an empirically based data-analysis method. Its basis of expansion is adaptive, so that it can produce physically meaningful representations of data from nonlinear and non-stationary processes [34]. Traditional data-analysis methods are all based on linear and stationary assumptions like Fourier transformation makes assumption of the signal period which creates spectral leakage. HHT’s basis of expansion is adaptive, so that it can produce physically meaningful representations of data from nonlinear and non-stationary processes. HHT was motivated by the need to describe nonlinear distorted waves in detail, along with the variations of these signals that naturally occur in non-stationary processes. As is well known, the natural physical processes are mostly nonlinear and non-stationary like EEG signals from brain, yet the data analysis methods provide very limited options for examining data from such processes. The available methods are either for linear

but non-stationary, or nonlinear but stationary and statistically deterministic processes, as stated above.

5.4 The empirical mode decomposition method:

As discussed by Huang et al. (1996, 1998, 1999), the empirical mode decomposition method is necessary to deal with data from non-stationary and nonlinear processes [35]. EMD decomposes signal $x(t)$ into a number of oscillatory component which is known as intrinsic mode function [main paper] through a shifting process. Each IMF has its own distinct time scale. Furthermore EMD does not consider the stationary and the linearity of the signal [16]. For an input signal $x(t)$ the process of calculating IMFs are given below:

1. Let's set $x(t) = x(t)_{old}$
2. Find all the maxima and minima in $x(t)_{old}$
3. interpolate between minima and maxima using cubic spline interpolation which will generate local maxima envelope $e_m(t)$ and local minima envelope $e_l(t)$
4. Calculate the mean of envelopes $e_m(t)$ and $e_l(t)$ using,

$$e_{mean} = \frac{e_m(t) + e_l(t)}{2}$$

5. Now subtract e_{mean} from $x(t)_{old}$ we will get $x(t)_{new}$ as,

$$x(t)_{new} = x(t)_{old} - e_{mean}$$

6. Now set $x(t)_{old} = x(t)_{new}$
7. repeat the process 2-4 until

$$\text{Standard deviation } sd = \frac{\sum |x(t)_{new} - x(t)_{old}|^2}{\sum x(t)^2_{old}} < \alpha$$

Where α is value between range of 0.2-0.3

The first IMF is defined as $c(t) = x(t)_{new}$ which is the smallest temporal scale of $x(t)$. By subtracting $c(t)$ from $x(t)$ we will get residual signal $r(t)$ which can be expressed as , $r(t) = x(t) - c(t)$. After acquiring $r(t)$ we put it in the same process above to get new IMF which means each IMF will have different frequencies against time. So the original signal can be rewritten like,

$$x(t) = \sum_{m=1}^M c_m(t) + r_M(t)$$

Now we will apply Huang Transformation to each IMF and we will get HHT of each channel.

Figure 4.1 – 5.0 represents the first ten IMFs of channel one of patient's preictal EEG data.

