

Lateralizing and Localizing Findings in Focal Epilepsies: A Concise Review

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Abstract

Knowledge of lateralizing and localizing value of seizure semiology and other clinical findings is necessary in the management process of patients with focal epilepsy, particularly with widespread use of surgery in the management of patients with refractory focal epilepsy. The advent of video-EEG monitoring has permitted careful analysis of semiologic features of seizures and their correlation with simultaneous EEG activities. The availability of new imaging and functional studies could be considered as a revolution in localization of the epileptogenic zone. In the current concise review, a list of well-documented lateralizing and localizing findings in focal epilepsies is prepared. This paper is designed as a practical tool for physicians, aiming to serve as a practical, problem-oriented reference. While I include the correlated symptomatogenic zone and the possible mechanism in generating the finding in the context of a focal seizure, this paper emphasizes how to localize the epileptogenic zone according to any given specific finding. I hope that this paper leads to improved patient care.

Keywords: EEG; Focal epilepsy; Lateralization; MRI; Semiology

Introduction

Knowledge of lateralizing and localizing value of seizure semiology (symptoms and signs) and other clinical findings is not only helpful, but also necessary in the management process of patients with focal epilepsy. The importance of these findings has specifically increased during the past three decades and with widespread use of surgery in the management of patients with refractory focal epilepsy. The advent of video-EEG monitoring has permitted careful analysis of semiologic features of seizures and their correlation with simultaneous EEG activities. The availability of new imaging and functional studies could be considered as a revolution in localization of the epileptogenic zone in patients with focal epilepsy [1].

One should consider that seizure semiology has several limitations in localizing and even lateralizing the seizure onset. Although, many semiologic features have high positive predictive values, none is perfect in determining the seizure onset or epileptogenic zones. Seizure semiology is sometimes dictated by the pathway of electrical seizure propagation [2] and can reflect only the symptomatogenic zone [3]. Therefore, video recordings of multiple seizures should be reviewed carefully to find as many useful semiologic features as possible. A seizure that is representative of the rest of the recorded seizures should be reviewed with the patient's relatives to verify that habitual seizures have been captured and analyzed. It is noteworthy to mention that age and brain maturation has some effects on seizure semiology and ictal features are less conclusive in children with focal epilepsy [4]. Concordance between seizure semiology and EEG activity increases the value of localization process and judgment [2,5]. Furthermore, concordance of the above-mentioned findings with imaging, functional studies, and neuropsychological studies increase the reliability of the localization and / or lateralization of the seizure onset zone or epileptogenic zone significantly [6-10].

In the current concise review, a list of presumably well-documented lateralizing and localizing findings in focal epilepsies is prepared (Tables 1-6). The purpose is to provide a complete and easy to use list of clinical findings, which are helpful in lateralization and localization of the epileptogenic zone in patients with focal epilepsy. This paper

is designed as a practical tool for physicians, aiming to serve as a practical, problem-oriented reference. While, I include the correlated symptomatogenic zone and the possible mechanism in generating the finding in the context of a focal seizure, this paper emphasizes how to localize the epileptogenic zone according to any given specific finding. These findings are organized in a series of tables (Tables 1-6). These tables are organized in a way, so that the reader can easily review the relevant information. I hope that this concise paper leads to improved patient care.

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| <i>Semiology</i> | <i>Lateralizing value (PPV)</i> | <i>Symptomatogenic zone</i> | <i>Mechanism</i> |
|--|---------------------------------|---|------------------------|
| Homonymous hemifield visual aura or defect | 100% | Brodmann areas 17-19 | Activation |
| Unilateral ictal paresis or immobile limb | 100% | Negative motor areas | Activation |
| Forced head version less than 10 second before secondary generalization | > 90% | Brodmann areas 6 & 8 (frontal eye and motor areas) | Activation |
| Unilateral ictal dystonia | > 90% | Spread from seizure onset zone to ipsilateral basal ganglia | Activation |
| Postictal (Todd's) palsy | > 90% | Brodmann areas 4 & 6 (primary motor area) | Exhaustion/ Inhibition |
| Fencing posture | 90% | SMA | Activation |
| Figure-of-4 sign (Asymmetric tonic limb posturing) | ~ 90% | SMA/ Prefrontal area | Activation |
| Unilateral tonic activity | ~ 90% | SMA/Brodmann area 6 | Activation |
| Unilateral sensory or painful aura | ~ 90% | Brodmann areas 1, 2, 3 (primary SSA) | Activation |
| Unilateral clonic activity | > 80% | Brodmann areas 4 & 6 (primary motor area) | Activation |
| Emotional facial asymmetry | > 80% | Unknown | Unknown |
| Epileptic nystagmus | N/A | Posterior head regions | Unknown |

PPV: Positive Predictive Value; SMA: Supplementary Motor Area; N/A: Not Assigned.

