Electroencephalography in epilepsy surgery planning

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Abstract

Objective To describe localizing value of surface EEG recording in the presurgical evaluation of medically intractable pediatric epilepsy patients.

Methods We review the relevant concepts required for understanding the role of surface EEG in the presurgical evaluation and in identifying the epileptogenic zone. The unique features of EEG and its limitations are discussed.

Conclusion Despite recent technological advancements, surface EEG continues to play a crucial role in defining the epileptogenic zone, and thus in presurgical planning.

Keywords Epilepsy · Surgery · Electroencephalography (EEG) · Localization · Epileptogenic zone · Pediatric

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Introduction

Epilepsy surgery continues to evolve as a valuable therapeutic option for the approximately 7–15% of pediatric epilepsy patients who are medically refractory [5, 7]. To maximize favorable surgical outcomes, we must strive to improve the selection process of surgical candidates, as well as to perfect the use of available traditional and emerging diagnostic studies. In most cases, clinical experience has shown that surface video-EEG recording of habitual seizures yields the most valuable data for determining surgical candidacy and initial surgical planning. This is in part due to the unique ability of video-EEG to characterize the ictal and interictal states clinically and electrographically. Video-EEG allows for the characterization of seizures, their frequency, and ictal semiology, in addition to a preliminary description of a potential epileptogenic focus. In combination with relevant clinical data, seizure semiology, neuroimaging, and other neurophysiologic studies, the video-EEG recording of seizures, thus, remains a critical component of the Phase I presurgical evaluation. In this study, we provide an overview of the relevant concepts and basic principles of surface EEG recording in the presurgical evaluation of epilepsy surgery.

The epileptogenic and other cortical zones

The role of surface EEG in surgical planning is to anatomically define the *epileptogenic zone*. This theoretical concept describes the region of cortex responsible for generating seizures. Thus, resection or disconnection of the epileptogenic zone will result in seizure freedom. The precision with which the epileptogenic zone can be identified is limited with existing technology. As well, preserving

Fig. 1 Relative attenuation of the posterior dominant rhythm in the right occipital region (*O2*) compared to the left occipital region (*O1*) in a 5-year-old girl with extensive right hemisphere cortical dysplasia and medically refractory seizures

eloquent cortical areas often limits the extent of a resection. Thus, only partial resection of the epileptogenic zone may be feasible at times. Whether this is sufficient to provide seizure freedom depends upon the location of the other cortical zones, on whether the known ictal generator has been resected, and whether there exist other potential generators of ictal activity within the remaining epileptogenic zone.

Clinically, we may define several cortical zones, which serve as markers for the epileptogenic zone. The location of each of these zones may be defined by various diagnostic techniques. When these zones overlap to describe the same cortical area, they localize the epileptogenic zone with increased confidence. In this study, we consider the ictal onset zone, the irritative zone, the symptomatogenic zone, and anatomic lesions.

The *ictal onset zone* defines the cortical region generating ictal onset. This does not define the additional regions of the epileptogenic zone necessary for seizure propagation and spread, nor other potential ictal generators within the epileptogenic zone. Surface EEG gives an approximate localization, with more precise definition possible via intracranial EEG recording. Though limited by the lag between seizure onset and tracer transport to the ictal focus, a prompt ictal single photon emission computed tomography also aims to define the ictal onset zone as well. The ictal onset zone is often believed to be smaller than the epileptogenic zone. In this case, resection of the entire epileptogenic zone must occur so that remaining potential ictal generators, or areas with potential epileptogenicity, are not left behind.

The *irritative zone* refers to the cortical region generating interictal epileptiform discharges. The irritative zone further defines the extent of the epileptogenic zone. This zone may be defined by surface EEG, or more precisely by intracranial EEG. Magnetoencephalography (MEG) and functional magnetic resonance imaging (fMRI) may also define this zone of interictal activity in deeper cortical or subcortical regions. These studies, however, sample a shorter time period than long-term surface video-EEG.

The *symptomatogenic zone* refers to the region of cortex that generates the clinical semiology of habitual clinical seizures when activated. This area may frequently be different than the epileptogenic zone; however, as ictal onset may occur in noneloquent or functionally "silent" regions of cortex. A seizure becomes clinically apparent only after spread has occurred to the symptomatogenic zone. Reviewing seizure semiology, by history or video-EEG capture, may localize the symptomatogenic zone. There is significant variability however in the localizing value of different ictal semiologies.

