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# Types of Epilepsies and Findings EEG- LORETA about Epilepsy

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***Abstract*—**Neural activity in the human brain starts from the early stages of prenatal development. This activity or signals generated by the brain are electrical in nature and represent not only the brain function but also the status of the whole body. At the present moment, three methods can record functional and physiological changes within the brain with high temporal resolution of neuronal interactions at the network level: the electroencephalogram (EEG), the magnet oencephalogram (MEG), and functional magnetic resonance imaging (fMRI); each of these has advantages and shortcomings. EEG recording with a large number of electrodes is now feasible in clinical practice. Multichannel EEG recorded from the scalp surface provides very valuable but indirect information about the source distribution. However, deep electrode measurements yield more reliable information about the source locations intracranial recordings and scalp EEG are used with the source imaging techniques to determine the locations and strengths of the epileptic activity. As a source localization method, Low Resolution Electro-Magnetic Tomography (LORETA) is solved for the realistic geometry based on both forward methods, the Boundary Element Method (BEM) and the Finite Difference Method (FDM)**.** In this paper, we review the findings EEG- LORETA about epilepsy**.**

***Keywords*—**Epilepsy, EEG, EEG- Loreta, loreta analysis.

## I. Introduction

EPILEPSY is one of the most common serious brain disorders, which can occur at all ages and has different possible presentations and causes. The two major categories of epilepsies and epilepsy syndromes are referred to as localization related (also: focal or partial epilepsy) or generalized [1]. Epilepsy is a brain disorder characterized by recurrent seizures stemming from abnormal electrical behavior of brain cells. Surgery that removes the epileptogenic region is the preferred treatment for some patients with intractable epilepsy [2] The idea of accurately localizing an epileptic focus using EEG is appealing due to the noninvasive nature of EEG and its low cost. Several methods have been studied in an attempt to solve the generic EEG inverse problem, and some work has been devoted to the specific clinical application of localizing an epileptic focus. These so-called source localization methods vary in the relevant featuresextracted and in the time scale assumed to be needed for correct localization of the source [3]-[5].

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Computerized source localization methods can use short episodes (50 milliseconds) of spatiotemporal EEG data to extract the relevant feature of a typical spike. These features are entered into an inverse algorithm. For ictal recording periods, spatiotemporal dipole modeling techniques can identify the epileptic activity at seizure onset and use either the characteristics of the spikes (e.g., minima and maxima), or the characteristics of the averaged data of similar spikes [5]-[7] The two pillars of the pathophysiological concept of idiopathic generalized epilepsy (IGE) are pathological thalamocortical interactions and the so-called mild diffuse epileptogenic state of the cortex. The latter term refers to the ictogenic property of the cortex, a persistent abnormality that was demonstrated in animal models, and neurochemical evidence suggested its existence in humans [8], [9] Ictal and interictal epileptic activity of the cortex can be best grasped by measuring its electromagnetic activity. In fact, a pioneering quantitative EEG (QEEG) observation [10] and a systematic interictal QEEG study [11] revealed that the regional distribution of spectral power is characteristically abnormal in untreated IGE patients.

## II. Localisation Related Epilepsies

### *A. EEG Source Imaging*

Electric Source Imaging (ESI) is increasingly being recognized as a valuable noninvasive technique to localize the epileptic focus in partial epilepsies. The method has mostly been applied to the analysis of interictal epileptic activity [11] but some studies have also looked into its ability to localize the onset of seizures [12] In combination with high resolution EEG systems (128-256 channels), source imaging in epilepsy is now possible with excellent localizing precision. Preoperative EEGs recorded from 19 to 29 scalp electrodes in 30 pediatric patients were reviewed, and interictal epileptiform activity was analyzed by using a linear source-imaging procedure in combination with statistical parametric mapping.

The ESI localization was considered as correct when the majority of the active voxels were within the resected area that rendered the patients seizure-free or almost seizure free. In 90% of the patients, the ESI result was correct. This number compared favorably with the results from the other functional imaging techniques in the same patients (PET correct in 82%; ictal SPECT correct in 70%). In the cases with extratemporal epilepsy, ESI was correct in all cases (100%), however, in those with temporal lobe epilepsy, only 10 of 13 (77%) cases were correct. In two of the three patients with incorrect ESI, a 128-channel recording was performed, leading to correct localization in both cases. Thus, while ESI analysis based on

routine long-term recordings with a limited electrode number provided already good localization in patients with extratemporal lobe epilepsy, patients with basal temporal foci benefit from larger electrode set-ups, since these foci are outside the standard electrode array. Further, we evaluated the question of how many electrodes are needed to correctly identify the epileptic focus. 14 patients with partial epilepsy were recorded with 123-channel EEG [13].

Epileptic discharges were determined on the basis of the 123-channel recordings and the electrode configuration was later down sampled to 63 and 31 electrodes. The correct underlying sources were then estimated for the three different electrode configurations, by determining the distance from the inverse solution maximum of each single spike to the epileptogenic lesion [14].

