

# Online brainmapping

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## EEG EVALUATION OF FOCAL CEREBRAL LESIONS

### • Role of Electroencephalography in the Era of Computed Tomography

The role of electroencephalography (EEG) in detecting focal cerebral disturbances has undergone a significant change in the last decade owing to the development and availability of computerized imaging techniques. EEG is generally complementary to these as, with the exception of positron emission tomography (PET) and single photon emission tomography (SPECT), only EEG evaluates changes in brain physiology. Furthermore, EEG provides the only continuous measure of cerebral function over time. Computerized transaxial tomography (CT) and magnetic resonance imaging (MRI) scanning are clearly the procedures of choice for delineating structural lesions. However, they do not currently reveal abnormalities unless anatomic alterations in brain tissue have occurred.

In this edition, we will review the major electroencephalographic changes that occur with focal cerebral lesions, describe how they relate to CT findings, and indicate the relevance of focal physiologic dysfunction in the absence of structural pathology. Although this article deals with conventional electroencephalographic techniques, newer developments such as topographic EEG mapping and magnetoencephalography suggest that the monitoring of spontaneous and evoked electrical brain activity will continue to play an important role in neurologic diagnosis.

## ELECTROENCEPHALOGRAPHIC ABNORMALITIES INDICATING FOCAL DYSFUNCTION

### • Focal Delta Activity

Focal delta activity is the classic electroencephalographic sign of a local disturbance in cerebral function. A structural lesion is most strongly suggested if the delta activity is continuously present, shows variability in waveform amplitude, duration, and morphology (so-called "polymorphic" or "arrhythmic" activity), and persists during changes in physiologic state. Delta waves that attenuate with eye opening (or other alerting maneuvers), or fail to persist into sleep, are less indicative of structural pathology.

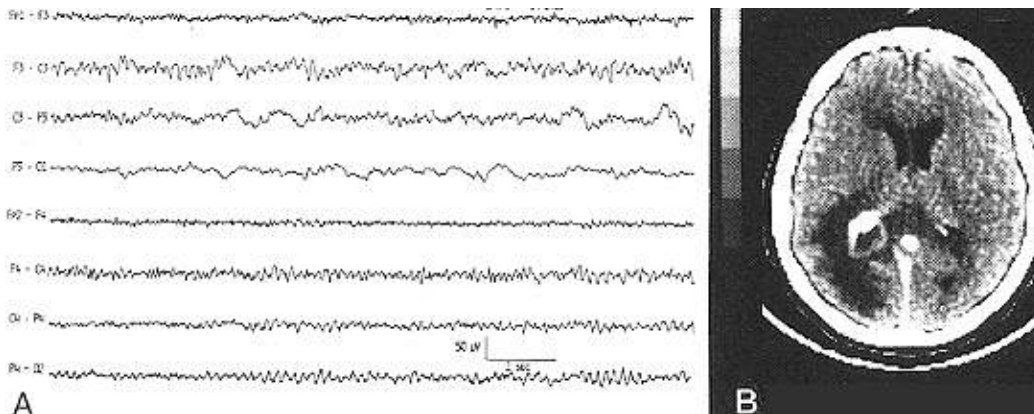


Figure 1. A, Continuous left parietal-occipital polymorphic delta activity. Note associated loss of alpha rhythm and attenuation of faster frequencies over the occipital region. The responsible lesion was a malignant glioma of the left parietal lobe (B).

The localizing value of focal delta is increased when it is topographically discrete or associated with depression of superimposed faster background frequencies. 3,19,34 Superficial lesions tend to produce more restricted EEG

changes, whereas deep cerebral lesions may result in hemispheric, or even bilateral, delta. Lesions involving the central and parietal areas are less likely to present with a circumscribed delta focus, and are also correspondingly more apt to produce delta activity falsely localized to the temporal areas.