An anatomic lesion identified by MRI often attracts suspicion as a potential epileptogenic focus. However, a lesion may not necessarily be epileptogenic. Furthermore, the epileptogenic zone may include only part of a lesion, or extend to cortex beyond the anatomic boundaries of a given lesion. Thus, surface video-EEG recording is still necessary to support the epileptogenicity of a given lesion, as well as to define the epileptogenic zone boundaries that may not be limited to the lesion itself.

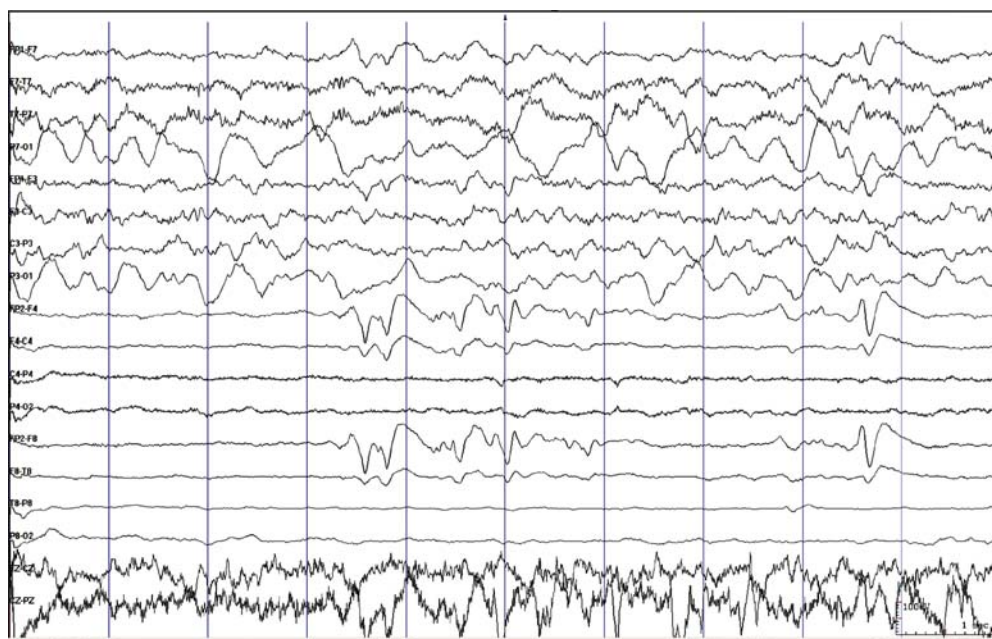## Methods to maximize the yield of surface EEG recording

The potential information which can be gathered from surface video-EEG recording can be maximized in several

Fig. 2 Four-month-old infant with right hemimegalencephaly and very frequent right-sided epileptiform discharges and seizures. Sleep spindles present over left hemisphere and entirely absent over right hemisphere
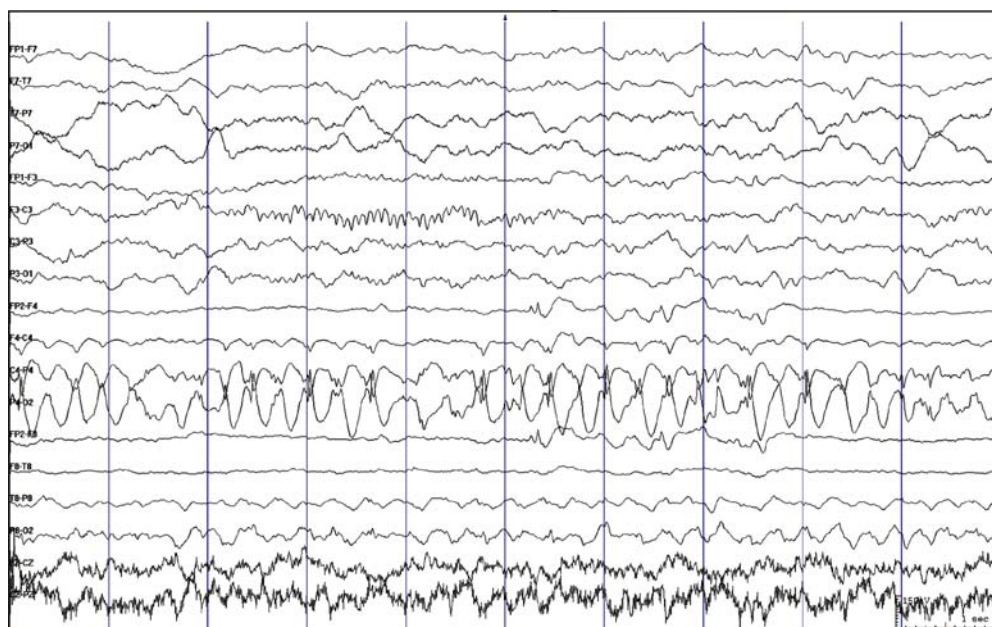
ways. Regarding basic monitoring techniques, the use of collodion rather than paste improves the quality of recordings lasting several days. For more precise localization, extra electrodes should be utilized when indicated by relevant clinical and electrographic data. In addition to the standard 10–20 international system electrodes, our institution routinely utilizes inferior anterior temporal electrodes (FT9, FT10) for presurgical evaluation. Supraorbital electrodes may aid in detecting orbital–frontal activity. Additional electrodes may be added for clarification of activity or lateralization. As well, chest EKG and extraorbital electrodes aid in the characterization of ictal behaviors, and also aid in identifying artifact.

