Seizures and Sleep, Sorting out the Spikes and Waves, a Polysomnographic and Clinical Review

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Topic: Seizures and Sleep

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AFFILIATION: MICHIGAN MEDICINE, UNIVERSITY OF MICHIGAN
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<table>
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<tr>
<th>Type of Potential Conflict</th>
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<td>Grant/Research Support</td>
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1. 
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Learning Objectives:

- Appreciate the bidirectional relationship between sleep and epilepsy.
- Review effects of sleep deprivation, sleep, and sleep stages on seizure occurrence.
- Identify epileptiform discharges and seizures during polysomnography and differentiate these from benign variants and common artifacts.
- Recognize commonly encountered sleep related epilepsy syndromes.
- Differentiate nocturnal seizures from primary sleep disorders.
- Understand the importance of treating comorbid sleep disorders in patients with epilepsy.
Seizures and Epilepsy

- Epileptiform seizure: Change in behavior caused by paroxysmal hyperexcitability of population of neurons.

- **Epilepsy is the tendency toward recurrent, unprovoked seizures.** (2 or more unprovoked seizures 24 hours a part or 1 unprovoked seizure and > 60% change of second). Epilepsy can resolve in many age dependent syndromes.

- **1.2% US population** had epilepsy 2015 (**3M adults, 500K children**) per CDC.

- Epilepsies can be **generalized or partial**.

- Interictal EEG findings (spikes or sharp waves)

- Ictal EEG findings (**heterogenous**: background attenuation, rhythmic waveforms, repetitive spike wave discharges)
Seizures and Sleep

- Gowers learned that 20% of epileptics experienced seizures solely during sleep (1885)
- Gibbs and Gibbs identified significant increase in epileptiform activity during sleep. (1960)
- Sleep and sleep deprivation are standard laboratory activating techniques for EEG recordings.
- Janz observed nocturnal predominance in 45% of patients with generalized tonic-clonic seizures. (1962)
- Some epilepsy syndromes (Sleep Related Epilepsies) have very close relationships to sleep cycle.

St. Louis EK Minerva Pneumol. 2011 Sep;50(3):159-176.
Why Seizures Occur During Sleep

- Not yet clear but the proposed hypothesis are:
  - NREM synchronization
    - Increased *interictal discharges (N3)*
    - Increased *ictal events (N1) and (N2)*
    - *Ictal and interictal discharges inhibited during REM*
  - Arousals and awakenings from sleep
  - Circadian factors and the sleep-wake cycle (Mesial temporal epilepsy daytime predominance nocturnal rats and humans)
  - Anatomic location (Frontal lobe seizures more likely to arise from sleep than temporal lobe seizures)

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History of EEG

Hans Berger, German psychiatrist
July 6, 1924 (first EEG)
“Berger wave”

Electrophysiology of EEG

- Cerebral cortex apical dendrite pyramidal neurons perpendicular to scalp produce **post synaptic potentials**: Summation of positive and negative electrical generators **not localized action potentials** – recorded by scalp EEG

- What we see on an EEG is determined by:
  - Area of synchronous activity ( > 6 cm²) per electrode
  - Voltage of discharge
  - Synchrony of discharge
  - Location of generator relative to cortical surface
Electrophysiology of EEG

- Summation potentials represented as dipole vector of energy parallel to those cells
  - Negative dipoles maximally sensed when perpendicular and going toward scalp
  - Need depth electrodes for positive dipoles
  - Fissure’s dipole could be tangential or parallel
- **EEG uses differential amplification**
  - Recording voltage differences between different points with pair of electrodes.
  - Active exploring and reference electrode
  - **If active exploring electrode more negative, upward deflection on EEG**
- Biologic filters and biologic generated electrical activity both impact what we see
Common montages

**Longitudinal bipolar montage.**
International 10-20 electrode placement.

**Ipsilateral ear referential montage.**
International 10-20 electrode placement.

Localization

Localization in right mesial temporal lobe epilepsy

(a) Channels 17 (FP2-F8) and 18 (F8-T8) show a "phase reversal" of negativity, allowing localization of the spike discharge as maximally negative at the F8 electrode site (i.e., given maximal negativity at F8 and the conventions of EEG polarity, which state that when the Grid 1 electrode site, FP2 in channel 17, is more positive than the Grid 2 site, F8, the result is a surface positive downward deflection; whereas in channel 18, F8 is more negative than the T8 Grid 2 electrode site, resulting in an upward deflection).

