



IN THE NAME OF GOD



Savadi Oskouee MD
Associate professor of neurology

**Tabriz University of Medical
Sciences**

Sleep in Epilepsy

Epidemiology of Epilepsy and Comorbidities in Sleep Disorders

About 1 % to 2% of the population has epilepsy, and 1 out of 10 people will have a seizure during their lifetime.

Approximately 20% of patients with epilepsy will have seizures solely while asleep .



Nocturnal seizures should be distinguished from other nocturnal events such as parasomnias .

The most difficult problem is to distinguish between frontal lobe seizures and parasomnias .

Sleep disorders such as sleep deprivation and sleep apnea may exacerbate seizures .



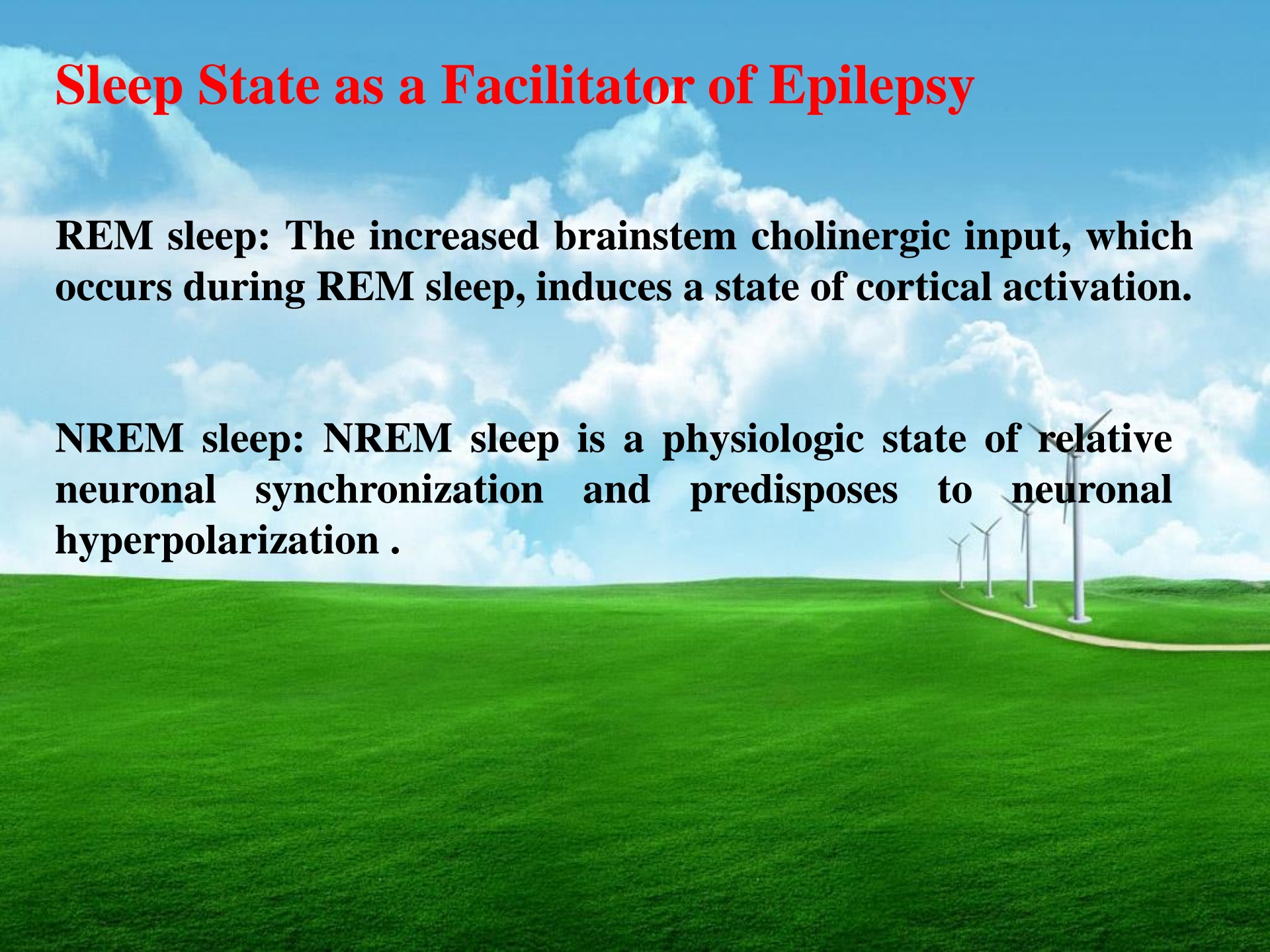
Conversely, seizures may affect sleep, as many of the current anti epileptic drugs produce undesirable side effects to sleep architecture and may predispose the patient to primary sleep disorders.



