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Focus on Diagnosis : Electroencephalography: A Primer

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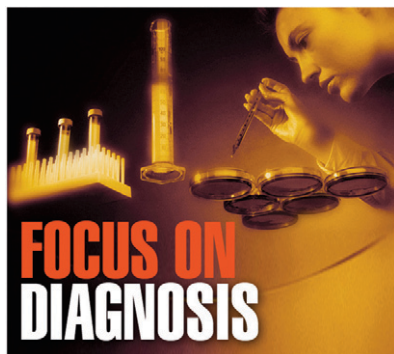
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Electroencephalography: A Primer

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Introduction

Electroencephalography is the recording of electrical activity along the scalp produced by the firing of neurons within the brain. The first recordings were made by Hans Berger in 1929.

Physiology

EEG activity is due to charge movement across neuronal membranes. This activity reflects the summation of excitatory and inhibitory postsynaptic potentials in apical dendrites of pyramidal neurons of the cortex.

Uses

EEG tracings can serve many functions:

1. To differentiate epileptic seizures from pseudoseizures, syncope, behavioral disorders, movement disorders, and migraine variants;
2. To distinguish organic or metabolic encephalopathy from primary psychiatric syndromes;
3. To define seizure semiology, prognosis, and characterization for treatment;
4. To localize the epileptic focus for possible respective surgery;
5. To monitor status epilepticus (especially nonconvulsive) in ICUs;
6. To determine whether to wean antiepileptic drugs;
7. To make the diagnosis of brain death; and
8. To investigate parasomnias (sleep disorders).

Twenty-one electrodes are usually placed on specific positions on the scalp to measure voltage fluctuations over time. The manner in which pairs of electrodes are connected to each amplifier of the EEG machine is called a montage. Primary generalized epilepsy is suggested by spike-and-wave discharges that are spread widely over both hemispheres of the brain. Spikes and sharp waves in a local area of the brain, such as the left temporal lobe, indicate that partial seizures are beginning in that area.

Case Vignette 1

A 6-year-old girl is brought to you by her mother, because her teacher has observed multiple daily staring episodes in which the child is unresponsive to verbal and tactile cues. These episodes last ~10 to 15 seconds and occur ~10 to 20 times a day. Immediately after the episode, she resumes normal activity with no recollection of the event. The teacher has also noted facial twitching and fluttering of the eyelids with some of these episodes. There has been a recent decline in school performance, and the diagnosis of attention deficit hyperactivity disorder is suggested. Results of physical examination are normal. EEG demonstrates 3 per second generalized spike-and-wave activity (Fig 1). This activity is provoked by hyperventilation.

The diagnosis in this case is absence seizures.

Absence seizures have an age of onset of ~5 to 6 years and are characterized by brief lapses in consciousness that occur multiple times per day. No aura or postictal state occurs with these seizures, and the spells can be provoked by hyperventilation or photic stimulation. EEG is diagnostic

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Figure 1. Three-hertz spike-and-wave discharges, the hallmark of absence epilepsy.

and is characterized by frequent 3 per second generalized spike-and-wave activity. The condition is treated with ethosuximide, valproate, or lamotrigine and typically is outgrown by the early teenage years.

Absence seizures may be mistaken for complex partial seizures. In the latter, spells usually last longer than 30 seconds, have a more gradual onset and termination, occur only once or twice daily, and commonly are associated with an aura or automatism or postictal confusion. EEG demonstrates focal epileptiform discharges, commonly in the temporal lobe. EEG changes are not precipitated by hyperventilation.

Case Vignette 2

A 15-year-old girl presents to the clinic with concerns of morning twitches.

These jerky spells occur within a few minutes of awakening. She becomes very tremulous, her arm jolts, and then she drops her toothbrush and comb. She remains conscious during these episodes. On the day of presentation, she has had a generalized tonic-clonic seizure. There is history of idiopathic primarily generalized epilepsy in her mother. The adolescent onset of morning myoclonus coupled with generalized convulsive seizures and an EEG study that demonstrates 4 to 5 cycles per second generalized polyspike and wave pattern (Fig 2) is consistent with juvenile myoclonic epilepsy (JME). Patients with JME also may have absence seizures in addition to their morning myoclonic seizures and generalized convulsions, some of which may be precipitated by photic stimulation.