### *B. EEG Imaging*

Absence seizures were recorded with dense-array256-channel scalp EEG in five subjects with primary generalized epilepsy [15]. Source analysis was applied to the spike components of each spike-wave burst in each seizure. The onset of seizures was typically associated with activation of discrete, often unilateral areas of the dorsolateral frontal or orbital frontal lobe. Although each patient showed unique features, the absence seizures of all patients showed rapid, stereotyped evolution to engage both mesial frontal and orbital frontal cortex sources during the repetitive cycles of spike-wave activity. This study indicates that absence seizures may not be "generalized" in the sense of simultaneous diffuse activation but involve highly localized activations from mesial frontal and frontopolar sources in both hemispheres. Focal, in particular frontal, seizures with rapid contralateral propagation may mimic primary generalized epilepsy. ESI should have the capacity to differentiate both conditions, which implies different treatment and prognosis. Rapid contralateral propagation, also termed secondary bisynchrony, has been investigated in patients with tuberous sclerosis (and multiple lesions ["tubers"] in both hemispheres). Seri et al. studied the topographic relationships between cortical and subcortical lesions shown on magnetic resonance images MRI) and sources of epileptiform activity in a series of nine children with intractable epilepsy and tuberous sclerosis complex [16]. Although video-EEG monitoring was suggestive of a unilateral frontal seizure onset, interictal EEG was, in seven of nine cases, in the form of apparently bisynchronous discharges. In all cases, the use of a short time lag estimation procedure based on a nonlinear correlation function between surface recorded EEG signals allowed the detection of a lateralized onset of EEG paroxysmal activity. Furthermore, ESI coregistered with the patient's MRI provided good topographic concordance between well-defined frontal cortical lesions shown on MRI and site of onset of "generalized" discharges. These findings are of major relevance for the patient's care, since focal onset in one tuber offers the possibility of surgical epilepsy treatment whereas the presence of diffuse, truly bilateral discharges does not [17].

Epilepsy is a common medical and social disorder or group of disorders with unique characteristics. Epilepsy is usually defined as a tendency to recurrent seizures. The word "epilepsy" is derived from Latin and Greek words for "seizure" or "to seize upon". This implies that epilepsy is an ancient disorder; indeed, in all civilizations it can be traced as far back as medical records exist. In fact, epilepsy is a disorder that can occur in all mammalian species, probably more frequently as brains have become more complex. Epilepsy is also remarkably uniformly distributed around the world [18].

## II. Encephalopathy Epilepsies

### *A. Early Myoclonic Epilepsy (EME)*

1) Seizure types: Erratic focal myoclonus, focal clonic or tonic seizures, tonic spasms; less commonly: massive bilateral myoclonus, epileptic spasms.
2) Ictal EEG: Onset of focal seizures are similar to neonatal seizures; generalized discharges are seen during massive myoclonus; no EEG correlate for erratic myoclonus.
3) Interictal EEG: Burst-suppression with loss of normal background features; silent periods last 3-10 sec.

### *B. Ohtahara Syndrome (OS)*

1) Seizure types: Tonic spasms, focal clonic or hemiclonic seizures; less commonly myoclonus.
2) Ictal EEG: Burst episodes may be accompanied by tonic spasms.
3) Interictal EEG: Burst-suppression with loss of normal background features; silent periods last 10-20 sec.

### *C. West Syndrome (WS)*

1) Seizure types: Epileptic spasms, tonic seizures, drop attacks; less commonly: focal clonic seizures
2) Ictal EEG: High voltage slow wave followed by electrodecrement during epileptic spasms (slow wave correlates with spasm); low voltage fast activity; focal spikes may associated with cluster of spasms
3) Interictal EEG: Hypsarrhythmia or its variants (modified hypsarrhythmia); multifocal spikes

### *D. Lennox-Gastaut Syndrome (LGS)*

1) Seizure types: Tonic seizures, atypical absences, atonic seizures, focal seizures, tonic status; less commonly tonic-clonic seizures, absence status; myoclonus in myoclonic variant of LGS
2) Ictal EEG: Paroxysmal fast activity or polyspikes during tonic seizures; various patterns including slow spike-waves, high-voltage spikes, low-voltage fast activity, electrodecrement, or no change during atypical absences
3) Interictal EEG: Slow spike-waves (<2.5 Hz); paroxysmal fast activity (10-25 Hz); low-voltage fast activity; slow and disorganized background in wake and sleep; multifocal slow waves; focal or multifocal spikes; atypical spike-waves, polyspike-waves

*E. Epilepsy with Continuous Spike-Waves during Sleep ECSWS*

The LKS form of ECSWS (formerly Landau-Kleffner syndrome) is characterized by aphasia and temporo-parietal location of epileptic activity whereas the non-LKS form is characterized by cognitive and behavioral changes and frontal location of epileptic activity. In both forms, seizures occur in the majority (but not all cases) before or after the onset of aphasia or neurobehavioral manifestations and frequent bilateral spikes and/or CSWS (detected with all-night EEG, polysomnography, or video-EEG monitoring) is required for diagnosis.