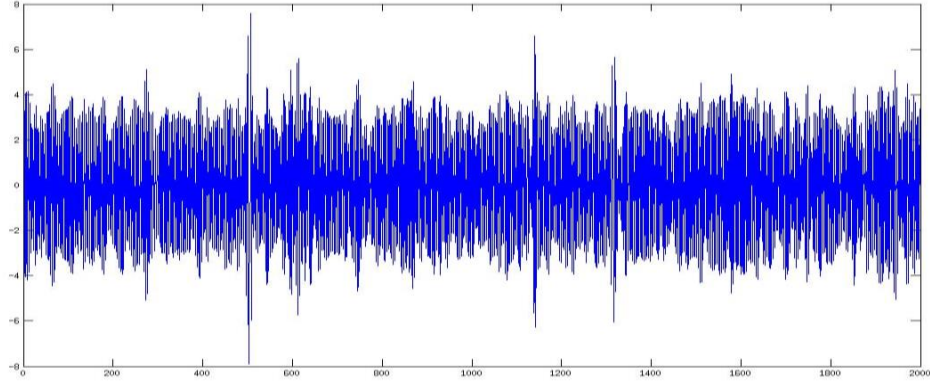


Figure 4.1 **First** IMF of the signal

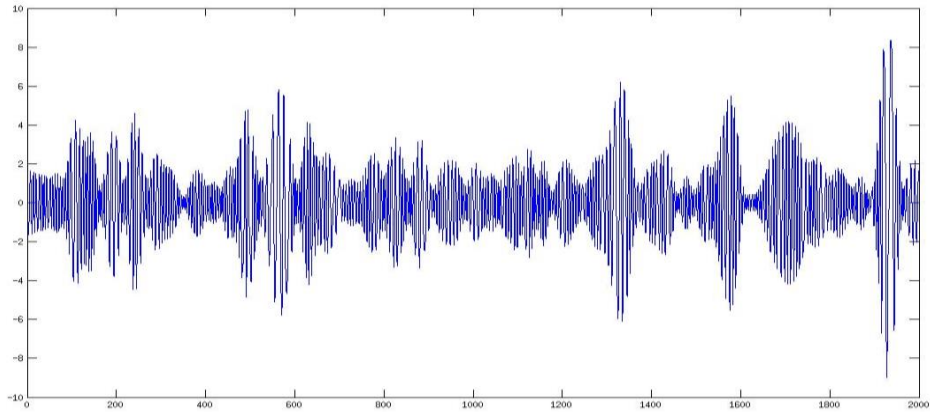


Figure 4.2: **Second** IMF of the signal

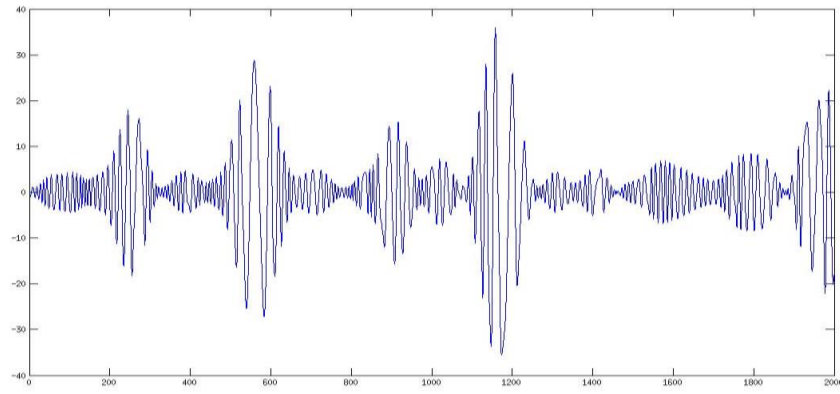


Figure 4.3: **Third** IMF of the signal

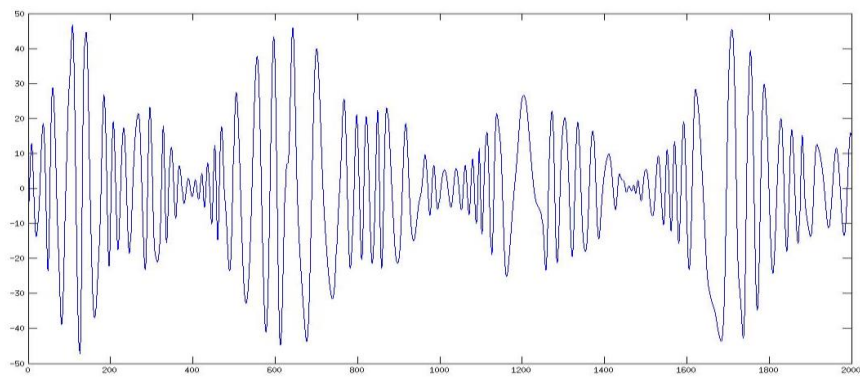


Figure 4.4: **Forth** IMF of the signal

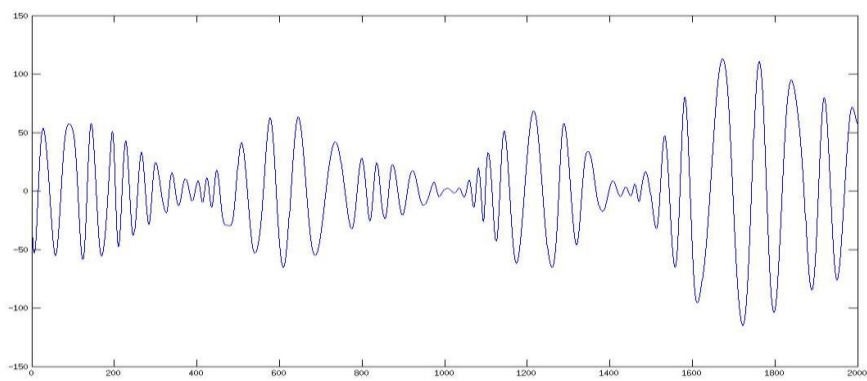


Figure 4.5: **Fifth** IMF of the signal

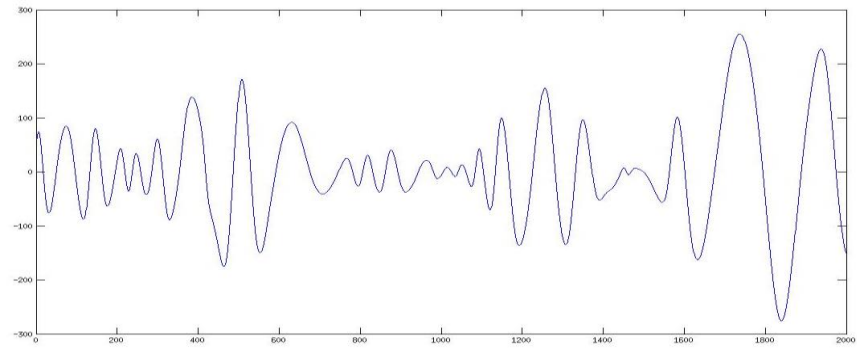


Figure 4.6: **Sixth** IMF of the signal

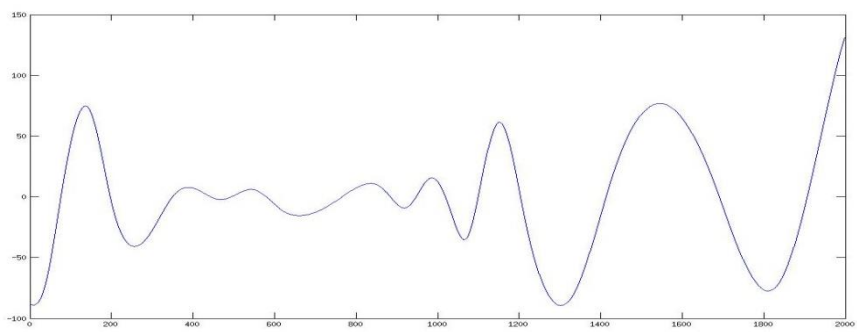


Figure 4.7: **Seventh** IMF of the signal

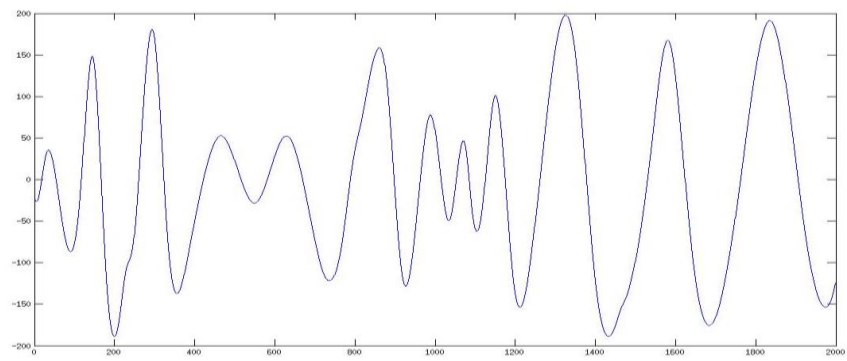


Figure 4.8: **Eight** IMF of the signal

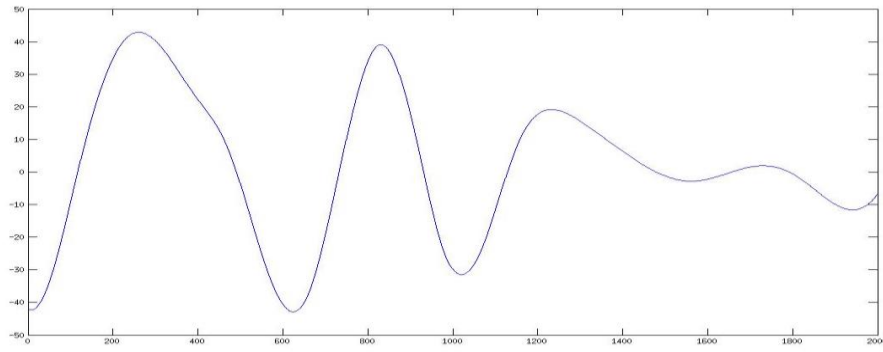


Figure 4.9: **Ninth** IMF of the signal

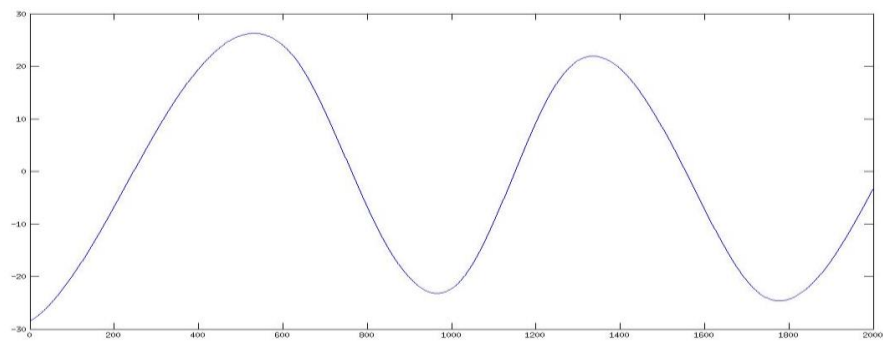


Figure 5.0: **Tenth** IMF of the signal

5.5 Statistical Analysis:

Studies shows that we can discriminate between normal EEG data and abnormal EEG by statistically analyzing the IMF. Their statistical use is motivated by the fact that the distributions of samples in the data are characterized by their asymmetry, dispersion and concentration around the mean. After analyzing visually we can see IMF obtained from normal and pathological EEG are quite different from one another. These differences can easily extracted by statistical methods.

5.5.1 Autocorrelation:

Autocorrelation can calculate non- randomness in the data and it can also calculate the right time series if the data is not random. To calculate autocorrelation we have to use the following equation

$$r_k = \frac{\sum_{i=1}^{N-k} (x(t_i) - \bar{X})(x(t_{i+k}) - \bar{X})}{\sum_{i=1}^{N-k} (x(t_i) - \bar{X})^2}$$

Here r_k is autocorrelation function and k is time lag. $x(t)$ Represents the signal and $\bar{X}$ is the mean of the signal and N is Number of sample.

5.5.2 Mean, Variance and Skewness:

Mean, variance and skewness of each IMF are calculate by the following equations

$$\mu_t = \frac{1}{N} \sum_{i=1}^N x(t_i)$$

$$\sigma_t = \sqrt{\frac{1}{N} \sum_{i=1}^N (x(t_i) - \mu_t)^2}$$

$$\beta_t = \frac{1}{N} \sum_{i=1}^N \left(\frac{x(t_i) - \mu_t}{\sigma_t} \right)^3$$

Where μ_t is mean, σ_t is variance and β_t is skewness of the corresponding IMF.

5.6 Spectral Analysis:

EMD has the ability to perform spectral analysis [36]. EEG signals from brain can create activities in certain frequency bands so frequency based analysis is useful for feature extraction. The main strength of EMD is to decompose a signal into number of patterns (IMF's) which are the responses to filter having narrow pass bands. Spectral features extracted from IMF could give feature that can distinguish between normal and pathological EEG signal.

