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A COMPREHENSIVE REVIEW ON EPILEPSY AND ITS DIAGNOSIS/ TREATMENT USING EEG SIGNALS

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Abstract
Epilepsy is a chronic functional disorder of the brain resulting in recurrent seizures due to hyper-synchronous neuronal activity. About 30% of those who suffer from epilepsy develop an incurable form of the disease that does not respond to medications, also known as drug-resistant epilepsy (DRE), which necessitates a careful evaluation of the diagnostic and therapeutic modalities. The foundation of epilepsy treatment remains electroencephalography (EEG), which continues to provide the best information regarding seizure localization, classification, and treatment follow-up. This review presents a comprehensive study of epilepsy, covering the historical development, epidemiology, and diverse etiology of epilepsy, with an in-depth study of EEG entailing the major contribution to the developments. The EEG techniques applied in this study embrace classical approaches as well as modern techniques, such as time-frequency techniques and machine learning (ML)- based automated seizure detection and deep learning (DL) based signal processing. Emphasis is made on the challenges common in elderly and pediatric age groups, together with specific EEG characteristics and timely treatment interventions. There is a detailed critical analysis of cutting-edge developments, including wearable EEG units, closed-loop neuromodulation systems, and AI-based individualized interventions.

Keywords: *Epilepsy; Electroencephalography (EEG); Seizure detection; Wearable EEG; Closed-loop neuromodulation; Automated diagnosis; Pediatric epilepsy.*

1. Introduction

Epilepsy is a neurological disorder characterized by the temporary occurrence of aberrant neuronal activity at essentially any point in the brain (Suruchi & Vaishali, 2013). According to an estimate made in 2022 by the World Health Organization (WHO, 2022), epilepsy affects around 50-70 million individuals globally: some of the most commonly experienced neurological disorders affect one out of every ten individuals. The primary epilepsy symptom is seizure, characterized as a brief alteration in movements behaviour, feelings, or awareness that lasts from a few seconds to minutes. If both the hemispheres of the brain are affected by such an illness, the condition is termed general seizure, whereas if only one hemisphere is affected, it is classified as partial seizure. These two types are classified as convulsive seizures, whereas absence seizures are classified as non-convulsive seizures. Of the 50 million individuals with epilepsy globally, 40% experience nonconvulsive episodes and 60% experience convulsive seizures. Continuous seizures are primarily caused by an imbalance between the excitatory and inhibitory actions of neurotransmitters in the temporal lobe, which can result from severe head injuries, brain infections, hereditary diseases, strokes, oxygen deprivation, or birth trauma. The person will feel self-conscious and unable to act as a result of this imbalance (Orosco et al., 2013).

The disease has been existing for the last 3000 centuries but would be discovered only within the few hundred years. Today, quite several researches are being carried out within the field in diagnosis, prevention, and treatment due to the availability of numerous medical facilities (Magiorkinis et al., 2014). The accurate diagnosis and classification of the seizure type are very important in optimizing treatment strategies and improving patient outcomes.

Electroencephalography (EEG) is a direct measure of the electrical activities of brain life, and the most important test for epilepsy diagnosis. Electroencephalography can confirm an epilepsy diagnosis by detecting interictal epileptiform discharges (Noachtar & Rémi, 2009); it can also localize the seizure onset zone before surgery (Schomer & Lopes da Silva, 2011). In the last few decades, developments in EEG hardware, signal digitalization technologies, and machine learning algorithms have improved sensitivity and specificity in EEG for epilepsy diagnosis and management (Acharya et al., 2013; Subasi & Gursoy, 2010).

2. Historical Overview of Epilepsy

The history of epilepsy joins with the human history as far back as antiquity. Few illnesses in the history of medicine can be traced back almost five or six thousand years. Various written documents, accounts, references, and philosophical treaties about epilepsy may be found in different civilizations such as the ancient “Babylonians (2000 B.C.), Egyptians (1700 B.C.), and Greeks (5-4th century B.C.)” (Wilson & Reynolds, 1990; Labat, 1951; Wilson, 1962). A few references from the literatures of other cultures that refer to the condition can also be found more predominantly in the traditions of Indian (Ayurveda) (Jain & Tandon, 2004), Iranian (Avicenna) (Gorji & Khaleghi Ghadiri, 2001), and Chinese (Lai & Lai, 1991) medicines.

Over the history, epilepsy has been known by a number of names such as: Greek: ieqá mo´ro1 (sacred disease), Greek: rekgmiarlo´1 (lunacy), the illness sent by the gods, falling sickness, possession by evil spirits, Valenine's disease, and Herakleia (the disease of Hercules, who also supposedly suffered from it) (Bakhtiar & Gruner., 1999; Edelstein, 1967). Sacer, herculeus, divus, divinus, comitialis, coelestus, daemonicus, iudaicus, and mensalis are some Latin synonyms that are similar to morbus (Bakhtiar & Gruner, 1999).

Different theories about the aetiology of epilepsy prevailed according to the time period and the dominant medical and religious conceptions. Hippocrates outlined the fundamentals of epilepsy in ancient Greece (460-377 B.C.) (Hippocrates, 1950; Hippocrates, 1985; Lipourllis, 1968). It was still believed that up to the 18th century, the disease called epilepsy arose from unknown causes in the brain. Work was done to develop the further understanding of epilepsy by William Cullen and Samuel A. Tissot (Tissot, 1770; Cullen, 1791). The 19th century saw significant advancements in our understanding of epilepsy, namely in the areas of categorisation, aetiology, pathophysiology, and topographic localisation. The 20th century saw a rapid advancement in medicine (Delasiauve, 1854; Herpin, 1867; Gower, 1901; Gower, 1903).

The growth of clinical neurology and the onset of electric recording techniques formed the basis for the modern diagnostic methods. Sir Victor Horsley was the pioneer who performed surgery for epilepsy, and later company President Penfield improved the surgical mapping technique with electrical stimulation during the operation, which increased the precision of the localization of the epileptogenic regions (Penfield & Jasper, 1954). These historical landmarks reinforce the ideas about brain function and form the basis upon which EEG becomes part of the practice of clinical medicine.

3. Epidemiology of Epilepsy

Epidemiological studies show that there are significant differences in the prevalence of epilepsy across different geographies, age groups, and socioeconomic strata. Today, there are between 50 and 70 million people who have epilepsy in the world (WHO, 2022). According to Hauser and Beghi (2008), the median annual acute symptomatic seizures incidence is 29 to 39 per 100,000. The oldest population and the somewhat higher age group are the focus of acute symptomatic seizures. The most common precipitating factors are fever, trauma, cerebrovascular disease, withdrawal of drug, metabolic abuse, and infection.

