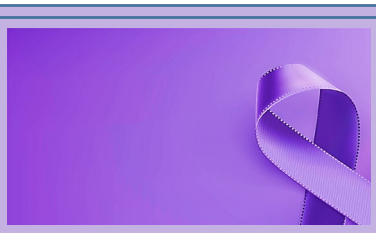

EPILEPSY

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Dear Doctor,

Greetings from Micro Labs. We are happy to bring you "EPILEPSY Newsletter" which contains latest and interesting news in the field of Neuropsychiatry.

This issue contains:

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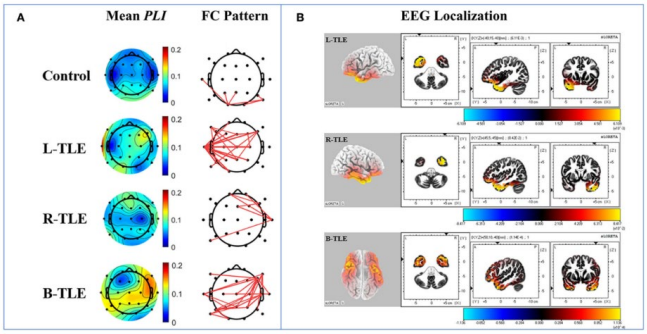
In patients with temporal lobe epilepsy, the fontotemporal phase lag index is correlated with the intensity of the seizures.

The most prevalent form of localization-related epilepsy, known as temporal lobe epilepsy (TLE), is characterised by recurrent spontaneous seizures and pervasive cognitive impairment. The idea that TLE is a disorder of large neural networks has been supported by compelling evidence from semeiology, histopathology, electrophysiology, and neuroimaging over the past few decades. These studies have shown that the neurobiological insults in TLE extend beyond the epileptogenic temporal lobe into extratemporal regions.[1] By using graph theory analysis, this study sought to identify neural network markers associated with the intensity of seizures in patients with temporal lobe epilepsy (TLE).[2]

A total of 151 TLE patients and 36 controls with video-EEG monitoring were included in the study. Data from the 90-s interictal EEG were collected. To quantify and localise their functional networks, we used a network analysis pipeline based on graph theory, which included the weighted classical network, minimal spanning tree, community structure, and LORETA. The seizure frequency, drug-resistant epilepsy (DRE), and VA-2 scores were used to assess the severity of the seizures.

Patients with TLE had ipsilateral frontotemporal activity, according to the network analysis process. Patients with more severe seizure types had greater frontotemporal phase lag index (PLI) values in the theta band (4–7 Hz) ($P < 0.05$). The frontotemporal PLI values in the theta band and age of onset were independently linked with the VA-2 scores, according to multivariate linear regression analysis ($P = 0.001$ and $P = 0.007$, respectively).



Diagrams of the functional network in patients with TLE and controls. The graph of the mean phase lag index, functional connection pattern

In patients with TLE, the study revealed independent relationships between frontotemporal functional connectivity and seizure severity. This study emphasised the critical function of the frontal lobe in TLE and offered a non-invasive, simple-to-use indicator for TLE patients who are drug-resistant.[2] The graph theory network results revealed enhanced frontotemporal connections ipsilaterally during interictal-resting periods, in contrast to negative findings using standard power spectrum analysis. The AF and UF are two examples of the different fibres that link the frontal and temporal cortices.[3] Other approaches, such as machine learning, were initially used to predict prognoses. Croce et al

This study showed that in patients with TLE, the frontotemporal PLI in the theta band independently linked with seizure severity. An approachable method to direct the treatment strategy in ordinary clinical practise was provided by the network analysis.

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A indicator of the effectiveness of Lennox-Gastaut syndrome deep brain stimulation is paroxysmal rapid activity.

One of the most severe epileptic and developmental encephalopathies is Lennox-Gastaut syndrome (LGS). Due to the prevalence of numerous drug-resistant seizure types as well as behavioural and cognitive impairments that may be altered by antiseizure drugs, this kind of epilepsy presents a therapeutic challenge to doctors (ASMs). The physical, emotional, social, and economical health of the family is eventually impacted by LGS, which also has a significant negative influence on the health-related quality of life of patients, their families, and their carers.[1] During a thalamic deep brain stimulation (DBS) therapy trial, we evaluated

generalised paroxysmal fast activity (GPFA), a crucial interictal electrographic hallmark of LGS, and associated GPFA burden with seizure diaries (Electrical Stimulation of the Thalamus in Epilepsy of Lennox–Gastaut Phenotype [ESTEL]).[2]

In the ESTEL research, a 12-month randomised clinical trial of DBS, seizure counts from 3-month diaries and GPFA from intermittent, 24-h electroencephalograms (EEGs) of 17 young adults with LGS (mean ageSD = 24.9 6.6) were compared (comprising a 3-month baseline and 9 months of postimplantation follow-up).