Focal delta is often, but by no means always, maximal over the actual lesion. If sufficient destruction of cortex has occurred, the voltage of delta activity may actually be reduced over the area of maximal cortical involvement and thus be higher in the areas bordering the lesion. 19 If two or more delta foci are present, the one that is most persistent and least rhythmic indicates the site of the major lesion, regardless of voltage. Few studies have correlated focal delta with CT abnormalities. Gilmore and Brenner, 17 examined 100 consecutive EEGs containing focal polymorphic delta activity and reviewed the CT findings in these patients. Sixty-eight patients had focal CT lesions, 10 had nonfocal abnormalities, and 22 had normal scans. Although peak delta voltage was not always directly over the lesion, laterality was invariably correct.

Normal CT scans occurred in patients with seizure disorders (12), concussion or contusion (5), ischemic strokes (3), viral encephalitis (1), and a progressive undiagnosed neurologic syndrome (1). Twelve patients with focal neurologic examinations had normal CT scans.

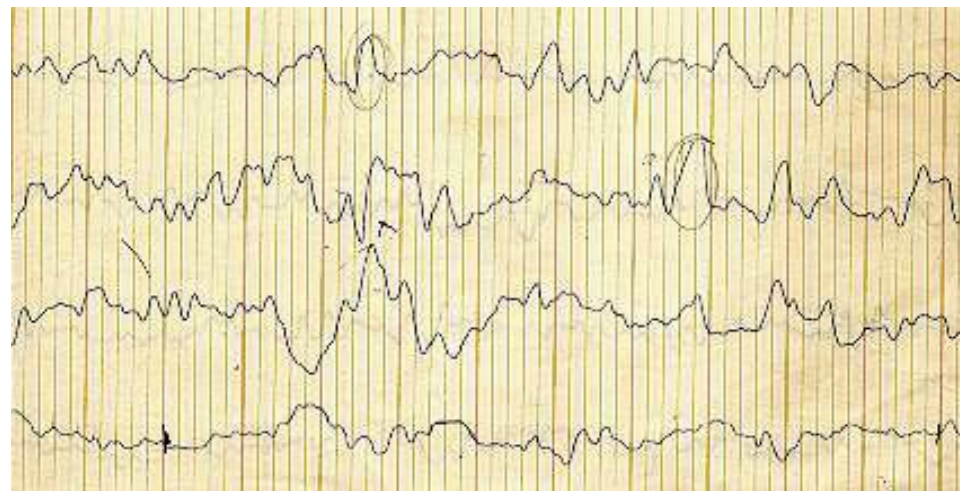
Weisberg and associated 57 studied 50 consecutive patients with a unilateral temporal delta pattern and neurologic signs. CT in these patients showed tumor (40 per cent), vascular lesions (20 per cent), diffuse atrophy (16 per cent), or hydrocephalus (4 per cent). Twenty per cent had normal CT scans, a figure similar to that of Gilmore and Brenner. 17 Half of the patients with normal CT had probable epilepsy; the remainder had no further evolution of their neurologic findings over a 4-year follow-up.

In another study, Weisberg and colleagues, 57 reviewed CT findings in 20 patients who had a "unilateral delta pattern" but normal neurologic examination, cerebrospinal fluid (CSF), and isotope brain scan. Six had abnormal CT scans: three showing diffuse atrophy and three with infarcts. The three patients with atrophy developed Alzheimer's disease within 1 year. The authors did not indicate why the remaining 14 patients with normal CT had been referred to the EEG laboratory.