Table 1: Localizing semiologic findings pointing to the contralateral location (for the epileptogenic zone).

| <i>Semiology</i> | <i>Lateralizing value (PPV)</i> | <i>Symptomatogenic zone</i> | <i>Mechanism</i> |
|--|---------------------------------|---|---|
| Unilateral automatisms with contralateral dystonic posturing* | > 95% | N/A | Release phenomenon / Activation |
| Postictal nose wiping | > 90% | Unknown | Unknown |
| Unilateral ictal eye blinking | > 80% | Unknown | Unknown |
| Ictal piloerection (goose bumps)* | > 80% | Unknown | Unknown |
| Last clonic jerk | > 80% | Brodmann areas 4 & 6 (primary motor area) | ? Exhaustion of the hemisphere of onset |

*In TLE.

PPV: Positive Predictive Value; TLE: Temporal Lobe Epilepsy; N/A: Not Assigned.

Table 2: Localizing semiologic findings pointing to the ipsilateral location (for the epileptogenic zone).

| <i>Semiology</i> | <i>Lateralizing value (PPV)</i> | <i>Symptomatogenic zone</i> | <i>Mechanism</i> |
|---|---------------------------------|--|----------------------------------|
| Preserved consciousness and automatisms* | 100% | Unknown | Unknown |
| Ictal speech preservation* | > 80% | N/A | Impairment of non-language areas |
| Ictal vomiting* | > 80% | Temporal lobe and Papez circuit | Activation |
| Ictal spitting* | 75% | Unknown | Unknown |
| Ictal urinary urge | N/A | Mesial frontal region/ Medial temporal gyrus | Activation |
| Orgasmic auras | N/A | Mesiotemporal / frontal / amygdala | Activation |
| Peri-ictal water drinking* | N/A | Lateral hypothalamus | Activation |

*in TLE.

PPV: Positive Predictive Value; TLE: Temporal Lobe Epilepsy; N/A: Not Assigned.

Table 3: Lateralizing semiologic findings pointing to the non-dominant hemisphere.

| <i>Semiology</i> | <i>Lateralizing value (PPV)</i> | <i>Symptomatogenic zone</i> | <i>Mechanism</i> |
|-----------------------------|---------------------------------|-----------------------------|------------------------------|
| Ictal speech arrest* | 67% | language areas | Impairment of language areas |
| Postictal dysphasia | > 80% | language areas | Impairment of language areas |

*in TLE.

PPV: Positive Predictive Value; TLE: Temporal Lobe Epilepsy

Table 4: Lateralizing semiologic findings pointing to the dominant hemisphere.

| <i>Semiology</i> | <i>Symptomatogenic zone</i> | <i>Mechanism</i> |
|--------------------------------------|-----------------------------|------------------|
| Auditory auras | Superior temporal gyrus | Activation |
| Ictal vocalization | Broca's area | Activation |
| Postictal Cough | N/A | N/A |
| Gelastic Seizure | Hypothalamus | N/A |
| Olfactory and Gustatory auras | Temporal lobe structures | Activation |

N/A: not assigned.

Table 5: Non-lateralizing semiologic findings in focal epilepsies.

| <i>Finding</i> | Lateralizing value (PPV) | Mechanism |
|------------------------------|---------------------------------|------------------|
| Interictal EEG in TLE | 75% | Activation |
| Ictal EEG in TLE | 80-92% | Activation |
| Ictal EEG + Semiology in TLE | 95% | _____ |
| Interictal EEG in ETE | 50-66% | Activation |
| Ictal EEG in ETE | Variable | Activation |
| CT scan abnormality | Variable | Lesion |
| MRI abnormality | 86-100% | Lesion |
| Interictal PET abnormality | ~ 90% | Hypometabolism |
| Ictal SPECT abnormality | > 95% | Hyperperfusion |
| Interictal SPECT abnormality | > 80% | Hypoperfusion |
| Wada test in TLE** | 85% | Memory asymmetry |

*Depends on the type of EEG recording. **Only in TLE.

PPV: Positive Predictive Value; TLE: Temporal Lobe Epilepsy; ETE: Extratemporal Epilepsy; EEG: Electroencephalography; CT: Computed Tomography; MRI: Magnetic Resonance Imaging; PET: Positron Emission Tomography; SPECT: Single Photon Emission Computed Tomography

Table 6: Lateralizing and localizing paraclinical findings in focal epilepsies.

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