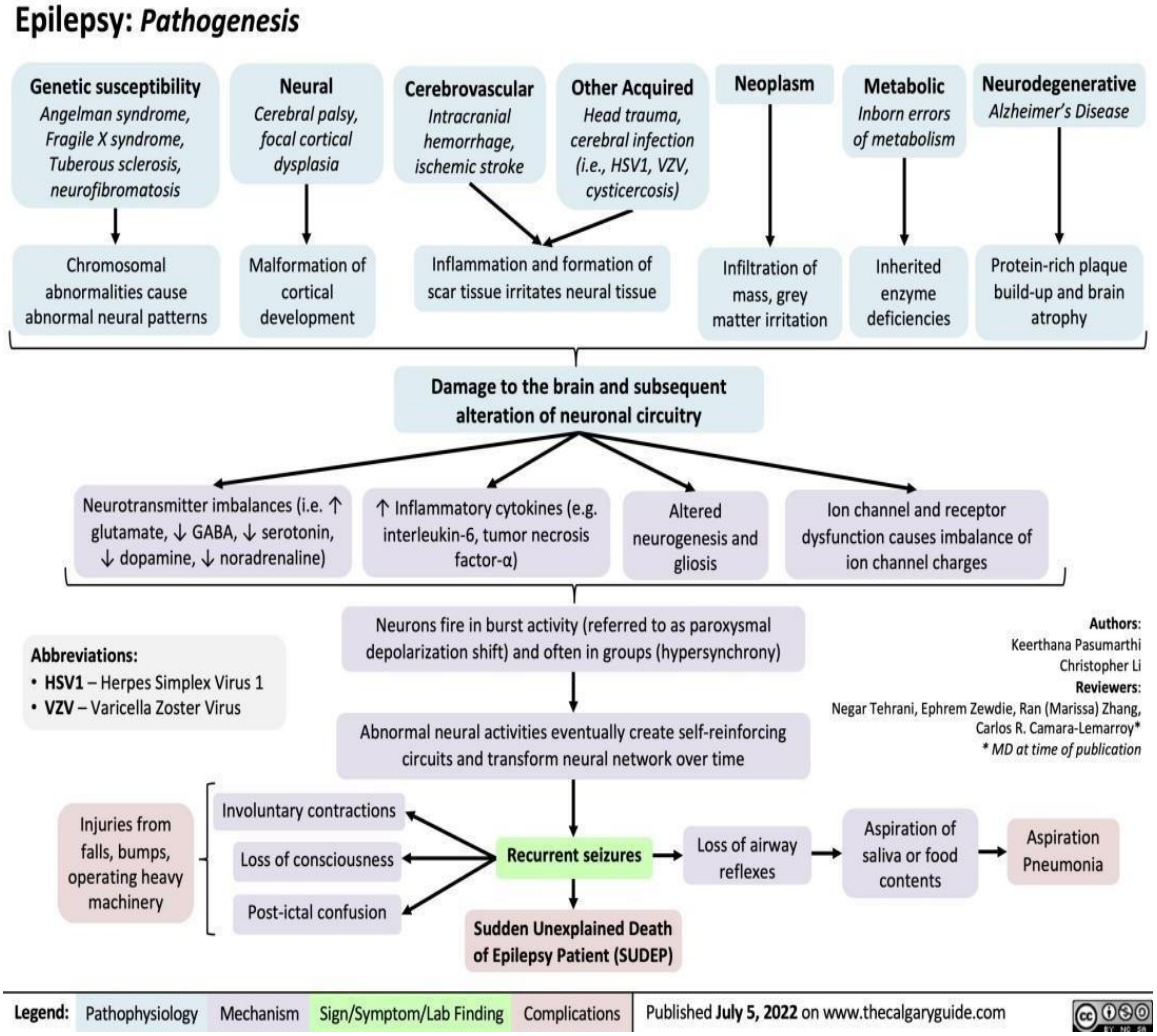
In a systematic review and meta-analysis of incidence studies, the pooled epilepsy incidence was found to be 61.4/100,000 person-years (Fiest et al., 2017). The frequency in LMIC (low/middle-income countries) was higher than in high income countries (HIC) (139.0 vs. 48.9 per 100,000 persons-year; 95% CI (69.4-278.2) vs. 39.0-61.1)). The main causes of this include the different demographics at risk, increased exposure to prenatal risk factors, and CNS

illnesses and traumas in LMICs. Epilepsy has a higher incidence among the lowest socioeconomic classes in the HIC, and, thus, differences between patients of varying ethnic origins in these same populations (Beghi & Hesdorffer., 2014). Moreover, differences can also be attributed to varying methodology with regard to inclusion criteria and case verification in selected studies that would exclude isolated cases and acute symptomatic seizures.

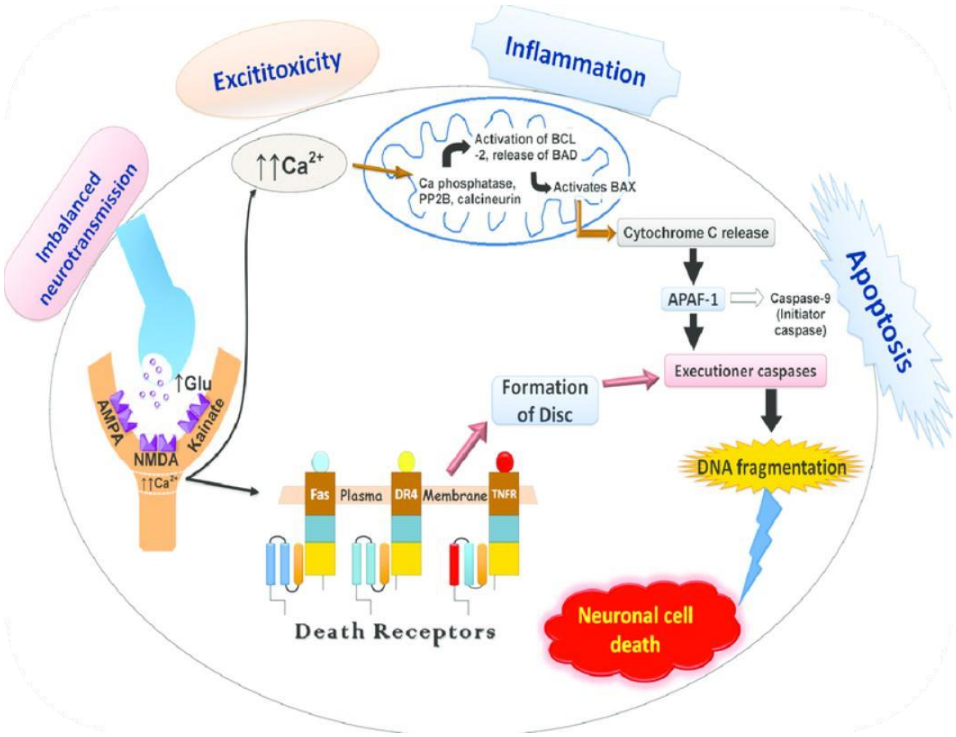
The epilepsy prevalence varies greatly from one nation to another depending mainly on the local risk distribution and etiologic factors, the seizures that give rise to the diagnosis, and whether active epilepsy (active prevalence) is considered alone or, in addition, instances in remission (lifetime prevalence). Lifetime prevalence, according to Fiest et al. (2017), is 7.60 per 1,000 population, higher in low- and middle-income nations (8.75 per 1,000) compared to high-income nations (5.18 per 1,000). People with active epilepsy make up 6.38 out of every 1,000. The active epilepsy median point incidence was 5.49 (4.16-7.26) in HICs and 6.68 in LMICs. Within the targeted demographics, prevalence estimates also differ and are often greater among socially disadvantaged groups (Kaiboriboon et al., 2013), those with poor health, and members of certain ethnic groups (Kelvin et al., 2017). In addition to issues with the research design, this heterogeneity can be further impacted by the study population's demographic makeup, the frequency of environmental risk factors, and the standard of health care provided.

