

# EEG-EMG Correlation for Parkinson's disease

Angana Saikia, Masaraf Hussain, Amit Ranjan Barua, Sudip Paul



**Abstract:** Parkinson's disease (PD) is a neurodegenerative disease in which disease symptoms progresses with time and mostly affected after 50 years of age. This disorder leads to loss of interconnectivity between brain and muscles. Human Biosignal like Electroencephalography (EEG) and Electromyography (EMG) acts as a tool for detecting various cognitive decline and muscle conditions in PD. Work reported here emphasized on a study of the EEG from the frontal and temporal brain in PD and healthy subjects as well as their muscle activities for the flexion and extension of the wrist. Signal acquisition was done using a dual channel Biosignal acquisition system from AD Instruments. Signals were acquired for 30 minutes each for all the subjects in the Out Patient Departments of various hospitals. Signal analysis was carried out in MATLAB platform. Various EEG and EMG features were extracted to determine brain and muscle conditions of patients. Classification was done using Artificial Neural Network for EEG and EMG features separately. A combination of EEG and EMG feature was also classified using ANN which gave a highest classification rate of 98.8% as compared to only EEG and EMG feature. This result proved that correlation and combination of various EEG and EMG features together provides more accuracy to the disease classifier. It gives an insight into the mechanism of muscle weakness and gait problems caused from the low production of dopamine in the brain of PD by giving a high classification rate as compared to others (Only EEG features and Only EMG features). Many studies have revealed the effect of EEG and EMG using various tools but here we focus mainly on correlation between them in the early stages. Detection of conditions of brain and muscle in early stage helps the clinician for proper medication to control disease progression. This work can also be further applicable for classifying the various stages of PD and also classifying them from the healthy subjects.

**Index Terms:** Neurodegenerative disease, Artificial Neural Network (ANN), Parkinson's disease (PD), Electroencephalography (EEG), Electromyography (EMG).

## I. INTRODUCTION

### Parkinson's disease (PD):

Parkinson's disease is neurodegenerative disease is due to loss of dopamine in the substantia nigra of the brain.

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Symptoms increases with the progression of the disease. Dopamine inhibits a person movement by establishing a link between brain and muscles [1]. It is a type of neurotransmitter produced in human brain. A person suffering from PD losses its coordination between brain and muscles, and results in disturbed limb movements [2].

Visible symptoms of PD are as follows [3, 4]:

- Bradykinesia, tremor, rigidity, postural instability.
- Sleep disorder, autonomous dysfunction, neuropsychiatric.

Environmental factors such as exposure to certain toxins substances (MPTP) and direct contact with some of the metal manganese by the workers in the mine fields may also lead to PD [5]. Villagers working with insecticides and pesticides are mostly prone to PD. Researchers and clinicians has also proved various genetic causes attached with PD. This included the alpha synuclein gene [6].

### Biosignal significance in PD:

Various brain functions are studied using EEG. It is a effective and important biomarker for diagnosis of various cognitive impairments in brain disorders like PD [7]. EEG carried out in resting state acts as an indicator of various spontaneous neuronal activities. It also detects specific cognitive tasks to establish brain behavior relationships [8].

EMG is used for the finding out the muscle strengths of any kind of diseases. PD also leads to weakening muscle due to loss of dopamine. Hence, EMG examines the various motor symptoms (tremor, bradykinesia) in PD [9]. Although these dysfunctions can be assessed by electromyography, it is still used rarely in the clinical evaluation of PD. The visible changes in the EMG signal of a PD patient is increased tonic background activities as well as bursts patterns are mostly alternating in nature [10,11].

Various Motor-cortical activities in PD can be identified using electroencephalography–electromyography correlation [12]. Synchronized activities of our neurons produce oscillatory activities whose frequencies reflect membrane properties of single neurons and interconnectivities of biological neural networks [13].

Hellwig et al in the year 2001, worked on EEG-EMG recordings, that detected cortico-muscular coherences at the tremor frequency. The outcome suggested that the sensory-motor cortex plays a vital role in the initiation of essential tremors, which is similar in Parkinsonian resting tremor [14].

The relationship between EMG and EEG gives the physiological information about how activities sensory-motor cortex, are related to the movement of limbs, [15]. In case of voluntary movement, EMG-EEG correlation is mainly studied to investigate cortical mechanisms which leads to central motor control and its associated disorders.