1) Seizure types (non-active phase): Focal clonic or hemiclonic seizures, complex partial seizures, secondary generalized tonic-clonic seizures; (active phase): atypical absences, atonic seizure, drop attacks, most seizures with onset before the active phase persist and become more frequent during the active phase; interictal CSWS pattern (see EEG below):
2) Ictal EEG: Non-active phase: focal seizures are infrequent; onset of focal seizures (including those that are secondarily generalized) usually temporal or parietal in LKS and frontal in non-LKS; active phase: atypical absences and atonic seizures occur with or without ictal EEG correlate; focal seizures are frequent; seizure onset usually temporal or parietal in LKS and frontal in non-LKS
3) Interictal EEG (non-active phase): In wake: Focal slow waves, spikes, and spike-waves, temporoparietal in LKS, frontal in non-LKS; infrequent bursts of bilateral 2-3 Hz spike-waves, absent in many LKS and some non-LKS; in sleep: increase amount of bilateral 2-3 Hz spike-waves but still < 85% of slow wave sleep; sleep spindles may be attenuated or absent in non-LKS patients; (active phase): in wake: abnormalities as in non-active phase but in greater amounts; In sleep: CSWS – nearly continuous bilateral 2-3 Hz spike-waves occupying > 85% of slow wave sleep; fragment or disappear in REM sleep; sleep architecture is preserved; intracranial EEG in LKS: focus of interictal activity in the posterior temporal lobe (often in superior temporal gyrus) and occasionally within the sylvian fissure near Heschl's gyrus.

*F. Migrating Focal Seizures in Infancy (MFSI*

1) Seizure types: Focal seizures with variable motor and autonomic manifestations, secondary generalized tonic-clonic seizures (later); less commonly: myoclonic seizures, epileptic spasms
2) Ictal EEG: Focal rhythmic theta appears at seizure onset decreasing in frequency as it spreads; subclinical seizures are common; as the number of independent seizure foci increase, seizures may begin before other seizures end and seizures may overlap as they spread producing a "migrating" picture.
3) Interictal EEG: Multifocal spikes, mainly temporal, occipital, and rolandic; background slowing at onset or develops later, usually shifting in laterality

*G. Dravet Syndrome (DS)*

1) Seizure types: Febrile seizures, clonic, hemiclonic or tonic-clonic seizures, erratic myoclonus, myoclonic absences, atypical absences, massive bilateral myoclonus, tonic seizures, myoclonic status, tonic-clonic status; non-epileptic segmental myoclonus
2) Ictal EEG: Electrodecrement followed by slow spike-waves are associated with tonic-clonic seizures; tonic-clonic seizures as in idiopathic epilepsies except initial tonic phase is vibratory due to high frequency clonic activity; polyspike-waves occur with myoclonic jerks; slow spike-waves (2-3.5 Hz) appear during atypical absences; no EEG correlate for multifocal erratic myoclonus
3) Interictal EEG: Slow spike-waves and polyspike-waves; focal or multifocal spikes, usually central or posterior; rhythmic central theta activity; background activity is usually normal at onset; may remain normal for years before becoming slow.

*H. Myoclonic Encephalopathy in Nonprogressive Disorders (MEND)*

1) Seizure types: Myoclonic status, erratic myoclonus, myoclonic absences, atypical absences, massive myoclonus, focal clonic seizures, hemiclonic seizures, generalized clonic or tonic-clonic seizures, febrile convulsions: non-epileptic paroxysms: massive startles, intentional tremor.
2) Ictal EEG: Slow spike-waves with bilateral myoclonus or myoclonic absences; bursts of bilateral spike-waves or slow waves alternating with periods of bicentral theta during myoclonic status; no EEG correlate for erratic myoclonus.
3) Interictal EEG: Multifocal slow waves and spike-waves; intermittent bifrontal delta and bilateral parieto-occipital spike-waves; background slowing is also present

*I. Progressive Myoclonus Epilepsy (PME)*

1) Seizure types: Focal or multifocal myoclonus, bilateral myoclonus, tonic-clonic seizures, atypical absence seizures, focal motor seizures, complex partial seizures, myoclonic status.
2) Ictal EEG: Low-amplitude focal myoclonic jerks usually have no EEG correlate (both in the conventional and back-averaged EEG); focal or multifocal spikes may be present but are often independent interictal phenomena with no definite relation to the myoclonic jerks; large-amplitude focal myoclonic jerks may be associated with focal slow waves, spike-waves, or polyspike-waves in the conventional EEG and jerk-locked EEG back-averaging often reveals a large positive-negative pre-myoclonic cortical potential even when conventional EEG fails to show any change; with myoclonic jerk of the hand, the cortical potential has a latency of about 20 ms and a voltage maximum in the contralateral central region; bilateral myoclonus is often associated with bifrontal slow waves, spike-waves, or polyspike-waves; distinguishing

the multifocal cortical myoclonus of PME from the thalamocortical myoclonus of idiopathic epilepsy can be difficult if there is rapid inter- and intrahemispheric spread of focal myoclonus.