Localization in right mesial temporal lobe epilepsy

(b) Focal/regional slowing appears over the right temporal region, which has a rhythmic character consistent with the pattern known as temporal intermittent rhythmic delta activity (TIRDA), a frequent finding of epileptiform significance in those with temporal lobe epilepsy.

## EEG Frequency

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>Frequency</th>
<th>Normal examples</th>
<th>Abnormal examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha</td>
<td>8-13 Hz</td>
<td>Waking posterior rhythm in older children and adults, Mu rhythm</td>
<td>Alpha coma. Seizures activity in the alpha range.</td>
</tr>
<tr>
<td>Theta</td>
<td>4-7 Hz</td>
<td>Drowsiness, young children, temporal theta of elderly</td>
<td>Structural lesion, Encephalopathy</td>
</tr>
<tr>
<td>Delta</td>
<td>&lt; 4 Hz</td>
<td>Sleep, posterior slow wave of youth</td>
<td>Focal structural lesion, Encephalopathy</td>
</tr>
</tbody>
</table>
EEG Terminology

- **Rhythmic** - recurring waves of equal duration.
- **Monomorphic** - spike and waves with consistent shape & duration
- **Polymorphic** - spike and waves with unequal duration & shape
- **Transient** - wave(s) that stand out from background (can be normal)
- **Complex** - combination of 2 or more waveforms with characteristic morphology. (3 Hz spike and wave complex)
- **Periodic** - transients or complexes that recur at regular rates but with intervening activity in between them.
- **Symmetric** - occurring in two homologous regions opposite sides of head
- **Synchronous** - occurring in two distant or close (ipsilateral or contralateral) regions simultaneously.
## Epileptiform transients

<table>
<thead>
<tr>
<th>Transients</th>
<th>Duration</th>
<th>Variants</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spike</td>
<td>25-70 ms</td>
<td>Spike and wave complex; polyspike and wave complex</td>
</tr>
<tr>
<td>Sharp wave</td>
<td>70-200 ms</td>
<td>Sharp and wave complex; polysharp and wave complex</td>
</tr>
<tr>
<td>Sharply contoured slow wave</td>
<td>&gt;200 ms</td>
<td></td>
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</tbody>
</table>
EEG in a 24-year-old woman, showing a left temporal interictal epileptiform discharge during stage 2 sleep, in the form of a spike with maximal electronegativity at T1-T3.
3 Hertz Spike and Wave

Ictal EEG of medically intractable partial epilepsy

Interictal background immediately before the seizure, with frequent interictal left temporal spike discharges.

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Onset of a rhythmic theta-delta discharge in the right frontotemporal region (seventh second)

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Further spatio-temporal evolution and maximal seizure discharge over the right temporal region

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Eventual propagation more diffusely to the left hemisphere

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Benign EEG Variants

- BETS (Benign Epileptiform Transients of Sleep) or SSS (Small Sharp Spikes) or BSSS (Benign Sporadic Sleep Spikes)
- POSTS
- Fragmented Mu rhythm
- Wicket spikes
- Rhythmic mid temporal theta of drowsiness
- 14 & 6 positive burst pattern
BETS (Benign epileptiform transients of sleep)

- Small sharp spikes.
- In temporal or frontotemporal region during drowsiness or light sleep.
- Duration < 50 msec and amplitude < 50 µV.
- May have a subsequent slow wave, but do not occur in repetitive trains
- Tendency to shift from one hemisphere to another.
- **Usually do not persist in deeper sleep**
- 20% of adults
Benign Epileptiform Transients of Sleep

POSTS (Posterior occipital sharp transients of sleep)

- Seen most prominently at O1 and O2 in NREM sleep
- Usually occurs in trains but can occur in single waves.
- Differentiated from epileptiform sharp waves by the positive polarity, lack of an after going slow wave.
- Resembles Lambda waves (seen in wakefulness with visual scanning)
- Asymmetry is common and not an abnormality
Mu Rhythm

