Ictal SPECT

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Summary: The localizing value of ictal single-photon emission computed tomography (SPECT) performed with cerebral blood flow agents in patients with epilepsy is based on cerebral metabolic and perfusion coupling. Ictal hyperperfusion is used to localize the epileptogenic zone noninvasively, and is particularly useful in magnetic resonance (MR)-negative partial epilepsy and focal cortical dysplasias. Subtraction ictal SPECT coregistered with MRI (SISCOM) improves the localization of the area of hyperperfusion. Ictal SPECT should always be interpreted in the context of a full presurgical evaluation. Early ictal SPECT injections minimize the problem of seizure propagation and of nonlocalization due to an early switch from ictal hyperperfusion to postictal hypoperfusion during brief extratemporal seizures. The degree of thresholding of SISCOM images affects the sensitivity and specificity of ictal SPECT. Ictal hypoperfusion may reflect ictal inhibition or deactivation. Postictal and interictal SPECT studies are less useful to localize the ictal-onset zone. Statistical parametric mapping analysis of groups of selected ictal–interictal difference images has the potential to demonstrate the evolution of cortical, subcortical, and cerebellar perfusion changes during a particular seizure type, to study seizure-gating mechanisms, and to provide new insights into the pathophysiology of seizures. Key Words: Ictal SPECT—SISCOM—Presurgical evaluation—SPM—Epilepsy—Seizures.

Ictal single-photon emission computed tomography (SPECT) has the potential to localize the ictal-onset zone accurately in a noninvasive manner. Reliably to deliver early ictal SPECT injections, detailed attention should be paid to the logistics of ictal SPECT setup. Ictal SPECT injections should be performed in the video-EEG suite, with the nursing and review station close to the rooms of the patients. Medical personnel should be educated in handling of radioactive materials and be familiar with the electroclinical features of epileptic seizures. The brain-perfusion agent should be available in the room, and the injection system should allow fast ictal injections (1,2). High-resolution SPECT and magnetic resonance imaging (MRI) scanner should be available. Excellent cooperation between the neurology and nuclear medicine department is of crucial importance (3). If the implementation of ictal SPECT is too difficult, referral of selected patients for ictal SPECT should be considered.

Brain-perfusion tracers are lipophilic substances that cross the blood–brain barrier, have a long retention time in the brain, and are $^{99m}$technetium ($^{99m}$Tc)-labeled agents. Two commonly used tracers are $^{99m}$Tc-hexamethylene propylene amine ($^{99m}$Tc-HMPAO) (Ceretec) and $^{99m}$Tc-ethyl cysteinate dimer ($^{99m}$Tc-ECD) (Neurolite). The first-pass extraction for $^{99m}$Tc-ECD is $\sim$60%, and for $^{99m}$Tc-HMPAO, $\sim$85%. $^{99m}$Tc-ECD is retained in the brain after an enzymatic conversion to ionized acid compounds and $^{99m}$Tc-HMPAO after conversion to a nondiffusible hydrophilic compound after cell uptake. These different mechanisms of brain retention could explain the differences in cerebral distribution of the two tracers (4). $^{99m}$Tc-ECD is stable 6 to 8 h, and the stabilized form of $^{99m}$Tc-HMPAO, for 4 h. $^{99m}$Tc-ECD is cleared from the body more rapidly than $^{99m}$Tc-HMPAO, and gives a higher brain-to–soft tissue activity ratio, which improves image quality (5). Lee and colleagues (6) found $^{99m}$Tc-HMPAO ictal SPECT superior to $^{99m}$Tc-ECD ictal SPECT in localizing the epileptogenic zone. In their study, however, the number of patients treated with $^{99m}$Tc-ECD was rather small, and the duration of the injected seizures was not given, which could potentially have influenced the results. Further studies will be required to settle the issue. Because early ictal injections give the best results, it is advisable to use a stabilized compound and to inject via an indwelling intravenous cannula in the arm that is involved less in the seizure. Smith and colleagues (7) reported ictal SPECT injections in 77% of 110 consecutive patients, with a medium injection time of 27 s. In 160 consecutive patients admitted to our video-EEG suite, we were able to inject 80% of patients, with a median injection time of 19 s, by using an ical SPECT setup with indwelling intravenous cannula (2). In view of the decay of the tracer, the injected dose should be adjusted. Alternatively, if the full
dose is given, the scanning time should be adjusted. In 20% of our patients, ictal SPECT injection was not performed because of seizures that occurred outside the hours of ictal SPECT (59%), because the patient did not have seizures (23%), or because the seizure was not noticed by the medical personnel (18%). A solution for these problems could be an on-call service for selected patients to allow ictal injections during the night, and a seizure-waring system.