On the other hand, the use of EEG-EMG correlation for the study of involuntary movements serves as a diagnostic supplement as well as a method for clarifying the physiological mechanism underlying the generation of each involuntary movement [16].

A person suffering from PD loses its coordination between brain and muscle, so evaluation of EEG and EMG correlation using various features is a significant tool for the early detection of the disease. The various features of EEG like Lyapunov exponent, Entropy etc gives the brain condition of the patient and the EMG features like Root mean square, Power etc gives the spastic background muscle activities of the patient. Comparison of these EEG and EMG features and classification gives a correlation between the brain and muscle of the patient from which we can detect the tremor and other PD symptoms which originated from the brain. As a result of which the person loss the coordination between its brain and muscle and results in disturbed gait pattern and tremor.

II. MATERIALS AND METHODOLOGY

The figure no 1 below show the methodology:

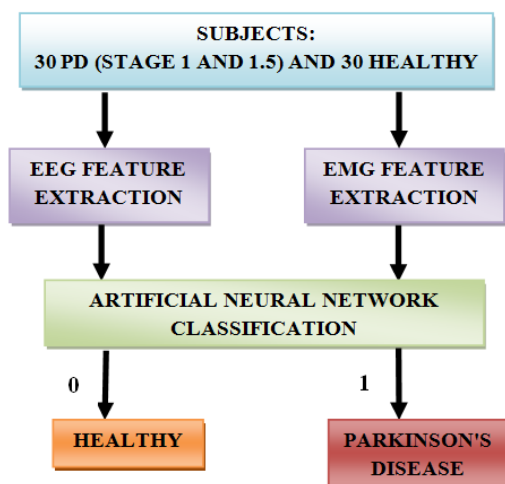


Figure no 1: Methodology

Participants:

This study was carried in various health centers in the city of Shillong, and Guwahati. Subjects were divided as:

- 30 subjects in the early stage (1 and 1.5) of PD
- 30 subjects were healthy (without PD)

Stage 1 refers to unilateral disease and Stage 1.5 refers to Unilateral and Axial involvement. Stages were selected according to modified Hoehn and Yahr scale. We have recruited subjects with upper limb tremor with unilateral damage. The age group of the subjects was between 50 to 70 years and weight is mostly between 45 to 60 kgs. Written consents were taken either from the subjects or the guardians before carrying out the experiment. All the EEG and EMG signals were recorded from a dual channel biophysical recording system (AD Instruments-Power Lab 26T acquisition system with Lab chart-7). Subjects were in sitting positions while data collection and were taken during the morning half's. The exclusion criteria of the study were:

- Patients with stages other than 1 and 1.5.

- Patients less than the age of 45 years.
- Patients with any kind of metallic implants.

Feature Extraction:

Various EEG and EMG features were extracted for the classification of PD subjects from those of healthy. These features acted as the input to the classifier.

Electroencephalography (EEG):

EEG signal was acquired from the temporal brain and frontal brain using the dual channel Electrode connected to the instrument. The time period of the data collection was 30 mints for all the subjects. Channel no 1 was connected in the frontal lobe while channel no. 2 in the temporal lobe. Both the channels were equipped with two types of electrodes where one acted as positive and the other as negative. The bone on the forehead was used as the ground. Some of the features analyzed from EEG are as follows:

a) Lyapunov and Inverse Lyapunov Exponent:

It is one of the most important chaotic measures. Chaotic processes are characterized by positive lyapunov exponent. The neuronal correlation of the human brain is determined using the lyapunov and inverse lyapunov exponent. Disorders like seizures can be easily detected [17]. In our experiment we have used Lyapunov Exponent as it can simultaneously also give both the features (Inverse Lyapunov Exponent and Lyapunov Exponent). There are other techniques to study the synchronization in brain but these features are very easy to understand and also give an accurate value. The positive and negative exponents determine the brain conditions. A positive high value shows a better neuronal oscillation where as negative value signifies less coordination between the neurons in the brain. Hence it can determine which area of the brain is more affected.

b) Autocorrelation Function:

It is correlation of a signal with a delayed copy of itself as a function of delay. The peaks found through autocorrelation function determine the various brain conditions of subjects in any neurological disorder [18].

c) Shannon Entropy:

Measure of uncertainty for the random variables is known as Claude Shannon entropy. Since EEG signals are random in nature, some information may be gathered if we use Shannon Entropy (ShEn) as one of the features to measure its complexity. When signal amplitudes are equally probable, ShEn is maximized. Then the Shannon entropy is determined and it is normalized by dividing log k where k is the length of the signal. Higher the values of these parameter corresponds to greater complexity in the data [19].

