Clinical Neurophysiology 129 (2018) 1056–1082

Contents lists available at ScienceDirect

Clinical Neurophysiology

journal homepage: www.elsevier.com/locate/clinph

Clinical utility of EEG in diagnosing and monitoring epilepsy in adults

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See Editorial, pages 1047–1048

ARTICLE INFO

Article history:
Accepted 9 January 2018
Available online 1 February 2018

Keywords:
EEG
Epilepsy
Epileptiform
Seizure
Guideline

HIGHLIGHTS

- This IFCN guideline assesses the categories of evidence for clinical utility of EEG in adults with epilepsy.
- EEG is useful for epilepsy diagnosis, classification and characterization before epilepsy surgery.
- EEG monitoring is helpful to detect and quantify nonconvulsive seizures, especially in critically ill patients.

ABSTRACT

Electroencephalography (EEG) remains an essential diagnostic tool for people with epilepsy (PWE). The International Federation of Clinical Neurophysiology produces new guidelines as an educational service for clinicians to address gaps in knowledge in clinical neurophysiology. The current guideline was prepared in response to gaps present in epilepsy-related neurophysiological assessment and is not intended to replace sound clinical judgement in the care of PWE. Furthermore, addressing specific pathophysiological conditions of the brain that produce epilepsy is of primary importance though is beyond the scope of

Abbreviations: aEEG, ambulatory electroencephalography; ACNS, American Clinical Neurophysiology Society; ASD, anti-seizure drug; CAA-EEG, computer-assisted aEEG; cEEG, continuous electroencephalography; ECG, electrocardiogram; ECS, electrical cortical stimulation; ECoG, electrocorticography; EEG, electroencephalogram; ESI, electroencephalographic source imaging; EMG, electromyography; EMU, epilepsy monitoring unit; EZ, epileptogenic zone; EE, epileptic encephalopathy; EPSP, excitatory postsynaptic potentials; ETLE, extratemporal lobe epilepsy; FCD, focal cortical dysplasia; fMRI, functional magnetic resonance imaging; GABA, gamma-aminobutyric acid; GGE, genetic generalized epilepsies; GPSW, generalized polyspike-and-waves; GSW, generalized spike-and-waves; GTC, generalized tonic-clonic; HFOs, high-frequency oscillations; HV, hyperventilation; IPSP, inhibitory postsynaptic potentials; ICU, intensive care unit; IED, interictal epileptiform discharge; IPS, intermittent photic stimulation; IFCN, International Federation of Clinical Neurophysiology; ILAE, International League Against Epilepsy; iEEG, intracranial EEG; LGS, Lennox-Gastaut syndrome; MRI, magnetic resonance imaging; NCSE, nonconvulsive status epilepticus; OLE, occipital lobe epilepsy; PLE, parietal lobe epilepsy; PWE, people with epilepsy; PPR, photoparoxysmal response; PNES, psychogenic nonepileptic seizure; QEEG, quantitative electroencephalogram; REM, rapid eye movement (sleep); SOZ, seizure-onset zone; SSW, slow spike-and-waves; SCORE, Standardized Computer-based Organized Reporting of EEG; TLE, temporal lobe epilepsies; VEM, video-EEG monitoring.

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https://doi.org/10.1016/j.clinph.2018.01.019

this guideline. Instead, our goal is to summarize the scientific evidence for the utility of EEG when diagnosing and monitoring PWE.

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1. Introduction and background

Epilepsy is a common, chronic, complex group of neurological disorders. Worldwide, more than 50 million people have epilepsy, affecting humans of all ages, ethnicity, social background, and geographic location (Perucca et al., 2014; England et al., 2012). It is characterized by an ongoing predisposition to recurrent seizures. Newer definitions of epilepsy include patients with reflex seizures, those with two or more unprovoked seizures greater than 24 h apart, and high-risk individuals with a single seizure and at least a 60% likelihood of experiencing recurrent seizures over the ensuing 10 years as compared to the general population (Fisher et al., 2014). A seizure is defined as a transient occurrence of clinical features produced by abnormal, excessive, and synchronous neuronal activity in the brain. Seizures are not synonymous with epilepsy, and epilepsy-related seizure types, frequency, and intensities vary greatly among individuals. When people with epilepsy (PWE) do not achieve seizure-freedom despite treatment, quality of life is impaired with a disability rating that ranks fourth among 220 health conditions and accounts for 1% of the global burden of disease (Salomon et al., 2012; Murray and Lopez, 1994).

Electroencephalography (EEG) is an essential tool in the evaluation and management of PWE, with a long and rich history as a clinical neurophysiological technique used in both hospital and outpatient community-based settings. PWE are more prevalent in poorer regions of the world where resources, including EEG-based studies for epilepsy evaluation and management, are limited (Newton and Garcia, 2012; Meyer et al., 2012). Guidelines exist for performing EEG according to European, Canadian, and American standards. The purpose of these guidelines is to serve as a standard throughout the world, which may require updating as technology evolves (Flink et al., 2002; Task Force of The Canadian Society of Clinical Neurophysiology, 2002; Tsuchida et al., 2016). While clinical application of EEG in the diagnosis of adult PWE is well-established, the interpretation of EEG recordings intended for clinical use has only moderate inter-rater reliability using visual analysis (Beniczky et al., 2013a). Various techniques used in EEG data acquisition can provide additional ways to identify epileptiform

abnormalities that may vary with age (Miskin et al., 2015), duration of recording (Losey and Uber-Zak, 2008), and clinical setting (Herman et al., 2015). Most pragmatic decision-making is based on standard scalp EEG recordings, usually 20–30 min (Pillai and Sperling, 2006; Airoldi et al., 1999). However, ambulatory EEG (aEEG), video-EEG monitoring (VEM), continuous EEG (cEEG) in the intensive care unit (ICU), and intraoperative and intracranial EEG (iEEG) monitoring are all important and emerging means of evaluating individual patients with epilepsy. Implementation of EEG as an important tool used in the diagnosis and management of PWE is supported by both level 1 and 2 evidence (Smith, 2005a). This guideline reflects a comprehensive overview of the evidence on a broad range of topics on the utility of EEG in diagnosing and monitoring adult PWE reviewed by an international panel of experts.

1.1. The role of EEG in epilepsy

The role of EEG in the diagnosis and classification of seizure types and epilepsy syndromes is well established (Leach et al., 2006; King et al., 1998; Sierra-Marcos et al., 2011; Cascino, 2001). More than eight decades after its discovery, EEG remains a safe, noninvasive, inexpensive, bedside test of neurological function. It is used in classification and characterization of seizures and seizure syndromes and to support the clinical diagnosis of a seizure, epilepsy, or epilepsy syndrome in PWE (Binnie and Prior, 1994). The process of extracting complex signals and applying them to clinically-relevant features is routinely achieved using visual analysis of the recording (Beniczky et al., 2013a). Standard scalp EEG represents the combined electrical activity of billions of neurons, but records only one-third of the cerebral cortex. Spatial limitations exist when attempting to record from the insular cortex; frontal-parietal opercular cortex; inferomedial temporal lobe; the interhemispheric fissure; and basal regions of the brain, such as the orbitofrontal cortex, inferior parietal-occipital cortices, and deep sulcal generators. In these regions, the standard EEG recorded from the scalp surface results in inherent underrepresentation of cortical generators. Most EEG is obtained using scalp electrodes in the interictal (asymptomatic) period. An *epileptiform discharge* has a high specificity for PWE when recorded during EEG. Interictal epileptiform discharges (IEDs) are distinctive waveforms or series of waveforms with features with similar import according to the International Federation of Clinical Neurophysiology (IFCN) formerly known as the International Federation of Societies for Electroencephalography and Clinical Neurophysiology (Chatrian et al., 1983). IEDs have various appearances depending on the type of epilepsy and associated cerebral pathology. Interictal spikes are characterized by high-amplitude, short duration waveforms with morphological characteristics of a spike lasting 20–70 ms or a sharp wave with a duration of 70–200 ms. The presence of IEDs is frequently followed by an after-going slow wave lasting 200–500 ms. In this way, EEG supports the operational classification of the type of seizures associated with various types of epilepsy (Fisher et al., 2017). Abnormal EEGs containing IEDs help classify seizures and identify epilepsy syndromes. Generalized spike-and-wave (GSW) patterns on EEG are the hallmark of generalized (genetic) epilepsies (GGE), with discharges within bursts characteristically repeating at 3 Hz or faster. The presence of *slow* (<3 Hz) spike-and-waves (SSW) is typical of an epileptic encephalopathy, such as Lennox-Gastaut syndrome (LGS). Focal anterior temporal spikes are often associated with mesial temporal lobe epilepsy (TLE). However, only some intracranial IEDs can be detected by standard scalp EEG recordings in PWE, limiting representation of underlying cortical epileptiform activity (Ray et al., 2007). Brain lesions have long been known to produce focal epileptiform and nonepileptiform abnormalities (Jasper and Van Buren, 1955). Nonepileptiform abnormalities such as focal, regional, or hemispheric delta slowing can fluctuate in morphology and amplitude, and have practical implications for cortical localization. Generalized theta and delta slowing result from diffuse white matter disturbances (Gloor et al., 1968). The etiologies are often nonspecific and include toxic-metabolic-systemic encephalopathies that may arise from a variety of disorders. The presence of continuous unreactive focal or bilateral independent polymorphic delta slowing is highly correlated with a structural lesion involving the subcortical white matter. This is especially true when coupled with attenuation of *fast* (>13 Hz) background activity, reflecting compromise of both gray and white matter. The presence of focal slowing is nonspecific for brain pathology and may be caused by tumors, stroke, encephalitis, trauma, and hematomas as well as by other lesions. In addition, slowing may occur as a consequence of a functional disturbance of the brain, such as during postictal recovery following a seizure, during a migraine, or with concurrent encephalopathies.

1.2. Study methods

An expert faculty was selected to represent the North American, European, Latin American, and Asian-Oceanic chapters of the IFCN. Upon approval by the executive committee, the charge to develop a new guideline on the utility of EEG in diagnosis and monitoring PWE was initiated. Published literature linking epilepsy and EEG was researched through September, 30, 2016, using search engines that included PubMed, Google Scholar, and Medline. Topical work groups were developed and comprised of one to three experts selected based on their area of expertise. A brief summary of one to three topics was then submitted for review following comprehensive compilation of the literature. A simplified categorization system for risk of bias was created for this project (Table 1). Broad spectrum classification was designated for studies, describing important confounders in their baseline population. The most relevant articles were identified, rated, and linked to recommendations predicated on the highest level of evidence obtained primarily from category 1 or 2 studies. Three to five of the most relevant papers containing the highest quality of evidence were classified into one of four categories and submitted for comparative grading by a methodologist (DG). Topic summary was based upon the best scientific evidence, focusing on yield and accuracy of the EEG in adult PWE relative to diagnosis and monitoring. We incorporated available, relevant guidelines, consensus statements, and task force proposals into summary statements reviewing the evidence in place of a formal grading system used in rigorous system-

Table 1
Categorization system used to define the levels of evidence in the literature on people with epilepsy.

Category 1: Individual randomized clinical trials (narrow confidence interval); systematic review with homogeneous randomized trials; prospective trials with a control group (all or none outcome)
Category 2: Individual cohort studies; broad-spectrum large retrospective trials; narrow spectrum prospective studies
Category 3: Narrow spectrum retrospective trials; systematic review of homogeneous case-control studies; individual case-control studies
Category 4: Case series; reviews; expert opinion

Table 2
Classification of seizure types.

Focal Onset[a]	Generalized Onset	Unknown Onset
Motor Onset	**Motor**	**Motor**
automatisms	tonic-clonic	tonic-clonic
atonic	clonic	epileptic spasms
clonic	tonic	
epileptic spasms	myoclonic	
hypokinetic	myoclonic-tonic-clonic	
myoclonic	myoclonic-atonic	
tonic	atonic	
	epileptic spasms	
Nonmotor Onset	**Nonmotor (absence)**	**Nonmotor**
autonomic	typical	behavioral arrest
behavior arrest	atypical	
cognitive	myoclonic	
emotional	eyelid myoclonia	
sensory		
Focal to bilateral tonic-clonic		**Unclassified**

Adapted from Fisher et al. (2017).
[a] Focal onset seizures are separated into focal aware, and focal impaired awareness.

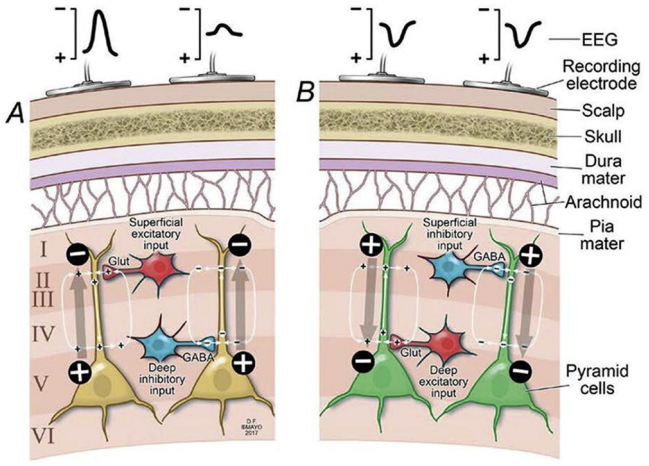

Fig. 1. Schematic drawing of the scalp EEG registering negative (A) and positive (B) deflections elicited from summated EPSPs and IPSPs derived from pooled pyramidal cells. Cells releasing glutamate and GABA provide excitatory and inhibitory superficial and deep synaptic connections resulting in an electrophysiological sink or source. EEG = electroencephalography; EPSPs = excitatory postsynaptic potentials; GABA = gamma-aminobutyric acid; IPSPs = inhibitory postsynaptic potentials. Figure courtesy of Anteneh Feyissa M.D. and Mayo Clinic.

atic guidelines (Oxford Centre for evidence-based medicine, 2009) and conforming to the style of guidelines used by *Clinical Neurophysiology*.

2. Anatomy and physiology

Epilepsies are clinically characterized by sudden changes in behavior associated with seizures. The seizure manifestations (semiology) are governed by the anatomic location of initiation and propagation. Generalized seizures affect both hemispheres of the cerebrum and are typically associated with loss of consciousness. Focal seizures present with symptoms related to the location or focal, regional, or hemispheric structures from which the seizures arise. Since the late 19th century, when Hughlings Jackson proposed that seizures were due to a focal neuronal discharge, the cerebral cortex has been considered the predominant anatomic site of origin for seizures in PWE (Jackson, 1890a, b, c).

In recent years, findings from histopathological, electrophysiological, and neuroimaging studies have provided ample evidence (mostly retrospective) demonstrating that seizures involve widespread network interactions among cortical and subcortical structures (Paz and Huguenard, 2015). Focal seizures can arise from very small and remote regions in the brain that defy detection by standard EEG. Seizures may originate within a single lobe, region, or hemisphere. In adults, the most common site of focal seizure onset is the temporal lobe, though frontal, parietal, and occipital lobes may be involved in descending order of frequency. Decades of experimental work have established the temporal lobe as the most epileptogenic region in the brain (Staley, 2015). The amygdalohippocampal complex is one of the key anatomic circuits involved in epilepsy. This is evidenced by TLE being the most common human adult form of epilepsy (Tatum, 2012). The hippocampus and dentate gyrus are composed of a three-layered cortex (the archipallidum), which is distinct from other regions of the brain where six layers are encountered. Hippocampal sclerosis is involved in a highly epileptogenic network, and is perhaps the most common pathology identified from surgical series in PWE (Walker, 2015). However, the process of seizure initiation appears to involve broad neuronal interconnections involving multiple independent limbic structures, with the thalamus acting as a physiological synchronizer of the cortex (Bertram et al., 1998). Other subcortical structures have been associated with seizures and demonstrate similar clinical, electrophysiological, and functional neuroimaging abnormalities, including the hypothalamus (Cascino et al., 1993; Kuzniecky et al., 1997; Berkovic et al., 2003) and cerebellum when dysplastic features have been encountered (Harvey et al., 1996).