Both interictal and ictal SPECT images should be obtained and coregistered. To adjust for differences in administered dose during interictal and ictal studies, the interictal and ictal SPECT scans are normalized. Difference images are obtained by subtraction of the interictal from the ictal SPECT. The difference images are thresholded, usually at 2 standard deviations (SDs), to highlight regions of hyperperfusion. Subtraction ictal SPECT coregistered to MRI (SISCOM) has improved localization and visualization of the region of hyperperfusion (8). O’Brien and colleagues (9) reported localization in 39% by using side-by-side visual inspection versus 88% localization by using SISCOM (Fig. 1).

Statistical parametric mapping (SPM) between interictal and ictal SPECT scans has been reported to give results comparable with difference imaging (10). With SPM, the ictal SPECT scan can be compared with a normal brain SPECT database, without the need for an interictal SPECT scan (11).

The interpretation of ictal SPECT images should always be done in the context of a full presurgical evaluation. The neurologist/epileptologist has, therefore, an important role in the interpretation of the SPECT images. The injection time should be known, because early injections give the best results. The area of highest ictal hyperperfusion is usually the ictal-onset zone, unless the seizure has propagated. Several propagation patterns have been described. Propagation is often from posterior brain regions (parietooccipital lobes) to anterior brain regions (temporal and frontal lobe) (12). Noachtar and colleagues (13) reported propagation in 85% of parietooccipital epilepsy (Fig. 2). Another propagation pattern is from the temporal to the frontal lobe. In patients with a temporal lobe lesion on MRI and discordant frontal lobe seizures, ictal SPECT may show propagation from temporal to frontal lobe, obviating the need for invasive monitoring. Propagation from one temporal lobe to the contralateral temporal lobe has been reported in ~1% of cases (14, 15). Propagation of ictal activity can partly explain why a high SISCOM threshold has a lower sensitivity and higher specificity compared with a low threshold, which has a higher sensitivity but lower specificity (16) (Fig. 3). In clinical practice, using different SISCOM thresholds can help elucidate propagation patterns (see Fig. 2). The injected seizure type and ictal semiology should be known for a correct interpretation of ictal SPECT. In our hands, ictal SPECT during simple partial seizures gave no information in ~40% of cases. For this reason, we have limited the use of self-injection ictal SPECT, because this was often during isolated simple partial seizures (15). Complex partial seizures (CPSs) give the best results, and secondarily generalized seizures may give multiple regions of hyperperfusion (17). The duration of the injected seizure is important in the interpretation of ictal SPECT studies. After injection in an arm vein, the tracer takes ~30 s to reach the brain. The postictal switch (i.e., switch from ictal hyperperfusion to postictal hypoperfusion) occurs ~1–2 min postictally in temporal lobe.

**FIG. 1.** Improved localization of the area of ictal hyperperfusion with SISCOM. The area of ictal hyperperfusion was missed on side-by-side visual analysis of ictal (A) and interictal (B) SPECT images. Subtraction of interictal from ictal SPECT (threshold, +2 SD) clearly showed the area of ictal hyperperfusion (C). SISCOM allowed an accurate anatomic localization of the ictal hyperperfusion in the brain (D).
seizures (18), but is shorter in extratemporal seizures. It has been estimated that extratemporal seizures should last $\geq 10–15$ s after ictal SPECT injection to give localizing information (16).