For spectral analysis we will calculate Spectral Centroid, Variance Coefficient and Spectral Skew and use them as our features

5.6.1 Spectral Centroid:

Centroid frequency can be used as a distinctive feature that can be used for characterization of EEG signals.

$$c_s = \frac{\sum_w p(w)}{\sum_w p(w)}$$

Where $p(w)$ is the amplitude of the w^{th} frequency bin in the spectrum

5.6.2 Variation Coefficient:

Variation coefficient is used as a Characterization of the signal because spectral variation is different for normal EEG and pathological EEG

$$\sigma_s^2 = \frac{\sum_w (w - c_s)^2 p(w)}{\sum_w p(w)}$$

Where σ_s^2 is Variation Coefficient.

5.6.3 Spectral skew:

Skewness is the third order moment and it measures the symmetry and asymmetry of the distribution. Skewness of the power of IMFs for the normal and pathological EEG signals differ from each other. So it can be a useful feature for classification. The equation of spectral skew is given below

$$\beta_s = \frac{\sum_w \left(\frac{w - c_s}{\sigma_s} \right)^3 p(w)}{\sum_w p(w)}$$

Here β_s is the spectral skew.

Our final feature vector will be $F = [\mu_t \ \sigma_t \ \beta_t \ c_s \ \sigma_s \ \beta_s]$

5.7 Neural Network:

Artificial neural network try to mimic human brain and its learning process. To understand neural network we have to know the structure of human brain cell neuron. Figure. 6 [37] shows a simple illustration of neuron.

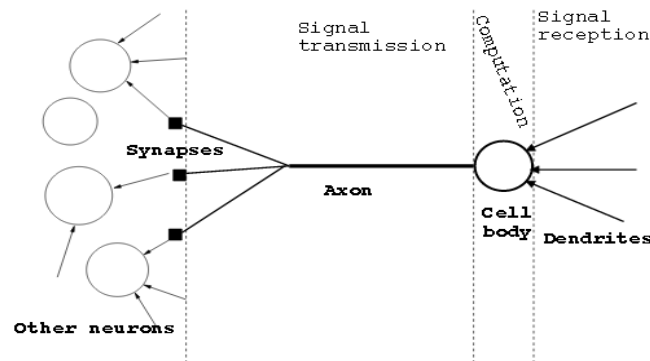


Figure 6.0: A simple illustration of neuron

We estimate around 10^{10} and 10^{11} the number of neurons in the human brain. They receive information from other neurons through their dendrites. Then they process that information in their cell body called soma. They send information from one neuron to another via a cable called axon. The point of connection between the axon branches and other neurons' dendrites are called synapses. The neuron communicates via an action potential which is an electrical impulse that travels through the axon. It generates a "spike" in the electric potential (voltage) of the axon. An action potential is generated at neuron only if it receives enough (over some threshold) of the "right" pattern of spikes from other neurons. Neural network try to mimic those process to make decision. Figure 6.1 shows an illustration of neural network

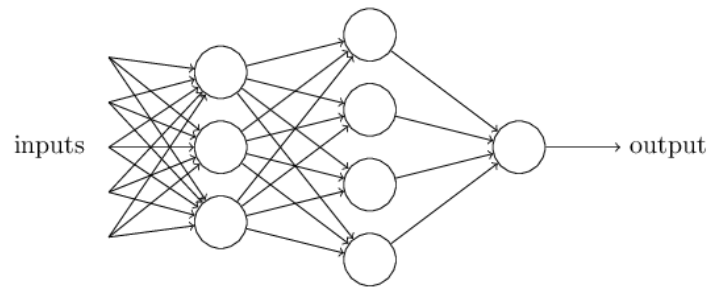


Figure 6.1: An illustration of neural network

The input represents dendrites of the neuron and the 2nd and 3rd layer called hidden layer and last one is output layer. Each node in hidden layer is called and activation functions. They process the information like soma. All the inputs are associated by some weights and these weights can change the value of activation function. The values from activation function are compared by some threshold value by the output layer and display us the appropriate results. Suppose we have our signal as a input vector $[X_1 \ X_2 \ X_3 \ X_4 \dots X_n]$ and there are some weights associated with them are $[W_1 \ W_2 \ W_3 \ W_4 \dots W_n]$ then the pre activation or input function for one activation function will be

$$a(X) = b + \sum x_i w_i$$

Here b is called the bias.