The centrencephalic theory of GGEs Penfield and Jasper in 1954 (Penfield and Jasper, 1954) proposed that subcortical structures were directly involved in initiating generalized epileptiform discharges in addition to generalized seizures via bilateral neocortical structures activated through thalamocortical connections (Nguyen et al., 2006; Badawy et al., 2013). The dorsal thalamus is considered to be chiefly responsible for synchronizing thalamocortical relay neurons with the neocortex. It addition, it has reciprocal branches that terminate in thalamic inhibitory interneurons (feedback servomechanism). The *nucleus reticularis thalami*, whose cells release the inhibitory neurotransmitter gamma-aminobutyric acid (GABA) in rhythmic bursts, depolarizes neurons of the dorsal thalamus and rostral brainstem, to serve as a primary pacemaker, resulting in EEG synchronization (Olejniczak, 2006). Generalized seizures arise from rapidly engaging bilaterally distributed networks, while focal seizures are limited to one hemisphere either discretely localized or within more widely distributed networks in one hemisphere (Berg et al., 2010). Disturbance in the networks involved with excitatory and inhibitory firing of subcortical thalamic reticular, thalamic relay, and neocortical pyramidal neurons is probably responsible for generating generalized 3 Hz spike-and-wave discharges that characterize typical absence seizures (Blumenfeld, 2003; Meeren et al., 2005; Koutroumanidis and Smith, 2005). Based on the evidence described above, a paradigm shift has emerged in recent years. Today seizures are believed to represent dysfunctional neural networks dominated by paroxysmal, excessive, or hypersynchronous activity (Paz and Huguenard, 2015). The recent classification of seizure types (Fisher et al., 2017) is listed in Table 2.

3. The cellular basis of seizures and epilepsy

Epileptogenesis refers to an alteration in the brain function that eventually results in recurrent seizures. The types of recurrent seizures depend on genetic mechanisms, alteration in the architectural anatomy of the brain, space-occupying structural lesions in the brain, and infectious-inflammatory conditions disturbing the

blood–brain barrier. Basic mechanisms of ictogenesis and epileptogenesis were significantly advanced when epileptiform discharges (spikes) in the EEG were first identified (Fischer, 1933). Abnormal electrophysiological activity underlying seizures in PWE results from neurobiochemical processes initiated at the level of the cell. Neuronal hyperexcitability and hypersynchrony underlie an altered seizure threshold. The expression of ictogenesis and epileptogenesis requires pooled neuronal involvement that intermittently results when large networks of neurons are recruited. The EEG recorded at the scalp is a complex electrophysiological signal generated by the brain composed of summated pools of neuronal excitatory postsynaptic potentials (EPSPs) and inhibitory postsynaptic potentials (IPSPs). The *spike* in EEG is due to recurrent synaptic excitation between pyramidal neurons. Intrinsic neuronal activity from action potentials is too brief in duration to contribute much to EEG (Merricks et al., 2015). Synaptic potentials are generated at the apical dendrites of large, vertically oriented pyramidal neurons located in cortical layers III, V, and VI (Olejniczak, 2006; Jackson and Bolger, 2014). Neurobiochemical phenomena involving voltage-gated ion channels and currents give rise to electrophysiological manifestations of epileptogenic activity. While inward (extracellular to intracellular) current flow of calcium or sodium ions causes depolarization resulting in an EPSP, activation of calcium mediated outward (intracellular to extracellular) flow of potassium ions or GABA mediates chloride influx and results in hyperpolarization that generates an IPSP at the dendritic synapses (Olejniczak, 2006). The EPSP at the dendrites create an extracellular voltage that is more negative than elsewhere along the neuron (an IPSP will have the reverse effect), thereby resulting in a dipole (Fig. 1). A region of positive charge (source) is thus separated from a region of negative charge (sink). Each neuron continually produces electrical activity relative to its own independent cellular basis. Astrocytes contribute to the spread of current flow by enhancing calcium influx into the neurons (Olejniczak, 2006). Neuronal pools continuously interact with other neurons in local neural networks and other networks in distant regions of the brain.

The IED is the hallmark of epilepsy. The paroxysmal depolarizing shift (PDS) is the cellular substrate for an IED. A large shift of the membrane potential from −85 mV to +30 mV makes the neuron fire rapid bursts of action potentials to characterize the PDS that occurs synchronously in neurons within a local region. The PDS has properties of a large EPSP and is driven by the simultaneous excitation from many other neurons within the same population. The PDS is initiated by neuronal depolarization from calcium and sodium influx and relatively synchronized potentials identified at the scalp EEG expressed as IEDs. Subsequently, an influx of negative charge at the axon appears as surface negative slow-waves. The essence of seizure generation involves an imbalance between excessive excitatory neurotransmitters and relative reduction of inhibitory neurotransmission. Multiple systems involving additional neurotransmitters are required to produce paroxysmal EEG activity involved seizure initiation, propagation, and termination in PWE.

Ictogenesis is due to augmented synchronization of neuronal epileptiform discharges due to increased neuronal activity. Generalized seizures associated with GGE involve a typical 3-Hz GSW pattern on EEG. The mechanisms may differ among seizure types. In absence epilepsy, it depends on synchronization of transient low-threshold T-type calcium channels in the thalamus via rhythmic activity of inhibitory neuronal networks (Jefferys, 2010). In focal seizures, mechanism for initiating a seizure involves excessive firing and synchronization of neurons. With a seizure, potassium concentration in the extracellular space is increased at the cellular level and results in breakdown in the local inhibitory mechanisms result (DC shift), excitation and depolarization of neurons, and seizure initiation.

The location of the epileptogenic zone (EZ) may play a role in different mechanisms of ictogenesis. More recently, high-frequency oscillations (HFOs) involving frequencies in the gamma range (30–80 Hz), ripples (80–250 Hz), and fast ripples (250–500 Hz), representing transient, very brief, high-frequency discharges stemming from neuronal synchronization, and have become increasingly important elements in the EEG. Pathological HFOs have been reported in PWE, though physiological HFOs have also been noted in people without epilepsy (Buzsaki et al., 1992). Standard scalp EEG has a reduced signal-to-noise ratio at higher frequencies because the scalp and skull attenuate high frequencies due to the low-voltage present with cortical potentials, thereby limiting their detection (Petroff et al., 2016). Discovery of frequencies above those traditionally used for clinical EEG (1–30 Hz) has had a profound impact on our understanding of brain function (Zijlmans et al., 2012). HFOs have been reported to be highly localized in pathologically connected neurons in the seizure-onset zone (SOZ) (Worrell et al., 2004), and appear to be good biomarkers for the EZ in patients with focal seizures who benefit from surgical resection (Zijlmans et al., 2012). Summated IPSPs generated at the pyramidal cell synaptic connections, nonsynaptic cellular interactions, and extracellular current caused by ephaptic interaction are implicated in generating pathological HFOs inherent in epileptogenesis (Menendez de la Prida and Trevelyan, 2011).

4. Recording concepts and reporting techniques

Summated dipoles from multiple neurons are required to become measurable at the scalp as a single dipole source. Thus, a combined synchronous cortical source composed of approximately 10^8 neurons is required to generate an IED in scalp EEG for the majority of individuals (Tao et al., 2005). The magnitude reflects the summation of the pooled number and synchronicity of neuronal dipoles. Computer-based technology is especially suited to recording, signal processing, and display of the EEG. Large amounts of data may be acquired and configured through remontaging, which may be used to accommodate various software applications, such as spike and seizure detection programs and quantitative EEG (QEEG) with trending according to some category 1 studies (Scherg et al., 2012) supported by older retrospective series (Young and Campbell, 1999). Routine EEG records signals from cortical generators displayed as amplified electrical potentials obtained from electrodes applied to the scalp. In category 3 and 4 studies of PWE, polygraphy was shown to be crucial in demonstrating the relationship between epileptiform activity on EEG, and modifications occurring simultaneously in a set of physiological parameters, including muscular activity and vital functions, such as heart rate and respiration (Rubboli et al., 2008; Tassinari and Rubboli, 2008). Simultaneous use of electromyography (EMG) and EEG are important to identify the type and topography of muscular contractions during an ictal event. For example, EMG and EEG can be used to identify positive and negative myoclonias, tonic and atonic seizures, spasms, and drop attacks, providing essential category 2 information for syndromic classification (Shibasaki and Hallett, 2005; Itoh et al., 2015; Oguni et al., 1994). Recording of polygraphic parameters (e.g., electrocardiogram [ECG] and EMG) is recommended for VEM based on category 3 and 4 studies (Rubboli et al., 2008; Tassinari and Rubboli, 2008). Detecting a cardiac arrhythmia in patients with recurrent seizures can be valuable in providing information regarding the differential diagnosis. The use of EMG can supplement information that might be missed on clinical inspection and on routine EEG (e.g., subtle myoclonic jerks associated with epileptic discharges or brief tonic seizures associated with a burst of paroxysmal fast activity) (Tinuper et al., 2001; Shibasaki and Hallett, 2005; Rubboli et al., 2008). Computer-

ized signal analysis techniques can be used to detect and characterize the EEG correlates of myoclonus (Barrett, 1992; Brown et al., 1999; Shibasaki and Hallett, 2005; Barrett, 1992). These techniques are based on identifying a time-locked relationship between EEG and the EMG signal associated with the muscle jerk to identify a consistent relationship with a cortical generator and eliminate inconsistent ones (Ugawa et al., 1989; Avanzini et al., 2016).

Outside the outpatient EEG laboratory setting, prolonging the duration of the EEG recording increases the yield of detecting and monitoring epileptiform activity. aEEG records EEG over a prolonged period of time in the patient's home environment. cEEG in special care units can quantify clinical and subclinical seizures for diagnosis and assist in the management of seizure emergencies, including status epilepticus. High-density EEG may provide complementary information (category 2) characterizing drug-resistant seizures in PWE, particularly in those with non-lesional brain magnetic resonance imaging (MRI) (Michel et al., 2004). High-density EEG uses a greater number of electrodes ($\geq$64), and when a higher sampling rate is used, high-density EEG is able to provide higher spatial resolution. For this reason, high-density EEG is increasingly used in the routine clinical investigation of patients undergoing presurgical evaluation (Gavaret et al., 2015). When repeated EEG recordings fail to demonstrate abnormality, magnetoencephalography (MEG) may provide additional diagnostic information (mostly category 3) by detecting and measuring tangential dipolar sources in PWE and being more sensitive to activity originating deep within the cortical sulci (Duez et al., 2016). Standard scalp EEG can detect radial dipoles which are located closer to the top of the cortical gyri. When EEG and MEG are used together, they may provide complementary information about the localization of IEDs. Coupling EEG with transcranial magnetic stimulation, and EEG with functional MRI (fMRI) are evolving techniques reported in category 4 studies that may have implications for the epilepsies (Kimiskidis et al., 2014).

An important aspect of EEG is the practice of structuring a report of findings obtained during standard scalp EEG. EEG has traditionally been thought of as a study associated with the qualitative description of a mixture of waveforms reported in text format. Moderate interobserver consistency of an EEG interpretation may be partly explained by differences in reporting styles (Beniczky et al., 2013a) and inconsistent terminology (Hirsch et al., 2013). The main consequence of nebulous reports is misinterpretation by the clinician of the import of the EEG, leading to inappropriate treatment. In addition, there is significant variability in EEG reporting (Tatum, 2013b) prompting guidelines endorsed by the American Clinical Neurophysiology Society (ACNS) (Tatum et al., 2016). Recently, a European consensus-statement has been published on the Standardized Computer-based Organized Reporting of EEG (SCORE). This has been supported by the European Chapter of the IFCN and the subgroup of the Commission on European Affairs for the Internal League Against Epilepsy (ILAE), where electroencephalographers assess and report the EEG findings using a forced-choice selection of predefined terms, presented in a context sensitive manner (Beniczky et al., 2013a). Electronic databases are emerging that may provide higher inter-rater agreement when specific EEG features are selected from a list of predefined terms (Gaspard et al., 2014; Beniczky et al., 2013a; Stroink et al., 2006).

5. Technical limitations

Limited evidence is available to address the optimal technical parameters of the EEG in PWE. Most information on the technical aspects of recording is based predominately on category 4 evidence. For example, both American (Sinha et al., 2016) and European guidelines (Flink et al., 2002) for a standard EEG propose durations of at least 20–30 min. Other reports propose a 20 min recording for standard awake EEGs and 30 min for sleep EEG recordings (Craciun et al., 2014). The IFCN recommends that the usual technical EEG requirements should also be applied when assessing comatose patients (Guerit et al., 1999). However, the comparatively brief recording time of a routine EEG might be inadequate for detecting IEDs that occur infrequently (Pedley, 1980; Pillai and Sperling, 2006). Minimal technical standards are essential in securing EEG tracings with a high signal-to-noise ratio and to ensure high-quality recording, storage, review, and exchange among collaborators for clinical use and collective research analysis (Sinha et al., 2016). Electrodes should be placed in accordance with the International 10-20 or 10-10 system of placement as recommended by the IFCN to ensure reproducibility from one recording to another (Jasper, 1958). In routine scalp EEG, electrodes access only one-third of the entire cortex; therefore, IEDs arising within sulci, in basal regions (e.g., orbitofrontal cortex), *buried regions* (e.g., amygdalohippocampal complex), and in interhemispheric regions (e.g., supplementary motor cortex) may not propagate to the scalp resulting in an attenuated signal and limited sensitivity of scalp EEG (Pillai and Sperling, 2006). The orientation of the dipole of IEDs must be perpendicular and close to the surface to be detectable by scalp electrodes. This accounts for the relatively moderate localizing value of interictal scalp EEG in addition to distortion of electrophysiological fields caused by different conductive properties of the brain, cerebrospinal fluid, meninges, skull, and scalp. Additionally, if too few electrodes are used, the chances of interpretative errors increase; conversely, when more channels are used, the likelihood of such errors decreases (Sinha et al., 2016). The modified 10-10 nomenclature replaces the T3/T4 and T5/T6 electrode positions with T7/T8 and P7/P8 and an increased number of electrodes (Acharya et al., 2016). Additional arrays selected for performing EEGs, depending upon the indication, have been used to further subdivide measurements from anatomical landmarks into 5%, 10%, and 20% (Oostenveld and Praamstra, 2001). When small areas of the cerebral cortex involve less than 10 cm^2, a considerable proportion of IEDs present at a cortical level are missed by routine scalp EEG recordings compared with iEEG recordings (Tao et al., 2005). In this instance, no IEDs were recorded with less than 6 cm^2, 10% were identified between 6 and 10 cm^2, and 90% were recorded when more than 10 cm^2 of surface area was involved. This work was extended with the use of waveform averaging with a small incremental detection of IEDs in less than 6 cm^2 of cerebral cortex (Ramantani et al., 2014).

Digital EEG recording equipment has many advantages over older paper-based EEG recording systems. Internationally recognized neurophysiological equipment standards, optimal EEG signal processing and electronic transfer capability, improved storage capacity, and accepted safety standards have enabled widespread access to EEG for PWE in developed countries (Velis et al., 2007; Nuwer et al., 1998). Amplifiers with greater sensitivity and waveform resolution, representation, and reliability are now available. These amplifiers use parameters that modify postrecorded EEG by altering the gain, filters, and montages and more efficiently analyzing large amounts of data. Improper use of digital filters may impair the technologist's ability to recognize technical difficulties and EEG abnormality and to eliminate artifact (Tatum et al., 2011). Low-frequency (high-pass) filter settings greater than 1 Hz may limit information about pathologic delta activity when present, whereas high-frequency (low-pass) filter settings less than 70 Hz (including 60-Hz notch filters) can distort or attenuate IEDs to become unrecognizable. Technical artifacts from overfiltering, electrode pops and myogenic discharges may lead to misidentification of artifact as pathological waveforms by the interpreter (Tatum, 2013a). The amplitude of a brain signal is inversely proportional to the square of the distance between the scalp electrode and source (Gloor,

1985). When the sensitivity is excessive (e.g., changed from 7 to 5 μV/mm), the amplitude of a given waveform increases on the EEG to the point where it may appear epileptiform.