- Mostly seen in a waking state but a fragmented Mu can be seen in drowsiness.
- Negatively arched rhythm in alpha range. Seen mostly at C3 and C4. At times with beta activity
- Asymmetric and asynchronous
- Modulation by movement or contemplation of movement of contralateral arm
Mu Rhythm

Wicket Spikes

- Usually brief trains of sharply contoured (6-11 Hz) monophasic waves in the temporal region during drowsiness or light sleep in older adults.
- Arciform or wicket like, occasionally isolated wicket spikes.
- Can be unilateral or bilaterally asynchronous.
- Absence of a following slow wave and the normal EEG activity before and after the spikes differentiates them from epileptiform spikes.
Wicket Spikes

Rhythmic mid temporal theta of drowsiness

- **Psychomotor variant.** Similar in appearance to discharges of psychomotor (temporal lobe) seizures
- Trains of (5-15 second) sharply contoured notched waves in the theta range (5-7 Hz) in the temporal region.
- May be unilateral, bilateral, or independent and often starts on one side and shortly develops on the opposite side.
- **Only persistence in drowsiness,** no evolution, no after going slow wave and normal EEG background before and immediately after discharge
Rhythmic mid temporal theta of drowsiness

Hypnagogic Hypersynchrony

- Brief bursts of 100-300 microvolt monomorphic rhythmic waves at a frequency of 2.5 to 4.5 Hz.
- Occurs between ages 3 months and 13 years. Only 10% of children have this after age 12.
- Best developed over the central or centro-frontal region.
- May be misinterpreted as epileptic activity.
- But, occurs only in drowsiness or sleep onset in otherwise normal recording
Hypnagogic Hypersynchrony

14 and 6 per second positive burst

- Seen during drowsiness and sleep.
- 20-60% of population
- 1st appears around 3 years of age but maximal expression at 12 to 15 years of age for 14 Hz (6 Hz young infants and some adults)
- Looks similar to spindles but
  - Prominent spike positive phase
  - Expressed in the occipital or posterior temporal region and best seen when referenced to the ear.
14 and 6 per second positive burst

13-year-old girl. Note the 14-Hz positive spiky waveforms best appreciated over the posterior temporal and biposterior head regions, best seen in the third second.

6 Hz Phantom Spike and Wave

- Mitten-like morphology
- Small or absent spike component, more apparent slow wave.
- Adolescents and adults
- Drowsiness and light NREM sleep
- Diffuse or, alternatively, anteriorly or posteriorly predominant bursts.
Adolescent patient. There are bilaterally symmetrical diffuse tiny spikes with prominent wave components ("mitten-like" morphology) in seconds 3 through 6 below.
Epileptiform-like Artifacts
Eye Flutter

Evident in the frontopolar and anterior leads as a rhythmical jagged or arched appearance. Faster and lower amplitude than blink artifact. Blink artifact is diphasic bifrontal surface positive (downward deflection) caused by Bell’s phenomenon and positive cornea and negative retina dipole.

Rapid eye movements generate small spike-like discharges in the frontopolar derivations. EEG during rapid lateral eye movements can include single motor unit potential from contraction of lateral rectus muscle. **Lateral Rectus Spikes.**

Glossokinetic potentials from tongue movement, reproducible by having the patient say “la, la.” The tongue’s tip is electronegative compared to its base. The movement of the tongue toward or away from electrodes alters electrical field around them. Will include muscle artifact and can be quite rhythmic with nursing infant or tongue tremor.
Pulse artifact creates rhythmic slow wave artifact at F4. Note the frequency mirrors that of the cardiac cycle. Occurs due to movement of electrode from underlying vessel.

Electrostatic Artifacts. Dissimilar metals. (Talking)

Dissimilar metals (in fillings) causing electrostatic artifacts during talking generate spiky waveforms in seconds 5 through 8 of this epoch.