Electromyography (EMG):

EMG was collected from two types of wrist muscles while performing the flexion and extension of the wrist by each subject. The muscles were Extension carpi ulnaris and Flexor digitorum superficialis. Data was collected using the same dual channel Electrode (as EEG) for 30 minutes. Some of the time and frequency features of EMG were analyzed [20, 21,22,23]:

- a) Power: A signals power signifies the muscle fatigue.

Mathematical expression:

$$P = \frac{1}{T(X_n)^2} \dots\dots\dots (1)$$

Where,  
T : time  
X<sub>n</sub> : EMG signal.

b) *Standard Deviation*: Standard deviation measures the variability between various muscles during any spontaneous activity.

Mathematically,

$$STD = \left[ \frac{1}{(N-1)(X_n - M)} \right]^{1/2} \dots\dots\dots (2)$$

where,  
N: number of signals  
X<sub>n</sub> : EMG signal  
M : mean.

c) *Root Mean Square (RMS)*: It is mostly the amplitude of a signal. After rectification of a signal, RMS represents the area under EMG signal. Mathematically,

$$RMS = \left[ \frac{1}{N(X_n)} \right]^{1/2} \dots\dots\dots (3)$$

where,  
N : number of signals  
X<sub>n</sub> : EMG signal.

e) *Variance(VAR)*: It uses the power of the sEMG signal as a feature. Mathematically,

$$VAR = \left[ \frac{1}{(N-1)} \right] X_{n2} \dots\dots\dots (4)$$

f) *Waveform Length (WL)*: It is the cumulative length of the EMG waveform over a period of time. Mathematically,

$$WL = (X_n - 1) - (X_n) \dots\dots\dots (5)$$

g) *Median and Mean Frequency(MNF, MDF)*: Both are derived from power spectrum. They are expressed as:

$$MNF = \frac{f_i P_j}{P_j} \dots\dots\dots (6)$$

$$MDF = \left( \frac{1}{2} \right) (P_j) \dots\dots\dots (7)$$

h) *Modified Mean Frequency*: Modified Median Frequency (MMDF) is the frequency at which the spectrum is divided into two equal half's having equal amplitude.

Mathematically,

$$MMF = \left( \frac{1}{2} \right) (A_j) \dots\dots\dots (8)$$

Where  
A<sub>j</sub> : sEMG amplitude spectrum at frequency at bin j.

i) *Modified Mean Frequency(MMNF)*: It is the average frequency.

Mathematically,

$$MMNF = \frac{f_j A_j}{A_j} \dots\dots\dots (9)$$

Where,  
A<sub>j</sub> : sEMG amplitude spectrum at frequency bin j.

**Artificial Neural Network (ANN) Classification:**

All the EEG and EMG features were classified using Artificial neural network. ANN is trained with input and target patterns. MLP (Multilayer Perceptron) network, back propagation (BP) learning algorithm is used [20]. Inputs data ( EEG and EMG features) is fed forward through the network. Weights were adjusted by backward propagation of the error during training. The error is minimized through unlimited training cycles called epoch till the time minimum error is obtained.

During learning of the network, all the data (features) were sub divided into three different data sets.

- Training :70%
- Validation :15%
- Testing: 15%.

Sigmoid transfer function was used for the hidden layer. System training parameters were:

- max\_epochs=10000
- show=50,
- performance goal=0.02.

'trainlm' was used as the training function as it much faster compared to other training function and mostly used in disease classification.

Classification rates were calculated for three different cases.

The cases were:

- Only EEG features
- Only EMG features.
- Both EEG and EMG feature as input to the network.

**III. RESULTS**

Table I shows Lyapunov and Inverse Lyapunov Exponents of the various groups. Root Mean Square, Standard deviation, Power, Variance, Waveform length, Mean frequency,

Median frequency and Modified mean frequency for two different muscles were calculated for all the subjects. Variance of all the features for different muscles is showed in Table II. Table III shows the Classification rates (%) of various cases of input features.