6. Standard scalp EEG in diagnosis

Previous observational studies, including two large randomized trials, demonstrate a recurrence risk of 40–50% within two years after a first seizure (Walczak et al., 1993; Marson et al., 2005). However, fewer than half of individuals have a readily apparent cause (Tao and Davis, 2016). The EEG demonstrates predictive value in determining the risk of seizure recurrence with category 1 evidence. In large multicenter studies, an abnormal EEG conferred a higher risk of recurrence (Kim et al., 2006) following a first seizure in children (Shinnar et al., 2000) and adults (Hauser et al., 1998; Krumholz et al., 2007). A recent evidence-based guideline reported 21–45% of unprovoked seizures recurred within two years following a first seizure in adults (Krumholz et al., 2015). The absolute risk reduction of seizure recurrence over two years is 35% (95% CI 23–46%) when comparing treated with untreated patients; however, treatment might not affect quality of life (category 2). The risk rises to 75% in people who have a second seizure (Musicco et al., 1997; Kim et al., 2006; Krumholz et al., 2015). In 2014, the ILAE revised the definition of epilepsy to include a single unprovoked seizure with the probability of recurrent seizures of 60% or more (Fisher et al., 2014). Therefore, an EEG containing IEDs following a first seizure will confer a clinical diagnosis of epilepsy in the appropriate clinical setting (Fisher et al., 2014) with implications for treatment with anti-seizure drugs (ASDs) (Krumholz et al., 2015). The risk of seizure recurrence compared to the general population doubled when IEDs were present in the EEG (category 1). Therefore, patients presenting with a first unprovoked seizure should be informed that they have a high chance of seizure recurrence over the next two years. Nevertheless, a normal interictal EEG does not exclude the diagnosis of epilepsy because there are spikes often present on electrocorticography (ECoG) that are not recognized on scalp EEG recordings (Ray et al., 2007).

In population-based studies, the yield of the first EEG to identify IEDs after a first unprovoked seizure has a relatively low sensitivity ranging from 32% to 59% in adults (Baldin et al., 2014), where the EEG was *positive* in 29–55% (Goodin et al., 1990). The EEG is persistently *negative* in roughly 10% of PWE. An EEG without IEDs is more likely to occur in patients with focal seizures than in patients with seizures associated with generalized epilepsy (Walczak et al., 1993). There is a slightly higher yield of recording IEDs if EEG is performed within 24–48 h of a new-onset seizure (King et al., 1998; Schreiner and Pohlmann-Eden, 2003). An abnormal nonepileptiform abnormality increases the risk of recurrence to more than 40%, and the presence of IEDs to more than 60% (Berg, 2008). In another study, a second seizure occurred in less than 30% of first seizure patients following a normal EEG (Shinnar et al., 1996, 2000). Therefore, the urge to treat a first seizure should be tempered and guided by the individual clinical situation, whereas those patients with an epileptiform EEG are often considered for ASD treatment given their greater risk for further seizures.

Standard outpatient EEG enables the diagnosis of epilepsy (category 1), even at a syndromic level in 77% of patients when performed within 24 h of the first seizure presentation (King et al., 1998). Sensitivity increases after several EEGs, rising to 80–90% when three or more serial EEGs are performed (Salinsky et al., 1987). Higher yields are also observed when EEG recordings contain sleep or follow sleep deprivation in category 1 studies (King et al., 1998; Leach et al., 2006). The specificity of EEG is better than its sensitivity, but varies from 78% to 98%, partly explained by the heterogeneity of case selection and differences in diagnostic criteria for epilepsy in the populations studied (Smith, 2005). The majority of evidence supports the use of EEG in assessing the risk of recurrence after a first seizure, but also helps establish the type of seizure (classification), and the epilepsy syndrome (van Donselaar et al., 1992; Hauser et al., 1982; King et al., 1998).

The frequency of IEDs does not accurately reflect seizure frequency nor predict the therapeutic response to ASDs (Binnie and Prior, 1994). Additionally, there is a high risk of epilepsy when IEDs are localized to the anterior temporal lobe or vertex as opposed to the frontal, centrotemporal, or occipital region where the risk is moderate (Fisch, 2003; Kutluay et al., 2001). Similarly, a high epileptogenic potential is observed when generalized paroxysmal fast activity (GPFA) and SSW are present compared with EEGs containing a photoparoxysmal response (PPR) (Hughes and Fino, 2003; Kellaway, 1981; St Louis and Cascino, 2016).

A risk of misdiagnosing epilepsy in patients with *spells* exists when the EEG is misinterpreted (Chowdhury et al., 2008; Benbadis and Tatum, 2003a). Focal and generalized IEDs may occur in people without seizures or epilepsy particularly in the occipital and central head regions. A very low false-positive rate of IEDs occurs in people without epilepsy (Table 3) [0.2–0.5% of normal adults; up to 3.5% in children without seizures] (Pillai and Sperling, 2006; Smith, 2005; Cavazzuti et al., 1980; Hendriksen and Elderson, 2001). Cavazzutti et al. identified IEDs in 3.5% of more than 3000 children ages 6–13 years, one-third with generalized IEDs (Cavazzuti et al., 1980). Hence a significant minority of people with generalized IEDs on EEG do not have clinical seizures, especially children. In large retrospective adult studies (category 2), there was failure to control for benign variants and lack of adequate follow-up. (Cavazzutti et al., 1980; Sam and So, 2001). In a community setting, 521 patients with a follow-up of 230 person-years had no history of unprovoked seizures, yet 64 (12.3%) had IEDs on their EEG (Sam and So, 2001); ~75% had acute or progressive disorders involving the brain (e.g., tumor, stroke, and subdural hematoma).

Inter-rater reliability based upon visual analysis of the EEG is only moderate (Beniczky et al., 2013a; Halford et al., 2011; Stroink et al., 2006). Over-interpretation of normal waveforms as abnormal (Benbadis and Tatum, 2003a), inappropriate pattern-recognition of normal variants (Krauss et al., 2005), and the use of subjective interpretation and reporting (Beniczky et al., 2013a) are pitfalls (Fowle and Binnie, 2000). There is high inter-rater variability with IEDs (Halford et al., 2011; van Donselaar et al., 1992).

Table 3
Summary of adult EEG studies (>1000) of epileptiform discharges of people without epilepsy.

Year-1st author	Population	Number evaluated	% IEDs	Follow-up
1943-Gibbs	Healthy adults (20 years old)	1000	0.4% (limited EEG reported)	No
1967-Bennett	Male aviators	1332	0.6% (limited EEG reported)	No
1968-Zivins	Tertiary care hospital	6497	2.0%	Yes-14.1% with epilepsy
1985-Iida	Outpatients nonepilepsy	10,473	8.1%	Yes-1.5% with epilepsy
1987-Bridgers[a]	Inpatients (11–85 yrs.) psychiatry	3143	1.0%	No
1993-Gregory[a]	Aircrew trainees (17–25 yrs.)	13,658	0.2%	Yes-2.3% with epilepsy

Abbreviations: EEG, electroencephalogram; IEDs, interictal epileptiform discharges.
[a] Excluded normal variants.

Normal variants (e.g., wicket spikes, 6-Hz GSW, benign epileptiform transients of sleep, etc.), neurological disorders (e.g., blindness, hypoxia), and systemic or drug effects (e.g., cefepime and lithium) may also produce IEDs in people without seizures and do not portend a significant risk of epilepsy (Table 3). Artifact created by movement (e.g., tremor, myoclonus) may also limit or obscure visualization of the underlying electrocerebral activity, and rhythmic tremor may serve to imitate an electrographic seizure leading to misinterpretation of the EEG (Benbadis, 2010).

6.1. Outpatient short-term video-EEG in diagnosis

Implementing video with EEG further increases the diagnostic yield beyond the yield of EEG alone when events are recorded (Cascino, 2002), with similar sensitivity when the two modalities are compared (Chen et al., 2008). The yield of IEDs and event detection is increased in standard EEGs extended up to 60 min compared to routine 30-min studies (Burkholder et al., 2016). In a prospective, cross-sectional, observational, category 2 study of 1803 patients, 19% of IEDs were first identified after 30 min. Longer recording times increased event capture rate by 30% (Burkholder et al., 2016). Short-term video-EEG, typically lasting less than 24 h (Bubrick et al., 2014), is emerging as a less costly and resource-demanding technique in comparison to VEM. Short-term video-EEG can differentiate among epilepsy and other paroxysmal disorders in more than 50% of patients supported primarily by category 3 and 4 studies (Tinuper et al., 2004; Tallawy et al., 2010). Prolonging the duration of outpatient EEG from 30 to 60 min increases the relative yield of new IEDs by approximately 20% (4.5% absolute increase in IEDs across all patients); irrespective of age, EEG indication, or IED type (Burkholder et al., 2016). These extended EEGs found new IEDs in ~1/22 studies regardless of the indication, and in ~1/13 studies when there was a high pretest probability (Burkholder et al., 2016). Outpatient short-term video-EEG with induction has been used to diagnose psychogenic nonepileptic seizures (PNES) and avert VEM (Benbadis et al., 2004). Polygraphy combined with video-EEG may be used as a dynamic short-term diagnostic test tailored to a patient-specific clinical process. EEG-fMRI may also be useful as a short-term outpatient VEM technique. EEG-fMRI combines the high temporal resolution of EEG abnormalities and the high spatial resolution of fMRI to noninvasively localize regional cerebral metabolic change (Stern, 2006; Gotman et al., 2004), and can be helpful in the presurgical evaluation of apparently ineligible candidates in a category 3 study (Zijlmans et al., 2007). When comparing the yield of short-term EEG and cEEG to detect seizures in patients with altered sensorium, one prospective study concluded that cEEG was superior to short-term EEG in this population (Rai et al., 2013).

6.2. Epilepsy classification and syndromes

Epilepsy classification is the key clinical tool in evaluating an individual who is presenting with seizures (Gastaut, 1969; Scheffer et al., 2017). A three-tiered approach should initially address the type of seizure involved (e.g., focal, generalized or unknown onset), followed by a diagnosis of the epilepsy type; focal epilepsy, generalized epilepsy, combined generalized and focal epilepsy, and unknown classification. Lastly, attempts to classify a specific syndromic diagnosis should be made. With significant advances in understanding the neurobiology of seizures and epileptic diseases, there have been major paradigm shifts in the concepts underpinning classification (Scheffer et al., 2017). Previously, etiological categories have divided the epilepsies into three main categories; genetic (aka idiopathic), structural (aka, symptomatic), and unknown (aka cryptogenic) (Berg et al., 2010; Scheffer et al., 2017), though additional categories (e.g., immune, metabolic, infectious) have become incorporated into recent classifications (Scheffer et al., 2017; Fisher et al., 2017). The purpose of classifying the epilepsies will hopefully improve diagnosis, provide greater understanding of etiology, and target therapies to the patient's disease. Computer-assisted-telephone-interview in an attempt to classify an epilepsy diagnosis in outpatients suspected to have seizures (D'Souza et al., 2010), suggests a potential role for automation in the future.

6.2.1. Generalized epilepsy

GGEs include many electroclinical syndromes based on specific clinical features and EEG abnormalities. Generalized IEDs during standard EEG recording are the common feature for GGEs. These discharges are usually an interictal finding when seizures are infrequent (Pillai and Sperling, 2006; Flink et al., 2002) though they are ictal in other situations especially when prolonged. Generalized IEDs may be seen in both genetic (idiopathic) and developmental and epileptic encephalopathies when seizures are a symptom of an underlying structural etiology (Fig. 2). In GGE, the discharges seen in the EEG are symmetrical, synchronous, frontally-predominant, GSW and generalized polyspike-and-waves (GPSW) recurring at 3 Hz or more in conjunction with normal background activity (Fig. 3A). None of the EEGs containing GSW and GPSW is seizure- or syndrome-specific for a particular GGE (Seneviratne et al., 2012). Instead, the features of the IEDs in the EEG are altered by age and the stage of sleep (Sadleir et al., 2009). However, while the characteristics of the IEDs vary in the individual GGE syndromes, some features can suggest a particular epilepsy syndrome. For example, absence epilepsy occurs with a typical 3-Hz GSW pattern (Panayiotopoulos et al., 1989), and the clinical utility of EEG in the diagnosis and classification of absence epilepsies has long been known (Sadleir et al., 2009). GGEs manifesting as generalized tonic-clonic (GTC) seizures alone have similar features in the EEG (Unterberger et al., 2001). Limited evidence from category 1 and 2 studies exists on epilepsy syndromes other than juvenile myoclonic epilepsy (JME). JME has a sensitivity and specificity ranging from 54% to 73.3% (Genton et al., 1995; Dhanuka et al., 2001). GPSW are associated with phenotypes that include myoclonic, GTC, and absence seizures (Rubboli et al., 1999). Performing EEG in the early morning hours (Sousa et al., 2005), obtaining N2 sleep (Bonakis and Koutroumanidis, 2009), and the use of activation techniques (e.g., sleep deprivation, intermittent photic stimulation (IPS) and hyperventilation [HV]) lead to significant activation of IEDs in GGEs (Penry et al., 1975; Degen and Degen, 1983; Panayiotopoulos et al., 1992; Koutroumanidis and Smith, 2005; Sousa et al., 2005; Bonakis and Koutroumanidis, 2009; Seneviratne et al., 2012). The presence of IEDs occurring upon awakening may serve as a specific indirect biomarker for GGE

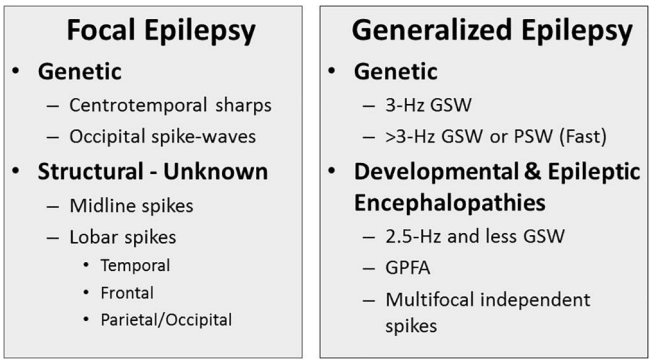

Fig. 2. Epilepsy classification and common type of interictal epileptiform discharges by etiology. GPFA = generalized paroxysmal fast activity; GSW = generalized spike-and-wave; PSW = polyspike-and-waves.