EMG-EEG polygraphy: Ballistic or tonic EMG pattern (50-300 msec agonist burst, asynchronous or synchronous antagonist burst) and absence of an EEG correlate indicates non-epileptic myoclonus; reflex EMG pattern (10-100 msec agonist burst, synchronous antagonist burst or silent period) and a cortical spike detected by routine EEG or burst-locked back-averaging indicates epileptic myoclonus.

3) Interictal EEG: Focal or multifocal spikes; the spikes are frequently independent interictal phenomena with no definite relation to the myoclonic jerks; vertex positive spikes suggest sialidosis; the background activity may be normal initially; with disease progression, mild slowing in the theta frequency range occurs in wake and the sleep background becomes disorganized; ultimately, both wake and sleep background activity become extremely slow and disorganized.

## III. Idiopathic Generalized Epilepsies

### *A. Benign Myoclonic Epilepsy in Infancy (BMEI)*

1) Seizure types: Bilateral myoclonus; simple febrile seizures preceding the myoclonus
2) Ictal EEG: Bisynchronous fast (>3-Hz) spike-waves and polyspike-waves with myoclonic jerks
3) Interictal EEG: Normal for age, central theta activity, bisynchronous spike-waves in REM sleep

### *B. Childhood Absence Epilepsy (CAE)*

1) Seizure types: Typical absences only in childhood; tonic-clonic seizures may begin after childhood
2) Ictal EEG: 3Hz spike-waves with typical absence (4–20 sec)
3) Interictal EEG: 3Hz spike-waves (<4 sec) including fragments, bioccipital 3-Hz slow waves

### *C. Juvenile Absence Epilepsy (JAE)*

1) Seizure types: Typical absences, tonic-clonic seizures, less commonly myoclonic seizures
2) Ictal EEG: 3Hz spike-waves with typical absence, slightly faster and longer than in CAE; EEG correlate of myoclonus or tonic-clonic seizures (see JME)
3) Interictal EEG: 3Hz spike-waves including fragments; background is usually normal

### *D. Juvenile Myoclonic Epilepsy (JME)*

1) Seizure types: Bilateral myoclonus, fragments of bilateral myoclonus, tonic-clonic seizures, atypical absences, reflex myoclonus
2) Ictal EEG: Polyspike-waves with the spike component time-locked to the myoclonic EMG burst; similar discharges with rhythmic myoclonic jerks in the early clonic phase preceding tonic-clonic seizures; 3Hz spike-waves or rarely slow spike-waves during absences
3) Interictal EEG: Atypical spike-waves (4-6 Hz) including fragments; 3Hz spike-waves (1/5 of cases)

### *E. Epilepsy with Generalized Tonic-Clonic Seizures Only (EGTCS)*

1) Seizure types: Tonic-clonic seizures (no other seizure type)
2) Ictal EEG: Polyspikes during myoclonus or clonus in the pre-tonic phase of tonic-clonic seizures
3) Interictal EEG: Atypical spike-waves (4-6 Hz) including fragments; 3Hz spike-waves (1/5 of cases)

### *F. Generalized Epilepsy with Febrile Seizures Plus (GEFS+)*

1) Seizure types: Typical febrile seizures (age < 6 years), febrile seizures plus (age > 6 years), afebrile tonic-clonic seizures; less commonly absences, myoclonus, atonic seizures; some members of GEFS+ families have EMAS or DS
2) Ictal EEG: Pattern consistent with tonic-clonic seizure during febrile seizures; 3Hz spike-waves with l absence seizures; polyspikes during myoclonus
3) Interictal EEG: 3Hz spike-waves; atypical spike-waves; polyspike-waves

### *G. Epilepsy with Myoclonic Absences (EMA)*

1) Seizure types: Myoclonic absences, tonic-clonic seizures, less commonly typical absences, myoclonic absence status, atonic seizures
2) Ictal EEG: 3Hz spike-waves with or without bifrontal delta waves at the end occur during myoclonic absences with the spike components time-locked to the myoclonic jerks (also 3 Hz)
3) Interictal EEG: 3Hz spike-waves, focal or multifocal spike-waves, normal background

### *H. Epilepsy with Myoclonic-Astatic Seizures (EMAS)*

1) Seizure types: Myoclonic-atonic seizures, myoclonic seizures, atypical absences, myoclonic status with obtundation, less commonly tonic-clonic seizures, tonic seizures
2) Ictal EEG: 3-Hz polyspike-waves followed by EMG silence (up to 500 msec) during myoclonic-atonic seizures: the spikes are time-locked with the jerks and the EMG silence coincides with atonia.
3) Interictal EEG: 3Hz spike-waves; parietal theta activity (4-7 Hz); occipital delta or theta (4-Hz).