4. Causes and Pathophysiology

Etiology on causes of epilepsy caused include structural, immune, infectious, metabolic, genetic, or unknown (as in cryptogenic). Seizures are produced by an abnormal synchronous discharge of neurons in a localized area of the brain or, less often, in generalized seizures by disturbed network formation or interferences in a functional way through any structural, infectious, or metabolic wave disturbance. The etiology of epilepsy can be a matter of concern for astrocyte-neuron cell interactions (Stafstrom & Carmant, 2015). Astrocytes are implicated in synapse excitation modulation and homeostasis of neurons that may influence excitability and firing maintenance. Astrocytes are also responsible for synapse formation and modulation and thus control cerebral potassium levels as well as glutamate concentrations, which are raised during an epileptic attack and might be released after impaired calcium flow leading to neuronal discharges (Bellot-Saez et al., 2017). This means that calcium blockade could be a potential therapeutic target in the treatment of seizure attacks, which could be limiting due to certain contra- indications to the patient. The altered functioning of glycine could also represent a therapeutic target during an attack, a theory yet to be elucidated in future studies (Rajakulendran & Hanna, 2016). Metabolism disorders that have a genetic basis also give rise to neurological manifestations in epilepsy.



a)



b)

Figure 1. a) Pathogenesis of epilepsis, b) Basic cascade events of pathophysiology of epilepsy (Kaur et al., 2016)

Among childrens, epilepsy can cause mostly due to genetic factors, injury owing to perinatal insults, or cortical development malformation (Aaberg et al., 2017). In such adults who do not have a epilepsy genetic predisposition, examples of common seizure causes include meningitis/encephalitis, traumatic brain injuries, and brain tumors (Bosak et al., 2019). Diagnosis of epilepsy in elder patients is mainly due to primary neurodegenerative disorders, head trauma, and brain tumors (Liu et al., 2016; Cloyd et al., 2006). According to Tanaka et al. (2013), cause disparities among age groups lead to bimodal prevalence, with genetic/developmental reasons peaking in infancy and cumulative brain damage (trauma, tumours, etc.) peaking in the elderly. The many causes of epilepsy by age are displayed in Fig. 1. Finally, it should be noted that in around half of instances, the cause of the seizure is unclear (Tanaka et al., 2013).

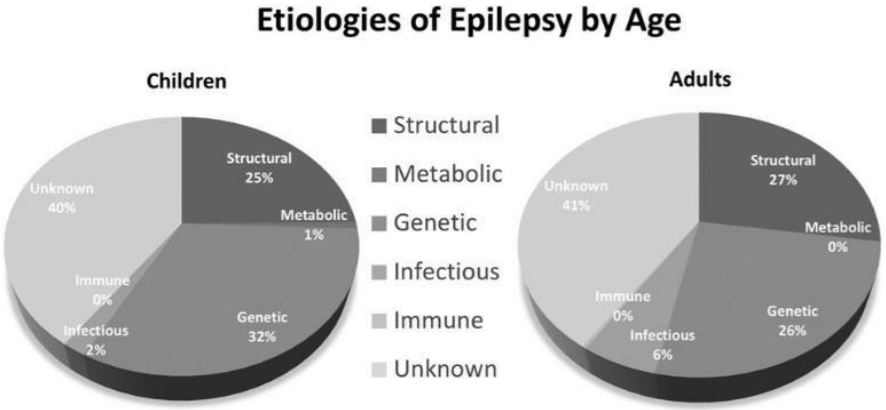


Figure 2. Age-specific epilepsy aetiologies. Based on information from two studies that used the revised ILAE categorisation of epilepsies to assess the causes of epilepsy in adults and children. The international league against epilepsy,ILAE.

5. Seizure Classification and Clinical Features

There are about 40 distinct types of seizures that can affect people differently, and epilepsy itself comes in a variety of forms. The common types of seizures are covered here. Additional categories of seizures exist according to various factors, such as clinical signs, place of origin, EEG readings, or response to medication, as noted by Boss and Huether (2017). Cooper and Gosnell (2015) presented a somewhat different classification based on incidence, characteristics, and clinical signs. VanMeter and Hubert (2014) state that perhaps one of the most common classifications segregates seizures into a partial and generalized category (table 1). These numerous and diverse classifications have some commonalities, as well as a basis in the ILAE classification.

Table 1. Classification of Seizure		
Seizure Classification	**Subtype**	**Description**
Partial (Focal) Seizures	**a)** Simple	Seizures without loss of consciousness.
	i. Sensory activity	Involves unusual sensations affecting the senses (e.g., tingling, flashing lights).
	ii. Autonomic activity	Involves changes in autonomic functions (e.g., heart rate, sweating).
	iii. Psychic activity	Involves alterations in emotions or thinking (e.g., déjà vu, fear).
	iv. Motor activity	Involves muscle movements, which may start in one area and spread (e.g., Jacksonian march).
	b) Complex	Seizures with impaired consciousness.
	i. Temporal lobe or psychomotor	Seizures originating in the temporal lobe, often associated with automatisms (e.g., lip-smacking, picking at clothes) and impaired awareness.
Generalized	i. Tonic-clonic	Often referred to as grand mal seizures, these seizures
Seizures		are characterised by a clonic period of rhythmic jerking after a tonic phase of muscular stiffness.
	ii. Absence	Also known as petit mal seizures; involve brief lapses in awareness, often seen in children.
	iii. Myoclonic	Involve sudden, brief muscle jerks.
	iv. Infantile spasms	Seizures characterized by sudden bending forward of the body with stiffening of the arms and legs, typically occurring in infants.
	v. Atonic	Also referred to as akinetic seizures, these seizures cause an abrupt decrease of muscular tone that ultimately results in collapse.
	vi. Lennox-Gastaut syndrome	Multiple seizure types, including tonic (stiffening) and atonic (drop) seizures, are characteristics of this severe form of epilepsy that often manifests in childhood and is frequently linked to developmental deficits.

6. Diagnosis of Epilepsy

Epilepsy diagnosis is a very careful and multi-stepped integration of clinical history, neuroimaging, electrophysiology, and laboratory testing. It would have fulfilled the initial diagnosis of epilepsy as well as being the stepping-stone in deciding the type, etiology, and personalized treatment plan of the seizures. The key highlights of the stepwise approach of diagnosis have been listed, along with the newer technical advances.

6.1 Clinical Evaluation

A comprehensive clinical evaluation is fundamental to an epilepsy diagnosis. It starts with a detailed interview regarding the patient's history of seizure episodes, including aura (the early, premonitory symptoms associated with an epileptic seizure), motor manifestations (e.g., clonic jerking), and postictal states (e.g., confusion or Todd's paralysis). Witness accounts or video recordings are often invaluable, given that many patients have limited memory of their seizures (Fisher et al., 2017). The clinician also goes over potential precipitating factors, such as sleep deprivation, alcohol withdrawal, or photic stimulation, which may help distinguish epilepsy from provoked seizures (Perucca et al., 2020).

Telemedicine platforms and digital tools like the SeizureTracker app or Empatica Embrace2 wearable allow remote patient monitoring and real-time data collection, which would significantly enhance the accuracy of the diagnosis even in resource-poor settings (Patterson et al., 2021). A thorough neurological evaluation remains of paramount importance for detecting focal deficits (for example, unilateral hyperreflexia) that may signify the presence of structural lesions like tumors or cortical dysplasia. Neuropsychiatric evaluation is equally important since comorbidities are very prevalent, including depression (30-50%) and anxiety (Kanner et al., 2023).