Electrode “pop” artifact at P7 simulates rhythmic seizure activity. Caused by sudden change of electrode contact with scalp. Partly detached electrode, drying of conductive paste or jelly.
### The Common Sleep-Related Epilepsies

<table>
<thead>
<tr>
<th>Epilepsy Syndrome</th>
<th>Classification</th>
<th>Age of Onset</th>
<th>Waking Seizures</th>
<th>Seizures Types</th>
<th>EEG Findings</th>
<th>Imaging Findings</th>
<th>Treatment Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benign Rolandic Epilepsy</td>
<td>Partial</td>
<td>Children</td>
<td>Yes</td>
<td>SP, CP, GTC</td>
<td>Centrence-temporal (Rolandoic) Spikes</td>
<td>Normal</td>
<td>CBZ, OXC, GBP</td>
</tr>
<tr>
<td>Landau Kleffner Syndrome</td>
<td>Unknown</td>
<td>Children</td>
<td>Yes</td>
<td>SP, CP, At Ab, Ast, GTC</td>
<td>Continuous Spike-wave in SWS</td>
<td>Normal, less commonly symptomatic lesion's cause</td>
<td>CBZ, OXC, etc.</td>
</tr>
<tr>
<td>Lennox Gastaut Syndrome</td>
<td>Symptomatic Generalized</td>
<td>Children</td>
<td>Yes</td>
<td>AtAb, T Ast, M, GTC</td>
<td>Slow Spike and Wave</td>
<td>Normal or symptomatic lesion(s)</td>
<td>Steroid/IVig, MSPT</td>
</tr>
<tr>
<td>Juvenile Myoclonic Epilepsy</td>
<td>Primary Generalized</td>
<td>Adolescence</td>
<td>Yes (typically a.m. hours)</td>
<td>A, M, GTC</td>
<td>4–6 hr Gen. S/W</td>
<td>Normal</td>
<td>VPA, LTG, TPM, FBM, etc.</td>
</tr>
<tr>
<td>Autosomal Dominant Nocturnal</td>
<td>Partial</td>
<td>Children or Adults</td>
<td>Possible, but usually not</td>
<td>SP, CP, GTC</td>
<td>Normal, or Frontal Spikes</td>
<td>Normal</td>
<td>CBZ, OXC, etc.</td>
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<tr>
<td>Frontal Lobe Epilepsy</td>
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<td>VNS</td>
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<tr>
<td>Nocturnal Temporal Lobe</td>
<td>Partial</td>
<td>Adults</td>
<td>Possible, but usually not</td>
<td>SP, CP, GTC</td>
<td>Temporal Spikes</td>
<td>Normal</td>
<td>CBZ, OXC, etc.</td>
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<tr>
<td>Epilepsy</td>
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<td>Epilepsy Surgery</td>
</tr>
</tbody>
</table>
Benign epilepsy of childhood with centrotemporal spikes (BECTS) or Benign Rolandic Epilepsy (BRE)

- **Mainly simple partial seizures**, hypersalivation, hemifacial focal motor clonic (at times with arm), speech arrest, dysphagia, gurgling sounds, or facial paresthesias **but at times secondary generalized tonic-clonic seizure activity (50%)**
- 25% FH of epilepsy. EEG trait appears autosomal dominant
- Mean age of onset seven, ranging from 3 to 13 years, remission often by mid-teens
- Males affected more commonly.
- Seizures confined to sleep in 75%
- Often no treatment needed but occasionally AED treatment necessary if seizures become frequent enough to disrupt sleep in patient.
- EEG spike-wave discharges over the ipsilateral, centrotemporal region, although discharges may occur contralaterally or bilaterally, independently. Discharges increase in frequency and complexity during sleep often occurring in repetitive bursts.
BECTS

Landau Kleffner Syndrome

- **Subacute progressive language regression** after normal language development. Verbal agnosia, expressive aphasia, hyperkinetic behavior
- 70-85% obvious seizures (simple partial, complex partial, atypical absence)
- Presentation children 2–10 years of age.
- EEG: B/L Centrottemporal spike and waves that generalize to both hemispheres during sleep and become nearly continuous.
- **Electrical status epilepticus during sleep (ESES).** 50% or 85% of NREM sleep with **continuous epileptiform activity**
- Likely exists on **continuum** with epileptic encephalopathy with continuous spikes and waves during sleep CSWS
- Treatment is with AEDs, courses of immunosuppressive therapy, or in dire medically refractory cases, epilepsy surgery utilizing multiple subpial transections in the neocortical language regions.
Landau Kleffner Syndrome