Healthy subjects				PD subjects			
Lyapunov Exponent		Inverse Lyapunov Exponent		Lyapunov Exponent		Inverse Lyapunov Exponent	
Frontal	Temporal	Frontal	Temporal	Frontal	Temporal	Frontal	Temporal
2.82	3.19	0.6599	0.27605	5.57	2.865	0.14221	0.1263

Table I: Variance of Lyapunov and Inverse Lyapunov Exponent

FEATURES	PD SUBJECTS	HEALTHY SUBJECTS
Standard Deviation	33.01	31.24
Variance	202.79	98.43
Root Mean Square	227.72	326.83
Waveform Length	3.33	46.44
Power	14.74	50.8
Median Frequency	22.12	4.6
Mean Frequency	33.98	14.33
Modified Median Frequency	278.37	356.03
Modified Mean Frequency	3.95	3.56

Table II: Variance of Time domain and Frequency Domain Features for hand muscles

Cases	No. of features	Classification Rate (%)
Only EEG features	3	62
Only EMG features	9	73
Both EEG and EMG features	12	98.8

Table III: Classification rates for various cases

IV. DISCUSSION

*Lyapunov and Inverse Lyapunov Exponent (Table I)* : The temporal part of the brain of PD patient showed is lesser lyapunov exponent value than that of the frontal while the inverse lyapunov exponent is higher. Fig 2,3,4,5 shows the Lyapunov exponent plots.

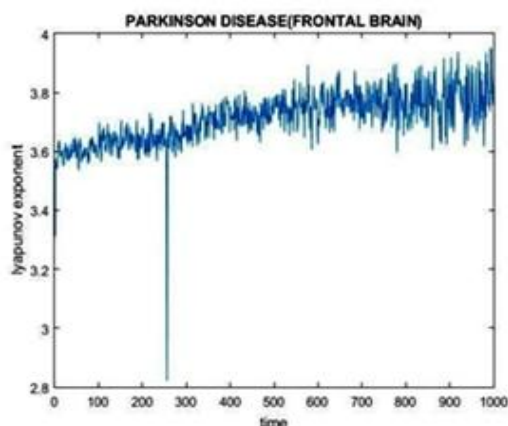


Fig 2: Plot for PD (Frontal)

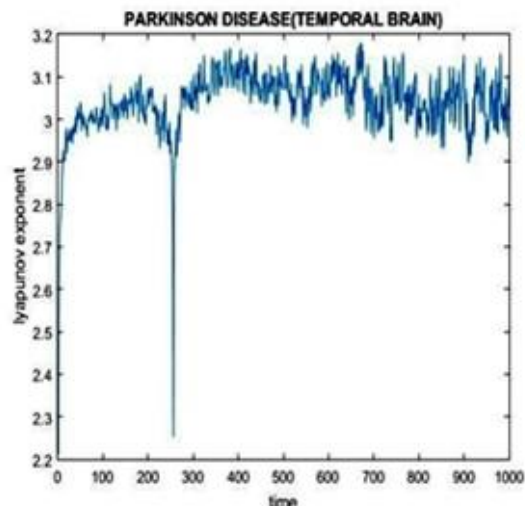


Fig 3: Plot for PD (Temporal)

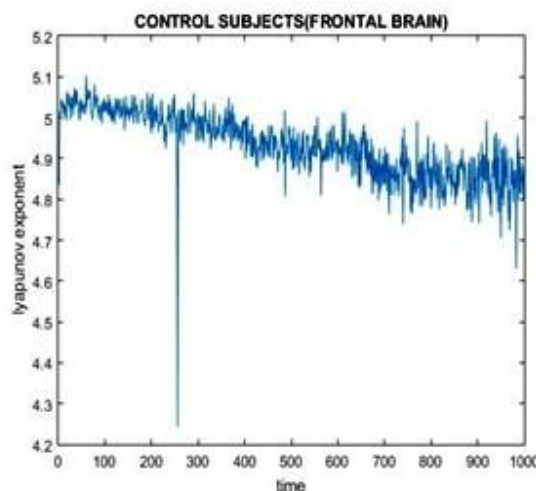


Fig 4: Plot for Control (Frontal)