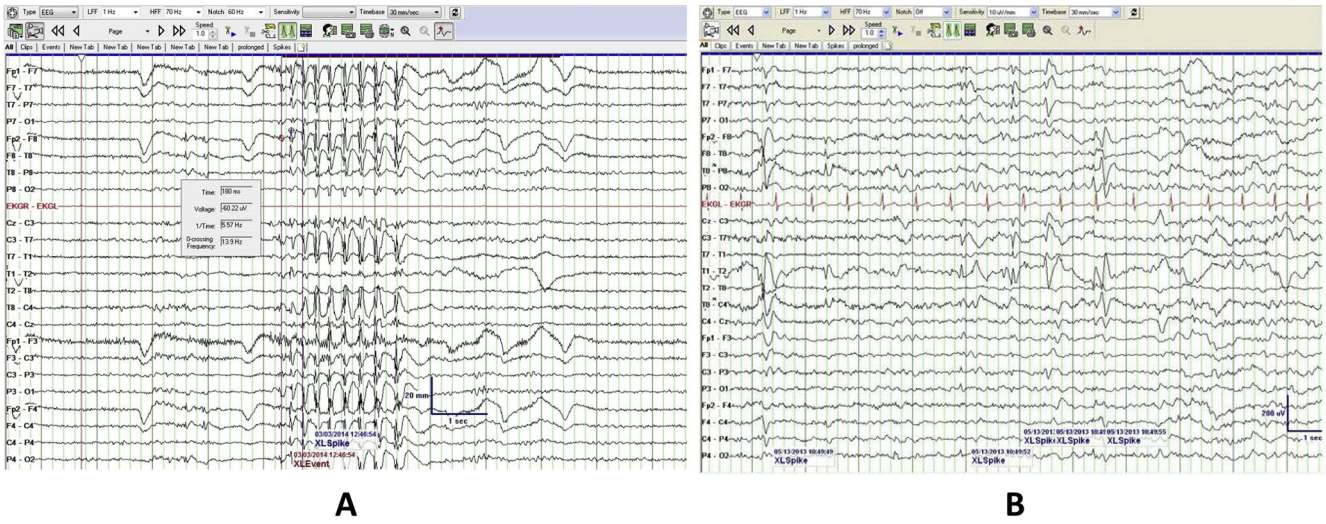

Fig. 3. (A) Fast spike-and-wave burst in an 18-year-old patient with GGE. (B) Frequent bitemporal IEDs in a 32-year-old undergoing a presurgical evaluation for drug-resistant focal epilepsy. GGE = genetic generalized epilepsies; IEDs = interictal epileptiform discharges.

(Fittipaldi et al., 2001), though specificity for individual syndromes remains limited. In GGE, focal abnormalities may occur and lateralized IEDs are often apparent as GSW and GPSW discharge *fragments* during sleep (Aliberti et al., 1994). Focal IEDs may be encountered in 30% or more of patients with JME (Jayalakshmi et al., 2010; Usui et al., 2005).

The term "epileptic encephalopathy" (EE) now replaces symptomatic generalized epilepsy as a term used where the epileptic activity itself contributes to severe cognitive and behavioral impairments above and beyond what might be expected from the underlying pathology alone (e.g., cortical malformation) (Scheffer et al., 2017). Many epilepsy syndromes associated with EE have an acquired and/or genetic etiology. Many of the severe seizure disorders also have developmental consequences arising directly from the effect of a genetic mutation, in addition to the effect of the frequent epileptic activity on development referred to as "developmental and epileptic encephalopathies (Scheffer et al., 2017), often associated with diffuse or multifocal cerebral pathology. Diffuse slowing of the background activity of the EEG and focal or multifocal abnormalities may be seen. There may be generalized IEDs with an interspike interval of 2.5 Hz or less (aka SSW). Some bursts of SSW may occur as prolonged runs that are asymptomatic. In other patients with seizures associated with EE, it may be difficult to differentiate an encephalopathy when it is acute, or even diagnose nonconvulsive status epilepticus (NCSE) when frequent generalized epileptiform discharges are present (Kaplan, 2007). Encephalopathy with status epilepticus during sleep (ESES) is an EE with heterogeneous clinical manifestations including cognitive motor, and behavioral disturbances in different associations, in addition to various seizure types that are related to a particular EEG pattern characterized by continuous paroxysmal spike and wave activity significantly activated during slow sleep (aka status epilepticus during sleep (Tassinari et al., 2000). Some patients exhibiting continuous generalized epileptiform discharges during sleep have an acquired age-related EE that presents with neurocognitive regression with or without overt seizures (Sánchez Fernández et al., 2012) or as acquired epileptic aphasia (Tassinari et al., 2000). It has become clear that electrical status epilepticus during non-REM sleep occurs at the time of clinical deterioration (Sanchez Fernandez et al., 2012). GPFA is another epileptiform abnormality typically seen in patients with EE occurring more frequently during non-REM sleep. It is manifest as generalized bursts of 15- to 25-Hz activity in the EEG, commonly associated with generalized tonic seizures though occasionally occurring during other seizure types (e.g., atonic seizures). In addition to the EEG features of SSW, abnormal background activity, and multifocal IEDs, GPFA is another characteristic feature seen in patients with LGS. Other symptomatic forms of the generalized epilepsies in adults include progressive myoclonus epilepsies (e.g., Unverricht-Lundborg disease and Lafora body disease). While some EEG features may suggest a specific type of EE (e.g., occipital IEDs in Lafora body disease, SSW in LGS), like GGE, none of the patterns of IEDs is syndrome-specific (Table 2).

6.2.2. Focal epilepsies

Focal seizures are the most common seizure type in adults with epilepsy. Initial EEGs uncovered focal epileptiform discharges in 44% of 116 patients (60% with sleep deprivation) clinically diagnosed with focal epilepsy in a category 1 study of 300 consecutive patients (King et al., 1998), the most epileptogenic region of the brain being the medial temporal lobe. Transient cognitive impairment was initially reported with occipital IEDs (Shewmon and Erwin, 1989) as brief cognitive dysfunction in direct relation to episodes of focal epileptiform activity in EEG. This must be distinguished from the chronic effect imposed during the interictal state, the postictal effect of seizures and from symptoms referable to the underlying etiology, with a clinical impact according to existing epidemiological data that is probably low (Aldenkamp and Arends, 2004). The clinical characteristics of medial TLE are well-described and fairly homogeneous including a blank stare, impaired awareness, and oroalimentary automatisms (French et al., 1993). The EEG may demonstrate focal slowing, which can occur in the form of temporal intermittent rhythmic delta activity often ipsilateral to seizure onset. Surface-negative anterior temporal spikes or sharp waves occur in more than 90% of patients (Williamson et al., 1993), and usually predict the side of seizure onset. Bilateral independent IEDs occur in one-third to one-half of cases (Fig. 3B) and are increased by long-term EEG recordings (Tatum, 2012). Neocortical (lateral) TLE has clinical features involving the neocortex (e.g., aphasia, visual dysfunction etc.) and have a greater predisposition to evolve to a bilateral convulsion. The interictal EEG more often has mid-temporal IEDs with a broad spatial distribution over the ipsilateral hemisphere.

Frontal lobe epilepsy (FLE) is the second most common form of focal epilepsy. The semiology may appear bizarre and present a diagnostic challenge for the clinician treating PWE. Seizure mimics include other paroxysmal neurological and non-neurological events (e.g., dystonia, syncope), sleep-related parasomnias, and

PNES, among others. Focal IEDs or focal paroxysmal fast activity is seen in 40–60% of EEGs in patients with FLE. Discharges may localize to the midline or appear as bifrontal or bilateral discharges more commonly than in patients with TLE. False localization of frontal lobe seizures to the temporal regions may also occur (Verma and Radtke, 2006). Generalized IEDs seen with FLE may be due to secondary bilateral synchrony with or without a structural lesion. This is often heralded by a rapid *lead in* of the IED from a focal region prior to the appearance of a generalized discharge. Sharply-contoured discharges may also be seen in midline electrodes during drowsiness and sleep. Midline spikes with an electrographic maximum on the EEG at Fz, Cz, or Pz may originate from either the mesial frontal or parietal cortices. The IEDs in epilepsy originating within the posterior quadrant that involve the parietal lobe may be elusive, and some reports suggest less than 15% manifest lateralized discharges (Salanova et al., 1995b). When IEDs are encountered in parietal lobe epilepsy (PLE), they may appear bilateral, be falsely localizing (often to the temporal region), or even be falsely lateralizing, with less than 10% of ictal EEGs being well localized. Similarly occipital lobe epilepsy (OLE) has well-defined IEDs over the occipital derivations in less than 20% of patients with IEDs. Furthermore, like other extratemporal lobe epilepsies, they may falsely localize to the temporal (especially posterior temporal) region (Usui et al., 2008a).

7. Activation techniques

The use of activation procedures during EEG is routine, and are used to elicit abnormalities in the EEG. They may be useful in patients where the diagnosis of underlying epilepsy is suspected, has already been made, or when the type of seizure or syndrome is unclear. HV and IPS are routine activation procedures used in each standard scalp EEG recording to enhance the likelihood of detecting IEDs, especially in GGE (Mendez and Brenner, 2006). Most category 1 and 2 evidence for increasing the yield with activation procedures exists in patients with GGE. Recording the EEG on awakening after a brief nap in conjunction with IPS and HV can increase the sensitivity of recording IEDs in up to 90% of patients with GGE when N1-N2 sleep is achieved (Unterberger et al., 2001; Leach et al., 2006; Koutroumanidis et al., 2008).

7.1. Hyperventilation

HV is the oldest activation procedure used during EEG (Gibbs and Gibbs, 1947) and is widely utilized in clinical practice (Binnie and Stefan, 1999). Numerous clinical studies (mostly category 3 and 4) exist, including two recent, large-scale, multicenter, prospective studies (category 1) that demonstrate efficacy and safety of HV during EEG (Kane et al., 2014; Craciun et al., 2015). HV should be performed routinely for a minimum of three minutes, unless medical or other reasons (e.g. pulmonary disease; raised intracranial pressure, cardiac disease) preclude its use (Sinha et al., 2016); it is recommended as part of the standard EEG recording by the ACNS (Sinha et al., 2016), United Kingdom (National Institute for Health and Care Excellence, 2012), and ILAE (Flink et al., 2002). In several category 2 studies, seizures in PWE were provoked by HV in 0.46–2.9% of patients (Holmes et al., 2004; Angus-Leppan, 2007; Kane et al., 2014; Craciun et al., 2015). HV and breath counting may help clarify the challenges involved in separating GSW along the interictal-ictal continuum by identifying a pause during counting associated with an absence seizure when accompanied by GSW. HV with breath counting may be helpful to distinguish epilepsy syndromes, such as GGE with GTC seizures alone from GGE with GTC seizures and absences when HV-induced absence seizures are precipitated (Koutroumanidis et al., 2008). Breath counting has also been a method for eliciting PNES in 0.95–1.1% of patients undergoing HV (Kane et al., 2014; Craciun et al., 2015). More than one-third (37%) of patients with PNES may have events precipitated by HV (Abubakr et al., 2010). Interictal EEG abnormalities were elicited or increased by HV in 11.9–12.2% of patients (Angus-Leppan, 2007; Kane et al., 2014; Craciun et al., 2015). A recent large-scale study showed that prolonging HV to five minutes increased the diagnostic yield (Craciun et al., 2015). More than one minute should be recorded in the post-HV period and usually two to three minutes are optimal (Sinha et al., 2016). Most of the seizures (85–88%) provoked by HV are generalized, especially absences, and in some rare cases, myoclonic seizures (Kane et al., 2014; Craciun et al., 2015). The role of HV in provoking focal seizures is less robust and more variable. In one category 2 study of patients with focal seizures, 4.4% had seizures precipitated by HV (Miley and Forster, 1977). However, in another category 2 study of patients with drug-resistant focal epilepsy, one-fourth of patients had seizures provoked by HV, with temporal lobe seizures more often provoked than frontal lobe seizures (Guaranha et al., 2005). The activation effect of HV was found to be potentiated in the subgroup of patients tapering ASDs in a category 2 study (Jonas et al., 2011). The rate of adverse consequences was very low; GTC seizure (0.03%), cardiovascular and respiratory adverse events (0.06%), tetany (0.001%), and nonspecific discomfort (0.003%) occurred in some people with HV (Kane et al., 2014; Craciun et al., 2015).

7.2. Sleep

Sleep is the most powerful and best documented modulator of seizures and of IEDs supported by category 2 and 3 studies (Gibbs and Gibbs, 1947; Guaranha et al., 2009; Beniczky et al., 2012a). There is evidence for the added diagnostic yield of sleep EEG recordings in patients suspected of epilepsy. Early category 1 studies by one investigator identified the diagnostic value of waking and sleep EEGs after sleep deprivation in PWE on ASDs (Degen, 1980) and of drug-induced sleep and sleep EEGs following sleep deprivation in patients with focal epilepsy (Degen and Degen, 1981). Spontaneous sleep, drug-induced sleep, and sleep deprivation to induce sleep are widely used in clinical practice. The complex relationship between seizures and sleep cycles or sleep-related oscillations was identified decades ago (Kellaway et al., 1980; Steriade and Amzica, 2003). Reports on the additional diagnostic yield of sleep-EEG vary widely (11–92%) due to differences in study design, patient population, and evaluation of confounders, with most investigating the additional yield of IEDs during sleep recording, in patients who previously had a normal standard recording. Sleep may activate IEDs in approximately one-third of PWE and up to 90% of those with epilepsies activated by sleep or awakening (Vaughn and D'Cruz, 2004; Pratt et al., 1968). There was an increase in IED frequency in non-REM sleep in 60% of patients, with the highest ratio of non-REM/wake, was 14:1 (Pavlova et al., 2009). When JME is excluded, sleep activated GSW in 57.2% of patients with GGE in routine EEGs (Giorgi et al., 2013). Several category 2 studies show sleep deprivation to activate IEDs independent of those found in sleep (Scollo-Lavizzari et al., 1977; Rowan et al., 1982; el-Ad et al., 1994; Fountain et al., 1998; Roupakiotis et al., 2000). and is an accepted practice adopted by European and American societies (Kasteleijn-Nolst Trenite et al., 2012; Sinha et al., 2016). The relative usefulness of different EEG protocols in patients with possible epilepsy has been explored in category 1 studies (Leach et al., 2006). Most of the studies used 24-h of sleep deprivation; however, two studies confirmed the activating effect of partial sleep deprivation (Carpay et al., 1997; Gustafsson et al., 2015). For drug-induced sleep, results are conflicting. Some studies found that melatonin

(Gustafsson et al., 2015; Wassmer et al., 2001) activated IEDs on EEG. Promazine hydrochloride (Degen and Degen, 1981) was found to be as effective as sleep deprivation in other studies but not by others (Rowan et al., 1982; Roupakiotis et al., 2000; Leach et al., 2006). Sleep may alter the morphology and location of IEDs. Approximately one-third of patients with typical GSW demonstrate polyspikes during stages 1 and 2 of sleep, and tend to have slower (<2.5 Hz) discharge rates during N3 non-REM sleep (Declerck, 1986; Veldhuizen et al., 1983). In deep sleep, IEDs become more irregular and disorganized (Sato et al., 1973). Activation of a contralateral focus during sleep is typically observed in patients with self-limited epilepsies such as benign childhood epilepsy with centrotemporal spikes (aka, rolandic epilepsy). In patients with JME, continued EEG recording after arousal may increase diagnostic yield (Janz, 1985).