Reports such as these demonstrate that although focal polymorphic delta is strongly correlated with localized anatomic pathology, EEG findings may occur in the absence of a demonstrable CT lesion. When focal delta is found without a corresponding CT abnormality, it is usually in the setting of seizures, nonhemorrhagic infarction, or trauma. 17, 57

Clinical, 46 and experimental, 18 observations indicate that polymorphic delta results primarily from lesions affecting cerebral white matter. Involvement of superficial cortex is not essential, and, indeed, lesions restricted to the cortical mantle do not generally produce significant focal delta. 18,46 It is likely that functional deafferentation of cortex, rather than a change in cortical metabolic rate, is critical. 18 Cerebral edema does not appear to make a substantial contribution to the production of delta waves. 16,18,46

Persistent polymorphic delta activity may not precisely match the true location of the lesion, particularly since it presumably arises from



physiological deranged neurons often lying on the margin of the destructive lesion. Persistent polymorphic delta activity is aetiologically nonspecific and is seen in a variety of subcortical (white matter) destructive lesions including neoplasms, infarctions, abscesses, trauma, and haemorrhage. It can also be seen in reversible processes such as focal ischemia in transient ischemic attacks or focal depression from a recent seizure.

Because the likelihood of a demonstrable structural change is strongly correlated with the degree of slowing, the clinical associations of focal theta activity are less striking, especially for acute or subacute lesions. Focal theta may be seen in the early stages of a slowly growing neoplasm or in the resolution of acute lesions caused by stroke or trauma.

Figure 2. Polymorphic slow wave activity in a patient with subcortical glioma, notice the marked variability in wave shape morphology, frequency and amplitude.

- **Beta Activity**

Abnormalities in beta activity are usually limited to voltage asymmetries. To be considered unequivocally abnormal, there should be a persistent amplitude difference of 35 per cent or greater (expressed as a percentage of the higher voltage). 32

Diminished beta activity results either from cortical dysfunction or from an increase in resistance of the medium separating cortex from scalp recording electrodes. Thus, local attenuation of beta may occur with a cortical infarction, for example, or in the presence of a subdural or epidural fluid collection. A beta asymmetry may also result from localized scalp edema caused by head injury or infiltration from an intravenous line. Similar considerations apply as well to the localized absence or attenuation of background rhythms other than beta.

Focally increased beta activity is usually associated with a skull defect. 7,25 Occasionally, localized enhancement of beta may occur over a tumor or as the manifestation of an epileptogenic focus. 20

- **Epileptiform Activity**

Focal epileptiform activity (spikes or sharp waves) may antedate the appearance of focal EEG-slowing or other clues to a tumor by months or years. 21 In a multicenter study of 1396 patients with epilepsy, 10 per cent had tumors detected by CT. 15 However, the incidence of tumor rose to 22 per cent when only patients with partial seizures were considered. Brain tumor did not occur in patients with primary generalized epilepsy and was found in only 5 per cent of those with secondary generalized seizures.

Periodic lateralized epileptiform discharges (PLEDS) usually occur in the setting of an acute or subacute destructive process. Impaired consciousness is virtually always present, and seizures are evident nearly 80 per cent of the time. The complexes are most often composed of di- or triphasic spikes or sharp waves recurring at approximately regular 1 to 2-second intervals. However, the distribution, morphology, voltage, and rate of repetition vary substantially among patients. Schwartz and coworkers, 53 studied 52 patients and found a typical electrographic evolution for PLEDs. Gradual simplification in morphology and progressive prolongation of the interval between discharges usually occurred within 4 weeks. In a few patients, however, clinical relapses were accompanied by reappearance of PLEDs.