## V. Idiopathic Focal Epilepsies

### *A. Benign Familial Neonatal Seizures (BFNS)*

1) Seizure types: Focal tonic or clonic seizures with or without automatisms, apnea is common and usually occurs first; seizure types are often mixed
2) Ictal EEG: Diffuse attenuation at the onset of focal seizure
3) Interictal EEG: Often normal; theta pointu alternant (~2/3 of cases); other non-specific abnormalities

*B. Benign Childhood Epilepsy with Centrotemporal Spikes (BCECTS)*

1) Seizure types: Rolandic seizures manifesting as paresthesia of oral mucosa, hypersalivation, and contractions of face, tongue, and pharyngeal muscles resulting in dysarthria, dysphagia, and drooling; spread to motor cortex result in jerking of the arm, rarely the leg; secondary generalization
2) Ictal EEG: Focal centrotemporal attenuation or low-voltage fast activity at the onset of seizure may evolve into spike discharge and spike-wave activity; onset may shift sides; may also generalize
3) Interictal EEG: Rolandic or centro-midtemporal spikes; less frequently: bisynchronous spike-waves, occipital spikes, frontal spikes, multifocal spikes; background activity is usually normal

*C. Early-Onset Benign Childhood Occipital Epilepsy (BCEOS-1)*

1) Seizure types: Autonomic seizures: ictal nausea ± emesis ± other autonomic changes, deviation of eyes ± head; visual seizures less common than BCEOS-2; rolandic and other focal seizures; secondary generalization; focal status epilepticus (common); ictal syncope (rare);
2) Ictal EEG: Occipital rhythmic theta or delta activity at the onset of seizure; bifrontal ictal onset
3) Interictal EEG: IED morphology resembles rolandic spikes but with variable foci including occipital, rolandic, frontal, and bifrontal; frequently multifocal; normal wake and sleep background activity

*D. Late-onset Childhood Occipital Epilepsy (BCEOS-2)*

1) Seizure types: Visual seizures; spread to rolandic area, motor cortex or temporal lobe; secondary generalization; focal status epilepticus; postictal headache common and misdiagnosed as migraine
2) Ictal EEG: Occipital and posterior temporal fast spike discharge at onset of seizure
3) Interictal EEG: Occipital spikes with stereotyped morphology and prominent occipital voltage peak; background activity is usually normal in wake and sleep

*E. Autosomal Dominant Nocturnal Frontal Lobe Epilepsy (ADNFLE)*

1) Seizure types: Frontal lobe seizures manifesting as paroxysmal arousal, nocturnal paroxysmal dystonia, episodic nocturnal wanderings (rare); secondary generalization
2) Ictal EEG: Often obscured by artifacts; no correlate (~1/2 of cases); seizure onset in NREM sleep: focal frontal/frontotemporal attenuation, rhythmic theta/delta, or fast activity; temporal-onset (~14%)
3) Interictal EEG: No IED (~1/2 of cases); wake IED (~1/3 of cases), sleep IED (~1/2 of cases); frontal or frontotemporal (~2/3), temporal (~1/3); wake-sleep background is usually normal

*F. Familial Temporal Lobe Epilepsy (FTLE)*

1) Seizure types: Mesial temporal lobe seizures, lateral temporal lobe seizures, secondary generalization; history of febrile seizures is common in mesial-FTLE; déjà vu is a common aura in some mesial-FTLE series and auditory auras are common in lateral-FTLE
2) Ictal EEG: The two FTLE phenotypes, mesial-FTLE and lateral-FTLE, share the electroclinical features of MTLE and LTLE, respectively
3) Interictal EEG: Frequently normal; occasionally, anterior temporal or posterior temporal spikes or sharp waves

*G. Familial Focal Epilepsy with Variable Foci (FFEVF)*

1) Seizure types: Frontal lobe seizures, temporal lobe seizures; rarely occipital and parietal lobe seizures; seizures types are constant for each individual, vary among members of the same family
2) Ictal EEG: Variable and depends on the seizure type
3) Interictal EEG: Frequently normal; occasionally, spikes or sharp waves are detected in the frontal, temporal, or other areas

## V. Symptomatic Focal Epilepsies

*A. Mesial Temporal Lobe Epilepsy (MTLE)*

1) Seizure types: Mesial temporal seizures, mesial temporal auras, mesial temporal seizures with spread to lateral temporal neocortex, mesial temporal seizures with extratemporal spread, mesial temporal seizures with secondary generalization; less commonly complex partial status, tonic-clonic status, or aura continua; history of febrile seizures
2) Ictal EEG: Rhythmic theta or alpha activity in one or both temporal regions within 30 sec of the clinical onset; initial change can be voltage attenuation or low-voltage fast activity; slower (2-5 Hz), more polymorphic, bilateral or diffuse patterns are possible but more common in LTLE
3) Interictal EEG: Temporal spikes with maximal anterobasal (FT9, FT10, or sphenoidal) negativity and broad vertex positivity; independent bitemporal spikes may be present (~ 1/3 of cases); middle or posterior temporal spikes are possible but more consistent with LTLE; spikes are absent in (~ 10% of cases); interictal abnormality may be limited to focal slow-waves or asymmetries; rarely, the interictal EEG is normal