7.3. Intermittent photic stimulation

Photosensitive epilepsy is the best known activation procedure. The signs and symptoms associated with the reflex photosensitive epilepsies include myoclonias, absences, eyelid fluttering, and cognitive impairment (Kasteleijn-Nolst Trenite et al., 1987; Kasteleijn-Nolst Trenite et al., 2001). Photosensitivity is detected on EEG as a paroxysmal epileptiform response to IPS. Paroxysmal photosensitive responses (PPR) occur in the EEG of patients with and without spontaneous epileptiform discharges. This reaction, is obtained by IPS performed before HV, or at least three minutes after HV when all HV-related EEG changes are expected to have resolved (Sinha et al., 2016). The middle flash frequencies, 10–20 Hz, are the most provocative, and there is a risk of evoking a GTC seizure during the stimulus. Therefore, frequencies in the lower flash frequency range should be used initially (Kasteleijn-Nolst Trenite et al., 2012). Photic stimulation should be performed in a room with low-level lighting. The lamp is placed at least 30 cm from the patient's face. Low-luminance, highly saturated red flickering lights with a wavelength around 700 nm has been reported (category 2) to induce a higher PPR provocation rate than standard white stroboscopic IPS in photosensitive patients (Takahashi et al., 1999). The proportion of cases with EEG evidence of photosensitivity varies substantially among studies when data are compared (Quirk et al., 1995b). Photosensitivity is age-dependent, being highest in late childhood and early adolescence, and is more common in females in some studies (Kasteleijn-Nolst Trenite, 1989). Overall, about 5% of PWE demonstrate a PPR, with most found among patients with GGEs (Kasteleijn-Nolst Trenite et al., 2011). In one study, 4.3% of PWE demonstrated a PPR elicited by IPS (Kasteleijn-Nolst Trenite, 1989); however, a range of 0.6% (Saleem et al., 1994) to 5.5% (Binnie and Jeavons, 1992) has been reported, with white patients having the highest prevalence (De Graaf et al., 1995). Both JME and GGE with GTC seizures alone are significantly overrepresented (Wolf and Goosses, 1986; Ricci and Vigevano, 1993). Photosensitive epilepsy is strongly associated with Jeavon's syndrome (Viravan et al., 2011). Focal epilepsy is rarely associated with a PPR, though OLE (Wolf and Goosses, 1986; Ricci and Vigevano, 1993; Shiraishi et al., 2001) and TLE (Gilliam and Chiappa, 1995) coexisting with a PPR have been reported in category 4 studies. Patients with EE, such as the progressive myoclonus epilepsies, may also demonstrate a PPR. Although photosensitive patients are most often encountered in an epilepsy population, a PPR can be an incidental finding in patients with other neurologic disease states. Parkinson disease, head trauma, alcohol and drug withdrawal, and dementia may coexist with a PPR, and migraine headache may be the only symptom (Parisi, 2009; Kasteleijn-Nolst Trenite et al., 2011). Furthermore, a PPR may exist as a genetic trait in normal people without seizures (Kasteleijn-Nolst Trenite et al., 2012). In families with a photosensitive parent, 50% of siblings may be affected, supporting the genetic basis for a PPR (Waltz and Stephani, 2000). Photosensitivity may relate to certain individual epilepsy syndromes.

7.4. Patient-specific activation

Specific activation methods may apply to some patients with reflex epilepsy. Therefore, use of an activation technique tailored to the individual with unique stimuli previously resulting in seizures can be informative as patients with reflex epilepsies may have normal EEG recordings unless individualized activation techniques are performed during the procedure. Patients with GGE share many of these reflex epilepsy traits, and patient-specific provocative methods (e.g., reading, talking, writing, mental arithmetic, and manipulating objects such as a Rubik's cube) can have important additional diagnostic yield, largely supported by category 4 studies (Koepp et al., 2016). Television epilepsy and electronic gaming-induced seizures (e.g., pocket monster in particular) play a role in reflex epilepsy associated with photosensitivity (Harding et al., 1994; Quirk et al., 1995a). Reading epilepsy is another recognized reflex epilepsy (Koutroumanidis et al., 1998). General and specific triggers together may be provocative in certain PWE (Woods and Gruenthal, 2006; El Bouzidi et al., 2010). Use of the more inclusive term, *provoked seizure*, reflects the specific factors involved with reflex seizures.

8. EEG and treatment

Predicting seizure recurrence in patients with a first seizure, and a successful response to a chosen ASD treatment is predicated on electroclinical features (Perucca and Tomson, 2011; Bergey, 2016). Provoked seizures (acute symptomatic seizures) impart a better prognosis and a lower risk for recurrence than seizures that are unprovoked. When a first seizure is unprovoked, there is category 1 evidence an epileptiform EEG increases the likelihood that seizures will recur (First Seizure Trial Group, 1993; Krumholz et al., 2015). An EEG with epileptiform abnormalities was associated with a relative rate increase for seizure recurrence at 1–5 years of 2.16 (95% CI 1.07–4.38) as compared with that in patients without such EEG abnormalities (Hauser et al., 1990). ASDs conferred a better prognosis to achieve seizure freedom in patients with a single unprovoked seizure and abnormal EEG when there was an existing neurological deficit, reducing the three-year risk of relapse from 72% to 50% (Kim et al., 2006). Focal abnormalities that were nonepileptiform did not portend seizure recurrence in one adult study (Hauser et al., 1982). However, other reports of nonepileptiform abnormalities in the EEG following a first seizure are conflicting (Hauser et al., 1982; Shinnar et al., 2000; Hirtz et al., 2003; Krumholz et al., 2015) suggesting a greater risk with normal neuroimaging (Shinnar et al., 1996, 2000).

The usefulness of EEG in predicting seizure recurrence before starting an ASD trial of taper in seizure-free patients is more controversial (Chadwick, 2006). When patients achieve long-term seizure control with ASDs (seizure-freedom for 2–5 years) and remission is suspected, tapering and possible discontinuing ASDs may avert chronic side-effects from long-term ASD use. In a patient with generalized seizures, the presence of GPSW in the EEG prior to contemplating a trial of ASD taper suggests a greater likelihood of seizure relapse and therefore may reinforce the need for continued therapy. Conversely, focal centrotemporal spikes in the EEG of healthy young adults are seen in self-limited epilepsies such as benign childhood epilepsy with centro-temporal spikes which usually has an excellent response to ASDs and prognosis for remission during adolescence. There is evidence (Peters et al., 1998) to support waiting at least two seizure-free years before discontinuing

ASDs, with recommendations derived from category 1 studies (Callaghan et al., 1988; Medical Research Council, 1991) favorable prognosis for seizure control was found when epileptiform activity regressed (Yamada et al., 2014). Some GGE patients (e.g., those with JME) require continued treatment despite prolonged seizure freedom. Even when epilepsy patients have been successfully controlled with ASDs for four to five years, they may be at significant risk for recurrence when persistent EEG abnormalities are present prior to drug withdrawal (Buna, 1998; Gavvala and Schuele, 2016). In patients with focal epilepsies before ASD reduction worsening EEG abnormalities resulted in 83% of patients relapsing compared to 69% with an epileptiform EEG, 60% with a normal EEG and 54% of patients with an EEG that was unchanged (Tinuper et al., 1996). Following successful epilepsy surgery, an abnormal EEG has been shown to predictseizure recurrence following ASD withdrawal (Jeha et al., 2006; Rathore et al., 2011). Prolonged EEG monitoring may increase the yield of IED recovery beyond a standard EEG and be better in predicting treatment relapse (Verrotti et al., 2000). Therefore, successful withdrawal of ASDs depends upon the timing of drug taper (Chadwick, 2006), an accurate classification of the epilepsy syndrome (Schmidt and Schachter, 2014), and the duration of EEG recording (Verrotti et al., 2000).

9. Emergency EEG

Emergent, or *stat*, EEG is frequently performed to exclude nonconvulsive seizures and NCSE (Leitinger et al., 2016) and influence management (Glauser et al., 2017). In a single-center study (category 3), 45% of cases suspected of subtle status epilepticus were confirmed by emergency EEG, and upon diagnosis, the treatment was modified in 46.6% of cases (Praline et al., 2007). A retrospective ICU study of the use of emergency EEGs showed that ASD changes occurred in 1/8 patients (Varelas et al., 2004). While single seizures did not predict a poor outcome, the presence of status myoclonus was associated with poor outcome and in-hospital death (Zandbergen et al., 2006; Wijdicks et al., 2006). In 236 comatose patients, Towne and colleagues found that 8% of patients in the ICU were in NCSE (Towne et al., 2000).

The presence of generalized epileptiform discharges on EEG predicted a poor outcome with insufficient accuracy in one North American study (Wijdicks et al., 2006). Similarly, emergency EEG in a neurocritical care population in Southeast Asia (Khan et al., 2005) revealed abnormalities in 62.1% of patients, including epileptiform discharges, discrete seizures, and NCSE (Khan et al., 2005). In one category 2 study, EEGs with epileptiform discharges predicted outcome after anoxic/hypoxic encephalopathy from cardiopulmonary arrest (Alvarez et al., 2013; Foreman et al., 2012). NCSE is increasingly diagnosed once periodic discharges at 2.5 Hz or greater was used as a criterion (category 2 study) (Leitinger et al., 2016). Periodic discharges predict subclinical (electrographic) seizures, clinical seizures, and NCSE in critically ill patients (Brenner and Schaul, 1990; Nei et al., 1999; Foreman et al., 2012; Rodriguez Ruiz et al., 2017). Specific discharge characteristics on the EEG, such as higher amplitude, longer duration, and higher background amplitude, suggest a more favorable prognosis (Husain et al., 1999) and may exhibit a more favorable response to ASDs (Nei et al., 1999). Some investigators suggest that the presence of periodic discharges on EEG are an independent marker for poor clinical outcome (Foreman et al., 2012); others suggest the opposite (Yoshimura et al., 2016). More patients developed epilepsy following an acute illness when they demonstrated periodic discharges on EEG (Pedersen et al., 2013). Persistence, duration, and frequency of periodic complexes provided prognostic outcome information and predicted subsequent epilepsy (Pedersen et al., 2013). cEEG has been shown to detect seizures and seizure emergencies significantly more often when compared to a standard 20- to 30-min EEG recording (Sutter et al., 2011).

10. Extended EEG in seizure detection and prediction

A variety of technologies and techniques have evolved to detect ongoing seizures in PWE (Ramgopal et al., 2014). EEG-based seizure detection systems exist, where signals are extracted, processed, quantified, modeled, and classified using a variety of computer algorithms with many developed for real-time application. Accurate detection systems must reliably determine if and when a seizure occurs. Currently available EEG systems use sensors applicable to all age groups, and include single and multi-channel recordings. Most can adapt to both scalp and intracranial EEG and can accommodate IED-based software detection algorithms and programs able to perform seizure modeling in both static and ambulatory environments. Most detection systems have imperfect predictive value with a substantial, but variable number of false positive detections (Aarabi et al., 2006; Kharbouch et al., 2011; Ramgopal et al., 2014). Electrophysiological signals from brain, scalp, skeletal muscle, cardiac muscle, electrodermal activity, and other vital signs in combination may enhance the ability to detect seizures (Jeppesen et al., 2010; Beniczky et al., 2013d; Ramgopal et al., 2014), and video-based detection may also be employed (Lu et al., 2013). Some sensors record signals directly from the brain, while most record signals indirectly from a noncerebral source (Narechania et al., 2013).

A variety of analytic techniques have been applied to EEG data for the purposes of seizure prediction. Most of this work has been hampered by lack of appropriate statistical validation (Mormann et al., 2007; Gadhoumi et al., 2016). To date, studies have been based on analysis of intracranial electroencephalographic data obtained during presurgical evaluation acquired over relatively short time intervals (days to weeks). Most datasets have been relatively small, and the analyses retrospective, with few seizures or limited interictal data. The critical importance of having large, long-term (months to years duration) datasets has been highlighted in a category 1 canine epilepsy study (Howbert et al., 2014) and by a first-in-man clinical trial (category 2) of a device that demonstrated for the first time that seizure prediction was possible in 15 patients (Cook et al., 2013) showing that seizure prediction is feasible, with prediction horizons of up to four hours (Cook et al., 2013). The highly individual nature of patient seizure patterns is another striking feature, suggesting that algorithms will need to be individually optimized. Seizure prediction techniques are being pursued and hold promise for notifying PWE in advance of a seizure (Kalitzin et al., 2005; Stypulkowski et al., 2013, 2014; Freestone et al., 2013), but are in the early stages of development and suffer from limitations related to cost and invasiveness.

11. Ambulatory EEG monitoring

Outpatient aEEG monitoring is 51–65% less expensive than inpatient 24-h VEM and less restrictive to patients, allows patient assessment in their natural environment with exposure to daily natural seizure triggers (Schomer, 2006). Overall, there is good evidence from early studies (most categories 3 and 4) that aEEG is feasible and can yield similar diagnostic information to inpatient EEG (Ebersole and Leroy, 1983). Concordance of aEEG with routine scalp EEG in detecting epileptiform abnormality was 77%; 79% of focal IEDs and 100% of generalized IEDs in the initial studies (Ebersole and Leroy, 1983). Prolonging the EEG recording has demonstrated a higher yield, with 95% of IEDs recorded within a two-day period (Faulkner et al., 2012a). Faulkner et al. (Faulkner et al., 2012a), showed that among 180 PWE, the median latency

to the first IED was about five hours with generalized IEDs occurring significantly earlier than focal IEDs. Another report noted that 68% of 324 consecutive aEEGs gave positive results (36% with IEDs and 52% with events) and changed management in 51%, established a diagnosis in 22%, and refined a previous diagnosis in 29% When compared with sleep-deprived EEG for ictal recording following an initial nondiagnostic EEG in PWE, a category 1 study found aEEG offered greater benefit (Liporace et al., 1998). Both the sleep-deprived EEG and aEEG improved detection of IEDs by a similar amount (24% versus 33%), but aEEG detected seizures in 15% of patients compared with no patients identified during sleep-deprived EEG. In one study, 58% of typical ictal events (seizure or nonepileptic event) occurred within 24 h, improving to 78% after 72 h, and reaching 100% by 96 h (Faulkner et al., 2012b). ILAE guidelines recommend the use of prolonged EEG when the diagnosis of epilepsy or the classification of seizure syndrome proves difficult with standard scalp EEG (Faulkner et al., 2012b) given the frequency of seizure mimics. One study found among 101 patients undergoing aEEG monitoring, 61% had psychogenic attacks (Shihabuddin et al., 1999). In a study of 6923 IEDs recorded in 105 abnormal 24-h aEEGs, the density of IEDs and the paroxysm durations were greatest in juvenile absence epilepsy, followed by JME, childhood absence epilepsy, and generalized epilepsy with GTC seizures on awakening. Generalized linear mixed models revealed that *pure* GSW discharges (without intervening polyspikes/polyspike-and-slow waves) were usually more frequent in absence epilepsies, suggesting the density and duration of IEDs can help differentiate among GGE syndromes (Seneviratne et al., 2017). In a large retrospective series (category 2) of 502 aEEG studies, 23.4% of events were not identified by the patient via push-button activation or bystander observation (Tatum et al., 2001). The inability for physicians to observe seizure semiology with aEEG in the past has been a major drawback. It is now possible with home video-EEG telemetry to extend the value of aEEG with the addition of video beyond seizure classification (supported by category 1 and 2 studies) to obtain information about nonepileptic diagnoses (Brunnhuber et al., 2014; Goodwin et al., 2014).

12. Long-term video-EEG monitoring

Long-term VEM facilities, including epilepsy monitoring units (EMU), seek to correlate the clinical seizure semiology and the corresponding EEG changes. The ILAE recommends long-term VEM in PWE when there is diagnostic uncertainty to classify seizure type or epilepsy syndrome, quantify seizures, and evaluate electroclinical seizure characteristics prior to epilepsy surgery (Velis et al., 2007). The diagnostic usefulness of VEM varies widely (19–75%) and depends on how utility is defined and on the selection of the patients evaluated (Alving and Beniczky, 2009; Deacon et al., 2003). Video alone may be a useful means of supplementing descriptions of a witnessed event (Beniczky et al., 2012b), but is unable to confirm a definitive diagnosis without EEG recording, adequately document seizure frequency (including status epilepticus), or characterize patients for epilepsy surgery (Velis et al., 2007). Long-term VEM records IEDs within the first 72 h in the majority of epilepsy patients according to prospective and large retrospective studies (Werhahn et al., 2015). In addition, documenting specific patterns in the occurrence of IEDs during sleep or of disruption of sleep architecture in PWE is essential (e.g., electrical status epilepticus in slow sleep). A system employing algorithms to detect the onset of seizures in PWE during scalp VEM showed a sensitivity in excess of 75% with low false positive detection rate in a category 1 study (Saab and Gotman, 2005). Combining polysomnography with EEG and utilizing full EEG montage and video is especially useful to assist in diagnosing paroxysmal nocturnal events and differentiate between the primary sleep disorders, seizures in PWE, or both. For the investigation of known PWE and sleep complaints or excessive daytime sleepiness, documenting diurnal or circadian variation is recommended to synchronize electroclinical patterns on EEG during polysomnography (Velis et al., 2007; Bubrick et al., 2014). The majority of the literature on VEM centers on cohorts of patients undergoing epilepsy surgery, including noninvasive and invasive presurgical evaluations (Sauro et al., 2014, 2016). In addition, cEEG monitoring in critically ill patients in the ICU has been increasingly used to identify and quantify subclinical seizures and electrographic status epilepticus and assist in management (Jirsch and Hirsch, 2007; Gavvala and Schuele, 2016).