JME is an inherited generalized epilepsy that is typically lifelong, as opposed to absence seizures, which are “outgrown” during adolescence. Several different genetic linkages have been discovered, many on chromosome 6. Patients with JME need to be particularly careful about avoiding sleep deprivation and alcohol. In general, treatment of JME is a broad spectrum antiepileptic drug (AED), such as valproate, levetiracetam, topiramate, or lamotrigine. Myoclonic seizures are precipitated with photic stimulation.

Case Vignette 3

A 6-month-old African American boy presents with a history of clusters of head nods and sudden forward flexion of the upper torso, along with flaring of arms outward, then inward.

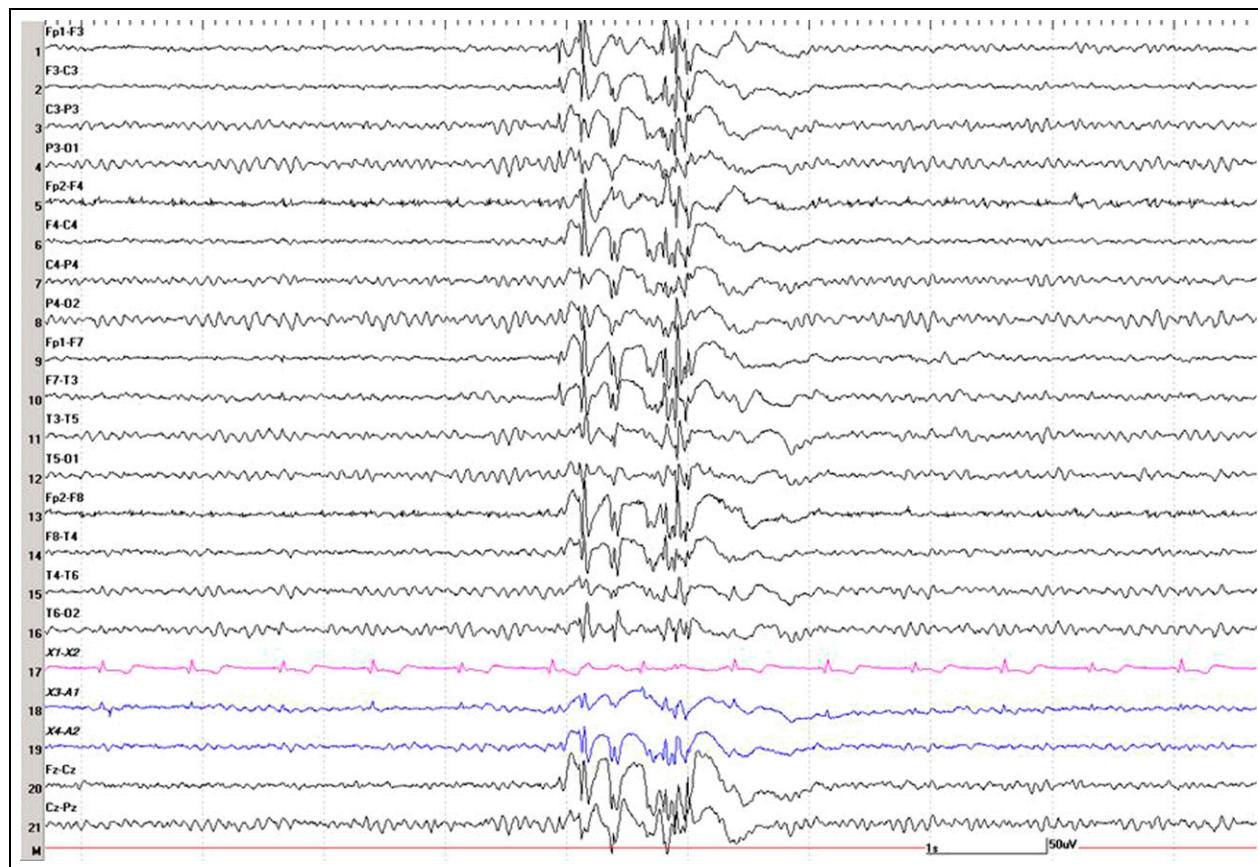


Figure 2. Four- to 6-Hz poly spike-and-wave activity is seen with generalized myoclonic seizures.