*B. Lateral Temporal Lobe Epilepsy (LTLE)*

1) Seizure types: Lateral temporal seizures, lateral temporal seizures with mesial temporal spread, lateral temporal seizures with extratemporal spread and/or secondary generalization; less commonly complex partial status, tonic-clonic status, history of febrile seizures is much less prominent than MTLE
2) Ictal EEG: Rhythmic theta or delta activity in one or both temporal regions appear 30 sec or more after the clinical onset; voltage attenuation or low-voltage fast activity may appear initially; slower (2-5 Hz), more polymorphic,

bilateral or diffuse patterns, and absence of EEG correlate are more common in LTS than in MTS.

3) Interictal EEG: Temporal spikes with broad temporal negativity and no vertex positivity; occasionally maximal mid-temporal (T7/T8) or posterior temporal (P7/P8) negativity; independent bitemporal spikes may be present; interictal changes may be limited to focal slow-waves or background asymmetry; interictal EEG can be normal

### *C. Frontal Lobe Epilepsy (FLE)*

1) Seizure types: Focal clonic seizures, jacksonian seizures, asymmetric tonic seizures, frontal lobe complex partial seizures, frontal lobe seizures (mixed type), frontal lobe seizures with spread outside the frontal lobe and/or secondary generalization; less commonly tonic-clonic status, focal status
2) Ictal EEG: Normal, obscured by artifact, non-localizing (due to rapid generalization), or any of these patterns: frontal low-voltage fast activity or rhythmic spike-waves with focal clonic seizures, low-voltage fast activity or attenuation near the vertex with supplementary motor area seizures, bifrontal voltage attenuation often followed by rhythmic theta/delta with mesiofrontal or orbitofrontal seizure, frontopolar rhythmic alpha/beta with orbitofrontal seizures
3) Interictal EEG: Normal, non-epileptiform EEG patterns, non-lateralizing/non-localizing IEDs, or localizing/ lateralizing IED (~1/2 of cases); secondary bilateral synchrony is common and often due to mesial-FLE; midline spikes also suggest a mesial frontal focus; lateralized frontal intermittent delta waves suggests an orbitofrontal focus

### *D. Parietal Lobe Epilepsy (PLE)*

1) Seizure types: Parietal lobe seizures, parietal lobe seizures with posterior spread, anterior spread, or inferior temporal lobe spread, parietal lobe seizures with secondary generalization; less commonly limbic or tonic-clonic status
2) Ictal EEG: Parietal lobe seizures often begin as a somatosensory aura with no EEG correlate; any subsequent discharge is often due to seizure spread to the temporal region, supplementary motor area, or other parts of the frontal lobe.
3) Interictal EEG: Parietal lobe spikes are elusive; patients with symptomatic PLE can manifest non-localizing or falsely-localizing temporal or frontal IED

### *E. Occipital Lobe Epilepsy (OLE)*

1) Seizure types: Occipital lobe seizures, occipital lobe seizures with infrasylvian or suprasylvian spread, occipital lobe seizures with secondary generalization; less commonly tonic-clonic or focal status
2) Ictal EEG: Occipital or temporo-occipital discharge may coincide with onset of visual seizure; a falsely-localizing EEG (e.g. temporal onset) is not uncommon.
3) Interictal EEG: A wide array of IEDs is found in symptomatic OLE, including occipital or bioccipital spikes, widely distributed IEDs, and falsely-localizing IEDs (e.g. temporal or frontal spikes).

### *F. Hemiconvulsion-Hemiplegia Syndrome (HHS)*

1) Seizure types: Hemiclonic status, hemiclonic seizures, tonic-clonic status, temporal lobe seizures, temporal lobe seizures with extratemporal spread, secondary generalization; extratemporal and other focal seizures
2) Ictal EEG: Focal onset (usually central or occipital) spikes or fast rhythms contralateral to the jerks with rapid spread; unihemispheric or lateralized (often posterior) high-voltage rhythmic 2-3 Hz slow waves or spike-waves.
3) Interictal EEG: Focal spikes or sharp waves; unihemispheric or lateralized slow waves.

### *G. Rasmussen Syndrome (RS)*

1) Seizure types: Focal motor seizures, hemiclonic seizures, EPC (focal myoclonic status epilepticus), complex partial seizures, tonic-clonic seizures, focal status other than EPC, tonic-clonic status.
2) Ictal EEG: Focal seizures are lateralized but not easy to localize; subclinical seizures are common; EPC consists of asynchronous jerks or twitches in different parts of the body; focal seizures and EPC are independent: activation occurs in the rolandic sulcus before the myoclonic jerk and in the neocortical convexity before the focal seizure.
3) Interictal EEG: Focal spikes are initially unilateral; with time, the number of independent spike foci increase; eventually spikes occur bilaterally but remain lateralized; bisynchronous spike-waves also occur (~ 50% of cases); background slowing and focal slow waves which are initially unilateral become bilateral but are always lateralized.