It will be easier for us if we represent the equation above as a vector notation. So our equation will be

$$a(X) = b + XW^T$$

So for single layer neuron our output function will be

$$h(X) = g(a(X)) = g(b + XW^T)$$

Here $g(.)$ is our activation function.

5.7.1 Activation Function:

There are many activation functions available for neural network with a good step function quality.

a. Linear activation function:

It does not perform any input squashing and does approximate a good out put

$$g(a) = a$$

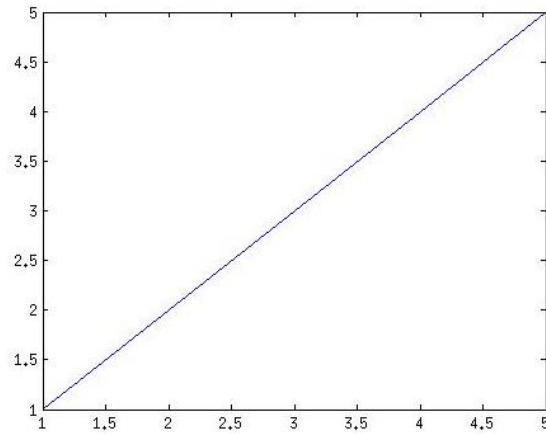


Figure 6.2: Linear activation function

b. Sigmoid Function:

Squashes the neuron's pre-activation between 0 and 1, always positive, bounded and strictly increasing.

$$g(a) = \text{sigm}(a) = \frac{1}{1 + e^{-a}}$$

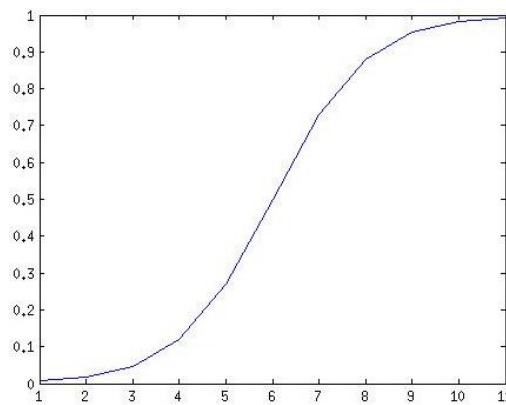


Figure 6.3: Sigmoid function

c. Hyperbolic tangent (“tanh”) activation function:

Squashes the neuron's pre-activation between -1 and 1, can be positive or negative, bounded and strictly increasing.

$$g(a) = \tanh(a) = \frac{e^a - e^{-a}}{e^a + e^{-a}} = \frac{e^{2a} - 1}{e^{2a} + 1}$$

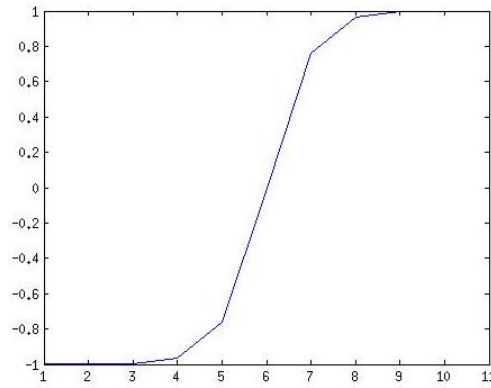


Figure 6.4: hyperbolic tangent activation function

d. Rectified linear activation function:

Bounded below by 0 so always non negative Not upper bounded. Strictly

increasing and tends to give neurons with sparse activities

$$g(a) = \text{reclin}(a) = \max(0, a)$$

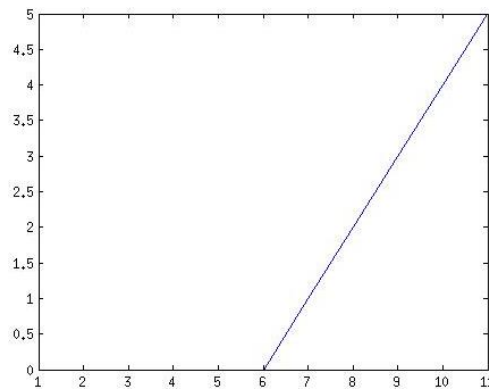


Figure 6.5: rectified linear activation function

In this paper we are going to use sigmoid function as it has a very good step like quality and it is very easy to differentiate and help us to calculate the gradient of the function for optimization.

5.7.2 Cost Function:

What we'd like is an algorithm which lets us find weights and biases so that the output from the network approximates $y(x)$ for all training inputs x . To quantify how well we're achieving this goal we define a cost function

$$C(w, b) = \frac{1}{2n} \sum_x |y(x) - a|^2$$

Here, w denotes the collection of all weights in the network, b all the biases, n is the total number of training inputs, a is the vector of outputs from the network when x is input, and the sum is over all training inputs, x . Here C the quadratic cost function; it's also sometimes known as the mean squared error. If we minimize the cost function then neural network will show better results. To optimize our cost function we used gradient decent.

5.7.3 Gradient Decent:

To minimize cost we have to adjust our weights to optimal. Gradient decent is an algorithm which will help use to re adjust our weights. It is an iterative process and in every iteration the cost function step downwards to global minima and when it reaches the global minima then our classifier is optimized. The gradient decent function for optimizing weights is given below

$$w := w - \alpha \frac{d}{dw} C(w)$$

Here w is the weight and we differentiate the cost function with respect to w . α is called learning rate which is actually how much steps we are going to take at each iteration.

Gradient decent function for optimizing bias is

$$b := b - \alpha \frac{d}{db} C(b)$$

Here b is the bias and we differentiate the cost function with respect to b . To calculate the gradient of the cost function we need to calculate the gradient of each node.

5.7.4 Backpropagation Algorithm:

Backpropagation also known as back propagation of error is an algorithm which is used to calculate the gradient of nodes of the neural network. To calculate the gradient of each node it cleverly differentiates the neural network with chain rule of differentiation. The equation is

$$\frac{\delta}{\delta w_{ij}^l} C(W, b) = \frac{1}{n} \sum_{i=1}^n \frac{\delta}{\delta w_{ij}^l} C(W, b; x^i, y^i)$$

$$\frac{\delta}{\delta b_{ij}^l} C(W, b) = \frac{1}{n} \sum_{i=1}^n \frac{\delta}{\delta b_{ij}^l} C(W, b; x^i, y^i)$$

Here i and j represent the column and row of input matrix, l represents the layer number and represents output layer.

Using the entire concept of neural network stated above we trained our feature vector obtained from each signal.

5.8 Overall processes:

1. Down sample the signal from 5000 Hz to 200 Hz
2. Apply Band-pass filter [1-60Hz]
3. Calculate empirically decomposed IMFs
4. Statistically analyze each IMF to extract feature
5. Apply spectral analysis to each IMF for spectral feature.
6. Feed each feature to neural network with one hidden layer and 100 activation function. For activation we have used sigmoid function with learning rate of 0.25.
7. Train the network and optimize the cost function
8. Find the performance parameter of the result

Chapter 6: Results and discussion

6.1 Performance Evaluation Parameters:

The performance is evaluated by using parameters such as Sensitivity, Specificity, and overall Accuracy which are defined as shown in equations (1), (2) and (3), respectively.

$$\text{Sensitivity} = \frac{TN_{CP}}{TN_{AP}} \dots \dots (1)$$

Where TN_{CP} represents the total number of correctly detected positive patterns;

TN_{AP} represents the total number of actual positive patterns; and a positive pattern indicates a detected seizure.

$$\text{Specificity} = \frac{T_{CN}}{T_{AN}} \dots \dots (2)$$

Where TN_{CN} represents the total number of correctly detected negative patterns; TN_{AN} represents the total number of actual negative patterns; and a negative pattern indicates a detected non-seizure.

$$\text{Overall Accuracy} = \frac{TN_{CDP}}{TN_{APP}} \dots \dots (3)$$

Where TN_{CDP} represents the total number of correctly detected patterns; TN_{APP} represents the total number of applied patterns; and a pattern indicates both seizure and non-seizure.