12.1. Differential diagnosis

The symptoms of seizures in PWE are diverse. Arriving at a definitive diagnosis of epilepsy may be challenging given the large differential diagnosis. A wide range of patients treated for epilepsy however, are ultimately discovered to not have epileptic seizures when VEM is performed. The differential diagnosis of recurrent spells is the most frequent reason why patients are referred for VEM (Binnie et al., 1981; McBride et al., 2002; Ghougassian et al., 2004; Alving and Beniczky, 2009). A meta-analysis of 135 published studies on VEM showed that 59% of the referrals were for diagnostic reasons (Sauro et al., 2014). In approximately 20–30% of patients, nonepileptic events were diagnosed based on VEM results (Alsaadi et al., 2004; Benbadis et al., 2004; Alving and Beniczky, 2009; Friedman and Hirsch, 2009). The vast majority of these patients had PNES. Many different terms have been used to describe them in the literature (Brigo et al., 2015). The most common nonepileptic paroxysmal events identified by VEM were syncope or near-syncope, sleep-disorders (cataplexy, parasomnia), and transient ischemic attacks in adults (McBride et al., 2002; Friedman and Hirsch, 2009). It can be particularly difficult to distinguish nocturnal FLE from parasomnias and movement disorders (Derry et al., 2009). The foundation for the efficacy of VEM in the differential diagnosis of PWE is based upon the EEG pattern recorded during a typical episode. In patients with seizure mimics, the EEG is unchanged from baseline during the event and occurs without an ictal EEG change characteristic of PWE. The habitual event captured in the setting of prolonged unconsciousness, despite the presence of a normal physiological alpha rhythm on the EEG with or without an atypical semiology, is essentially diagnostic of a nonepileptic event (Benbadis et al., 2004). Category 2 studies suggest interictal EEG abnormalities alone are unable to differentiate between seizures in PWE and PNES (McBride et al., 2002; Friedman and Hirsch, 2009). In addition, historical reporting by a witness may be misleading (Deacon et al., 2003). Seventeen to 26% of patients eventually diagnosed with non-epileptic events had IEDs recorded during the VEM session (McBride et al., 2002; Friedman and Hirsch, 2009). In a category 1 study involving review of video and EEG independently to establish the diagnosis of nonepileptic events, only moderate accuracy ($\kappa = 0.57$, 95% CI 0.39–0.76) in the inter-rater reliability occurred (Benbadis et al., 2009). Outpatient VEM can be performed successfully with activation to provide the diagnosis of PNES in many patients otherwise in need of inpatient VEM, according to a randomized, controlled trial involving simple suggestion techniques (McGonigal et al., 2002; Benbadis et al., 2004).

Video-EEG telemetry can be a crucial tool for neurologists experienced in epilepsy when diagnosing seizure disorders. The reported inpatient diagnostic yield according to several category 2 studies varies considerably (Binnie et al., 1981; Alsaadi et al., 2004; Ghougassian et al., 2004; Alving and Beniczky, 2009). This

is due to the differences in the patient-populations studied, and how the diagnostic yield was defined. A large, prospective category 1 study demonstrated the utility of VEM in clarifying the diagnosis for 56.3% of patients (Sauro et al., 2014). A meta-analysis indicated that VEM changed the diagnosis in 35.6% of patients (Sauro et al., 2016). There was no significant difference in diagnostic yield between reasons to referral for VEM (Alving and Beniczky, 2009). Even in patients extensively investigated before monitoring (Alving and Beniczky, 2009), and in patients who previously had aEEG recordings (Alix et al., 2014), VEM was useful in 41–77% of patients. The diagnostic yield of the monitoring did not differ among age groups (Alving and Beniczky, 2009), and it also proved to be useful in elderly patients in retrospective trials (McBride et al., 2002; Kipervasser and Neufeld, 2007) despite different neurological disorders compared with younger adults (Keranen et al., 2002). The duration of diagnostic VEM is typically shorter when compared with a presurgical evaluation, typically lasting two to three days (Alving and Beniczky, 2009; Friedman and Hirsch, 2009). While VEM is the *gold standard* for diagnosis, psychiatrists often view VEM results as inaccurate in the diagnosis of PNES compared with neurologists (Harden et al., 2003).

12.2. Classification and quantification of seizures

Classification must take into account the results of investigations such as EEG and neuroimaging studies together with other studies exploring the underlying etiology of the epilepsy, as it often carries significant treatment implications (Scheffer et al., 2017). The impact of EEG interpretation on treatment was demonstrated in one retrospective study (category 4) where 70% of patients with GGE following VEM were found to be taking inappropriate ASDs capable of adversely affecting seizure control (Benbadis et al., 2003b) capable of resulting in pseudo-drug-resistance (Schmidt, 2009). Although EEG is routinely used to support a clinical diagnosis of epilepsy, VEM may be needed to classify the type of seizure, epilepsy, or epilepsy syndrome. Semiological classification of seizures is a reliable method applicable for everyday use during outpatient visits (Blume et al., 2001), and a descriptive semiology classification system has been suggested (Luders et al., 1998). However, in a study of 90 patients, some seizure types had excellent semiology consistency between description before and after VEM (e.g., myoclonic and hypermotor seizures), while others did not (e.g., auras and focal seizures) (Hirfanoglu et al., 2007). Certain forms of epilepsy have special clinical and EEG characteristics, regardless of their heterogeneous etiologies. Electroclinical classification of epilepsies into focal and generalized seizures (Berg et al., 2010) is recognized as a continuum (Engel, 2006), and some seizures cannot be classified. The concepts of generalized and focal when used to characterize seizures now explicitly references involvement of neuronal networks (Berg et al., 2010). In generalized epilepsies, GSW and GPSW may with measurable behavioral changes, such as impairment of cognition, motor and autonomic manifestations on video and polygraphy, or appear to be subclinical unless appropriate testing is performed. Some epilepsy syndromes activate the EEG during deep stages of sleep, not seen with standard scalp recording. During overnight VEM, GSW discharges may occur more frequently, acquire a polyspike component, and become shorter, incomplete, or fragmented during deep sleep. Typically, the appearance of IEDs is inhibited during REM sleep.

Quantification of seizure frequency and severity is indispensable in validating reported seizure frequency. In addition, EEG may be used to assess the efficacy of therapy in PWE. Prolonged outpatient VEM more reliably quantifies seizure frequency compared to historical reporting by PWE (Stefan et al., 2011). Generalized epilepsies and patients with severe epilepsy may benefit from objective seizure quantification to optimize treatment inpatient and outpatient VEM (category 2) studies, show that 20–30% of patients were not aware of their seizures compared with historical reporting (Blum et al., 1996; Tatum et al., 2001). Blum et al. found that patients with convulsions associated with GGE were always aware they had a seizure, while patients with focal seizures evolving to bilateral convulsions produced variable post-ictal self-awareness. Many patients exhibit variable seizure awareness after the event (Blum et al., 1996; Kerling et al., 2006; Langston and Tatum, 2015) and some seizures only present with transient epileptic amnesia (Butler et al., 2007; Zeman and Butler, 2010). In one study, using a patient questionnaire in the EMU 44.2% of VEM-proven seizures went unnoticed (Kerling et al., 2006). In several studies, more seizures involving the left temporal lobe went unrecognized (Kerling et al., 2006; Heo et al., 2006; Langston and Tatum, 2015). Seizure detection algorithms provide high values for sensitivity and specificity in addition to push-button activation of patient or witness-identified seizures. A study of 159 patients (composed predominantly of patients with TLE) analyzed 794 seizures with a sensitivity of 87.3% and 0.22 false detections per hour (Hopfengartner et al., 2014). Automated seizure detection systems using algorithms which extract and classify features of EEG and other signals may improve outcomes in patients and allow more tailored therapies and potentially decrease morbidity and mortality (Ramgopal et al., 2014).

12.3. Presurgical evaluation

Epilepsy surgery has been the only treatment (category 1) that can result in seizure freedom in patients with drug-resistant focal epilepsy (Wiebe et al., 2001; Engel et al., 2003). A scalp-based (phase 1) VEM evaluation is an integral component in the presurgical evaluation of drug-resistant epilepsy. Concordant IEDs in the interictal EEG and brain MRI focal abnormality are often sufficient in lesional TLE to predict a seizure-free outcome following resective epilepsy surgery (Polkey, 1994; Diehl and Luders, 2000; Blume, 2001). Therefore, scalp presurgical evaluation is often used to identify patients with concordant patterns. During scalp VEM, different sources may produce the same scalp epileptiform potentials and not reflect the site of epileptogenesis (Ebersole, 1999; Tao et al., 2007). Only 20–30% of focal seizures without impaired consciousness are visible on scalp EEG during the seizure due to the limited volume or distance from the scalp recording electrodes (Palmini and Gloor, 1992; Sirven et al., 1996). On the other hand, scalp EEG changes are present in 85–95% of focal seizures with impaired consciousness (Verma and Radtke, 2006). In one study, approximately two-thirds of seizures were localized, while one-third was not (22% non-localized/non-lateralized and 4% and 6% were incorrectly localized or lateralized respectively (Foldvary et al., 2001). Rarely, seizures may be falsely lateralized to the incorrect hemisphere involved in seizure onset during scalp ictal EEG recording (Spencer et al., 1985). This may be anticipated when it occurs in the hemisphere opposite a structural lesion (Mintzer et al., 2004; Foldvary et al., 2001). Localized seizure onsets using standard scalp EEG recording were more common when comparing mesial TLE, dorsolateral FLE, and PLE to lateralized seizures in neocortical TLE, and generalized seizure onset in mesial FLE and OLE (Foldvary et al., 2001). False localization and lateralization were not uncommon features in parietal and occipital lobe epilepsies (Foldvary et al., 2001). Seizure onset with gamma and other HFOs are more limited by scalp recording than iEEG. Detection of low-amplitude fast activity, small or remote generators, and the presence of myogenic artifact during seizures are a few examples why invasive EEG recording may be necessary to identify HFOs beyond the bandwidth of 1–30 Hz (aka Berger band) used in routine scalp-based EEG.

13. Scalp EEG monitoring in temporal lobe epilepsy

TLE is the most common form of the adult focal epilepsies. Although seizures originate from the mesial structures of the temporal lobe in the majority of cases, about 10% of patients with TLE have seizures arising from the lateral neocortex (Schramm et al., 2001). The role of scalp VEM confirms the diagnosis of TLE and plays a crucial role in surgical management as patients are frequently drug-resistant (Engel et al., 2003). Category 3 studies in TLE confirmed the presence of unilateral temporal IEDs or slowing can predict localization of ictal scalp EEG changes with a high degree of reliability (Blume et al., 1993; Pataraia et al., 1998). Bitemporal spikes are present on standard scalp EEG in approximately one-third of patients with TLE, though invasive EEG recordings demonstrate seizures arise primarily from a single temporal lobe in the majority of cases (Engel et al., 2003; Tatum, 2012). A more recent retrospective study (category 3) in mesial TLE patients with mesial temporal sclerosis found 47.1% of standard scalp EEG recordings were normal or had nonspecific features, though sleep deprivation improved the yield (Dericioglu et al., 2010). In an early study, ictal EEG changes were rarely detected at the time of clinical seizure onset, but lateralized buildup of rhythmic activity on the EEG during a seizure occurred in 80% of patients (Risinger et al., 1989). In 13%, the scalp EEG seizure buildup was, however, contralateral to the side of seizure origin as subsequently determined by depth EEG and resection. In another study of 44 patients, seizures originated exclusively or with a strong predominance in one temporal lobe in 77% when recording iEEG with depth electrodes. Seizures originated in a single temporal lobe in 44%, and in an additional 33% of cases, more than 80% of seizures arose from one side (So et al., 1989). In a scalp-intracranial ictal EEG study (category 3), Ebersole and Pacia found that a regular 5- to 9-Hz inferotemporal rhythm in the anterior-basal temporal scalp electrodes (Fig. 4) was most specific for hippocampal-onset seizures (Ebersole and Pacia, 1996). Another study found that more than 70% of patients had low-voltage fast activity as the initial pattern on iEEG and when it was visible at the scalp, rhythmic delta or theta activity was then identified (Pelliccia et al., 2013). Neocortical TLE has rhythmic delta slowing at seizure onset, a wider hemispheric distribution, rapid propagation, and a shorter duration on the ictal EEG. Postictal slowing may be encountered in 70% of seizures and is less consistent than seizure onset in reflecting the site and side of seizure onset (Ebersole and Pacia, 1996). In contrast, consistent postictal slowing was found to be a very reliable lateralizing finding in other studies (Williamson et al., 1993). The usefulness and reliability of scalp EEG in contributing to identifying and selecting patients with drug-resistant mesial TLE eligible for surgery has been further established by two randomized controlled trials designed to compare effectiveness of medical therapy with surgical treatment (Wiebe et al., 2001; Engel et al., 2012). The EEG pattern in mesial TLE due to hippocampal sclerosis showed that rhythmic theta activity was the most frequent pattern seen at seizure onset and correlated with successful postsurgical outcome (Sirin et al., 2013). In one category 3 study, rhythmic ictal theta activity predicted a seizure-free postsurgical outcome in patients with TLE and a normal MRI (Tatum et al., 2008). Dericioglu and Saygi (Dericioglu and Saygi, 2008) showed that ipsilateral temporal rhythmic theta-delta activity occurred in 85.2% of the seizures in mesial TLE an average of 13.4 s after onset and was associated with Engel I postsurgical outcome at five years. Conversely, a study of ictal scalp EEG patterns in 284 patients with TLE associated with hippocampal sclerosis (HS) found that ictal EEG patterns did not affect Engel outcome after temporal lobectomy (Monnerat et al., 2013). Patients with nonlocalized/nonlateralized seizure onset or independent bilateral seizure onsets were more likely to have postoperative seizures than patients with unilateral seizure onset on EEG (Dericioglu and Saygi, 2008). A prospective (category 1 study) of 82 patients with bilateral mesial TLE t (King-Stephens et al., 2015) using intracranial ambulatory electrocorticography (ECoG) captured a first electrographic seizure contralateral to the initial seizure on average, at 41.6 days (median 13 days, range 0–376 days) in about one-third of the patients. This illustrates the variable seizure lateralization in patients with bitemporal epilepsy.