## VI. Reflex Epilepsies

### *A. Idiopathic Photosensitive Occipital Lobe Epilepsy (IPOLE)*

1) Seizure types: Reflex occipital lobe seizures; rarely with temporal spread or secondary generalization
2) Ictal EEG: Photic-induced occipital lobe seizures: onset in Oz and shifting laterality; PPR followed by buildup of occipital ictal discharge; exaggerated driving evolving to a self-sustained ictal activity
3) Interictal EEG: Spontaneous focal occipital spikes, rarely bisynchronous spike-waves; no spontaneous interictal spikes in some; wake and sleep background is usually normal (including alpha rhythm)

### *B. Visual Pattern Sensitive Epilepsy (VPSE)*

1) Seizure types: Reflex tonic-clonic, myoclonic, or absence seizures
2) Ictal EEG: Seizures evoked by pattern stimulation
3) Interictal EEG: Pattern-induced PPR; other responses are not specific can be confined to posterior regions; no PPR in some; wake and sleep background is usually normal (including alpha rhythm)

*C. Primary Reading Epilepsy (PRE)*

1) Seizure types: Reflex myoclonic jaw jerks, bilateral myoclonus, aphasic seizures, absence seizures, tonic-clonic seizures
2) Ictal EEG: Bisymmetric, lateralized, or focal left temporoparietal or frontocentroparietal spikes, spike-waves, or rhythmic theta during myoclonic jaw jerks; bisynchronous spike-waves during bilateral myoclonus or absences
3) Interictal EEG: Without activation: normal (80%), spontaneous bisynchronous spike-waves (10%) or focal temporal spikes (5%); wake and sleep background is usually normal

*D. Epilepsy with Startle-Induced Seizures (ESIS)*

1) Seizure types: Reflex tonic, tonic-atonic, or myoclonic seizures; in patients with hemiparesis, the weak side is preferentially involved; infrequent spontaneous seizures occur in all cases
2) Ictal EEG: Scalp vertex spikes followed by flattening or low-voltage activity during reflex seizures; intracranial EEG: motor evoked responses followed by ictal discharge in the motor-premotor areas
3) Interictal EEG: Spontaneous or startle-induced focal spikes or spike-waves; often lateralized especially when unilateral lesions and hemiparesis are present; background asymmetries and slowing are common [19].

Some methods of seizure detection were based on detecting strong rhythmic movements of the patient, but these methods had a limitation: seizures do not always present strong movements. This limitation led the detection problem to methods based on EEG signal analysis, for example, detection of large seizures discharges in several EEG channels by amplitude discrimination was described [21], [20] designed an electronic circuit for seizures detection from intracraneal electrodes. However, some seizures do not present EEG changes, therefore seizure detection only based on EEG analysis was not at all reliable and it was necessary to combine it with other methods. For example [22] identified on the EEG signal a large increase followed by a clear decrease in the amplitude and at the same time by large electromyogram (EMG) activity; [23] described a method based on spectral parameters and discriminant analysis. New alternatives for this detection problem are addressed from the point of view of pattern recognition. [24] presented an automatic detection system based on seizure patterns. The drawback of this method is the necessity of traditional visual inspection of the patterns, being necessary a careful examination of them by a specialist. Presently, EEG epileptic detectors have evolved including new techniques such as neural networks, non-linear models, independent component analysis (ICA), Bayesian methods, support vector machines and variance-based methods, as described in [25] Other group of methods potentially useful for detecting and analyzing non-stationary signals are time-frequency distributions (TFDs) [26] These methods allow us to visualize the evolution of the frequency behavior during some non-stationary event by mapping a one dimensional (1-D) time signal into a two-dimensional (2-D) function of time and frequency. Therefore, from the time-frequency (TF) plane it is possible to extract relevant information using methods such as peak matching, filter banks, energy estimation, etc. On the other hand, most of the detection methods proposed in the literature assume a clean EEG signal free of artifacts or noise, leaving the preprocessing problem open to any denoising algorithm such as digital filters, independent component analysis (ICA) or adaptive schemes using the electrooculogram (EOG) as reference signal, as in [27].