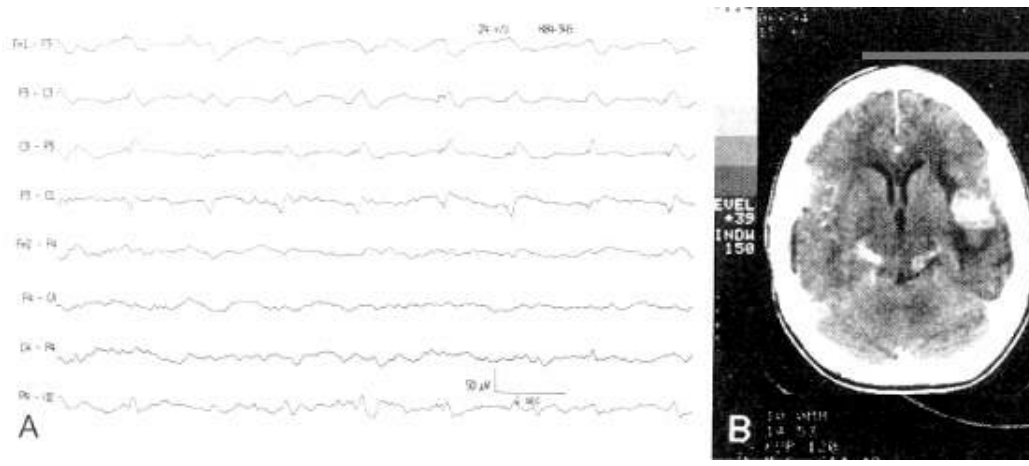


Figure 3. A, Left-sided PLEDs, maximally involving the left parietal-occipital region. Background rhythms are slowed bilaterally, and there is a slight reduction in faster frequencies on the left. The patient had an intracerebral tuberculoma on that side.

PLEDs may also occur independently over both hemispheres, a situation referred to as BIPLEDs. 11 In patients exhibiting BIPLEDs, diffuse diseases, rather than focal lesions, are the rule. Thus, BIPLEDs are most often seen with infections (particularly herpes simplex encephalitis), anoxic encephalopathy, epilepsy, and sickle cell anemia. 01,11

**LATERALIZED AND GENERALIZED ELECTROENCEPHALOGRAPHIC FINDINGS**

- Lateralised EEG changes

The character and distribution of the electroencephalographic changes produced by a focal lesion depend on size of lesion, its distance from the cortical surface, and the specific structures involved. A small lesion critically located in the thalamus, for example, may produce widespread hemispheric slowing and alteration in sleep spindles and alpha rhythm regulation. The same discrete lesion, however, located at the cortical surface, may produce few, if any, electroencephalographic findings. Indeed, cortical lesions must involve relatively large areas to produce

attenuation of background rhythms in the relative absence of slowing. Examples include subdural hematomas and meningiomas. Large infarcts (due to middle cerebral or carotid artery occlusions, for instance) involve extensive areas of cortex as well as adjacent white matter, thus producing both hemispheric polymorphic delta and loss of overriding faster frequencies. Lesions that produce hemispheric depression of background rhythms affect both normal and abnormal patterns, as illustrated by the case of a subdural hematoma causing an interhemispheric asymmetry of triphasic waves in an alcoholic with hepatic failure.

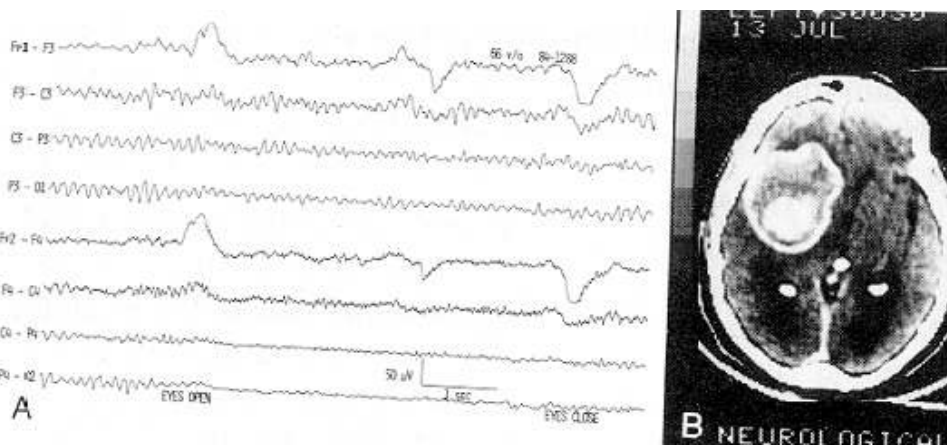


Figure 4. A, Failure of alpha rhythm to attenuate normally with eye-opening on the left. The patient had a giant aneurysm of the left internal carotid-middle cerebral artery bifurcation with compression of the frontal and temporal lobes from below (B).