These clusters occur >20 times per day. Skin examination reveals ash leaf macules. Infantile spasms are suspected. The patient is placed on continuous EEG monitoring, which demonstrates modified hypsarrhythmia with low-amplitude and less fast activity (Fig 3). MRI of the brain reveals the presence of tubers suggestive of tuberous sclerosis. Tuberous sclerosis complex presents often as infantile spasms. Treatment choices include corticotropin, corticosteroids, and vigabatrin or other broad-spectrum AEDs.

Case Vignette 4

A 7-year-old girl experiences three episodes of seizures within a span of 1 month characterized by paresthesias of the mouth followed by clonic movements of the mouth, face, and right

arm. The seizures last for ~30 seconds to 1 minute without loss of consciousness. There is no family history of febrile seizures, epilepsy, or any other neurologic illness. The results of her neurologic examination are normal. An EEG shows bilateral centrotemporal spikes (Fig 4). A gentle tapping on the volar aspect of her thumb with a tendon hammer produces spikes above the contralateral centrotemporal regions.

Benign rolandic epilepsy, or benign childhood epilepsy with centrotemporal spikes, is a common partial epilepsy that causes oropharyngeal, motor, and somatosensory symptoms confined to the face and ipsilateral extremity. The condition begins at an average age of 7 to 9 years (range, 2–14) and occurs mostly during sleep at night. Neurologic examination

results are normal, and intelligence is unaffected. The EEG pattern is diagnostic and demonstrates bilateral spikes in the centrotemporal or rolandic area with normal background activity. Neuroimaging results are normal. Anticonvulsants usually are not indicated because the seizures are infrequent and resolve spontaneously by ~15 years of age. Carbamazepine may be prescribed if seizures are frequent.

Case Vignette 5

A 13-month-old boy is referred for refractory seizures. He is having intermittent episodes of extension of his neck associated with rolling of his eyeballs. These episodes occur ~5 to 10 times a day and are not associated with loss of consciousness or postural tone.