When compared to 284 (interquartile range [IQR] = 120.5-360) electrographic seizures per day, the baseline median number of seizures recorded in diaries was 2.6 (interquartile range [IQR] =1.4-5) per day, demonstrating that diaries only record a tiny portion of the seizure load. The average number of GPFA discharges per hour of sleep across all patient EEGs was 138 (IQR =72-258). (Table 1) When measured throughout 2-hour windows of sleep, GPFA duration and frequency were substantially correlated with seizure counts recorded in diaries over 3-month intervals ($p<.001$, 2 p =.30-.48). Over the course of three months, 20–25 diary seizures were seen for each GPFA discharge. However, within specific patients, the ratio was similar over time, so that the percentage change from pre-DBS baseline in seizure diaries was strongly correlated with the percentage change in GPFA. There was a high degree of interpatient variability in the ratio between diary seizure burden and GPFA burden.

Characteristics of identified EEG abnormalities within and across all study timepoints

	EEG timepoint				
EEG abnormality	Baseline, 17	Week 12, 17ª	Week 24, n = 17b	Week 36, n = 17c	Combined timepoints, n = 68
GPFA, median (IQR)					
Events per minute, n	2.6 (1.7-5)	2.9 (.8-3.8)	2.3 (1.4-4.7)	2.1 (1.1-3.5)	2.3 (1.2-4.3)
Event duration, S	1.2 (.9-1.8)	1.4 (1-1.6)	1.1 (.8-1.6)	1.2 (.8-1.4)	1.2 (.9-1.6)
Burden, s/2 h	471 (268-724)	415 (140-591)	390 (222-539)	288 (156-459)	370 (173-560)
EEG-clinical seizures, median (IQR)					
n/h	1.3 (.8-2.6)	1.4 (.5-2.2)	1.0 (.3-2.0)	(.3-1.3)	1.1 (.4-2.2)
Duration, S	28.3 (14.7-69.1)	27.2 (16.5-47.4)	33.2 (16.7-73.9)	26.9 (13.9-57.5)	36.7 (13.5-83)

EEG-seizures, median (IQR)					
n/h	11.8 (5-15)	12.1 (5.2-15.1)	8 (5.2-14)	7.4 (3.6-11.6)	9.4 (4.6-14)
Duration, S	26.1 (17.4-49.3)	22.9 (14.5-43)	29.2 (17.6-53.6)	28 (14.9-39.6)	26.7 (17.4-49.3)

The study demonstrate using data from the ESTEL DBS study that changes in GPFA duration and count, assessed from sporadic 2-hour sleep EEG recordings, track changes in the daily average number of seizures reported during three-month intervals of seizure diaries. The ratio of GPFA to diary seizures varied significantly from patient to patient, but it appeared constant over time within the same patient, indicating that percentage decreases from baseline in GPFA were highly correlated with comparable percentage reductions in diary seizures. This shows that GPFA load could be a quick and accurate way to assess a patient's response to treatment for LGS.[2] As most patients with LGS are unable to accurately self-report seizures, caregivers are left to handle this responsibility while also managing the patient's seizures, associated injuries, and cognitive and behavioural comorbidities.[3]

Instead of waiting for feedback from seizure diaries, monitoring changes in GPFA may enable quick adjustment of therapy parameters when trying to optimise treatment for patients with LGS.

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An Explanatory Statistical Method for Predicting Seizures Using EEG-Based Brain Functional Connectivity

Epilepsy is a condition that affects the central nervous system (CNS) and is brought on by an imbalance in neuronal electrical activity, which can lead to seizures or cognitive dysfunction.[1] A category of long-term neurological conditions known as epilepsy are characterised by frequent and sudden seizures. The hardships associated with this illness can be lessened by effective seizure prediction. Currently, studies classify patients' preictal or interictal stages using neural network properties, enabling seizure prediction. The majority of prediction algorithms, however, rely on deep learning methods, which are difficult to understand and have a high level of computational complexity. In this study, we suggested a novel two-stage statistical procedure that is straightforward to compute and interpret in order to overcome these problems.[2]

The well-known public dataset CHB-MIT was one of two datasets utilised in the study to assess the effectiveness of the suggested methodology. For each epoch, we first evaluated the dynamic brain functional connectivity network. Tenth, we employed the developed network predictor for seizure prediction in the second stage.