Focal lesions may slow or attenuate the alpha rhythm unilaterally. A particularly striking abnormality of the alpha rhythm is unilateral failure to attenuate normally with eye opening (Bancaud's phenomenon) or other alerting maneuvers. These changes are reliable indicators of an ipsilateral, usually posterior, cerebral lesion, but they do not provide more specific localizing information.

The photic driving response to repetitive flash stimulation may be consistently lateralized in normal individuals. 9 When it is the only finding in an otherwise normal record, an asymmetry of photic driving may usually be ignored. It is clear that a cortical lesion may depress the photic response unilaterally, but under these circumstances, the asymmetric photic response occurs in conjunction with other indications of focal dysfunction. Occasionally, focal lesions (especially subcortical or epileptogenic ones) may enhance the photic response on one side. 9 Hyperventilation will often enhance localized, low-amplitude polymorphic delta or convert intermittent slowing into a continuous focal abnormality. Focal spikes, or even seizures, sometimes appear only during hyperventilation. 42 A consistently asymmetric response to hyperventilation is always abnormal.



- **Generalized EEG changes**

Generalized electroencephalographic abnormalities do not contribute to localization of a focal lesion or by themselves even suggest the presence of localized structural pathology. They do, however, provide information about the extent of dysfunction resulting from a focal lesion or about a coexisting abnormality (metabolic encephalopathy, for example).

Subfrontal, diencephalic, or infratentorial lesions may produce generalized electroencephalographic changes, usually a combination of intermittent bursts of rhythmic delta waves and continuous, widespread polymorphic theta and delta slowing. In the absence of obstructive hydrocephalus, electroencephalographic abnormalities are more frequent with rostral than caudal brain-stem lesions. Schaul and coworkers, 51 reviewed the EEGs of 154 patients with diencephalic or posterior fossa lesions. Only 12 per cent of patients with diencephalic lesions had normal EEGs, whereas 60 per cent and 73 per cent of patients with lower brain-stem or cerebellar pathology respectively had normal EEGs. If the EEG abnormality was clearly lateralized, an infratentorial lesion was unlikely.

Paroxysmal bursts of rhythmic delta waves with frontal or occipital predominance (the latter especially common in children) have been associated with subfrontal, deep midline, or posterior fossa lesions. In fact, however, intermittent rhythmic delta activity (IRDA) is nonspecific and is seen much more often in the setting of metabolic disorders or other encephalopathies affecting the brain diffusely than with focal lesions, regardless of location.

IRDA may appear against an otherwise normal background. In contrast to polymorphic delta, IRDA is usually reactive to alerting maneuvers, disappears in sleep, and is augmented by hyperventilation or drowsiness. Correlative studies using CT and PET 16,29,41,46 have failed to demonstrate a particular anatomic structure responsible for generating IRDA. Physiologic investigations, 18,46 implicate dysfunction of thalamocortical interactions.

Rhythmic delta activity consists of sinusoidal waveforms of approximately 2.5 Hz that occur intermittently in the EEG recording. It is most often symmetric but can be lateralized. In adults, the delta activity has a frontal predominance (frontal intermittent rhythmic delta activity [FIRDA]). In children, it is maximal posteriorly (occipital intermittent rhythmic delta activity [OIRDA]). Intermittent rhythmic delta activity is associated with structural lesions, most commonly diencephalic, infratentorial or intraventricular tumors, or with diffuse encephalopathies. FIRDA occurring in patients with a normal EEG background suggests that the pattern is due to a structural lesion; when associated with EEG background abnormalities, it is likely to be due to encephalopathy. In cases of encephalopathy with FIRDA, the pathophysiologic processes are believed to involve cortical and subcortical gray matter. OIRDA is associated with absence epilepsy in children aged 6-10 years.

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