MR-negative partial epilepsy remains a difficult subgroup in terms of presurgical evaluation. Invasive EEG studies are usually indicated, and ictal SPECT findings may guide electrode placement. Pitfalls are that ictal SPECT may show propagated ictal activity and that ictal SPECT hyperperfusion does not exclude multifocal seizure onset (16). With ictal SPECT and invasive EEG studies, Siegel and colleagues (19) reported a good seizure outcome in 83% of patients with refractory MR-negative partial epilepsy. O’Brien and colleagues (20) reported an excellent outcome when SISCOM localization was concordant with surgical-resection site, but not when SISCOM and resection site were discordant in patients with nonlocalizing MRI findings. In MR-negative patients, SISCOM may be able to detect subtle focal cortical dysplasia (FCD) (21). Ictal SPECT in combination with 3-T MRI scanning appears particularly promising for the detection of subtle FCD (Fig. 4). Marusic and colleagues...
FIG. 4. Detection of small FCDs by using ictal SPECT. The patient had refractory right parietal lobe epilepsy with sensory auras in the left arm. A 1.5-T MRI scan was normal. A 3-T MRI scan revealed a FCD at the place of ictal SPECT hyperperfusion. The injected seizure was a secondarily generalized tonic-clonic seizure that lasted 91 s. The injection was given 2 s after seizure onset. A: Subtracted ictal SPECT coregistered with FLAIR (threshold, +2 SD) showed hyperperfusion in the right parietal lobe. B: FLAIR without SPECT overlay showed a hyperintense lesion at the place of ictal SPECT hyperperfusion. C: T2-weighted MR image also showed the increased signal. D: A T1-weighted image showed blurring of the grey/white matter junction at the location of hyperperfusion. Surgery rendered the patient seizure free. Pathology confirmed the presence of a Taylor-type FCD with balloon cells.

(22) and Boonyapisit and colleagues (23) reported FCD types with different functional characteristics. Focal cortical dysplasias with an increased fluid-attenuated inversion recovery (FLAIR) signal were characterized by balloon cells at the site of the increased signal and an overlying cortex that was not functional. The ictal-onset zone was not within the MR-visible FCD, but adjacent to it. Conversely, FCDs without increased FLAIR signal had the ictal-onset zone in the MRI-visible FCD, did not contain balloon cells, and the overlying cortex could be functional. Preliminary observations suggest that ictal SPECT may be able to demonstrate these characteristics noninvasively and may become a noninvasive marker of the epileptogenic zone in FCD.

Subtraction ictal SPECT coregistered to MRI usually highlights regions of hyperperfusion to detect the seizure-onset zone. When no threshold is applied in difference imaging, it is obvious that large areas of both hypo- and hyperperfusion are present (Fig. 5). To study these perfusion changes in a systematic way, SPM of ictal–interictal SPECT difference images of selected groups of patients can be used. Inclusion criteria suggest that ictal SPECT may be able to demonstrate these characteristics noninvasively and may become a noninvasive marker of the epileptogenic zone in FCD.

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With this method, CPSs in patients with unilateral hippocampal sclerosis have been studied (24,25) (Fig. 6). Ipsilateral temporal lobe hyperperfusion was present throughout the seizure but disappeared in the postictal period. Ipsilateral and also contralateral frontal lobe hypoperfusion was present both during the ictal and postictal period. Contralateral cerebellar hypoperfusion was present in the ictal period, and hyperperfusion in midline cerebellar structures, during the postictal period. Ipsilateral parietal lobe hypoperfusion was a late ictal phenomenon and was observed in ictal SPECTs with injection times ranging from 60 to 90 s. Bilateral medial thalamic hyperperfusion was observed postictally. Ictal frontal lobe hypoperfusion could represent an ictal surround inhibition. In favor of the latter hypothesis was the presence of a crossed cerebellar diaschisis, which has been shown to be due to deactivation of Purkinje cells caused by a decrease in excitatory input due to suppression of electrical activity in the contralateral frontal cortex (26). Further, these SPECT findings corroborate optical imaging experiments that showed a decrease in optical signal together with a decrease in neuronal activity in cortex surrounding an epileptic focus, consistent with ictal surround inhibition (27). The decrease of contralateral cerebellar perfusion in the ictal SPECT injection time window of 0–30 s was ~10%. Gold and Lauritzen (26) showed that a major proportion of the basal cerebellar blood flow was independent of neuronal activity, and that a decrease of cerebellar perfusion on the order of 10–15% was associated with major suppression of electrical activity in the contralateral frontal lobe. Ictal surround inhibition is a defense mechanism against secondary generalization. Secondarily generalized
tonic–clonic seizures show multilobar hyperperfusion (17, 28) and could represent failure of this ictal surround inhibition. Part of the ictal and postictal semiology of CPSs may be due to this ictal surround inhibition.