The study used two distinct datasets to show the outcomes of our technique for seizure prediction. Our method produced an AUC value of 0.963 for the FH-PKU dataset, a prediction sensitivity of 93.1%, and a false discovery rate of 7.7%. Our strategy outperformed current state-of-the-art techniques for the CHB-MIT dataset, achieving an AUC value of 0.940, a prediction sensitivity of 93.0%, and a false discovery rate of 11.1%.

Seizure prediction results for the patients in CHB-MIT database via STS-HGCN method

Patient ID		BN data			Raw data			BN + raw data			STS-HGCN-AL	
	AUC	SENS	FDR	AUC	SENS	FDR	AUC	SENS	FDR	AUC	SENS	FDR
chb01	0.968	95.0	0.060	0.496	47.5	0.374	0.970	95.0	0.088	0.996	100	0.000
chb02	0.984	100	0.102	0.634	75.0	0.473	0.973	90.0	0.059	0.897	100	0.145
chb03	0.992	95.5	0.022	0.691	50.0	0.193	0.989	95.5	0.029	0.928	83.3	0.173
chb04	0.867	86.7	0.290	0.595	50.0	0.247	0.825	93.3	0.362	NA	NA	NA

chb05	0.835	91.5	0.173	0.458	100	0.946	0.778	60.0	0.135	0.875	100	0.000
chb06	0.804	84.3	0.344	0.505	22.9	0.154	0.779	65.7	0.205	0.906	100	0.162
chb07	0.978	93.3	0.067	0.583	43.3	0.221	0.975	93.3	0.057	NA	NA	NA
chb08	0.953	85.0	0.078	0.548	40.0	0.200	0.933	85.0	0.100	0.999	100	0.000
chb09	0.963	90.0	0.043	0.553	96.7	0.815	0.950	90.0	0.069	0.843	100	0.092
chb10	0.947	86.7	0.073	0.544	25.0	0.110	0.922	85.0	0.127	0.977	83.3	0.171
chb11	0.990	95.0	0.039	0.696	80.0	0.459	0.989	95.0	0.036	0.940	100	0.123
chb12	0.911	76.8	0.095	0.479	96.4	0.919	0.904	87.5	0.162	NA	NA	NA
chb13	0.998	96.3	0.000	0.506	22.2	0.100	0.996	96.3	0.025	0.915	85.7	0.109
chb14	0.872	95.0	0.015	0.490	42.5	0.335	0.826	92.5	0.369	0.976	100	0.104
chb15	0.836	99.8	0.231	0.433	84.6	0.838	0.793	73.6	0.306	NA	NA	NA
chb16	0.950	93.3	0.189	0.486	40.0	0.322	0.904	96.7	0.211	0.954	87.5	0.187
chb17	0.960	95.0	0.080	0.542	75.0	0.587	0.954	90.0	0.073	0.826	100	0.237
chb18	0.912	100	0.300	0.553	30.0	0.143	0.901	87.5	0.259	0.992	75.0	0.138
chb19	0.976	91.0	0.046	0.610	63.6	0.390	0.974	100	0.167	0.991	100	0.038
chb20	0.987	100	0.077	0.386	33.3	0.319	0.980	100	0.117	0.982	100	0.184
chb21	0.957	90.0	0.085	0.451	5.0	0.002	0.923	85.0	0.115	0.833	100	0.156
chb22	0.924	96.3	0.245	0.485	40.7	0.283	0.860	85.2	0.219	0.997	100	0.000
chb23	0.994	100	0.011	0.409	10.0	0.043	0.990	100	0.031	0.990	100	0.047
chb24	1.000	100	0.000	0.599	87.0	0.637	1.000	100	0.000	NA	NA	NA
Average	0.940	93.0	0.111	0.530	52.5	0.380	0.920	89.3	0.138	0.938	95.5	0.109

This study suggested a statistical technique that can predict epileptic seizures and is useful for sounding the warning prior to seizures. More specifically, our methodology employs time-varying precision matrix estimation to create a dynamic brain functional connectivity network using scalp EEG data. Tenth, we use these assessments of brain functional connectivity as variables that can be predicted in the ensemble prediction model. 2 This study is not the first to derive brain connection characteristics for seizure prediction from scalp EEG data. 3

Numerous cutting-edge methods have been developed, including those by Gemein et al., Tsiouris et al., and Truong et al. As far as we are aware, Yang et al.4's STS-HGCN-AL technique has the best seizure prediction performance among these approaches.

This study provided a comprehensible statistical technique that uses a scalp EEG approach to measure the brain network and a network predictor to forecast epileptic episodes.