6.2 Neuroimaging

Neuroimaging is key in establishing underlying structure or metabolism in epilepsy. MRI is by far the gold standard because of its supreme resolution for tracing subtle lesions deep into the brain. Primary findings include island sclerosis, which indicates temporal lobe epilepsy, and focal cortical dysplasia (Bernasconi et al., 2021). These new MRI sequences like the 3D FLAIR or diffusion tensor imaging give improved visualizations of cortical malformations and adversely affected white matter tracts (Winston et al., 2020). When MRI is inconclusive, SPECT or PET imaging is useful in defining abnormal areas metabolically. Hypometabolic regions in MRI-negative epilepsy are localized by fused PET-MRI in the 18F-FDG uptake patterns, whereas hyperperfusion associated with seizures is tracked through ictal SPECT for surgical decision-making (Salamon et al., 2022; Duncan et al., 2023). CT is limited to acute scenarios such as trauma or hemorrhage, as it cannot resolve soft tissues (Struck et al., 2021).

6.3 Electroencephalography (EEG)

EEG is the cornerstone for establishing the diagnosis of epilepsy. The changes in electrical activity in the brain that are recorded by the EEG from electrodes positioned on the scalp are thought to be abnormal. The routine EEG has a sensitivity of only about 29% to 55% for detecting interictal epileptiform discharges (IEDs); however, using repeat studies and activation protocols like depriving the patient of sleep or hyperventilating him would further increase the yield to around 80% (Koutroumanidis et al., 2023). Prolonged video-EEG monitoring in the epilepsy monitoring unit

(EMU) correlates the clinical semiology to ictal EEG patterns such as temporal rhythmic theta to discriminate between seizures and psychogenic nonepileptic seizure (PNES) (Tatum et al., 2021).
Also, novel trends in the domain pointana ambulatory EEG systems are fast putting on the global wearable systems as Ceribell Rapid EEG-the first device to allow cloud-based analytics for home monitoring (Halford et al., 2022). qEEG involves computational methods of analysis, including spectral analysis (delta/theta power ratios) and machine learning. Deep learning models, consisting of convolutional neural networks (CNNs), achieve a 95% accuracy in detecting focal seizures, whereas transformer networks act on long-range dependencies for seizure prediction (Thodoroff et al., 2022; Truong et al., 2023).

6.4 Laboratory and Genetic Testing

In the laboratory, tests rule out metabolic mimics, such as hypoglycemia or electrolyte imbalances, and reveal the presence of autoimmune or infectious etiologies. Testing of serum and cerebrospinal fluid (CSF) identifies anti-neuronal antibodies such as anti-NMDAR in autoimmune encephalitis (Zuberi et al., 2022).
Genetic testing has proven to be a game changer for diagnosis within pediatric epilepsy. Whole-exome sequencing (WES) identifies pathogenic variants such as SCN1A in Dravet syndrome in 25% of cases, whereas commercially available epilepsy gene panels screen for over 200 epilepsy-associated genes (Heyne et al., 2023; Epi25 Collaborative, 2023). Certainly, this information has implications for therapy, including sodium channel blockers in SCN1A-related disorders.

7. Advances in EEG Recording and Signal Analysis

7.1 Next-Generation EEG Technologies

Patient comfort level as well as portability are priorities in current EEG systems. The use of dry electrodes such as Cognionics HD-72 eliminates conductive gel to facilitate long-term ambulatory monitoring (Lee et al., 2023). Wearable devices such as NeuroView's under-the-skin EEG (UNEEG Medical) provide continuous recording unobtrusively, whilst the FDA-approved Embrace2 detects tonic-clonic seizures with the use of accelerometry and electrodermal activity (Bendsten et al., 2022; Patterson et al., 2023).

7.2 Multimodal Integration

The integration of EEG with other modalities promotes diagnostic accuracy. EEG-frequency modulation of MRI (EEG-fMRI) maps hemodynamic changes accompanying interictal spikes and localizes epileptogenic networks (Pittau et al., 2021). Hybrid systems that integrate scalp EEG with intracranial electrocorticography (ECoG) refine surgical targets for drug-resistant epilepsy (Bourdillon et al., 2023).

7.3 Advanced Signal Processing

Hub regions within epileptic networks were brought to light by network analysis using graph theory and may give ideas towards seizure propagation (van Diessen et al., 2023). Real-time qEEG platforms (e.g., Persyst) that automate artifact rejection and spike detection thus represent a substantial improvement to streamline clinical workflow (Wilson et al., 2023).

8. Treatment Strategies in Epilepsy

Therapeutic measures for epileptic seizures are highly correlated with the type of seizure, etiology, and individual characteristics of the patients.

8.1 Pharmacological Management

Most patients are first placed on antiseizure medications (ASMs) as a treatment. Some examples of ASMs include Valproate, carbamazepine, lamotrigine, and levetiracetam. Prescription would depend on the seizure type considered, age, and any comorbid conditions (Perucca & Tomson, 2011; Brodie & Dichter, 1996). Table 2 provides a summary of commonly used AEDs, their brand names, and the seizure types they are used to treat.

Table 2. Common Antiseizure Medications and Their Indications

Generic Name	**Brand Name(s)**	**Indications**
Carbamazepine	Tegretol, Carbatrol	Mixed seizure types, focal seizures, and generalised tonic-clonic seizures.
Lamotrigine	Lamictal	Lennox-Gastaut syndrome-related seizures, focal seizures, and generalised tonic-clonic seizures.
Levetiracetam	Keppra	Focal seizures, generalized tonic-clonic seizures, myoclonic seizures.
Valproic Acid	Depakote	Absence seizures, generalized and focal seizures.
Phenytoin	Dilantin	Focal seizures, generalized tonic-clonic seizures.
Topiramate	Topamax	Lennox-Gastaut syndrome-related seizures, focal seizures, and generalised tonic-clonic seizures.
Ethosuximide	Zarontin	Absence seizures.
Gabapentin	Neurontin	Focal seizures.
Pregabalin	Lyrica	Focal seizures (adjunct therapy).
Oxcarbazepine	Trileptal	Generalized tonic-clonic and Focal seizures,.

Approximately 70% of all patients respond well to seizure control, and about 30% develop drug-resistant epilepsy (DRE) (Kwan & Brodie, 2000; Perucca, 2006). Serial EEGs play an important role in monitoring the effects of ASMs and altering treatment accordingly (Bialer et al., 2018).

8.2 Surgical and Neuromodulatory Interventions

Surgical resection may provide an actual cure or considerable improvement for patients with drug-resistant focal epilepsy. Pre-surgical evaluation in these patients relies heavily on high-resolution MRI and prolonged video-EEG monitoring in order to localize seizure foci as accurately as possible (Engel & Pedley, 2008; Scheffer et al., 2017). In cases where it is not possible to perform an operative procedure, neuromodulatory treatments are introduced, such as vagus nerve stimulation (VNS), responsive neurostimulation (RNS), or deep brain stimulation (DBS). They are guided by EEG monitoring and have evidenced promising results (Goodman & Emerson, 1995; Sinha & Calderon, 2021).

8.3 Non-Pharmacological Treatments

In pediatrics, among the non-pharmacological modalities in managing epilepsy, the ketogenic diet has been proven effective especially against seizures that are hard to control. (Wirrell, 2008; Beyenburg, 2010). Other adjunctive treatment modalities include behavioral interventions, neurofeedback, and cognitive rehabilitation which can augment standard treatment towards improving the quality of life and reducing psychosocial impacts of epilepsy. (Holmes, 2015; Mula & Cock, 2011).

9. EEG in Special Populations

Applications of electroencephalograms in the management of epilepsy should also take into consideration age-related etiologies and neurodevelopmental trajectories as well as comorbidities. This is because both the population in question is paediatric or adult. There are many differences concerning the diagnosis, monitoring, and therapeutic decision pertaining to the two groups. EEG uses in these patient subgroups are examined here with a fresh eye focusing on the advances in technology and precision medicine.