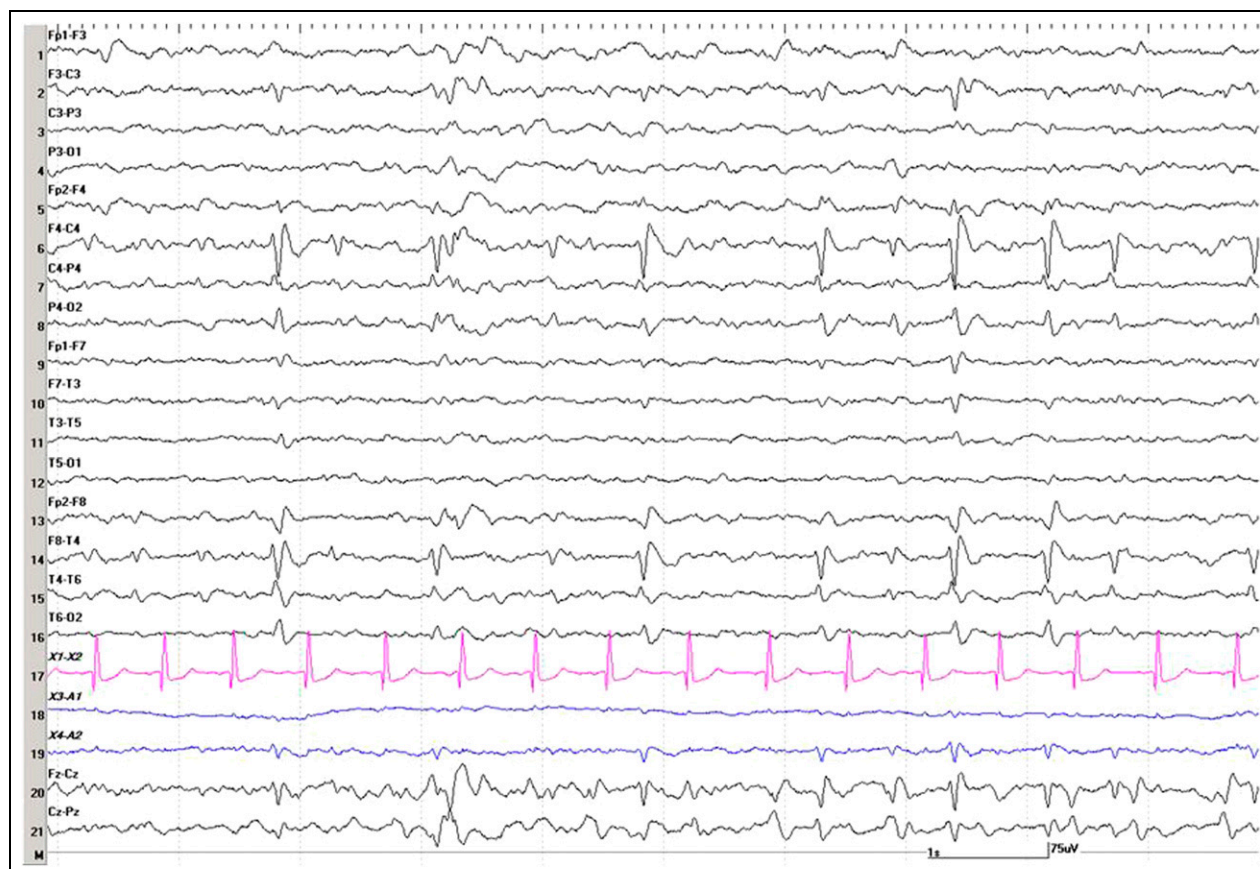


Figure 3. Repetitive spike focus localized in the centrotemporal or rolandic area with a normal background activity, typical of benign rolandic epilepsy.

There is no postictal confusion. These episodes are more frequent after feeding. There is a family history of seizures. The boy's birth and development have been normal. Neurologic assessment is within normal limits. The results of a previous EEG were normal, and the boy has been treated with valproate and levetiracetam with no effect. He is admitted for a video EEG. Videotaping of these episodes shows recurrent episodes of dystonic movements of the neck. Sandifer syndrome is suspected and confirmed by esophageal pH probe studies. His symptoms resolve completely with antireflux therapy.

Sandifer syndrome is characterized by spasmodic torticollis and dystonia associated with gastroesophageal reflux

or hiatal hernia. The episodes may be confused with infantile spasms. A temporal relationship with feeding and a neurologic examination with normal results may help in arriving at the diagnosis. Treatment is antireflux therapy or correction of the hiatal hernia.

Case Vignette 6

An 8-year-old white girl has presented with intractable seizures since infancy. She was born at term, and global developmental delay was noted early in childhood. She experienced a multiplicity of seizure types, including atypical absence, generalized tonic clonic, myoclonic, and drop attacks. Multiple antiepileptic drugs, including valproate, clonazepam, clobazam, gabapentin, and levetiracetam, were used,

but to no avail. Interictal EEG reveals generalized slow spike-and-wave abnormality. The diagnosis is Lennox-Gestaut syndrome.

Lennox-Gestaut syndrome is characterized by a triad of intractable seizures of multiple types, interictal EEG abnormalities, and mental retardation. The condition usually begins at 2 to 4 years of age and may be preceded by infantile spasms. Seizures are resistant to therapy. Valproate, clonazepam, vigabatrin, lamotrigine, felbamate, and topiramate have been used. A ketogenic diet should be considered in patients refractory to AEDs.