## VII. LORETA Analysis

LORETA is a recently developed method to localize multiple distributed cortical sources of bioelectric activity in the three-dimensional space [28]. In other words LORETA demonstrates the synchronously activated neuronal populations underlying EEG activity by computing their cortical localization from the scalp distribution of the electric field. This is called "solving the inverse problem of the EEG." The LORETA inverse solution is based on the "smoothness" assumption, which means that neighboring neuronal generators show highly correlated activity in terms of orientation and strength ،The smoothness assumption is based on neuroanatomical and electrophysiological constraints as described in [29]. In order to mathematically mitigate the disturbing effects of the electrically conducting layers between the cortical surface and the electrodes, LORETA computes the inverse solution within a three-shell spherical head model including scalp, skull, and brain [30] The brain compartment of this model was restricted to the cortical gray matter and hippocampus, according to the Talairach Brain Atlas that was digitized at Montreal Neurological Institute [31] The consistency of LORETA with physiology and localization has been validated by several authors [32] LORETA and functional MRI localized the changes to the same cortical region in a language-processing task [33] Concerning epilepsy, LORETA-defined localization of circumscribed epileptic activity corresponded to the localization of the epileptic discharges given by functional MRI, subdural and intracerebral EEG recordings, and the MRI-defined epileptogenic lesion [34]-[36].

## VIII. The Possible Relationship of the LORETA Results to Prior Findings in IGE

In theory, some relationship between our LORETA results and the structural alterations described in IGE [37], [38] is predictable, because altered cortical microstructure gives rise to altered EEG activity [39], [40]. However, unlike the LORETA findings, most structural abnormalities are diffuse, and not clearly delineated. The only example is abnormal thickness of the inferior frontomedial gray matter which anatomically corresponds to the prefrontal LORETA-cluster. Concerning functional neuroimaging, the intimate relationship between excitatory neurotransmitters, cortical excitability, and increased neuronal synchronization [41], [42] implies that

comparing the topographic patterns of neurotransmitters and bioelectric activity may be worthy. In fact, LORETA-defined increased activity of the prefrontal delta and theta generators topographically coincided with the prefrontal excess of glutamate and glutamine [43].

From the pathophysiological point, increased neuronal synchronization is the sine qua none of epileptic malfunctioning [44] Thus, it seems plausible that cortical areas where LORETA showed increased (i.e., pathologically synchronized) activity may have ictogenic property. In fact, intracranial EEG recording disclosed that unilateral stimulation of medial frontal sites triggered absences and generalized tonic–clonic seizures [45] Furthermore, a human ictal LORETA study showed that the bilaterally organized EEG onset of absence seizures ("by the second to fourth spike after onset") was invariably localized to the medial and orbital frontal cortex [46] LORETA-defined parieto occipital hyperactivity recalls the unexplained results of some prior studies. Besides the well-known frontal negative maximum of the potential field, voltage mapping studies disclosed circumscribed parietooccipital negativity in the early course of the ictal GSW complex [47]-[50]. Understanding that recognizes the likely contribution of multiple factors, rather than a single explanation. Future research and advances in technology will continue toincrease understanding of the human brain and its fascinating abilities and potential. The brain, once considered to be a fixed and stable organ, is now viewed as dynamic, flexible, and adaptive. Efforts are beginning to focus on ways to harness the plastic qualities of the brain for treatment and recovery. There is much that is still unclear about the relationship between neuroplasticity and mental health. Research capabilities for human studies are limited, so most questions must be addressed by study of animal models. This makes disentangling genetic, environmental, and experiential influences much more challenging. Although there is not yet consensus, it appears the field is moving toward a more multifaceted, nuanced understanding that recognizes the likely contribution of multiple factors, rather than a single explanation. Future research and advances in technology will continue to increase understanding of the human brain and its fascinating abilities and potential.

## IX. Conclusion

Electroencephalographic evidence indicates preferential spread of epileptic activity between the parietal cortex and a wide range of other limbic and isocortical areas [51]. The dichotomic developmental distribution of increased and decreased activity may be confounded by other factors like the anatomical and functional diversity of some cortical subregions. For example, the lack of increased activity in the middle part of the cingulate gyrus (as opposed to the increased activity in its anterior and posterior parts) may be related to the greatly dissimilar connectivity and function of these areas [52]. In any case, our results suggest that the demonstrated cortical abnormality is a highly organized condition. The ontogenetically dichotomic distribution of the bioelectric abnormalities clearly differs from the topographically unstructured cortical dysfunction of the acquired diffuse encephalopathies. Similarly, it differs from the very inconsistent topographic distribution of the bioelectric abnormalities in generalized epilepsies caused by single gene defects [53]. Our findings seem to support the recently developed hypothesis stating that IGE results from a complex and organized maldevelopmental cascade of events [54].

105 deep electrodes provide detailed information about the activity of the epileptic sources without a significant noise corruption and signal distortion. However, their exploitation as EEG datafor the inverse modeling requires more precise registration and forward modeling. FDM gives us the opportunity to compute the electrical potential values inside the brain tissue within the millimeter resolution. Also FDM based deep EEG localization is much more sensitive to the sources inside the brain. We also observe that the weakest source among the multiple sources corresponds to the activity obtained from the BEM based surface EEG localization. It is well known that when we use the scalp EEG, LORETA has a poor sensitivity to the deep sources than to the superficial ones. The results indicate that using the deep electrode measurements even the deep sources can be captured by LORETA [55].

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