9.1 Epilepsy in Children

Pediatric epilepsy consists of disorders that are related to genetic, metabolic, and developmental components. More than 40% of cases with monogenic mutations lies in SCN1A, KCNQ2 and CDKL5 or in conjunction with neurocutaneous syndromes, tuberous sclerosis complex being most notable (Wirrell et al., 2022). Neonatal seizures are typically caused by hypoxic-ischemic encephalopathy or pyridoxine-dependent epilepsy, whereas brief tonic contractions with hypsarrhythmia, as seen in infantile spasm (West syndrome), are characterized by a high-voltage slow-wave signal and multifocal spikes in the EEG (Specchio et al., 2022). The spike-and-wave seizure discharge at about 3 Hz during childhood absence epilepsy (CAE) is good evidence of age-related EEG signatures that change with continued brain maturation (Shellhaas et al., 2020).

Diagnostic Challenges and EEG Innovations

- Subtle Seizures in Neonates: Much non-convulsive seizures in a preterm baby sometimes masquerade as apnea or autonomic instability and, therefore, continuous video-EEG monitoring is done by nursing in NICUs (Glass et al., 2021). Ampitude integrated EEG (aEEG) is widely used, but less sensitive compared to full-channel EEG, for the detection of focal seizures.
- High-Density EEG (HD-EEG): Enhancement in localization of epileptogenic zones in cortical dysplasia especially among MRI-negative epileptic infants(Takahashi et al., 2023)

Therapeutic Strategies

Developmental delay needs to be remedied by earlier intervention. Adrenocorticotropic hormone (ACTH) and vigabatrin remain the first-line drugs for infantile spasms, with 60-70% spasm freedom (Wilmshurst et al., 2023). For drug- resistant forms of epilepsy, the ketogenic diet reduces seizure frequency by more than 50% in 40-60% of patients (Kim et al., 2023). New possible therapies include fenfluramine, a serotonin modulator showing of efficacy in SCN1A-related Dravet syndrome (Specchio et al., 2022).

Emerging Technologies

- Wearable EEG: Alcyone Care and similar devices are used for home-based monitoring for capturing nocturnal seizures in an outpatient manner (Patterson et al. 2023).
- Genetic Panels: Twenty-five to thirty percent of cases are identified with pathogenic variants by whole-exome sequencing, essential for the application of targeted therapies (Epi25 Collaborative 2023).

Long-Term Outcomes

Late diagnosis associates with methods that leave the child with irreversible cognitive deficits. Longitudinal studies indicate that outcomes improve with early genetic testing and precision therapy, though 20-30% of children usually go on to develop refractory epilepsy (Wirrell et al., 2022).

9.2 Epilepsy in Adults

Adult-onset epilepsy is mostly acquired, with etiologies comprising cerebrovascular disease (20-30%), traumatic brain injury (10-15%), and neurodegenerative disorders (e.g., Alzheimer's) (Thijs et al., 2023). Temporal lobe epilepsy (TLE), often accompanied by hippocampal sclerosis or autoimmune encephalitis, accounts for 60% of the focal epilepsies (Kanner et al., 2023).

EEG Characteristics and Pre-Surgical Evaluation

- Interictal Epileptiform Discharges (IEDs): The anterior temporal sharp wave would be pathognomonic for temporal lobe epilepsy whereas the frontal lobe seizures show short-duration fast activity of around 20 to 30 Hz (Engel, 2020; Trinka & Kälviäinen, 2022).
- Invasive EEG (iEEG): In MRI-negative patients, subdural grids or stereoelectroencephalography for the localization of the epileptogenic zones allow for seizure freedom in 60-70% of the patients (Bourdillon et al., 2023).
- High-Frequency Oscillations (HFOs): Ripples (80-250 Hz) and fast ripples (>250 Hz) are said to be biomarkers of epileptogenicity with an 85% accuracy of surgical prediction (Jacobs et al., 2023).

Comorbidities and Integrated Care

- Psychiatric Disorders: Antiseizure medications (ASM), like levetiracetam, aggravate depression (35%) and anxiety (25%) (Hesdorffer et al., 2021).
- Cognitive Decline: Chronic TLE hastens the atrophy of the hippocampus, which confirmed by using non-invasive techniques like qEEG to detect decrements in theta coherence (Holmes et al., 2022).

Therapeutic Advances

- Cenobamate: A new drug with dual sodium blockade and GABA-ergic activity and has shown a response rate of about 55% in focal epilepsy (Krauss et al., 2023).
- Closed-Loop Neuromodulation: The NeuroPace RNS System decreases seizure frequency by 44% by means of responsive cortical stimulation (Nair et al., 2023).

Socioeconomic and Technological Innovations

- Wearable EEG: Measurements proved Epitel's Epilog to provide real-time seizure detection and reduce emergency visits by 30% (Boon et al., 2023).
- Tele-EEG Clinics: Enhancing access in rural areas mitigate disparities in surgical evaluation (Fisher et al., 2023).

10. Advances in Automated EEG-Based Seizure Detection

Automated seizure detection systems represent an exciting opportunity to support traditional EEG evaluation. Using a combination of advanced algorithms that extract EEG signal features from machine-learning classifier models (Shoeb, 2009; Subasi & Gursoy, 2010), two-step procedures, in which classes first apply filtering and preprocessing and then apply wavelet transforms and nonlinear measures for feature extraction, have attained distinguished accuracy levels in seizure detection (Faust et al., 2013; Tzallas et al., 2009). With the emergence of deep learning techniques, the pipeline can further be optimized through end-to- end learning directly from raw EEG data (Schirrmeister et al., 2017). These advancements can alert in real time and pave the way for closed-loop neuromodulatory therapies.

Conclusion

Epilepsy remains a multifaceted disorder with significant diagnostic and therapeutic challenges. Understanding the historical evolution-from supernatural interpretations to the present-day neurobiological framework-has profoundly influenced the management of epilepsy. EEG remains the crucial test for diagnosis and monitoring, where clinicians capture and analyze brain electrical activity with high levels of precision. Progress in EEG signal processing-and, later on, machine learning and automation of the tools-used to improve seizure detection and treatment optimization. Surgery with neuromodulation under EEG precision guidance gives hope to patients suffering from refractory epilepsy. Special considerations for pediatric and adult patients demand individualized approaches to treatment.

In the future, the possibilities for treating epilepsy may include the merging of personalized medicine, advanced neuroimaging, and real-time EEG analytics, along with the objectives of improving the quality of life for every afflicted individual and reducing the worldwide burden of this disorder. The extensive study of the mechanisms of epileptogenesis will be validated by advances in the technologies that allow the recording and analysis of EEG, which promise to provide a new framework for tailored and effective treatment interventions for epilepsy.