Case Vignette 7

A 10-day-old girl born at term presents with multiple episodes of

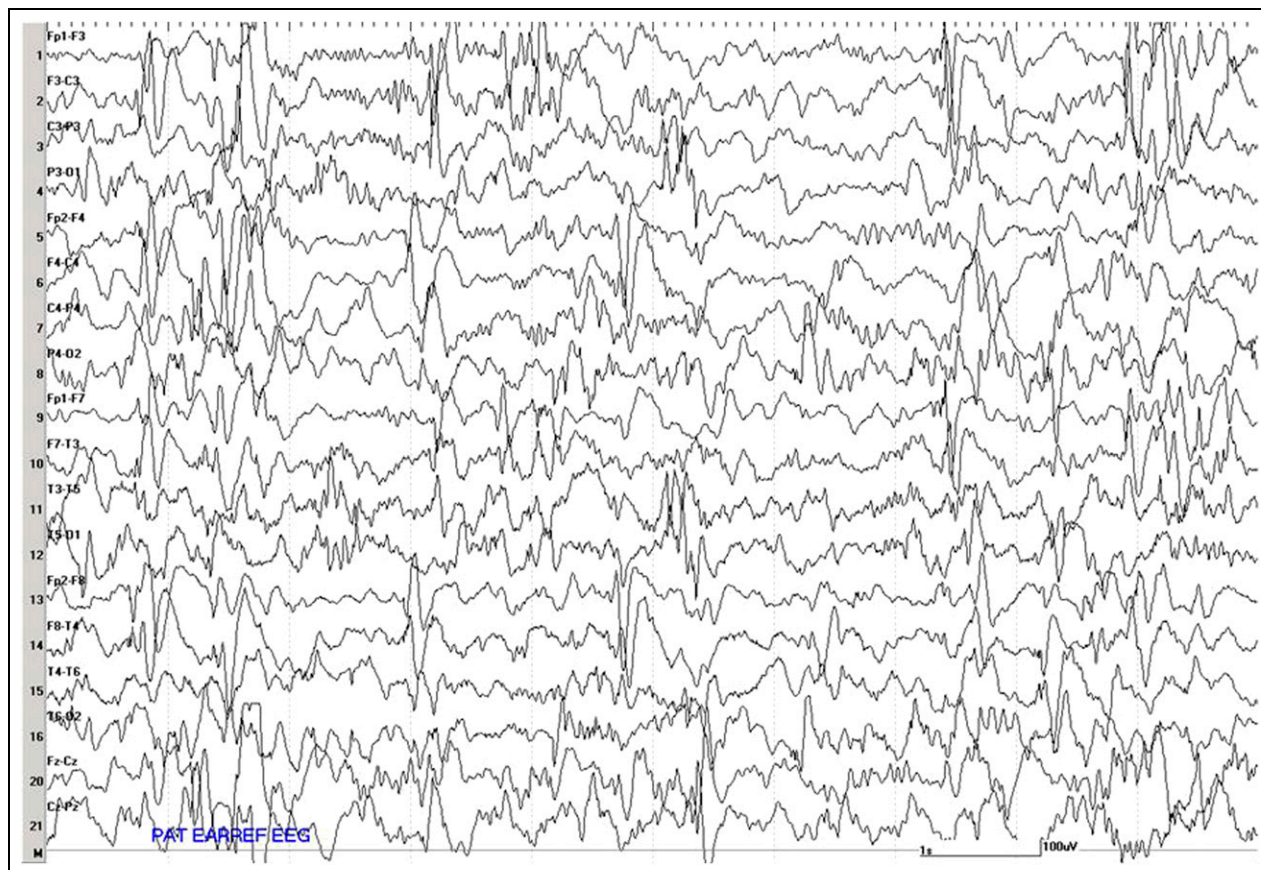


Figure 4. Hypsarrhythmia, which consists of a chaotic pattern of high voltage, bilaterally asynchronous, slow wave activity, characteristic of infantile spasms.