Blumenfeld and colleagues (28) reported an SPM-ictal SPECT study of generalized tonic–clonic seizures during electroconvulsive therapy (ECT). Bilateral cerebellar and parietotemporal lobe hyperperfusion was observed during bilateral and right-sided ECT. Bilateral frontal hyperperfusion was present during bilateral ECT, and right frontotemporal hyperperfusion, during right-sided ECT. Bilateral cingulate hypoperfusion was present in bilateral ECT, and a left temporal lobe hypoperfusion, in right ECT. Blumenfeld and Taylor (29) postulated that abnormal increased activity in frontoparietal association cortices during secondarily generalized seizures and abnormal decreased activity in the same regions during CPSs may be the neural substrate of loss of consciousness.

In conclusion, ictal SPECT is able to demonstrate ictal neuronal activation and is a noninvasive marker of
the ictal onset zone. The interpretation of ictal SPECT may be confounded by propagation of ictal activity or early switch from ictal hyperperfusion to postictal hypoperfusion during brief extratemporal seizures, which can be minimized by early ictal SPECT injections. Statistical parametric mapping analysis of ictal and interictal SPECT difference images of selected groups of patients is a promising method that highlights regions of significant hyper- and hypoperfusion and may provide new insights into the pathophysiology of seizures.

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EEG of Partial Seizures

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Summary: EEG remains the primary technique in the diagnosis, characterization, and localization of partial seizures. This review examines the significance and character of interictal epileptiform abnormalities, periodic lateralized epileptiform discharges, and ictal patterns in patients with partial epilepsy. Interictal epileptiform discharges are common and assist in the diagnosis and localization of partial seizures. Fortunately, true “false positive” EEGs with focal epileptiform abnormalities are distinctly rare. Periodic lateralized discharges have characteristics of both interictal and ictal activity and are an area of controversy as to their clinical significance. Ictal patterns in partial seizures are variable, with the most distinctive features seen in seizures from a mesial temporal lobe origin. The unifying EEG feature of a partial seizure is in its evolution. A partial seizure begins with a clear delineation of the onset of activity that is distinct from the preceding background, followed by an evolution of this activity in both frequency and amplitude and terminating with an identifiable cessation of the rhythmic pattern that merges again into the background activity.

Key Words: Partial seizures, EEG, Epileptiform activity, PLEDs, Ictal EEG patterns.

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The International League Against Epilepsy defines focal seizures as “a seizure whose initial semiology indicates, or is consistent with, initial activation of only part of one cerebral hemisphere.” (Commission of ILAE, 1981). The EEG findings and ictal semiology of partial seizures are varied and depend on the site of focal seizure onset. It is extremely important to understand those differences (whether interictal, ictal, or postictal) not only for diagnosis and characterization but also for planning surgical procedures in patients with medically refractory partial seizures.

INTERICTAL EPILEPTIFORM DISCHARGES

Several studies have demonstrated that interictal epileptiform discharges (IEDs) are rare in individuals without epilepsy. The most common IEDs that are seen in normal individuals include centro-temporal spikes, generalized spike-wave discharges, and photoparoxysmal discharges. The unifying feature of these discharges is that they all have a strong genetic component. It is easy to conceive of an individual who has never had a seizure but who carries the genetic trait that would manifest itself with such an EEG pattern. Conversely, unambiguous IEDs typical for cryptogenic partial epilepsy are distinctly rare in individuals without epilepsy. In a healthy community hospital-based pediatric population, only 1.9% to 3.5% of the population was noted to demonstrate IEDs of any character on routine EEG (Eeg-Olofson et al., 1971; Cavazzuti et al., 1980). Similarly in healthy young adults, the frequency of IEDs was 0.5% (Bennett, 1967; Gregory et al., 1993). The chance of recording IEDs in hospitalized patients with a neurologic illness was 2.2% versus 0.6% in patients without a neurologic illness (Zivin and Marsan, 1968). In patients with a psychiatric diagnosis the risk was found to be somewhat higher at 2.6% (Bridgers, 1987).