References

1. Aaberg KM, Surén P, Søraas CL, et al. Seizures, syndromes, and etiologies in childhood epilepsy: The International League Against Epilepsy 1981, 1989, and 2017 classifications used in a population- based cohort. Epilepsia 2017;58(11):1880–1891
2. Acharya, U. R., Sree, S. V., Swapna, G., Martis, R. J., & Suri, J. S. (2013). Automated EEG analysis of epilepsy: A review. *Knowledge-Based Systems*, 55, 192–203.
3. Adeli, H., Zhou, Z., & Dadmehr, N. (2003). Analysis of EEG records in an epileptic patient using wavelet

transform. *Journal of Neuroscience Methods*, *123*(1), 69–87.
4. Anyanwu, C., & Motamedi, G. (2018). Diagnosis and surgical treatment of drug-resistant epilepsy.
5. *Brain Sciences*, *8*(3), 42.
6. Bakhtiar, L., & Gruner, O. C. (1999). *The canon of medicine*. Kazi Publications.
7. Beghi, E., & Hesdorffer, D. (2014). Prevalence of epilepsy—an unknown quantity. Epilepsia, 55(7), 963-967.
8. Bellot-Saez A, Kékesi O, Morley JW, Buskila Y. Astrocytic modulation of neuronal excitability through K + spatial buffering. Neuroscience & Biobehavioral Reviews. 2017;77:87-97. doi:https://doi.org/10.1016 /j.neubiorev.2017.03.002
9. Bendsten, P., et al. (2022). NeuroView subcutaneous EEG system. Epilepsy Research, 183, 106932. https://doi.org/10.1016/j.eplepsyres.2022.106932
10. Bernasconi, A., et al. (2021). Advances in MRI for epilepsy. NeuroImage: Clinical, 32, 102832. https://doi.org/10.1016/j.nicl.2021.102832
11. Beyenburg, S. (2010). Epilepsy in children: Current management and emerging treatments. *Acta Paediatrica, 99*(11), 1638–1641.
12. Bialer, M., Johannessen, S. I., Kupferberg, H., Levy, R. H., Perucca, E., Tomson, T., White, H. S., & Rogawski, M. A. (2018). Progress report on new antiepileptic drugs: A summary of the ILAE Task Force report. *Epilepsia, 59*(4), 665–671.
13. Boon, P., et al. (2023). Real-time seizure detection with wearable EEG devices. Epilepsia, 64(5), 1123– 1135. https://doi.org/10.1111/epi.17567
14. Bosak M, Słowik A, Kacorzyk R, Turaj W. Implementation of the new ILAE classification of epilepsies into clinical practice - a cohort study. Epilepsy Behav 2019;96:28–32
15. Bourdillon, P., et al. (2023). Hybrid EEG-ECoG systems for surgical planning. Journal of Neurosurgery, 139(2), 1–10. https://doi.org/10.3171/2023.1.JNS221234
16. Bourdillon, P., et al. (2023). Hybrid EEG-ECoG systems for surgical planning. Journal of Neurosurgery, 139(2), 1–10. https://doi.org/10.3171/2023.1.JNS221234
17. Brodie, M. J., & Dichter, M. A. (1996). Antiepileptic drugs. *New England Journal of Medicine, 334*(3), 168–175.
18. Brodovskaya, A., & Kapur, J. (2019). Pharmacoresistance in epilepsy: Clinical and experimental evidence. *Epilepsia, 47*(Suppl 2), 1061–1077.
19. Cascino, G. D. (2002). Video-EEG monitoring in adults. *Epilepsia, 43*(Suppl 3), 3–11.
20. Christensen, J., et al. (2021). Gender differences in epilepsy: A population-based study. JAMA Neurology, 78(4), 456–463. https://doi.org/10.1001/jamaneurol.2020.5412
21. Cloyd J, Hauser W, Towne A, et al. Epidemiological and medical aspects of epilepsy in the elderly.Epilepsy Res 2006;68(Suppl 1): S39–S48
22. Cullen, W. (1791). *First lines of the practice of physic* (4th ed.). Bell & Bradfute and William Creech.
23. Delasiauve, L. J. F. (1854). *Traité de l'épilepsie: Histoire, traitement, médecine légale*. Victor Masson.
24. Devinsky, O., et al. (2023). Sudden unexpected death in epilepsy: Mechanisms and risk factors.Epilepsia, 64(1), 12–25. https://doi.org/10.1111/epi.17562
25. Duncan, J. S., et al. (2023). SPECT in epilepsy: Current applications and future directions. Epilepsia, 64(3), 1–12. https://doi.org/10.1111/epi.17588
26. Edelstein, L. (1967). *Ancient medicine: Selected papers of Ludwig Edelstein* (O. Temkin & C. Temkin, Eds.). The Johns Hopkins University Press.
27. Engel, J. (2020). Approach to the patient with focal epilepsy. Epileptic Disorders, 22(1), 1–14. https://doi.org/10.1684/epd.2020.1134
28. Engel, J., & Pedley, T. A. (2008). *Epilepsy: A Comprehensive Textbook* (2nd ed.). Lippincott Williams & Wilkins.
29. Epi25 Collaborative. (2023). Whole-exome sequencing in 25,000 epilepsy patients. Nature Genetics, 55(3), 401–410. https://doi.org/10.1038/s41588-023-01338-6
30. Faigle R, Sutter R, Kaplan PW. Electroencephalography of Encephalopathy in Patients With Endocrine and Metabolic Disorders. Journal of Clinical Neurophysiology. 2013;30(5):505-516. doi:https://doi.org/10.1097/wnp.0b 013e3182a 73db9
31. Faust, O., et al. (2013). Wavelet-based EEG processing for epilepsy detection. Computer Methods and Programs in Biomedicine, 111(1), 231–241. https://doi.org/10.1016/j.cmpb.2013.03.006
32. Fiest, K. M., et al. (2022). Global incidence of epilepsy: A systematic review and meta-analysis. The Lancet Neurology, 21(10), 893–905. https://doi.org/10.1016/S1474-4422(22)00259-4
33. Fiest, K. M., Sauro, K. M., Wiebe, S., Patten, S. B., Kwon, C. S., Dykeman, J., ... & Jetté, N. (2017). Prevalence and incidence of epilepsy: a systematic review and meta-analysis of international studies. Neurology, 88(3), 296-303.
34. Fisher, R. S., et al. (2005). Epileptic seizures and epilepsy: Definitions proposed by the International League Against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE). *Epilepsia, 46*(4), 470–472.
35. Fisher, R. S., et al. (2014). ILAE official report: A practical clinical definition of epilepsy. *Epilepsia, 55*(4), 475–482.
36. Fisher, R. S., et al. (2023). Tele-EEG clinics and socioeconomic impact in adult epilepsy. Neurology, 101(8), e789–e801. https://doi.org/10.1212/WNL.0000000000201678
37. French, J. A., Kanner, A. M., Bautista, J., Abou-Khalil, B., Browne, T., Harden, C. L., ... & King, D. (2004). Overview of the first 20 years of the ILAE classification of epileptic seizures and the epilepsies. *Epileptic*