14. Scalp EEG monitoring in extratemporal lobe epilepsy

Interictal scalp EEG of extratemporal lobe epilepsy (ETLE) patients is more complex and heterogeneous than those encountered with TLE. It is often times normal or nonlocalizing (Westmoreland, 1998). The ictal scalp EEG in ETLE demonstrates shorter seizure duration, lack of postictal EEG changes and more

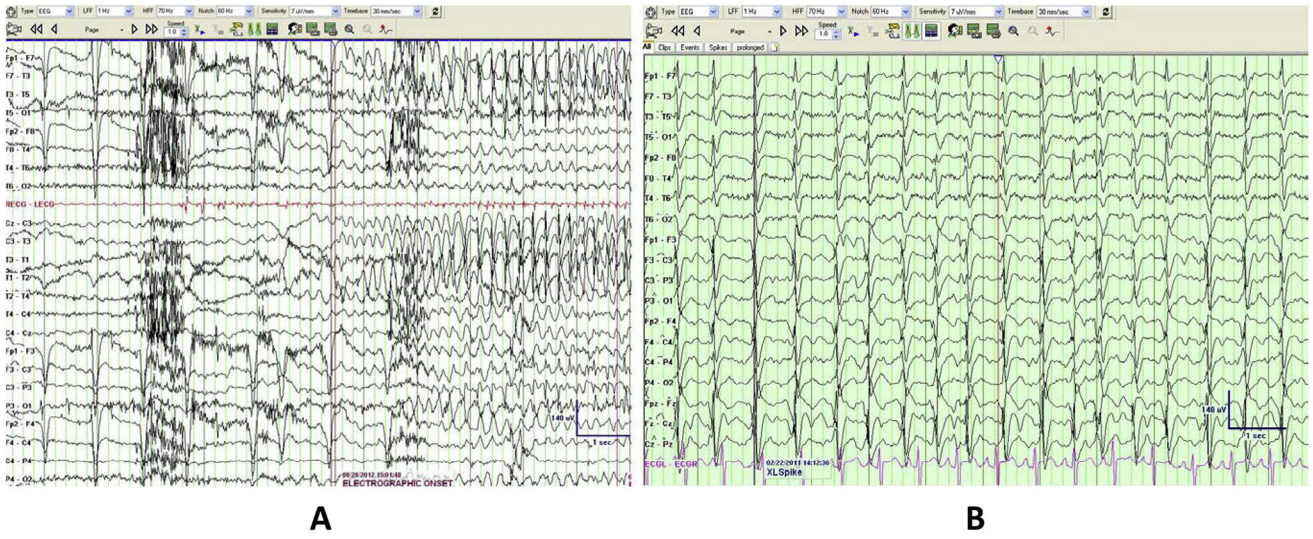

Fig. 4. (A) Focal seizure recorded during video-electroencephalography monitoring as part of a comprehensive presurgical evaluation. (B) Generalized periodic discharges during continuous EEG monitoring following anoxia in a comatose survivor after cardiac arrest.

difficult lateralization as compared to temporal lobe epilepsies (Walczak et al., 1991). Furthermore, the absence of IEDs in standard EEG and even prolonged VEM has been reported as a feature of ETLE that can be observed in over 30% of patients, according to category 2 reports (Stuve et al., 2001; NOE et al., 2013). When IEDs are detected in mesial FLE, they may be falsely lateralized. When localized repetitive epileptiform activity was seen, it was highly predictive of lateral FLE (Foldvary et al., 2001). In a category 2 study of patients with FLE and TLE, interictal rhythmic midline activity was found to occur in up to 48% of patients with FLE (62% of those patients lacked IEDs). This observation is frequently seen in FLE (Beleza et al., 2009). In category 2 and 3 studies, when unifocal IEDs were detectable in ETLE, they reliably predicted the SOZ and a favorable post-surgery outcome (Holmes et al., 2000; Liava et al., 2012). A recent category 3 study has shown that valuable lateralizing and localizing information (such as lateralized or regional slow activity, or lateralized or regional spikes) can be obtained by the analysis of the postictal EEG in about 40% of FLE patients (Whitehead et al., 2016). The presence of residual IEDs in the postoperative scalp EEG portended poor outcome, when IEDs occurred in both awake and asleep EEG. This observation was seen as early as two months after surgery (Di Gennaro et al., 2012).

The diagnostic yield of scalp EEG in patients with FLE during seizure monitoring is often limited compared to patients with TLE due to difficulty detecting mesial and basal cortical seizure onset. The ictal EEG in FLE is nonlocalized in more than 50% of patients. Bilateral attenuation, bilateral slow waveforms (theta and delta), and generalized epileptiform activity may be encountered without lateralization when artifact does not intercede to obscure the tracing. Category 3 studies have shown diffuse electrodecremental ictal EEG patterns may result from a regional cortical high-frequency ictal discharge in a single frontal lobe on intracranial recordings (Arroyo et al., 1994). Focal beta activity at seizure onset is seen in about 25% of FLE patients and predictive of excellent seizure outcome (Worrell et al., 2002). Such localized ictal fast activity suggests seizure onset in the dorsolateral prefrontal cortex, and have a good surgical prognosis (Zakaria et al., 2012; Liava et al., 2012). Patients with seizures originating in the medial frontal region often show either no IEDs or multifocal epileptiform discharges (Bautista et al., 1998; Vadlamudi et al., 2004). Absence of generalized EEG signs was associated with seizure-freedom (category 2 study) (Janszky et al., 2000); conversely generalized or nonlocalized ictal EEG patterns predicted seizure recurrence (Jeha et al., 2007); not all studies confirm these findings (Ferrier et al., 1999), and the different sample size and methodology may explain these differences.

The interictal and ictal EEG in PLE is typically nonlocalized. Focal ictal rhythms are seen in a minority of cases and often with diffuse rhythmic ictal activity that may be falsely localizing or lateralizing (Williamson et al., 1992a). In PLE patients, IEDs may possess variable topographies (such as fronto-centro-parietal, parieto-posterior-temporal, parietal, parieto-occipital, fronto-centro-temporal, fronto-temporal-parietal), which may be detected in more than 90% of subjects (Salanova et al., 1995a). However, regional or localized ictal EEG changes were detected in only 10–35% of PLE patients (Lee et al., 2000; Kim et al., 2004). These category 3 findings have been confirmed in a recent category 2 study showing there is a more variable scatter of IEDs and a lower localizing value of ictal recordings in patients with PLE when compared to TLE and FLE patients (Ristic et al., 2012).

Similar to PLE, in OLE, a well formed ictal rhythm is infrequently localized to one occipital lobe. Electroclinical manifestations, evaluation, and outcome have largely come from smaller series of category 3 and 4 data due to the lower prevalence and operative morbidity associated with OLE. Patient with OLE who have undergone iEEG have demonstrated rapid propagation from the occipital lobe to the temporal lobe or even the frontal lobe (Usui et al., 2008b). The interictal scalp EEG in OLE is consistently abnormal, but frequently misleading. Posterior temporal IEDs are the most common interictal finding on scalp EEG (Williamson et al., 1992b). Posterior cortex epilepsy with posterior temporal-occipital epileptiform abnormalities have been detected in 46% of patients (Salanova et al., 1992), whereas ictal EEG has shown non-localizing value in up to 90% of patients (Lee et al., 2000). In another study in patients with lesional OLE who underwent surgery, ictal EEG provided correct localization of the lesion in 13 of 16 post-surgery seizure-free patients and 5 of 10 nonseizure-free patients (Kun Lee et al., 2005).

15. EEG source localization

There is good evidence (category 1 and 2 studies) for the accuracy of electroencephalographic source imaging (ESI) in presurgical evaluation of both temporal and extratemporal foci (Brodbeck et al., 2011; Megevand et al., 2014; Rikir et al., 2014; Lascano et al., 2016). The accuracy ranged from 67% and 88%, with a positive predictive value between 80% and 97% and a negative predictive value between 43% and 61%. One accuracy study analyzed ictal discharges (Beniczky et al., 2013c), the others analyzed IEDs. Averaging was used to increase the signal-to-noise ratio of the IEDs in most studies (Maliia et al., 2016). EEG source imaging was obtained from head models and based on individual brain MRI (Megevand et al., 2014; Rikir et al., 2014; Lascano et al., 2016; Maliia et al., 2016), with both individual and templated head models (Brodbeck et al., 2011) and a template head models alone (Beniczky et al., 2013c) evaluated (Fig. 5). Several studies used distributed source models (Brodbeck et al., 2011; Beniczky et al., 2013c; Megevand et al., 2014; Lascano et al., 2016), one used equivalent current dipoles (Maliia et al., 2016), and one used both techniques (Rikir et al., 2014). The accuracy of ESI was higher when high-density EEG arrays (128–256 electrodes) and an individual head model was used relative to low-density EEG arrays involving fewer than 32 electrodes and a template head model (Brodbeck et al., 2011). The combination of MRI and high-density ESI offered the highest predictive value for postoperative seizure-freedom (Lascano et al., 2016). Interictal ESI co-localized with the intracranial electrode contacts recording IEDs, and the median distance from the ESI maximum to the nearest electrode involved in the SOZ was 17 mm (Megevand et al., 2014). The clinical utility of

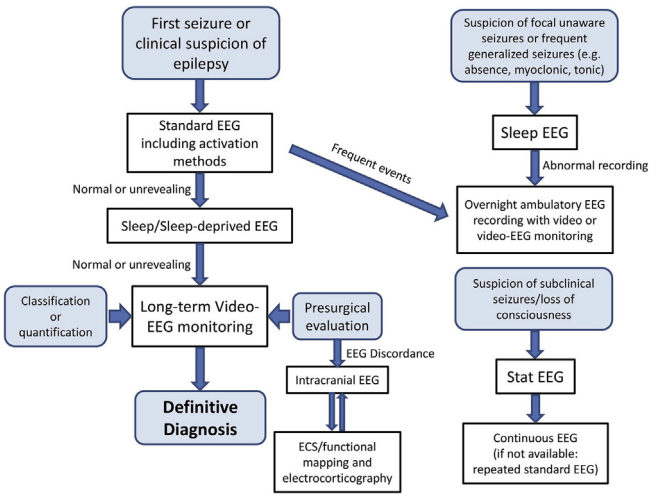

Fig. 5. Algorithm developed to illustrate the approach to using EEG for diagnosis and seizures monitoring.

ESI in presurgical evaluation was addressed by one category 1 study (Boon et al., 2002) and one category 2 study (Maliia et al., 2016). In a prospective series of 100 presurgical candidates, ictal ESI could be performed in 31%, and in 14%, it proved to be a key element in the surgical decision-making process (Boon et al., 2002). Moreover, it influenced decision-making in none of the patients where ictal-EEG was completely concordant with MRI, in 50% of patients where ictal-EEG was neither completely concordant or was discordant with MRI, and in 83% of patients where ictal-EEG was discordant with MRI. Typically, the results of ictal ESI helped avoid iEEG recordings in patients who appeared to be unsuitable candidates for resection. ESI had high added value in the MRI-negative subgroup, where it correctly restricted sublobar localizations in 73% of patients (Rikir et al., 2014). Therefore, the use of ESI is a well-established method in PWE and is no longer considered an experimental technique. A recent survey of the European Epilepsy Surgery centers showed only 38% of these centers include this modality in the presurgical evaluation (Rikir et al., 2014), indicating a gap in the clinical utilization of ESI and the need for qualified training programs to address this adjunctive method in presurgical evaluation of PWE.

16. Intracranial EEG monitoring

iEEG is able to detect brain signals with an exceptionally high signal-to-noise ratio, and therefore, is less susceptible to artifact and has a high spatial and temporal resolution, resulting in a greater likelihood of earlier seizure detection compared with scalp-based EEG recording (Hill et al., 2012). iEEG methods of recording include depth electrodes, subdural grids and strips, foramen ovale electrodes, and epidural pegs used independently or in combination to clarify hemispheric lateralization and interlobar or intralobar localization. The purpose of iEEG is to differentiate regional, lobar, and multi-lobar involvement; determine the side of seizure onset (e.g., mesial temporal involvement); elucidate *dual* pathology by demonstrating more than one SOZ; and define deep seated or interhemispheric cortical sources when a lesion is not evident on brain MRI (Brna et al., 2015; Jayakar et al., 2016). iEEG also serves to resolve discordant noninvasive data and perform electrical cortical stimulation (ECS) studies that functionally map eloquent cortical function. iEEG is used infrequently to provide value for diagnostic information (e.g., bitemporal seizure onset during neuromodulation), prognosticate in epilepsy surgery, and provide treatment via thermocoagulation (Tonini et al., 2004; Jayakar et al., 2014, 2016). Favorable seizure outcomes are possible and frequently seen in the complex epilepsy population requiring iEEG studies. Mislocalization of the EZ and incomplete resection are frequently cited to explain early postoperative recurrences in PWE who fail resective epilepsy surgery (Bulacio et al., 2012). Failure to completely remove the SOZ is considered a risk factor for surgical failure usually discussed with the patient preoperatively (Rosenow and Luders, 2001; Cohen-Gadol et al., 2004; Jeha et al., 2007). Stereo-electroencephalography is a stereotactic surgical methodology that permits accurate three-dimensional placement of intracerebral recording electrodes for electroclinical recording ictal epileptiform activity and rarely for ECS (Gonzalez-Martinez, 2016). Other advantages include access to deep cortical structures, ability to localize the EZ when subdural grids have failed to do so, and utility in multilobar bihemispheric evaluations (Serletis et al., 2014). Flow charts and tables defining when iEEG techniques should be considered, and which technique is indicated relative to individual patient needs, are available (Tonini et al., 2004; Jayakar et al., 2014) in addition to a recent ILAE guideline for the use of intracerebral EEG (Jayakar et al., 2016). Since iEEG is used only as a technique to identify the SOZ as a surrogate marker for the EZ preoperatively in patients with drug-resistant focal seizures, localization is ultimately related to the surgical outcome. Several prospective studies have looked at short- and long-term surgical outcomes in patients undergoing iEEG (McGonigal et al., 2007; Asano et al., 2009; Bulacio et al., 2012). Outcomes using iEEG have been separated into MRI + (lesional) and MRI – (nonlesional) patients (McGonigal et al., 2007; Perucca et al., 2014). Much of the data represent category 3 evidence being retrospective reports, or reports that did not control for pathology (Bulacio et al., 2012; Perucca et al., 2014; Brna et al., 2015). One study (Bulacio et al., 2012) looked at short and very long-term outcomes in 414 patients evaluated with iEEG; 336 underwent surgery, and 61% were seizure free at one year, 47% at three years, 42% at five years, and 33% of patients were seizure-free at 10 years postoperatively. In a prospective report of MRI + versus MRI – patients (McGonigal et al., 2007), the authors found that there was no difference in iEEG ability to identify the EZ and no differences in outcomes at one and two year after surgery between the two groups. HFOs may appear as an interictal feature in addition to gamma, ripples, and fast ripples at seizure onset (Worrell et al., 2004). When present on iEEG recording at seizure onset, they have a positive predictive value in localizing the SOZ in neocortical-onset epilepsies with resection of HFOs predicting a favorable surgical outcome (Worrell et al., 2004; Wu et al., 2010; van 't Klooster et al., 2015). A large meta-analysis, composed mostly of category 3 and 4 studies, found that using iEEG actually had a negative predictive value for a long-term seizure-free outcome (Tonini et al., 2004). One very important concept and limitation of all iEEG studies is that they only allow the recording of a very small fraction of the brain. Therefore, a clear and focused hypothesis about the SOZ is needed to justify their use and anticipate localization (Jayakar et al., 2016). While the patient population with iEEG obtaining seizure-freedom after epilepsy surgery is very different from the patient population that does not require iEEG, the outcomes from both approaches are typically more favorable than continued use of ASDs alone when the patient has proven to be drug-resistant.