Activating procedures, including hyperventilation and photic stimulation, are useful in some cases. Sleep deprivation and medication reductions often increase the probability of recording interictal and ictal epileptiform activity. Medication reduction, however, may change the electrographic and clinical features of habitual seizures.

Digital EEG recordings allow for enhanced clarification of EEG phenomena by allowing the reformatting of data in different montages, and minimizing artifacts. As well, the ability to easily change filters, paper speed, gain, and review video simultaneously allows for clarification of electrographic activity, when done appropriately by a capable electroencephalographer.

Fig. 3 Four-month-old infant with right hemimegalencephaly. Right hemisphere voltage attenuation seen

Fig. 4 Left hemisphere hypsar-
rhythmia in a 14-month-old boy
with a history of premature birth
at 23 weeks with left-sided
severe intraventricular hemor-
rhage, and infantile spasms
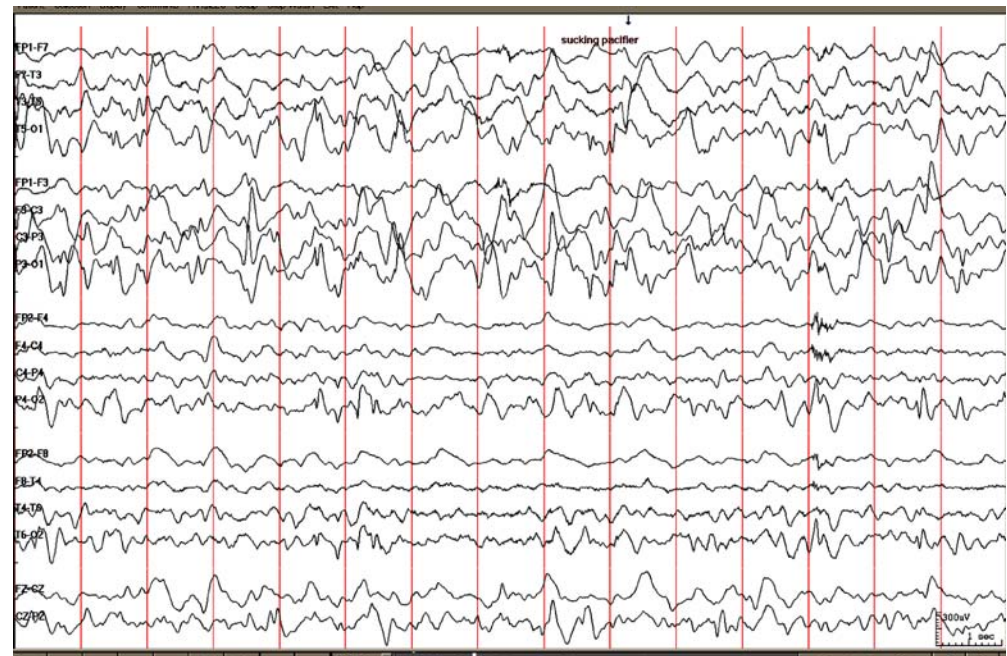
Interictal EEG patterns

In identifying useful data from interictal recording, several concepts serve useful. Distortions of normal EEG rhythms may aid in localizing or lateralizing abnormalities. This may include asymmetry or poor architecture of the posterior dominant alpha rhythm or mu rhythms in waking or asymmetric spindles or vertex waves in sleep. (Figs. 1 and 2) EEG reactivity may also be impaired. Likewise, normal EEG recording may be consistent with a focus in deeper cortical regions, if suspected [4].