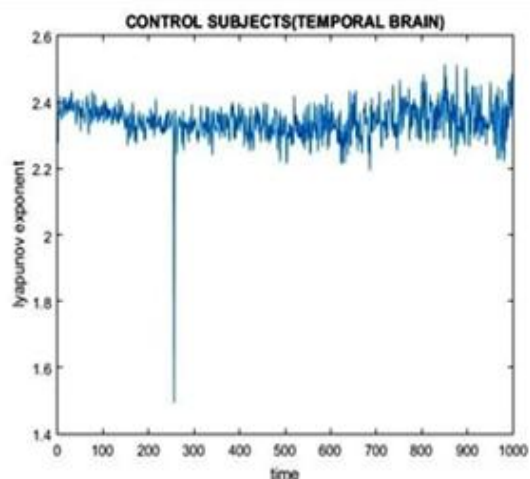


Fig 5: Plot for Control (Temporal)

Autocorrelation function:

•Frontal region: It is observed that there are many negative peaks present and the correlations also disturbed for the PD compared with the control subjects (Fig 6 and 7).

The negative peaks indicate that the neuronal oscillations are completely 180 degree reverse and synchronization breaks. So, one can say that the neuronal insult occurs in the frontal region for the PD cases.

•Temporal Region: Here the most important consideration is the peak amplitude changes rapidly; which signifies that the brain potential especially the action potential changes rapidly compared with the control one (Fig 8 and 9).

CONTROL SUBJECTS (FRONTAL BRAIN)

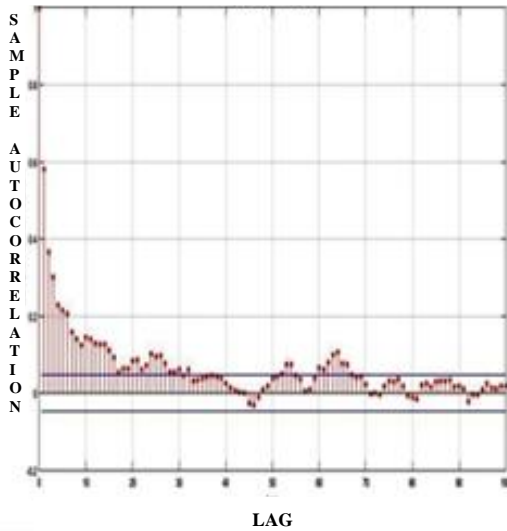


Fig 6 : Autocorrelation Function for Control (Frontal)

PD SUBJECTS (FRONTAL BRAIN)

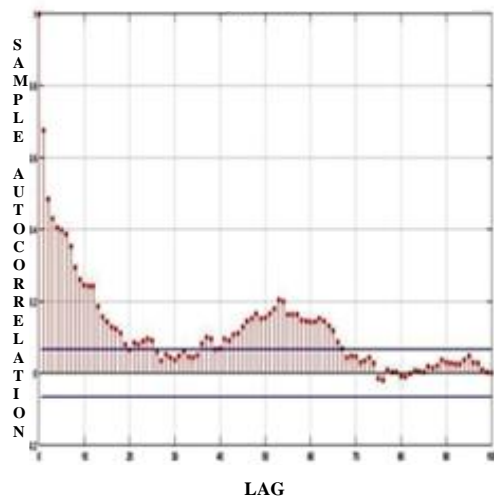


Fig 7: Autocorrelation Function for PD(Frontal)

PD SUBJECTS (TEMPORAL BRAIN)

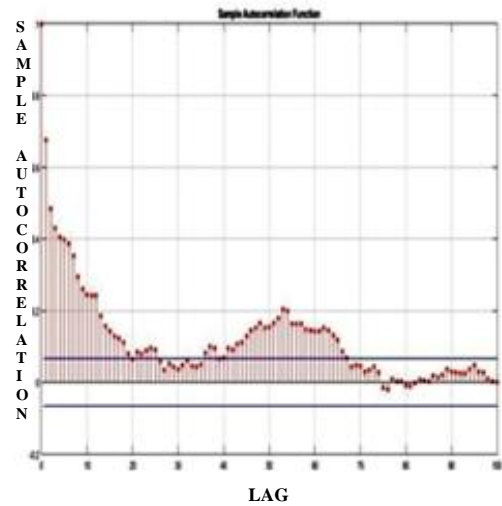


Fig 8: Autocorrelation Function PD(Temporal)