17. Electrocorticography

ECoG is used in the presurgical evaluation of patients with drug-resistant epilepsy. A major advantage of ECoG is that it avoids the discomfort, risks, and costs of staged implantation and extraoperative iEEG monitoring, and hence, the need for a second surgery (placing and removing the electrodes for surgery). The purpose of ECoG is to record EEG from sensors surgically placed directly on the surface of the brain or implanted into deep-seated areas of cortex. The goal is to identify preoperative IEDs in the operating room when ECoG is used or identify the ictal onset zone during investigations where chronic invasive electrodes are utilized during VEM. In addition to recording the EEG, it is use as an adjunct during functional brain mapping with ECS. Additionally, it has been used to aid in defining postsurgical prognosis with regard to postoperative seizure outcome. This has been based on recording persistent and de novo IEDs after resection of epileptogenic tissue. The use of ECoG to assess interictal spike frequency using subdural electrodes suffers from the sampling bias imposed by limitations on where the electrodes are placed. In addition, IEDs have a limited ability to identify the SOZ (Asano et al., 2009). While ECoG may be performed extraoperatively with chronic intracranial electrodes, it usually refers to intraoperative recording. Intraoperative ECoG has been used to tailor resection of epileptogenic tissue in epilepsy surgery (Cascino et al., 1995; Tran et al., 1995). However, the ability of post-resection ECoG containing residual IEDs to predict the effect of surgical outcome on postoperative seizure frequency, presents conflicting results (Wyllie et al., 1987). Few studies have

investigated surgical outcome of patients submitted to ECoG-tailored resection compared to those who underwent resections without ECoG, with mixed results that do not allow for a firm conclusion (Jooma et al., 1995; Rassi-Neto et al., 1999; San-juan et al., 2011; Fernandez and Loddenkemper, 2013; Qiu et al., 2014). A few studies (category 3 and 4) have shown an association between residual spikes and surgical outcome in temporal lobe surgery (McKhann et al., 2000; Sugano et al., 2007), suggesting the use of ECoG for tailoring resections (McKhann et al., 2000). Overall, in TLE surgery, there is not enough evidence to support the routine use of intraoperative ECoG to help guide the extent of neocortical resection during temporal lobectomy, as residual spikes after resection do not seem to correlate with surgical outcome (Tuunainen et al., 1994; Cascino et al., 1995; Tran et al., 1995; Kanazawa et al., 1996; San-juan et al., 2011). In addition to focal spiking with or without propagation, focal slowing in the theta or delta range and ictal-appearing ECoG patterns occurred in 40% of the patients with TLE in one study (Stefan et al., 2008). When the anterior mesial temporal resection included the interictal spikes, ictal-appearing activity, and focal slowing, the postoperative outcome was good (Stefan et al., 2008). In children, a large retrospective series who underwent TLE surgery investigated with intraoperative ECoG the results did not correlate with a more favorable surgical outcome (Benifla et al., 2006). The role of intraoperative ECoG in extratemporal epilepsy surgery may be greater as resections in these cases are less standardized. However, in studies including both patients with temporal and extratemporal lesions, results have also been conflicting. In a series of 74 patients with drug-resistant epilepsy undergoing surgical treatment, including 34 with focal cortical dysplasia (FCD), different ECoG patterns were observed during surgery, and some studies found an association between residual spikes indicated a worse surgical outcome (Palmini et al., 1995). The same association was found by subsequent investigators (Wennberg et al., 1998a; Tripathi et al., 2010; Greiner et al., 2016), but others have failed to confirm these findings Histopathology has been correlated with the degree of epileptogenicity using ECoG spiking. One study found increased neocortical spiking after selective amygdalohippocampectomy (Wennberg et al., 1998b). A relationship was seen between the type of ECoG abnormal feature detailed by continuous spiking, epileptiform bursts, and recruiting discharges in patients with FCD and glioneuronal tumors. Continuous spiking was seen significantly more often in patients with FCD (Ferrier et al., 2006). In addition, a greater degree of abnormality was present in higher grade FCD with a greater degree of cellular disarray when compared to FCD with lower histopathological grades (Ferrier et al., 2006). Other investigators assessed the clinical utility of ECoG during callosotomy for treatment of drug-resistant seizures in patients with LGS, and failed to confirm a useful association (Kwan et al., 2005). More recently, the presence of residual HFOs (ripples and fast ripples) in the post-resection ECoG has been associated with worse surgical outcome in both children and adults (Wu et al., 2010; van 't Klooster et al., 2015).

18. Functional mapping

Electrocortical stimulation is performed in waking patients to identify functional areas of cerebral cortex. Stimulation can be performed using intracranial electrodes during VEM or by a surgeon using a hand-held stimulator in the operating room. Electrical stimulation of the brain is used to elicit a functional map of eloquent cortex to outline safe boundaries for resective surgery. The procedure may be performed intraoperatively or outside the operating suite and is usually performed through subdural electrodes imbedded in silastic grids or strips. Penfield and Jasper delineated the motor and sensory homunculus using ECS in awakened patients during the initial years when epilepsy surgery was being performed (Penfield and Jasper, 1954). Combining ECoG with ECS is useful for several reasons; it reflects the state of wakefulness by delineating the ECoG background activity, similarly assesses the effects of anesthesia, validates the integrity of ECS by monitoring stimulation-induced artifact, and provides recording for the presence of afterdischarges that may be incurred due to excessive stimulation. Identifying afterdischarges on ECoG during ECS is important to ensure electrographic seizures do not go unnoticed or become provoked by repetitive stimulation. Intraoperative ECS requires the patient to be cooperative and fully alert (Luders et al., 1991). Physiological and morphological complexity from ECS produces heterogeneity of behavioral effects, which range from evoking excitatory (e.g., motor movement) to inhibitory responses (e.g., speech arrest). These seemingly opposite effects sometimes even occur when ECS is applied at the same cortical site (Borchers et al., 2011). During the ECS procedure, a preset brief electrical stimulus is applied through an implanted electrode or hand-held wand for several seconds using a train of biphasic pulses. Incremental current intensities are applied to the selected site until a clinical response is identified, an afterdischarge occurs on the ECoG, or a predetermined machine-specific maximal intensity of current is reached. An afterdischarge appears as repetitive burst or run of spikes or as a brief sustained rhythmic discharge generally unassociated with clinical signs or symptoms. Functional cortical maps include sensory, motor, and language areas and should also include electrode sites where no function is elicited and no afterdischarge occurs (noneloquent cortex). Afterdischarge threshold maps have more limited usefulness as do reproduction of an aura or seizures during ECS. Additionally, findings from ECS are not an absolute guide to the end result after surgical resection, and variability in outcomes have been identified postoperatively despite the presence or absence of presumed functional tissue (Seeck et al., 2006).

19. Continuous EEG monitoring for seizures in critical illness

There is category 2 evidence that cEEG yields diagnostic information valuable for identifying clinical involvement from seizures. These data likely provide clinically meaningful information in detecting nonconvulsive seizures in critically ill patients. One prospective study assessed the incidence of NCSE after the control of convulsive status epilepticus and found 48% of patients had ongoing electrographic seizures, and 14% had NCSE (DeLorenzo et al., 1998). Seizures and periodic discharges have been found to occur more often with sepsis than other conditions in the medical ICU (Oddo et al., 2009). In the surgical ICU, nonconvulsive seizures were found in 16% and NCSE in 5% of patients. In the largest category 2 investigation of electrographic seizures in acutely ill adults, seizures were detected in 19% of 570 patients (Claassen et al., 2004); 56% with seizures had their first event within one hour of initiating cEEG monitoring, and this percentage increased to 82%, 88%, and 93% within 12, 24, and 48 h, respectively. Another large retrospective category 2 study (n = 625) in acutely ill adults found a seizure incidence rate of 27% during cEEG monitoring, with seizures more likely to occur early (<30 min) during monitoring (Westover et al., 2015). Other large cohort studies of neurocritical care patients admitted with altered mental status found a similar number of patients with nonconvulsive seizures (Young and Campbell, 2005). Thus, prospective and large retrospective studies (category 1 and 2) indicate that cEEG monitoring has a clinically meaningful and valuable yield in detecting nonconvulsive seizures, especially in critically ill patients.

In essentially all patients, NCSE can be diagnosed when monitored by cEEG (Swisher et al., 2015). One large retrospective study (n = 537) of patients with suspected status epilepticus compared the rates of diagnosis before and after the introduction of a cEEG monitoring protocol and found a significant increase in diagnostic yield with the use of cEEG (Sutter et al., 2011). NCSE is a challenging neurological problem with the diagnosis solely supported by EEG and occurring in about 20% of critically ill patients. cEEG monitoring has become the gold standard for identification of seizures in hospitalized patients as seizures in this population are frequently nonconvulsive (Brophy et al., 2012). Newer definitions and classifications of NCSE incorporate the EEG to reflect its diagnostic accuracy (Trinka et al., 2015; Leitinger et al., 2016). Newer terminology for describing EEG patterns (Hirsch et al., 2013; Beniczky et al., 2013b) helps provide unambiguous instruction and a significant increase in specificity for clinical and research use of cEEG monitoring. The modified Salzburg criteria for NCSE include the presence of more than 25 epileptiform discharges/10-s epoch or more than 2.5 discharges/s. To meet the criteria for NCSE, when periodic discharges are less than or equal to 2.5/s (Fig. 4b) or rhythmic delta/theta activity exceeds 0.5/s, the presence of additional criteria must be met to confirm the diagnosis of NCSE. In the latter situation, at least one of the additional criteria must also exist; clinical and EEG improvement due to ASD administration, subtle ictal clinical features are also present, or typical spatiotemporal evolution of the epileptiform activity must occur. When refined to include the ACNS terminology, this definition resulted in clinically relevant and statistically significant reduction of false positive diagnoses of NCSE with minimal loss in sensitivity (Leitinger et al., 2015). During the event, fluctuation of the ongoing activity was present without evolution or when EEG improvement without clinical improvement occurred following administration of ASDs, they recommended a diagnosis of possible NCSE.

The use of cEEG is a helpful adjunct to diagnosis of epileptiform activity, including nonconvulsive seizures in various acute neurological conditions, such as traumatic brain injury (Vespa et al., 1999; Ronne-Engstrom and Winkler, 2006; Amantini et al., 2009), subarachnoid hemorrhage (Claassen et al., 2003; Claassen et al., 2005), intracranial hemorrhage (Vespa et al., 2003; Claassen et al., 2007), hypoxia following cardiac arrest (Rossetti et al., 2007), monitoring treatment with therapeutic hypothermia (Legriel et al., 2009; Rittenberger et al., 2012; Knight et al., 2013), and central nervous system infection (Claassen et al., 2004; Carrera et al., 2008). Every hour of seizure burden may increase the odds of disability or death at three months in patients with subarachnoid hemorrhage (De Marchis et al., 2016), though outcome information following cEEG has been limited. For diagnosis or quantification, cEEG review of the entire raw EEG record or facilitated by trending observed on QEEG analysis is performed to locate and count seizure events and monitor the effects of antiseizure treatment. Two category 2 studies evaluated the diagnostic yield of QEEG and amplitude-integrated EEG in detecting seizures. One study assessed the sensitivity of QEEG versus visual inspection of raw EEG showing a mean sensitivity for seizure identification ranging from 51% to 67% (Haider et al., 2016). Using anesthetic drugs, titration to reach EEG background suppression was associated with a lower risk of breakthrough seizures than titration to seizure suppression. In comatose survivors of cardiac arrest, cEEG helped to detect epileptiform activity and predict prognosis (Alvarez et al., 2013). The literature is confounded by the use of different classification systems to identify degrees of encephalopathy, with variable intervals of time until the EEG was recorded after cardiopulmonary resuscitation.

Other patterns on EEG may assist with prognosis; however, there is a notable lack of outcome validation after detection of seizures in the critically ill patient using cEEG. In a small prospective study of 34 patients, standard EEG with background reactivity evaluation performed at two separate time points, during therapeutic hypothermia and normothermia, seemed to be as efficient as cEEG in the setting of coma after cardiac arrest (Alvarez et al., 2013). Others have found features present on cEEG capable of predicting outcome (Crepeau et al., 2013). Sleep was found to improve prognosis in a category 3 study performed to predict the rehabilitation outcome after severe traumatic brain injury (Sandsmark et al., 2016). In another study (category 2) of poor grade subarachnoid hemorrhage, cEEG provided independent prognostic information with unfavorable outcome when any form of periodic discharges, electrographic status epilepticus, absence of reactivity, or absence of sleep architecture was encountered (Claassen et al., 2006). Using cEEG, a category 1 study of electrographic seizures after traumatic brain injury resulted in a delayed, prolonged increase in intracranial pressure and metabolic crisis that affected outcome (Vespa et al., 2007), and provides information about the potential for seizures to affect morbidity.

20. Conclusions and final remarks

For most patients, the diagnosis of epilepsy is based upon a thorough history and physical examination. However, description of the semiology provided by witnesses of a seizure (including family members) can be misleading (Deacon et al., 2003). A standard scalp EEG is the most useful test when evaluating possible epilepsy though in developing countries EEG services may be limited (Meinardi et al., 2001). In patients with a first, unprovoked seizure, there is strong evidence that an EEG with unequivocal IEDs helps establish a diagnosis of epilepsy. Standard EEG recordings should be of at least 20 min duration, with activation methods to increase the diagnostic yield. When standard EEG recordings are unrevealing, additional methods of EEG recordings can enhance its usefulness. Obtaining a sleep EEG is recommended to enhance the possibility of capturing an abnormality. Long-term VEM is able to document the relationship between the paroxysmal semiology and the EEG and synchronize signals from multiple generators including EEG, ECG, and EMG. VEM is especially useful for clarifying the differential diagnosis of patients with spells, classifying seizure types, quantifying seizure frequency, and characterizing the electroclinical manifestations in PWE during a presurgical evaluation. More sophisticated methods, such as source localization in

Table 4
Summary statements.

The presence of IEDs in a standard EEG predicts a high risk of recurrence following a first seizure
The presence of IEDs in a standard EEG in patients with controlled epilepsy may predict a higher risk of seizure relapse following ASD taper
EEG helps classify seizure type (focal or generalized) when IEDs are encountered in the recording
Video-EEG monitoring can provide a definitive diagnosis in most PWE when seizures are recorded
Video-EEG monitoring is useful in an epilepsy surgery evaluation
Continuous EEG monitoring is a useful adjunct to diagnosing and quantifying seizures, especially in critically ill patients

Abbreviations: ASD, antiseizure drugs; EEG, electroencephalogram; IEDs, interictal epileptiform discharges.

the extratemporal epilepsies may influence surgical decision-making and have added value in the evaluation process of patients with drug-resistant focal epilepsy. In critically ill patients, cEEG monitoring is an indispensable means for diagnosing electrographic seizures and identifying NCSE.

Recommendations based upon high-level evidence are summarized in Table 4. The approach to the use of EEG in diagnosis and monitoring of PWE is provided by the algorithm in Fig. 5. With advances in aEEG monitoring, the use of telemedicine (Bingham, 2002), computer-assisted telephone interviews (D'Souza et al., 2010), and home video review (Chen et al., 2008) are new approaches to diagnosis and management of PWE and epilepsy syndromes that may strengthen the routine approach to clinical diagnosis in developed and underdeveloped countries. Similarly, reporting is likely to become automated to enhance reproducibility of EEG findings, and in turn improving patient management and clinical research (Beniczky et al., 2013a; Kaplan and Benbadis, 2013; Tatum et al., 2016). Technical barriers are being breached as computing power and engineering techniques advance, allowing recordings from larger numbers of electrodes, smaller contacts, and recording frequencies beyond those routinely used in standard scalp EEG for the past 80 years (Bragin et al., 2002).

Conflict of interest

Dr. Donald Schomer is the editor of the Niedermeyer's Electroencephalography textbook.

Dr. David Gloss is an evidence-based medicine consultant to the AAN. The other authors do not have any significant conflict of interest.

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Acknowledgements

We wish to recognize the many fine citations and collaborators not included in this guideline that contributed substantially to our understanding of the yield and utility of EEG in patients with epilepsy. Thanks to Alison Dowdell at Mayo Clinic for editing this manuscript.

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