

# EEG: Interictal Patterns

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- 1) Understand and apply the principles of identifying abnormal focal sharp waves and spikes
  - a. Sharply contoured deflections are artifact until proven otherwise.
  - b. Pathological sharp waves and spikes must be distinguished from physiologic (normal) sharp waves.
  - c. Sharp waves and spikes must have a definable electrical field.
  - d. Sharp waves and spikes usually have surface negative polarity.
  - e. Sharp waves and spikes are usually followed by a slow wave.
  - f. Sharp waves and spikes must deviate from the background pattern.
  
- 2) Identify temporal intermittent rhythmic delta activity (TIRDA) and understand its association with seizures
  - a. TIRDA is often, but not always, associated with focal spikes in the same region.
  - b. TIRDA is most often observed during drowsiness.
  - c. TIRDA has a high positive predictive value for temporal lobe epilepsy (it is very rarely seen in a general EEG lab setting, but is a common feature of EEGs among people being evaluated for temporal lobectomy).
  
- 3) Explain the implications of other patterns of focal slowing
  - a. Focal slowing should be described according to its location, frequency, and pattern.
    - i. Abundance: rare, intermittent, frequent, continuous
    - ii. Location: as precisely as possible (e.g. left anterior temporal, T3-maximal).
    - iii. Frequency: frequency range in Hz
    - iv. Patterns:
      1. Polymorphic (constantly changing morphology, frequency, and voltage)
        - a. Continuous polymorphic delta activity → localized structural lesion (tumor, stroke, abscess)

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- b. Intermittent polymorphic delta activity → less specific association (but suggestive of underlying focal neuronal dysfunction)
      - 2. Rhythmic (slow waves of uniform frequency and morphology)
        - a. Frontal intermittent delta activity (FIRDA) must be distinguished from vertical eye movements or blinks, hypnagogic hypersynchrony, or hyperventilation response. FIRDA is most often associated with encephalopathies but can also occur in association with deep midline lesions, subcortical white matter abnormalities, or increased intracranial pressure.
        - b. Occipital intermittent rhythmic delta activity (OIRDA) is associated with childhood absence epilepsy.
        - c. See above for temporal intermittent rhythmic delta activity (TIRDA)
  - 4) Recognize and accurately describe generalized spike-wave and generalized polyspike-wave discharges
    - a. Generalized spike-wave (GSW) discharges are the hallmark of many genetic generalized epilepsies (in which GSW discharges punctuate an otherwise normal EEG background).
    - b. Typical GSW occurs at a frequency of around 3 Hz and is synchronous and symmetric. The frequency should be measured in the first countable second of the discharge, as frequency may slow over the course of a burst of GSW.
    - c. GSW in juvenile myoclonic epilepsy is typically faster (3.5-6 Hz) than that of childhood absence epilepsy (2.7-5 Hz).
    - d. To distinguish between generalized single-spike-wave and generalized polyspike-wave, it is often helpful to view the EEG in an average reference montage. Polyspikes have two or more surface-negative spikes prior to the first slow wave.
    - e. Atypical GSW are synchronous discharges with variable rates, amplitudes, and morphology within and between the bursts.
  - 5) Recognize photoparoxysmal responses (PPR) and understand their associations with specific epilepsy syndromes
    - a. Spikes or complexes of spike-wave elicited by photic stimulation.

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- i. Self-limited – the discharges do not persist past the end of photic stimulation
      - ii. Self-sustaining – the discharges continue after the end of stimulation
    - b. Description of the PPR should include the flash frequency at which it was provoked, the morphology of the EEG discharge, and whether there was an associated clinical manifestation (e.g. subclinical, myoclonus, or convulsion).
    - c. Generalized PPR is the most common pattern, but posterior-predominant, bi-occipital, or focal (unilateral) discharges can also be provoked by photic stimulation.
    - d. Up to three quarters of people with generalized PPR have a generalized epilepsy syndrome.
  - 6) Recognize hypsarrhythmia and its association with infantile spasms
    - a. Classic hypsarrhythmia is characterized by chaotic, very high amplitude ( $>200\mu\text{V}$ ; often  $>300\mu\text{V}$ ) slow waves with abundant multifocal and diffuse epileptiform discharges (may be a combination of sharps, spikes, spike-wave, and/or paroxysmal fast activity). There is no background organization. This background pattern may be interrupted intermittently by abrupt voltage attenuation for several seconds (electrodecrement) which may be focal or diffuse.
    - b. There are multiple forms of modified hypsarrhythmia:
      - i. hypsarrhythmia with increased interhemispheric synchrony
      - ii. hypsarrhythmia with preserved stage II sleep architecture
      - iii. hemi-hypsarrhythmia (also known as asymmetric hypsarrhythmia)
      - iv. hypsarrhythmia with a consistent focus of abnormal discharges
      - v. hypsarrhythmia with high voltage slow activity with few sharp-waves or spike-wave discharges
      - vi. Treatment response for infantile spasms does not seem to vary according to classic versus modified hypsarrhythmia designation.
    - c. Although the EEG pattern appears “generalized”, focal brain lesions can result in hypsarrhythmia (and resection of an epileptogenic focus, when present, can be curative).
    - d. Infantile spasms are typically associated with hypsarrhythmia. The spasm coincides with a high amplitude slow wave, followed by a 1-2 second electrodecrement which may have over-riding low amplitude fast activity.

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- e. Hypsarrhythmia may be present only during sleep; when infantile spasms are suspected, an EEG of sufficient duration to capture wakefulness and sleep should be obtained urgently.
  - f. Early, successful treatment of infantile spasms with standard medications (ACTH, oral steroids, or vigabatrin) offers the best chance of protecting neurodevelopment.
- 7) Identify asymmetric beta activity and explain the implications of this pattern
- a. Persistent asymmetry of beta activity (>35% voltage difference between homologous regions) implies focal cortical dysfunction.
  - b. Breach rhythm is the most common cause of focal enhanced beta activity.
  - c. Focally attenuated beta activity is a sign of cortical dysfunction. The asymmetry may be accentuated by barbiturates or benzodiazepines (the abnormal region does not generate the expected normal beta activity when exposed to these medications).
  - d. Extra-cerebral fluid collections (e.g. subdural or epidural hematoma) can also result in focal attenuation of beta activity.

#### References

- 1) Current practice of clinical electroencephalography, 4<sup>th</sup> Edition. Ebersole JS, Husain AM, and Nordli DR, editors. 2014. Wolters Kluwer. Philadelphia, PA.
- 2) Mulsby RL. Some guidelines for assessment of spikes and sharp waves in EEG tracings. Am J EEG Technol. 1971; 11:3-16.
- 3) Hirsch LJ, et al. American Clinical Neurophysiology Society's Standardized critical care EEG Terminology: 2012 version. J Clin Neurophysiol. 2013;30:1-27.