CONTROL SUBJECTS (TEMPORAL BRAIN)

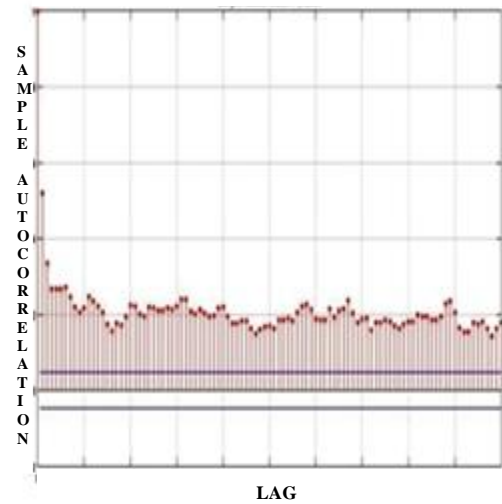


Fig 9 : Autocorrelation Function for Control(Temporal)  
**Shannon Entropy:** It has been seen that Shannon entropy for PD is higher than healthy subjects (Fig 10). It signifies that increase in complexity value for PD subjects is due to neural complexity.

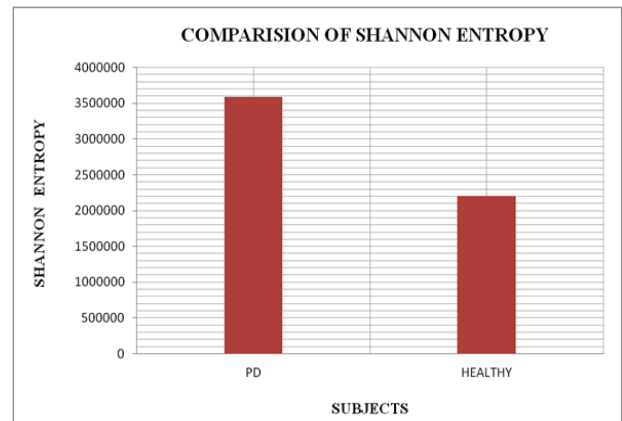


Fig 10: Shannon Entropy comparison graph

**Root Mean Square, Standard deviation, Power, Mean, Variance, Waveform length, Mean frequency, Median frequency and Modified mean frequency (Table no II):**

It was found that among all these parameters, root mean square, waveform length, power and modified mean frequency showed a higher value for healthy subjects than a PD subjects. It signifies that the person from PD always suffer from weakening of their muscle and as result disturbs their gait. Fig 11 shows the comparison graph for all the features.

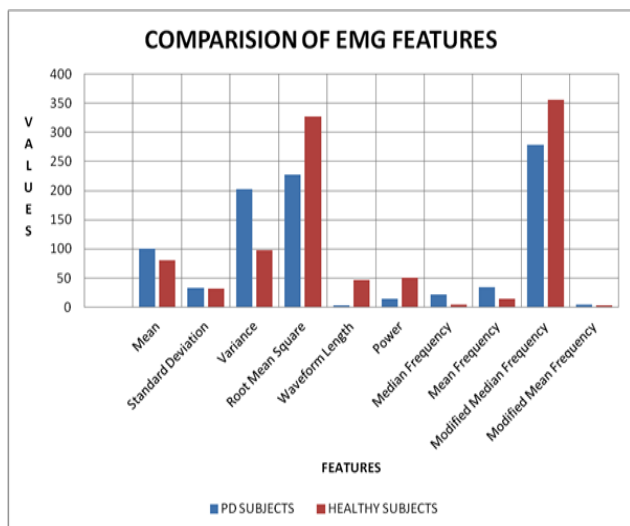


Fig 11: EMG Features comparison Graph

V. CONCLUSION

In this work the EEG and EMG signals were acquired from 30 no of PD subjects and 30 control subjects using Biophysical acquisition device (AD Instruments) from various hospitals in Assam and Meghalaya. Some of the EEG and EMG features were analyzed for each subject in MATLAB platform and also statistical comparison was done between the groups (PD and Healthy) .