6.2 Results:

An average probability of prediction or block sensitivity of 56.5% was achieved with an average block false positive rate of 0.201 FP predictions/hour, corresponding to 84.35% specificity. The focus channel, historically used for evaluation in seizure-prediction research, was not selected as the best channel for predicting seizures in any patient but we have selected all the channels. The neural network training program was obtained from neural network toolbox of MATLAB8.4 (2012a). Training was done for a significant amount of time to enhance results, which was possible using GPU calculations which proved to be much faster than using the CPU alone. The calculations were done on a ASUS PC with an Intel Core i7 4770 CPU @ 3.40GHz, 16 GB of memory @ 1600 MHz, and the graphics card used for GPU calculations was an NVIDIA GeForce GTX 780 with 2304 CUDA cores @ 324 MHz, with 2 GB DDR5 of video memory.

6.3 Discussion:

All implanted IEEG channels were used in this study. At first we tried to classify raw EEG data which required lots of computation power as our data size was 56 GB. After classification result was not that good as raw contained a lot of noise and over fit the classifier with lot of unnecessary information. But some other studies researchers found good result using high resolution EEG data using deep belief network [38] but that requires a lot of memory and computational power. But in this paper we try to reduce the memory and computational power as well as speed up the process. In our second we use PCA to reduce dimensionality of our data and after feeding the PCA data to neural network the results are not satisfactory as we observed that PCA is not a good feature PCA from normal EEG and pathological EEG are somewhat similar. Fourier transformation without window was not performing well because of wrong periodic assumption about FFT creates a lot of spectral leakage. After applying some window

with 50% overlaps increase performance at a good level. But in our feature set we also wanted to capture temporal data as well as special data. So we choose discrete wavelet transform for its temporal resolution as it saves the information about location information [39] which Fourier transformation doesn't keep track of. There were little bit of improvement after using DWT. Furthermore we know that natural signal like EEG have nonlinear and non-stationary property and need an algorithm which takes account of these things. We choose recent and relatively new signal processing algorithm which called Hilbert Huang transformation. As Hilbert describe this process is capable of extract relative information from nonlinear and non-stationary signals [40]. This is an empirical method is necessary to deal with non-stationary and nonlinear data as it

Divide the signal into number of oscillatory components called intrinsic mode function which is quite different from normal EEG data from pathological EEG data. So we apply EMD to get IMFS from each signal for further analysis. At first we apply EMD to raw EEG data but taking too much computational time as our signal sampled at a very high frequency rate. For that new need to down sample our data to get faster computational time. After down sampling the data we realize most of EEG signals lie between [1-60HZ] so we apply band-pass filter to it. As EMD is different from normal and pathological EEG we decided to apply some statistical analysis for our feature extraction. After doing some further study we found that spectral plot for normal EEG and pathological EEG is also different so we have also apply some spectral analysis for feature extraction. Putting all together we got a pretty good result then other processes.

Chapter 7: Further Research

We will try to apply more machine learning approach to improve our results such as support vector machines, deep belief network, random forest and relatively simple algorithms like K-nearest neighbors and logistic regression. We also want to devise a semi supervised learning mechanism for prediction of seizure.

Chapter 8: Conclusion

The methods we tried to apply proved good enough results but it is still far from state of the art. We have to look at slightly different mechanisms trying to exploit the time series component of the EEG data. If experts well versed in this field can be consulted and understand their perception of the data detection of anomalies using their instinct, we could try some more fancy neural networks and deep learning techniques to solve this problem.

Chapter 9: References

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