Nonepileptiform abnormalities, which may help localize the epileptogenic zone, include slowing and suppression of normal activity (Fig. 3). Focal slowing indicates dysfunction in a particular region, which may support the case for identifying the epileptogenic zone [3]. Persistent slowing supports an underlying structural abnormality. Rhythmic temporal theta activity supports a temporal focus. As well, focal background attenuation is suggestive of an epileptogenic focus [20].

Epileptiform abnormalities such as spikes, sharp waves, and spike-wave complexes during the interictal record are supportive of epilepsy, and help to localize the irritative zone. Their appearance is dependent upon the epileptogenic zone being on the surface, rather than inferior or mesial. Given the presence of interictal spikes, however, these serve useful in localizing or lateralizing the epileptogenic zone. Persistent focal interictal spikes and frequent rhythmic spikes are useful in localizing the epileptogenic zone (Fig. 4). Multifocal or generalized interictal abnormalities are less helpful in localization and require additional data for the determination of the epileptogenic zone.

Ictal EEG patterns

To more confidently describe a patient's habitual seizures, recording at least three and preferably five seizures has been advocated. The exact number required is unclear, and appears to vary dependent upon location and seizure type [2, 25]. The first habitual seizure recorded appears predictive of the epileptogenic zone [28]. Our practice is to capture at least three habitual seizures [23]. Once recorded, seizures may then attempt to be lateralized/localized. If they cannot, then recording results may prove inconclusive and surgical candidacy thus less likely. Surface ictal EEG recording has been shown to be accurate and reliable in lateralizing seizures [29]. Electrographic onset before clinical onset of a seizure increases the confidence of localizing the epileptogenic zone before spread to the symptomatic zone. When EEG findings are seen after clinical onset, then a mesial or inferior focus not initially seen on the surface may be present.

Focal rhythmic bursts of higher frequency discharges with evolution to increasing amplitude and slowing is the most commonly observed pattern in focal seizure onset [12, 19]. Faster frequencies seen at ictal onset may indicate closer proximity to the ictal focus. A focal electrodecrement pattern with a reduction in interictal activity may support a deeper region of ictal onset [1, 17, 22]. Consistent focal repetitive spikes or rhythmic slowing at onset also aids in localization and lateralization.

Specific ictal onset patterns have been seen to occur more frequently within particular regions of cortex (e.g., temporal vs extratemporal); however, these patterns alone cannot implicate origin from a specific region. The

temporal lobe ictal patterns appear to be more consistent, however, than extratemporal patterns [12]. These include rhythmic theta activity in temporal seizures, repetitive epileptiform activity in lateral frontal seizures, suppression or paroxysmal fast activity in mesial frontal seizures, and bilateral changes and tendency towards false localization or lateralization with parietal and occipital lobe seizures [6, 9, 17, 31]. Postictal focal slowing aids in lateralizing the epileptogenic zone, though is of limited localizing value within a hemisphere [15, 29].

Limitations of surface EEG recording

Though surface EEG remains fundamental in the presurgical evaluation, it has several limitations in its ability to define the epileptogenic zone. Higher than lower frequency cerebral activity is attenuated by the intervening structures between scalp and cortex, thus, limiting the sensitivity of surface EEG recording. As well, the sensitivity of detecting an epileptic discharge by surface EEG is dependent upon the depth, size, orientation, and duration of a discharge [13]. Deeper cortical and subcortical seizure onset and propagation may not have a surface EEG correlate.

Small areas of seizure activity, even if on the surface, may not have a surface correlate until larger regions of cortex are involved. It has been estimated that ictal activity must spread to approximately 6 cm^2 of cortex to be detected by surface EEG, thus, limiting definition of the precise ictal onset zone from the surface [8].