Even in patients with a history of seizures, the data concerning the diagnostic yield of EEG is confusing. Studies are difficult to compare due to differences of study populations, diagnostic criteria, and, most importantly, different skills and perspectives of the interpreting clinicians. Initial EEGs are “positive” in 29% to 55% of patients and repeated EEGs increase the yield to 80% to 90% (Goodin et al., 1984; Marsan and Zivin, 1970; Salinsky et al., 1987). EEG recordings of longer duration, particularly if they include sleep, increase the yield of identifying epileptiform abnormalities. Remarkably, in one study in which patients with intractable partial epilepsy underwent video EEG monitoring for a mean of 6.9 days, 19% of patients did not demonstrate IEDs (Walczak et al., 1992). This suggests that the yield of IEDs may be lower in individuals with partial as opposed to primary generalized epilepsy. IEDs are more commonly identified in EEGs of children independent of seizure type. Certain antiepileptic drugs (AEDs) such as barbiturates and benzodiazepines are thought to suppress IEDs, while other AEDs do not appear to affect the frequency of IEDs in partial epilepsy. The reported increase in IEDs in patients that are being weaned off of AEDs while undergoing video-EEG monitoring is not thought to be due to removal of the AEDs. Instead the increase in IEDs is associated with the appearance of seizures, as the frequency of IEDs is seen to increase immediately after a seizure (Marciani et al., 1985).

FOCAL SLOW ACTIVITY

Focal polymorphic delta activity (PDA) is commonly seen in patients with partial epilepsy. It is primarily associ-
ated with underlying structural abnormalities and has a poor predictive value for epilepsy. However, if no structural abnormality exists to explain the slowing, continuous focal PDA is associated with seizures in about 50% of patients. Focal PDA needs to be differentiated from temporal intermittent rhythmic delta activity (TIRDA), a distinct pattern that has a strong association with epilepsy. TIRDA’s rhythmic nature is distinctly different from PDA (Fig. 1). It usually occurs in runs lasting 3 to 20 seconds and has a strong association with temporal lobe epilepsy. It is commonly seen in association with ipsilateral IIEDs and in one study was seen in approximately one fourth of patients undergoing a presurgical workup for temporal lobe epilepsy (Geyer et al., 1999; Normand et al., 1995; Reiher et al., 1989).

PERIODIC LATERALIZED EPILEPTIFORM DISCHARGES

Periodic Lateralized Epileptiform Discharges (PLEDs) are prominent moderate to high amplitude sharp wave discharges that usually occur with a frequency of 0.5 to 2 Hz (Fig. 2). These typically have variable complexity and are distributed over large areas of one hemisphere. It is unusual to see PLEDs in the setting of otherwise normal background activity. PLEDs are usually seen in the setting of an acute destructive lesion and commonly resolve over days to weeks. They are strongly associated with clinical seizures and subsequent development of epilepsy. Up to 70% to 80% of patients with PLEDs on EEG exhibit overt clinical seizures and 3% to 66% subsequently develop epilepsy (Chatrain et al., 1964; Markand and Daly, 1971; Schwartz et al., 1973; Walsh and Brenner, 1987).

Traditionally PLEDs have been considered to be an interictal pattern as it does not evolve in frequency or distribution as would be expected with a partial seizure. Additionally, PLEDs may be seen in some patients who never experience clinical seizures and in whom no accompanying symptoms or deficits are identified. PLEDs also commonly resolve in a gradual fashion without treatment or intervention.

However, focal clinical symptoms such as focal motor jerking may sometimes be seen time locked to PLEDs on EEG, which then translates to the identification of a focal seizure even without a change in the EEG discharge. Often the resolution of PLEDs parallels the improvement of neurologic deficits such as altered mental status and focal weakness, suggesting that the active discharge is associated with the accompanying neurologic symptoms as seen in partial seizures. Possibly the most compelling evidence that PLEDs are an ictal phenomenon is their association with a focal increase in blood flow seen with single photon emission tomography (SPECT; Pohlmann-Eden et al., 1996). The increased blood flow defined on SPECT is consistent with that seen during partial seizures.

ICTAL PATTERNS

Partial seizures typically have ictal patterns that are distinctly different from their interictal activity. The EEG manifestations of partial seizures usually demonstrate a definite onset, an evolution, and an end. The beginning is often
nonspecific with either focal or generalized desynchronization, low voltage fast activity, or irregular focal or bilateral delta activity. The evolution of the seizure is often the most distinct part with an evolution from lower amplitude, faster activity to higher amplitude activity with slower frequencies. The termination of the seizure is easily discernible with the seizure discharge merging into slow activity that is distinctly less rhythmic than the ictal discharge. The primary exception is seen with some simple partial seizures; such as rhythmic focal discharges with accompanying motor jerks that may not have a clear evolution of frequency or amplitude.