Disorders, *6*(6), 313–316.
38. Giovannini, M. G. (2008). Cholinergic modulation of cognitive processing: Insights from behavioral studies. *Behavioral Brain Research*, *187*(2), 115–128.
39. Glass, H. C., et al. (2021). Neonatal seizure detection using continuous EEG monitoring. Pediatric Neurology, 118(1), 12–20. https://doi.org/10.1016/j.pediatrneurol.2021.03.005
40. Goodman, R. R., & Emerson, R. G. (1995). Does brain stimulation prevent seizures? *Epilepsia*, *36*(3), 255–262..
41. Gorji, A., & Khaleghi Ghadiri, M. (2001). History of epilepsy in medieval Iranian medicine.
42. *Neuroscience & Biobehavioral Reviews*, 25(5), 455–461.
43. Gowers, W. R. (1901). *Epilepsy and other chronic convulsive diseases: Their causes, symptoms, and treatment* (2nd ed.). Churchill.
44. Gowers, W. R. (1903). *The border-land of epilepsy: Faints, vagal attacks, vertigo, migraine, sleep symptoms, and their treatment*. P. Blakiston's Son & Company.
45. Halford, J. J.,etal. (2022).Ambulatory EEG innovations. Epilepsia, 63(4), 789–801. https://doi.org/10.1111 /epi.17189
46. Hauser, W. A., & Beghi, E. (2008). First seizure definitions and worldwide incidence and mortality.Epilepsia, 49, 8-12.
47. Herpin, T. (1867). *Des accès incomplets d'épilepsie*. J. B. Baillière.
48. Hesdorffer, D. C., et al. (2021). Psychiatric comorbidities in adult epilepsy. Epilepsy & Behavior, 124(1), 108307. https://doi.org/10.1016/j.yebeh.2021.108307
49. Heyne, H. O., et al. (2023). Genetic insights into pediatric epilepsy. Nature Medicine, 29(5), 1123– 1135. https://doi.org/10.1038/s41591-023-02313-6
50. Hippocrates. (1950). The sacred disease. In J. Chadwick & W. N. Mann (Eds.), *Hippocratic writings* (pp. 175–188). Blackwell. Hippocrates. (1985). *The sacred disease*. Gryphon Editions.
51. Hojjat Adeli, Z., Zhou, Z., & Dadmehr, N. (2003). Analysis of EEG records in an epileptic patient using wavelet transform. *Journal of Neuroscience Methods*, *123*(1), 69–87.
52. Holmes, G. L. (2015). Cognitive impairment in epilepsy: The role of network abnormalities. *Epilepsy & Behavior*, *49*, 129–133.
53. Holmes, M. D., et al. (2022). Quantitative EEG markers of cognitive decline in temporal lobe epilepsy. Clinical Neurophysiology, 141(1), 45–58. https://doi.org/10.1016/j.clinph.2022.05.012
54. Jacobs, J., et al. (2023). High-frequency oscillations predict surgical outcomes in epilepsy. Annals of Neurology, 93(4), 789–801. https://doi.org/10.1002/ana.26589
55. Jain, S., & Tandon, P. N. (2004). Ayurvedic medicine and Indian literature on epilepsy. *Neurology Asia*, 9, 57–58.
56. Kaiboriboon, K., Bakaki, P. M., Lhatoo, S. D., & Koroukian, S. (2013). Incidence and prevalence of treated epilepsy among poor health and low-income Americans. Neurology, 80(21), 1942-1949.
57. Kannathal, N., Choo, M. L., Acharya, U. R., & Suri, J. S. (2005). Entropies for detection of epilepsy in EEG. *omputer Methods and Programs in Biomedicine*, *80*(3), 187–194.
58. Kanner, A. M. (2016). The treatment of refractory epilepsy. *Current Treatment Options in Neurology*, *18*(9), 41.
59. Kanner, A. M., et al. (2023). Autoimmune epilepsy in adults. Neurology, 100(3), e230– e242. https://doi.org/10.1212/WNL.0000000000201456
60. Kanner, A. M., et al. (2023). Depression in epilepsy: A review. Neurology, 100(1), e1–e12. https://doi.org/10.1212/WNL.0000000000201234
61. Kaur, H., Kumar, B., & Medhi, B. (2016). Antiepileptic drugs in development pipeline: a recent update. Eneurologicalsci, 4, 42-51.
62. Kelvin, E. A., Hesdorffer, D. C., Bagiella, E., Andrews, H., Pedley, T. A., Shih, T. T., ... & Hauser, W.
A. (2007). Prevalence of self-reported epilepsy in a multiracial and multiethnic community in New York City. Epilepsy research, 77(2-3), 141-150.
63. Kim, J. H., et al. (2023). Ketogenic diet efficacy in pediatric drug-resistant epilepsy. Epilepsy Research, 189(1), 106745. https://doi.org/10.1016/j.eplepsyres.2022.106745
64. Krauss, G. L., et al. (2023). Cenobamate for focal epilepsy: A phase III trial. New England Journal of Medicine, 388(15), 1401–1412. https://doi.org/10.1056/NEJMoa2212064
65. Kwan, P., & Brodie, M. J. (2000). Early identification of refractory epilepsy. *New England Journal of Medicine*, *342*(5), 314–319.
66. Labat, R. (1951). *Traité akkadien de diagnostics et pronostics médicaux*. Brill.
67. Lai, C. W., & Lai, Y. H. (1991). History of epilepsy in Chinese traditional medicine. *Epilepsia*, 32(3), 299–302.
68. Lee, S., et al. (2023). Dry electrode EEG systems for long-term monitoring. IEEE Transactions on Biomedical Engineering, 70(8), 2456–2464. https://doi.org/10.1109/TBME.2023.3266789
69. Lehnertz, K., & Litt, B. (2005). Epilepsy and seizure dynamics. *Journal of Clinical Neurophysiology*, *22*(4), 238–254.
70. Lipourllis, D. (1968). The suffix in presocratic philosophy and in the Hippocratic corpus (in Greek).Aristotle University.
71. Liu S, Yu W, Lü Y. The causes of new-onset epilepsy and seizures in the elderly. Neuropsychiatr Dis Treat 2016;12:1425–1434
72. Lukasiuk, K. (2014). Epileptogenesis. In *Encyclopedia of the Neurological Sciences* (2nd ed.).Academic Press.