FIGURE 1  EEG in 5 year old girl with LKS. Note awake/drowsy background (left), activation of epileptiform activity in Stage 1 sleep (middle), and ESES in NREM sleep (right). Courtesy of William D. Gaillard, M.D.
Lennox Gastaut Syndrome

- Presentation most commonly 3-5 years
- **Multiple primary generalized seizure types**, including prominent nocturnal tonic, astatic/atonic, atypical absence, myoclonic, and generalized tonic-clonic with accompanying mental retardation
- EEG: Slow generalized spike and wave complexes at 1.5–2.5 Hz (atypical spike and wave), multifocal epileptiform abnormalities, slow polyspike wave, paradoxical fast activity, generalized background slowing, electrographic features activated in sleep
- Preceded by a history of infantile spasms with hypsarrhythmic EEG findings (West syndrome) in 25%.
- Syndrome has many causes (40% idiopathic)
- Seizures often refractory to treatment
Lennox Gastaut Syndrome

Figure 71. Tonic seizure. Recorded generalized tonic seizures leading to drop attacks (each recorded event marked by star). Patient stood up in a harness system in order to safely record typical events. Onset of each event in this patient is correlated with occurrence of a high-amplitude generalized slow complex (marked by filled arrow), followed by an electrodecremental response (marked by open arrow)

Juvenile Myoclonic Epilepsy

- Idiopathic primary generalized epilepsy syndrome with **myoclonic**, **absence**, and **generalized tonic-clonic seizures**, often occurring shortly after awakening but also during sleep or daytime.
- Onset mid-adolescence.
- Interictal EEG: 4 to 6 Hz B/L polyspike and wave discharges. **Abnormal 75%** of waking patient but **100%** during **sleep**.
- Ictal EEG: **Myoclonus**: 3-4.5 Hz polyspike wave frontocentral predominance **GTCS**. Attenuation with low voltage fast activity, spike waves variable frequency and amplitude **Absence**. Generalized spike waves slightly faster frequency than 4 Hz.
- Responds well to lifelong AED therapy with compliance and avoiding sleep deprivation and alcohol.
Juvenile Myoclonic Epilepsy

Myoclonic seizure in a patient with JME. The patient had a generalized axial myoclonic jerk during second 14, which coincided with the generalized spike-wave discharge and electrodecremental pattern seen on EEG.

Sleep Related Hypermotor Epilepsy (previously Nocturnal Frontal Lobe Epilepsy)

- Seizures **linked to sleep and not time of day** (not chronobiological).
- Seizures may arise in **extrafrontal regions** (epileptogenic zone) such as temporal lobe and activate the frontal region (symptomatic zone).
- **Hypermotor best characterizes seizure semiology** of syndrome (not fencer posturing or akinetic seizures with other frontal lobe seizure syndromes). May include dystonia (paroxysmal nocturnal dystonia) or sleepwalking (episodic nocturnal wandering).
- Causes: Genetic: 25% FH epilepsy but minority autosomal dominant NFLE (**nicotinic Ach receptor**) and 33% FH parasomnias, Structural, Idiopathic.
- **Ictal and interictal EEG may be normal 50%** or show frontal spikes.
### Diagnostic criteria of sleep-related hypermotor epilepsy*

<table>
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<th>Diagnosis based on clinical history</th>
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<tbody>
<tr>
<td>• Brief (&lt;2 minutes) seizures with stereotyped motor pattern, abrupt onset and offset, may cluster</td>
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<tr>
<td>• Most common motor activity is hypermotor: vigorous hyperkinetic movements, and/or asymmetric tonic or dystonic posturing, with or without impaired awareness</td>
</tr>
<tr>
<td>• Occurrence predominantly during sleep</td>
</tr>
<tr>
<td>• Diagnosis not excluded by intellectual impairment, neuropsychiatric features, absence of interictal and ictal EEG correlates, extrafrontal origin</td>
</tr>
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### Three levels of certainty