Poor detection of oscillations in brain activity represents another limitation of scalp EEG. This limitation has been analyzed in the context of cognitive research that uses EEG to examine event related oscillations—brain activities that correspond to specific functional tasks. For example, cognitive researchers have found that scalp EEG does a poor job in localizing both gamma and theta oscillations [14, 21]. Such deficient oscillatory detection largely occurs because scalp electrodes can only record oscillations that are synchronized, have high amplitudes, and are spread out over large areas of the cortex [14], and only a fraction of the brain's oscillatory activity exhibits these stringent characteristics. Furthermore, oscillations originating in structures deep inside the brain cannot be detected easily with scalp EEG [14, 24]. Because epilepsy and seizures are both rhythmic events exhibiting oscillations across all frequency bands [9, 32], the inability of scalp electrodes to capture oscillations represents a significant flaw in the use of scalp EEG to localize epileptogenic areas.

Another limitation of scalp EEG is that the optimal number and spacing of surface electrodes is mathematically uncertain [30]. The spacing of surface electrodes is referred to as the spatial sampling frequency. Mathematically, the Nyquist theorem is used to determine the requirements for the sampling process of EEG signals that ensure that a sampled signal will allow for adequate reconstruction of the original analog signal. In the spatial domain, this theorem generally states that the interelectrode distance must be small enough to prevent the highest spherical harmonics of the EEG signal from aliasing back onto the lower harmonics. The naturally occurring low pass filter of the skull fortunately eradicates insignificant high level harmonics; thus, ensuring that a finite number of electrodes can reproduce a nondistorted signal. Yet, the Nyquist theorem does not provide a way to determine exactly how many electrodes are necessary to fully prevent aliasing.

Empirical studies concerning the optimal number and spacing of electrodes have varied. For a complete covering of the scalp, Srinivasan et al. [27] have suggested that 19 and 32 electrode channel maps represent a gross undersampling of EEG activity. Srinivasan et al. and Lantz et al. [16] have instead suggested that 128 channel recordings often improve topographical mapping significantly compared to a 64 channel electrode scheme. Lantz et al. also concluded that the increase of electrodes from 31 to 63 showed the sharpest increase in topographical mapping. Furthermore, Spitzer et al. [26] recommended a 3-cm minimum interelectrode distance. Freeman et al. [10] reasoned later that an intersensor distance of 5–8 mm was necessary when utilizing EEG to ascertain "nonlocal information in the EEG that is cognitively related" [10]. However, Freeman also concluded that such a tight packing of electrodes does not significantly help the localization of "spatial sources for cortical signals, such as epileptic foci or components of event related potentials" [10].

A low signal to noise ratio represents another possible limitation of scalp EEG. The noise in an EEG signal can originate from biological sources (artifacts from muscular changes, background EEG, eye movements, etc.) or electrical sources (60 or 50 Hz line noise, electrode motion, etc.). Possible solutions to correct electrical noise are notch filters, bipolar montaging, adaptive filtering, and independent component analysis (ICA) [11]. In general, notch filters are most commonly implemented; both adaptive filtering and ICA algorithms tend to be too computationally burdensome. Biological noise and artifacts are most easily recognized and removed by the electroencephalographer-automated detection of such signals is also computationally complex.

Future directions

The basis of surface EEG recording has remained essentially unchanged since first described, and newer technologies have yet to prove more valuable in the presurgical evaluation. More importantly, improving old and further

exploring new technologies will increase the sensitivity and specificity with which the epileptogenic zone may be described, thus improving surgical outcomes.

MEG is an increasingly utilized technology, able to map eloquent cortical areas of interest, such as language and somatosensory function, as well as to help define the irritative zone. MEG has the additional capability to detect activity from deeper cortical and subcortical structures not otherwise evident on surface EEG. However, the limited study time, compared to long-term video-EEG, may not describe the extent of the irritative zone as well as surface EEG. MEG cannot be performed in very young children, and seizures cannot be routinely captured, thus, limiting its ability to define the ictal onset zone. Nonetheless, MEG appears to be promising, and in at least one series appears to be as efficacious as surface EEG recording [18].

Conclusion

Surface EEG continues to prove indispensable in the presurgical planning of medically refractory epilepsy patients. This is largely due to its unique capability of describing seizure onset and evolution over the time. At present, newer technologies have yet to prove as effective as surface EEG in defining the epileptogenic zone. Through continued improvement of EEG techniques and newer technologies, and understanding the strengths of each, we strive to increase the power of localization by using these new methods in a complementary fashion.

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