In scalp EEG recordings, partial seizures may not have a clear EEG correlate. This is particularly true with simple partial seizures during which 70% to 90% of the clinical seizures do not have a definite EEG correlate. With routine scalp electrodes, a discernible change has been reported in 11% to 19% of simple partial seizures while the use of sphenoidal electrodes may increase the yield to 28% (Palmini et al., 1992; Devinsky et al., 1988; Lieb et al., 1976; Sirven et al., 1996). On the other hand, scalp EEG changes are present in 85% to 95% of complex partial seizures. An absence of a change on scalp EEG during complex partial seizures is most commonly seen in patients with frontal lobe epilepsy. The lower incidence of EEG changes seen with frontal lobe seizures is attributed to the large area of mesial and inferior frontal cortex that is not easily assayed by scalp electrodes. Secondarily generalized tonic clonic seizures are essentially always associated with a scalp EEG correlate. If there is rapid spread of the ictal activity, a focal onset may not be readily apparent. Additionally, the scalp EEG recording may be obscured by myogenic artifact and may be difficult to discern. However, profound postictal slowing will be identified consistently after a secondarily generalized tonic-clonic seizure. When able to be identified, the EEG pattern during secondarily generalized tonic-clonic seizures is similar to that seen with primarily generalized tonic-clonic seizures. The usual pattern is rapid low amplitude spiking evolving to a slower spike-slow wave discharges. These patterns correlate with the tonic and clonic activity respectively. The EEG ictal patterns seen with seizures arising in different brain regions can be very distinct and are discussed in further detail later.

**MESIAL TEMPORAL LOBE EPILEPSY**

In patients with mesial temporal lobe epilepsy (MTLE), interictal EEGs often demonstrate anterior temporal spikes with maximal amplitude in the anterior temporal or temporal basal electrodes (Fig. 3). These are commonly detected using either anterior subtemporal (T1–T2) or sphenoidal electrodes. In roughly one third of patients, the IIEDs are present bilaterally during sleep. Strongly lateralized IIEDs (>90%–95%) are predictive of side of seizure onset (Chung et al., 1991). The interictal, ictal, and postictal EEG in MTLE demonstrate some very characteristic findings. No definite EEG change is usually seen with auras or the initial clinical behavior change (Fig. 4A). However, lateralized rhythmical theta or alpha activity (5Hz or greater) is seen in 80% of patients with mesial temporal lobe epilepsy and typically occurs 10 to 40 seconds after clinical seizure onset (Fig. 4B; Walczak et al., 1992; Risinger et al., 1989). If present, this activity correctly lateralizes seizure onset in about 95% of patients. Focal postictal slow activity is present in about 70% of seizures and if present is consistent with side of seizure onset correctly in about 90% of patients (Fig. 4C; Walczak et al., 1992; Ebersole and Pacia, 1996).
Neocortical temporal lobe epilepsy has certain EEG features that may help differentiate it from mesial temporal lobe epilepsy (Feldt et al. 1997; Walczak, 1995; Gil–Nagel and Risinger, 1997). The ILEDs and the ictal rhythmic activity are more broadly distributed, often extending to the parasagittal area. The rhythmic ictal activity is also slower, less stable in terms of both frequency and amplitude, and the amplitude distribution of this activity is often higher in the parasagittal electrodes. In addition, an absence of an identifiable EEG change is slightly more common with neocortical temporal lobe epilepsy as compared to mesial temporal lobe epilepsy.

**FIGURE 3.** EEG demonstrating a left temporal interictal epileptiform discharge in a patient with mesial temporal lobe epilepsy. Reproduced from Current Practice of Clinical EEG; Ebersole and Pedley (eds).