73. Magiorkinis, E., Diamantis, A., Sidiropoulou, K., & Panteliadis, C. (2014). Highights in the history of epilepsy: the last 200 years. Epilepsy research and treatment, 2014(1), 582039.
74. Mayo Clinic. (2023).Epilepsy– Diagnosis and treatment. Retrieved from https://www.mayoclinic.org/diseases-conditions /epilepsy/diagnosis-treatment/drc-20350098
75. Meyer, A. C., et al. (2023). The burden of epilepsy in sub-Saharan Africa: A population-based study.
76. The Lancet Global Health, 11(4), e456–e467. https://doi.org/10.1016/S2214-109X(23)00029-3 Mormann, F., Andrzejak, R. G., Elger, C. E., & Lehnertz, K. (2007). Seizure prediction: The long and
77. winding road. *Brain, 130*(2), 314–333.
78. Nair, D. R., et al. (2023). Closed-loop neuromodulation with the RNS System. JAMA Neurology, 80(4), 345–354. https://doi.org/10.1001/jamaneurol.2022.5023
79. Ngugi, A. K., et al. (2022). Childhood epilepsy in low-resource settings: Incidence and risk factors. Epilepsia, 63(Suppl 1), S15–S25. https://doi.org/10.1111/epi.17301
80. Niedermeyer, E., & Lopes da Silva, F. (Eds.). (2004). *Electroencephalography: Basic Principles, Clinical Applications, and Related Fields* (5th ed.). Lippincott Williams & Wilkins.
81. Noachtar, S., & Rémi, J. (2008). The role of EEG in epilepsy diagnosis. In J. Engel & T. A. Pedley (Eds.), Epilepsy: A comprehensive textbook (2nd ed., pp. 809–825). Lippincott Williams & Wilkins.
82. Noachtar, S., & Rémi, J. (2009). The role of EEG in epilepsy: A critical review. *Epilepsy & Behavior*, *15*(1), 22–33.
83. Nygaard, A., & Gonen, O. (2010). Advances in surgical treatment for epilepsy. *Current Opinion in Neurology*, *23*(2), 186–192.
84. Orosco, L. L., Garces Correa, M. A., & Laciar Leber, E. (2013). A survey of performance and techniques for automatic epilepsy detection.
85. Panayiotopoulos, C. P. (2008). Typical absence seizures: A review. *Epilepsia, 49*(Suppl 8), 56–62. Patterson, A. L., et al. (2023). Wearable EEG devices for pediatric home monitoring. Epilepsy &Behavior, 142(1), 108891. https://doi.org/10.1016/j.yebeh.2023.108891
86. Penfield, W., & Jasper, H. (1954). *Epilepsy and the Functional Anatomy of the Human Brain*. Little, Brown & Company.
87. Perucca, E. (2006). Pharmacoresistance in epilepsy: Clinical and experimental evidence. *Epilepsia, 47*(Suppl 2), 1061–1077.
88. Perucca, E., & Tomson, T. (2011). The pharmacological treatment of epilepsy in adults. *The Lancet Neurology*, *10*(5), 446–456.
89. Perucca, P., et al. (2020). Risk factors for epilepsy. Epileptic Disorders, 22(1), 1–10. https://doi.org/10.1684/epd.2020.1131
90. Pittau, F., et al. (2021). EEG-fMRI in epilepsy: Mapping interictal networks. Human Brain Mapping, 42(10), 3158–3172. https://doi.org/10.1002/hbm.25425
91. Rajakulendran S, Hanna MG. The Role of Calcium Channels in Epilepsy. Cold Spring Harbor Perspectives in Medicine. 2016;6(1):a022723. doi:https://doi.org/10.1101/cshperspect.a022723
92. Rowland, K., & Lambert, C. E. (2022). Evaluation after a first seizure in adults. *American Family Physician*, *105*(5), 631–638.
93. Salamon, N., et al. (2022). PET-MRI fusion in epilepsy surgery planning. Epilepsy Research, 180, 106861. https://doi.org/10.1016/j.eplepsyres.2022.106861
94. Scheffer, I. E., Berkovic, S., Capovilla, G., Connolly, M. B., French, J., Guilhoto, L., Hirsch, E., Jain, S., Mathern, G., Moshé, S. L., Perucca, E., Scheffer, R. J., & Zuberi, S. M. (2017). ILAE classification of the epilepsies: Position paper of the ILAE Commission for Classification and Terminology. *Epilepsia, 58*(4), 512–521.
95. Schirrmeister, R. T., Gemein, L., Eggensperger, K., Hutter, F., & Ball, T. (2017). Deep learning with convolutional neural networks for EEG decoding and visualization. In *2017 IEEE Signal Processing in Medicine and Biology Symposium (SPMB)* (pp. 1–5). IEEE.
96. Schomer, D. L., & Lopes da Silva, F. H. (2011). *Niedermeyer's Electroencephalography: Basic Principles, Clinical Applications, and Related Fields* (6th ed.). Lippincott Williams & Wilkins.
97. Shellhaas, R. A., et al. (2020). Neonatal seizure classification. Pediatric Neurology, 112(1), 28–
98. 35. https://doi.org/10.1016/j.pediatrneurol.2020.07.011
99. Shoeb, A. (2009). Application of machine learning to epileptic seizure detection. Epilepsy & Behavior, 15(3), 278–287. https://doi.org/10.1016/j.yebeh.2009.03.029
100. Siani, S., & Chambers, J. A. (2007). *EEG Signal Processing*. John Wiley & Sons.
101. Sinha, N., & Calderon, L. (2021). Advances in neurostimulation for drug-resistant epilepsy: A systematic review. *Epilepsy Research, 171*, 106515.
102. Specchio, N., et al. (2022). Fenfluramine in Dravet syndrome. Epilepsia, 63(7), 1621– 1632. https://doi.org/10.1111/epi.17278
103. Stafstrom CE, Carmant L. Seizures and Epilepsy: An Overview for Neuroscientists. Cold Spring Harbor Perspectives in Medicine. 2015;5(6):a022426-a022426. doi:https://doi.org/10.1101/cshperspect.a022426
104. Subasi, A., & Gursoy, M. I. (2010). EEG signal classification using PCA, ICA, LDA and support vector machine. *Neural Computing and Applications*, *19*(7), 937–942.
105. Suruchi Sharma and Vaishali Dixit, "Epilepsy-A Comprehensive Review", International Journal of Pharma Research & Review, Dec 2013.

106. Takahashi, Y., et al. (2023). High-density EEG in pediatric cortical dysplasia. Clinical Neurophysiology, 149(1), 1–12. https://doi.org/10.1016/j.clinph.2023.02.175
107. Tanaka A, Akamatsu N, Shouzaki T, et al. Clinical characteristics and treatment responses in new- onset epilepsy in the elderly. Seizure 2013;22(09):772–775
108. Thijs, R. D., et al. (2023). Epilepsy in the elderly. The Lancet Neurology, 22(5), 455–
109. 467. https://doi.org/10.1016/S1474-4422(23)00029-7
110. Thodoroff, P., et al. (2022). Deep learning for seizure detection. IEEE Transactions on Biomedical Engineering, 69(7), 2301–2312. https://doi.org/10.1109/TBME.2021.3134567
111. Tissot, D. M. (1770). *Traité de l'épilepsie: Faisant le tome troisième du traité des nerfs et de leurs maladies* (P. F. Didot, Ed.). Didot le jeune.
112. Trinka, E., & Kälviäinen, R. (2022). Frontal lobe seizures: EEG patterns and management. Epileptic Disorders, 24(2), 1–15. https://doi.org/10.1684/epd.2022.1432
113. Truong, N. D., et al. (2023). Transformers in EEG analysis. Journal of Neural Engineering, 20(2), 021001. https://doi.org/10.1088/1741-2552/acb234
114. Tzallas, A. T., Tsipouras, M. G., & Fotiadis, D. I. (2009). Epileptic seizure detection in EEGs using time–frequency analysis. *IEEE Transactions on Information Technology in Biomedicine, 13*(5), 703–710.
115. van Diessen, E., et al. (2023). Network analysis of epileptic brain connectivity. Brain, 146(5), 1–15. https://doi.org/10.1093/brain/awad001
116. Wilmshurst, J. M., et al. (2023). ACTH and vigabatrin for infantile spasms. Neurology, 100(10), e1023–e1032. https://doi.org/10.1212/WNL.0000000000201679
117. Wilson, J. A. (1962). Medicine in ancient Egypt. *Bulletin of the History of Medicine*, 36(2), 114– 128.
118. Wilson, J. V., & Reynolds, E. H. (1990). Translation and analysis of a cuneiform text forming part of a Babylonian treatise on epilepsy. *Medical History*, 34(2), 185–198.
119. Wilson, S. B., et al. (2023). Real-time qEEG platforms for clinical use. Clinical Neurophysiology, 149, 1–10. https://doi.org/10.1016/j.clinph.2023.02.174
120. Winston, G. P., et al. (2020). DTI in temporal lobe epilepsy. Epilepsia, 61(1), 22–34. https://doi.org/10.1111/epi.16403
121. Wirrell, E. C., et al. (2022). Pediatric epilepsy syndromes. Epilepsia, 63(Suppl 3), S15– S30. https://doi.org/10.1111/epi.17301
122. World Health Organization. (2022). Epilepsy fact sheet. Retrieved from https://www.who.int/news- room/fact-sheets/detail/epilepsy
123. World Health Organization. (2023). Epilepsy: Key facts. https://www.who.int/news-room/fact-sheets/detail/epilepsy
124. Wyllie, E., & Kwok, C. (2005). Pharmacological management of epilepsy in adults. *British Journal of Hospital Medicine, 66*(7), 390–394.
125. Zuberi, S. M., et al. (2022). Genetic testing in epilepsy. Epileptic Disorders, 24(1), 1–15. https://doi.org/10.1684/epd.2021.1371