Lyapunov, Inverse Lyapunov exponent and Shannon entropy features for both the groups were calculated. Lyapunov exponent for the frontal brain is higher than that of the temporal lobe while the inverse lyapunov exponent is higher in frontal lobe all the PD patients. This proves that in the PD brains the correlations between neurons are stronger in temporal region. The various sensory organs of a human being is located in temporal lobe that initiates human movements and maintains a person's balance. As the correlation between the neuronal parts is less, the person suffering from PD suffers from unbalancing and some motor symptoms like tremor. Radiological investigations like Computerized Tomography or Magnetic Resonance Imaging may help to diagnose the brain conditions of PD, but it's quite costlier than EEG and EMG investigation and sometimes unavailable at the remote locations.

From the autocorrelation function we can conclude that the neuronal excitation and the systemic oscillation hampered in PD compared with the control one. The increase in Shannon entropy in PD subjects shows that there is a neural complexity in them due to which they have a poor coordination and as a result their gait is disturbed.

From EMG, it was found that the healthy persons have a

higher value of root mean square, waveform length, power and modified mean frequency than a Parkinson's patient.

Hence, as the disease progress the muscles become more weakened and it becomes difficult for the person to move its limbs. The result provides an insight into the mechanism of muscle weakness and gait problems.

ANN classification rate using all the EEG and EMG features together as input to the network showed a highest percentage of 98.8% as compared to other combination such as only EEG feature/ only EMG feature as input to the network. The result signified that a correlation and combination of both EEG and EMG together can classify accurately the PD subjects from the healthy one. More the number of input features better is the classification rate.

Correlation of the EEG and EMG features provides an insight of the brain and muscle conditions of the patient. It gives the proper justification why the patient's loss its coordination between brains signals and muscles signals. In near future this type of brain and muscle investigations will help in the development of rehabilitation programs for patients with PD. This will add to their healthy and happy life ahead. The novelty of the work is that it gives the disease condition in the early stages. Hence this would be a novel finding which would help the clinicians and PD caregivers to know the disease conditions at the onset of the disorder. As a result of which the patient will receive the treatment from the onset of the disease.

**Conflict of Interest:** None declared

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**Ethical approval Committees:**

- 1) North Eastern Hill University, Shillong (vide no: IECHSP/2017/42)
- 2) North Eastern Indira Gandhi Regional Institute of Health & Medical Science, Shillong (vide no: NEIGR/IEC/M6/F13/18).

REFERENCES

1. L.M. De Lau, M.M. Breteler, " Epidemiology of Parkinson's disease," The Lancet Neurology, 2006, vol.5(6),pp.525-35.
2. H. Checkoway, L.M.Nelson, " Epidemiologic approaches to the study of Parkinson's disease etiology," Epidemiology, 1999, pp. 327-36.
3. S. Chapuis, L. Ouchchane, O. Metz, L. Gerbaud, F. Durif, " Impact of the motor complications of Parkinson's disease on the quality of life," Movement disorders: official journal of the Movement Disorder Society, 2005, vol.20(2), pp. 224-30.
4. A.J.Hughes, S.E.Daniel, L. Kilford, A.J.Lees, "Accuracy of clinical diagnosis of idiopathic Parkinson's disease: a clinico-pathological study of 100 cases," Journal of Neurology, Neurosurgery & Psychiatry, 1992, vol.55(3), pp.181-4.