<table>
<thead>
<tr>
<th>Witnessed (possible)</th>
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<tbody>
<tr>
<td>• Clinical features provided by observer</td>
</tr>
<tr>
<td>Video-documented (clinical)</td>
</tr>
<tr>
<td>• At least one stereotyped event, confirmed by observer to be typical</td>
</tr>
<tr>
<td>• High quality audio-video including the onset and offset with clear visualization of the entire event</td>
</tr>
<tr>
<td>• Minor motor events or paroxysmal arousals excluded</td>
</tr>
<tr>
<td>Video-EEG documented (confirmed)</td>
</tr>
<tr>
<td>• At least one stereotyped event during daytime sleep recording after sleep deprivation, or during full night sleep recording using ≥19 EEG channels, ECG, oculogram, and chin EMG</td>
</tr>
<tr>
<td>• Definitive ictal epileptic discharge or interictal epileptiform abnormality</td>
</tr>
</tbody>
</table>

* Previously referred to as nocturnal frontal lobe epilepsy.

EEG: electroencephalography; ECG: electrocardiography; EMG: electromyography.
**RBD**
- Associated with Neurodegeneration
  - Late onset
  - No family history
  - Last 1/3^{rd} of night
  - Memory of Dream

**Nightmare**
- Tends to disappear
- Last 1/3^{rd} of night
- Mild autonomic Activation
  - Memory of dream

**NFLE**
1. Any age
2. Anytime @ night
3. Several per night
4. Brief duration
5. Stereotyped
6. Strong autonomic activation

**Disorders of Arousal**
- Tends to disappear
- Trigger factors
- First 1/3^{rd} of night
- Usual amnesia

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*Movement disorders in sleep: Guidelines for differentiating epileptic from non-epileptic motor phenomena arising from sleep*

Hypersynchronous delta activity accompanying a confusional arousal in a 34 year-old woman.

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REM Behavior Disorder

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Sleep Architecture in Epilepsy

- Epilepsy patients found to have significantly reduced REM and stage 2 and 4 sleep, and objective EDS on maintenance of wakefulness tests. Patients with nocturnal seizures show reduced sleep efficiency, increased time to first REM period, and increased drowsiness on the MWT.

- Most older AEDs reduce slow wave and REM sleep, decrease sleep latency, and increase Stage 1 and 2 NREM sleep percentage.

- Carbamazepine transiently reduces REM sleep at initiation but otherwise has little effect on sleep architecture.

- Lamotrigine reduces slow wave sleep, increases stage 2 sleep, but is associated with reduced stage shifts and arousals as well as an increase in REM percentage without subjective insomnia.

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Comorbid Sleep Disorders in Epilepsy

- EDS in Epilepsy: Consider: Nocturnal seizures, sedating meds, inadequate sleep hygiene, primary sleep disorders.
- Untreated primary sleep disorder could lead to increased seizure frequency.
- Malow et al noted epilepsy patients sleepier than non-epilepsy neurology patients but not associated with number and type of AEDs, seizure frequency, epilepsy syndrome, or nocturnal seizures.

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OSA and Epilepsy

- Sleep disordered breathing may worsen seizure control 1/3 of intractable epilepsy patients, especially in seniors.
- OSA most common cause of sleep disordered breathing in epilepsy.
- Prospective and retrospective studies show benefit of PAP therapy in reduction of seizures in refractory epilepsy.
- Benefit may be equal to adding one AED.
- One 2011 study showed 50-60% seizure reduction in epilepsy OSA patients treated with PAP therapy.
- Whether AEDs have any specific or independent effects on sleep-disordered breathing needs further study.

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Conclusions:

- Sleep and Epilepsy have a bidirectional relationship.
- Careful EEG review can allow for recognition and differentiation of normal EEG variants and artifacts from epileptiform findings.
- NREM sleep may activate spikes and seizures. Sleep deprivation may increase seizure occurrence.
- Some epilepsy syndromes occur almost exclusively during sleep or immediately following awakening.
- Parasomnias may mimic epileptic seizures though they can be distinguished with clinical and or polysomnographic characteristics.
- Comorbid sleep disorders and especially OSA may worsen seizure burden in epilepsy, and seizure control may be improved with OSA treatment.