abnormal jerks affecting all four limbs. In between the episodes, she is active and feeding well. There is no history of fever. The pregnancy and delivery were uneventful. The infant is breastfed exclusively. The physical examination is within normal limits. Blood levels of glucose, electrolytes, calcium, and magnesium are normal. The results of a septic evaluation are negative. Computed tomography scan of the brain and EEG have normal results. A tentative diagnosis of benign sleep myoclonus is made. The myoclonic jerks occur during sleep only and disappear when the infant is awake. This finding is corroborated by the nurse. Parents are reassured, and the infant discharged from the hospital. The

myoclonus disappears by 3 months of age. Follow-up at 1 year of age reveals normal neurologic findings and development.

Benign neonatal sleep myoclonus is a nonepileptic, self-limited parasomnia that begins typically during the neonatal period. It occurs during sleep and stops when the baby is awake. The myoclonus may be induced by repetitive sound stimuli or rocking the baby. These episodes may be confused with infantile spasms but can be distinguished from the latter by the absence of EEG findings. The prognosis is good, with no sequelae and cessation of myoclonus by 2 years of age. Antiepileptic therapy is not indicated.

Case Vignette 8

A 16-year-old girl is referred for refractory seizures. She was admitted for video EEG monitoring. Results of her neurologic examination are normal. One of the episodes captured during video EEG reveals a gradual onset of facial grimacing followed by forward pelvic thrusting. She appears unresponsive. No urinary incontinence, frothing from mouth, cyanosis, or tongue bite is noted. Her pupils remain normal, and she has normal muscle tone and flexor plantar response. She recollects the episode and had no lethargy or confusion after the episode. EEG shows excess muscle artifacts with a normal background activity devoid of epileptic discharges.

Detailed psychological evaluation reveals a deep fear of her mathematics teacher and possibility of sexual abuse. She experienced two such episodes at school just before mathematics class. She is diagnosed with nonepileptic seizures (pseudoseizures or psychogenic seizures) and anticonvulsants are stopped.

Pseudoseizures are more frequent in adolescent girls. A more gradual onset; bizarre and variable pattern of episodes; verbalizations; lack of cyanosis; normal pupillary light reflex; flexor plantar responses; absence of sphincter incontinence, tongue bite, or body injury; response to noxious stimuli; and memory for the episode may help distinguish a pseudoseizure from a true seizure. Often these patients have a past history of epilepsy. Serum prolactin level is unchanged from baseline after a pseudoseizure,

unlike in a true seizure in which case it is increased.

Video EEG has a high yield in distinguishing seizures from nonepileptic events, in classifying seizures, and in determining candidacy for epileptic surgery. In one study, video EEG resulted in an alteration of clinical management in 45% of pediatric patients. (1)

Limitations

The sensitivity and specificity of EEG in diagnosing epilepsy is ~25% to 56% and 78% to 98%, respectively. EEG is less sensitive to pick up signals generated from the deeper structures (eg, hippocampus, basal ganglia). Normal EEG results are seen in 10% to 20% children with epilepsy. EEG abnormalities may be found in 2% to 4% of healthy children with

no known history of seizures. Approximately 50% patients with epilepsy have a normal first EEG.

Reference

1. Chen LS, Mitchell WG, Horton EJ, Snead OC III. Clinical utility of video-EEG monitoring. *Pediatr Neurol.* 1995;12(3):220–224

Suggested Reading

- Ebersole JS, Pedley TA, eds. *Current Practice of Clinical Electroencephalography.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins;2003
- Guerrini R. Epilepsy in children. *Lancet.* 2006;367:499–524
- Major P, Thiele EA. Seizures in children: laboratory diagnosis and management. *Pediatr Rev.* 2007;28(11):405–414
- Smith SJM. EEG in the diagnosis, classification, and management of patients with epilepsy. *J Neurol Neurosurg Psychiatry.* 2005;76:ii2–ii7

Condolences

The staff of *Pediatrics in Review* would like to express our sorrow at the death of Dr. Donald Lewis, co-author of this article, a highly respected colleague, and valuable member of our Editorial Board.

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