5. T.T. Warner, A.H. Schapira, "Genetic and environmental factors in the cause of Parkinson's disease," *Annals of Neurology: Official Journal of the American Neurological Association and the Child Neurology Society.*, 2003, vol.53(S3), pp.S16-25.
6. P. Surathi , K. Jhunjhunwala, R. Yadav , P.K. Pal , "Research in Parkinson's disease in India: A review," *Annals of Indian Academy of Neurology*, 2016, vol.19(1), pp.9.
7. C.J. Stam , B. Jelles, H.A. Achtereekte , S.A. Rombouts, J.P. Slaets, R.W. Keunen, "Investigation of EEG non-linearity in dementia and Parkinson's disease," *Electroencephalography and clinical neurophysiology*, 1995, vol. 95(5), pp. 309-17.
8. J.N. Caviness , J.G. Hentz, V.G. Evidente, E. Driver-Dunckley, J. Samanta , P. Mahan , D.J. Connor, M.N. Sabbagh, A.H. Shill, C.H. Adler, "Both early and late cognitive dysfunction affects the electroencephalogram in Parkinson's disease," *Parkinsonism & related disorders*, 2007, vol.13(6), pp.348-54.
9. I. Milanov, "Clinical and electromyographic examinations of Parkinsonian tremor," *Parkinsonism & related disorders*, 2000, vol.6(4), pp. 229-35.
10. Y.Z. Huang, F.Y. Chang, W.C. Liu, Y.F. Chuang, L.L. Chuang, Y.J. Chang, "Fatigue and muscle strength involving walking speed in Parkinson's disease: insights for developing rehabilitation strategy for PD," *Neural plasticity*, 2017.
11. L. Arendt-Nielsen, K.R. Mills, A. Forster, "Changes in muscle fiber conduction velocity, mean power frequency, and mean EMG voltage during prolonged submaximal contractions," *Muscle & nerve*, 1989, vol. 12(6), pp.493-7.
12. Y. Hashimoto, J. Ushiba, A. Kimura, M. Liu, Y. Tomita, "Correlation between EEG-EMG coherence during isometric contraction and its imaginary execution," *Acta Neurobiol Exp (Wars)*, 2010, vol. 70(1), pp.76-85.
13. W. De Clercq, A. Vergult, B. Vanrumste , W. Van Paesschen, S. Van Huffel, "Canonical correlation analysis applied to remove muscle artifacts from the electroencephalogram," *IEEE transactions on Biomedical Engineering*, 2006, vol. 53(12), pp.2583-7.
14. B. Hellwig, S. Häussler, B. Schelter, M. Lauk, B. Guschlbauer, J. Timmer , C.H. Lücking, "Tremor-correlated cortical activity in essential tremor," *The Lancet*, 2001, vol. 357(9255), pp.519-23.
15. J. Chiang, Z.J. Wang, M.J. McKeown, "A multiblock PLS model of cortico-cortical and corticomuscular interactions in Parkinson's disease," *NeuroImage*, 2012, vol. 63(3), pp.1498-509.
16. R. Bortel, P. Sovka, "EEG-EMG coherence enhancement," *Signal Processing*, 2006, vol. 86(7), pp.1737-51.
17. D.J. Kim , J. Jeong, J.H. Chae, S. Park, S.Y. Kim , H.J. Go, I.H. Paik , K.S. Kim, B. Choi, "An estimation of the first positive Lyapunov exponent of the EEG in patients with schizophrenia," *Psychiatry Research: Neuroimaging*, 2000, vol.98(3), pp.177-89.
18. D. Michael, J. Houchin, "Automatic EEG analysis: a segmentation procedure based on the autocorrelation function," *Electroencephalography and clinical neurophysiology*, 1979, vol.46(2), pp. 232-5.
19. B. Thilakvathi, S.S. Devi, K. Bhanu, M. Malaippan, "EEG signal complexity analysis for schizophrenia during rest and mental activity," *BIOMEDICAL RESEARCH-INDIA*, 2017, vol. 28(1), pp.1-9.
20. A. Phinyomar, C. Limsakul, P. Phukpattaranont, "A novel feature extraction for robust EMG pattern recognition," *arXiv preprint arXiv:0912.3973*, 2009.
21. K. Englehart , B. Hudgins, P.A. Parker, M. Stevenson, "Classification of the myoelectric signal using time-frequency based representations," *Med Eng Phys*, 1999, vol. 21, pp.431-438
22. A. Saikia, M. Hussain, A.R. Barua, S. Paul, "Significance of Lyapunov Exponent in Parkinson's Disease Using Electroencephalography", 6th International Conference on Signal Processing and Integrated Networks, IEEE, 2019, pp. 791-795.
23. A. Saikia, S. Mazumdar, N. Sahai, S. Paul, D. Bhatia, "Comparative study and feature extraction of the muscle activity patterns in healthy subjects", 3rd International Conference on Signal Processing and Integrated Networks, IEEE, 2016, pp. 147-151.

Assam, India in 2014. She has many research publications in reputed journals and attended various international conferences in USA. Recently she wrote a book on "Overview of Parkinson's disease and its Relevance" published by LAP LAMBERT Academic Publishing, Germany. She also has many research publications and attended many International conferences.



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