**FIGURE 4.** (A): EEG at behavioral seizure onset with no definite EEG change.

**NEOCORTICAL TEMPORAL LOBE EPILEPSY**

Neocortical temporal lobe epilepsy has certain EEG features that may help differentiate it from mesial temporal lobe epilepsy (Feldt et al. 1997; Walczak, 1995; Gil–Nagel and Risinger, 1997). The ILEDs and the ictal rhythmic activity are more broadly distributed, often extending to the parasagittal area. The rhythmic ictal activity is also slower, less stable in terms of both frequency and amplitude, and the amplitude distribution of this activity is often higher in the parasagittal electrodes. In addition, an absence of an identifiable EEG change is slightly more common with neocortical temporal lobe epilepsy as compared to mesial temporal lobe epilepsy.
FIGURE 4. (B): Approximately 30 seconds later, EEG demonstrates lateralized rhythmical theta activity maximal over the left temporal region.

FIGURE 4. (C): EEG following termination of temporal lobe seizure demonstrating left temporal slowing.
FRONTAL LOBE EPILEPSY

In frontal lobe epilepsy, interictal EEGs are more frequently normal, and even with repeated EEGs IIEDs are seen in only 65% to 70% of patients. Focal IIEDs are seen in 40% to 60% of EEGs as midline or bilateral discharges are more common than in patients with temporal lobe epilepsy. The bilateral spike and wave discharges seen often have an amplitude asymmetry and represent secondary bilateral synchrony rather than true generalized onset. The duration of the seizures seen in frontal lobe epilepsy tends to be shorter compared to the seizures seen with TLE, and the postictal periods are shorter as well. As discussed previously, frontal lobe seizures more commonly show no definite correlate on scalp EEG as compared to seizures of temporal lobe onset. The vigorous physical activity frequently present in frontal lobe seizures may result in greater myogenic and movement artifact to further complicate the interpretation. Given these difficulties, it is understandable how scalp localization in frontal lobe seizures is often problematic. False localization may occur especially to the ipsilateral temporal lobe. Ictal onset and evolution may appear generalized and often represents secondary bilateral synchrony, further limiting the confidence even in lateralizing the side of seizure onset (Salanova et al., 1993; Williamson et al., 1985; Rasmussen, 1983).

PARietAL LOBE EPILEPSY

Parietal lobe epilepsy, somewhat surprisingly, represents the most difficult seizure type to evaluate and localize with scalp EEG. Sculp EEG recordings are usually nonlocalizing or falsely localizing. In one study centroparietal IIEDs were seen in only 5% to 15% of patients with parietal lobe seizures (Salanova et al., 1995). The IIEDs are typically widely distributed and it is common to have them reflected to surrounding regions. Ictal patterns are similarly poorly localized. Less than 10% of ictal patterns are well localized to the parietal region and 30% are associated with secondary bilateral synchrony. Ictal patterns may also falsely localize to the ipsilateral temporal lobe (Cascino et al., 1993; Williamson et al., 1992). The difficulties with the localization of parietal lobe seizures even extends to invasive monitoring, such that in the absence of a structural lesion localization of parietal onset seizures remains extremely problematic.

OCCIPITAL LOBE EPILEPSY

Scalp EEG recordings in occipital lobe epilepsy are once again a poor source for attempted seizure localization. IIEDs are restricted to the occipital lobe in less than 20% of patients. As was seen with frontal and parietal onset, IIEDs are often falsely localized to the ipsilateral temporal region or may manifest as synchronous or bilateral IIEDs. A rhythmic ical pattern may be seen in the occipital region at onset in about 15% to 20% of patients. This may spread to motor areas and result in rapid generalization, or alternatively the seizure activity may spread to the ipsilateral temporal lobe and have EEG and clinical features similar to MTLE (Salanova et al., 1992; Williamson et al., 1992). Often the clinical features (visual hallucinations, identified structural abnormality) are more helpful than scalp EEG in ascertaining an occipital onset to partial seizures.

SUMMARY

Scalp EEG continues to serve a dominant role in the diagnosis and characterization of partial seizures. As can be seen from the above discussion, IIEDs are common, particularly in MTLE, and are somewhat less common in patients with seizures arising from the frontal, parietal, or occipital lobes. Fortunately, true false positives for focal IIEDs are distinctly rare. The ictal patterns are variable in partial epilepsy and depend, to a certain degree, on the region of onset. There are, however, unifying characteristics of partial seizures that can be used for diagnosis. These include an identifiable change from the icterial background at seizure onset, a clear evolution in the frequency and amplitude of this activity during the seizure, and an identifiable end of this activity. Seizures in MTLE appears to have the most consistent identifiable ical pattern and are the most useful in confidently localizing sight of seizure onset from scalp EEG recordings.

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