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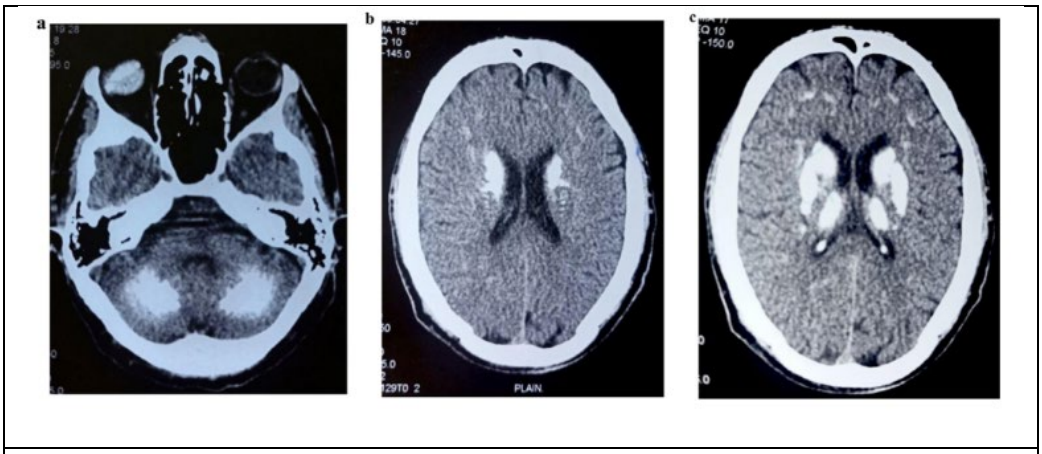
Bilateral striopallidodentate calcinosis (BSPDC) is the name given to Fahr's disease, a rare sporadic genetic neurodegenerative condition that has also been referred to by 35 other names in previously documented instances. It is characterised by calcium deposition that penetrates the blood-brain barrier and causes the cerebral cortex, dentate nuclei, basal ganglia, and other brain structures to become calcified. It is now possible to characterise the condition more precisely thanks to the discovery of numerous gene alterations linked to it.[1] It is unusual to see seizures as a presenting symptom in this case. In this article, we give the case of a 43-year-old man who experienced his first Fahr's illness symptom, an epileptic seizure.[2]

Case Presentation:

A 43-year-old man's wife took him to the emergency room after he saw an incident of strange whole-body motions. Upon general inspection, the patient showed signs of fatigue and confusion. He had a fever, a blood pressure of 120/80 mmHg, a respiratory rate of 16 breaths per minute, a heart rate of 80 beats per minute, and a room air oxygen saturation of 97%. He was also febrile. Initial laboratory examinations turned up no anomalies. The NS1 antigen fast test was ordered due to an increase in dengue fever cases in that area, and the results were nonreactive. A lumbar puncture performed as part of further studies revealed normal cytology and biochemistry. The results of a head computed tomography (CT) scan and magnetic

resonance imaging (MRI) showed that the caudate nuclei, putamen, globus pallidus, thalami, cerebellar dentate nuclei, centrum semiovale, and subcortical white matter were all bilaterally symmetrically calcified (Figs. 1 and 2). The brain MRI revealed no ischemia or hemorrhagic abnormalities. The study eliminated all potential secondary causes of calcification. The data collected pointed to Fahr's disease. He was put on oral carbamazepine medication and discharged after being monitored for three months with no additional seizure occurrences being noted.



A computed tomography (CT) scan and magnetic resonance imaging (MRI) of the head was performed and they revealed bilateral symmetrical calcification of the head of caudate nuclei, putamen, globus pallidus, thalami, cerebellar dentate nuclei, centrum semiovale, and subcortical white matter

Fahr's disease is a neurological illness that can be familial or sporadic. It is primarily transmitted through the autosomal dominant gene, though it can also arise through the autosomal recessive gene. It affects the genetic loci SLC20A2, PDGFB, and PDGFB, and a locus at 14q (IBGC1) is frequently affected, according to a number of studies. On chromosome 8 there is a second locus, and on chromosome.[2] there is a third. 2 Therefore, being able to recognise Fahr's disease from other movement diseases is important. One of the least common signs of Fahr's disease is seizures. Aldawsari et al. described a case of Fahr's disease in which the patient had an uncommon primary sign of widespread tonic-clonic seizures.

Overall, the focus of the treatment plan should be on improving the quality of life and treating patients' symptoms. Despite its rare, Fahr's disease needs to be considered in the differential diagnosis of adult patients having their first seizure, especially when mental problems are present. The issue must be properly recognised because it can mirror other movement problems, especially in people with underlying neurological illnesses that might make the diagnosis more difficult.

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