Sleep State as a Facilitator of Epilepsy

REM sleep: The increased brainstem cholinergic input, which occurs during REM sleep, induces a state of cortical activation.

NREM sleep: NREM sleep is a physiologic state of relative neuronal synchronization and predisposes to neuronal hyperpolarization .





“Seizure Promotor”

“Seizure Protector”

NREM Sleep:

Synchrony of thalamocortical synaptic activity

Greater tendency for propagation of epileptiform discharges

REM Sleep:

Increased cholinergic tone

Desynchronized neuronal discharge patterns

Relative resistance to propagation of EEG potentials

Skeletal muscle paralysis

SYNCHRONIZED SLEEP

Excessive diffuse cortical synchronization.

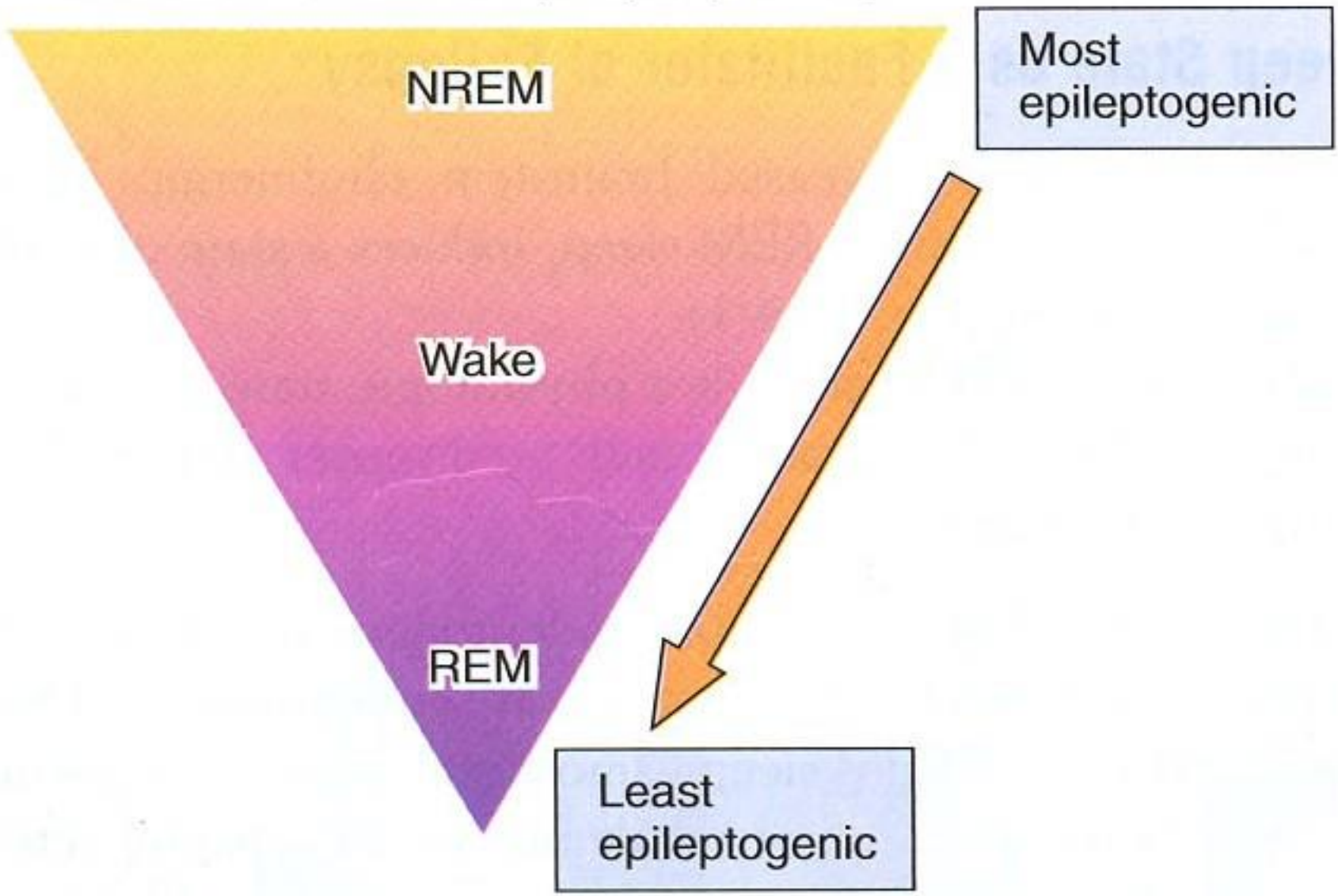
Enhancement in interhemispheric impulse traffic.

DESYNCHRONIZED SLEEP

Inhibition of thalamocortical synchronization.

Tonic reduction in interhemispheric impulse traffic.

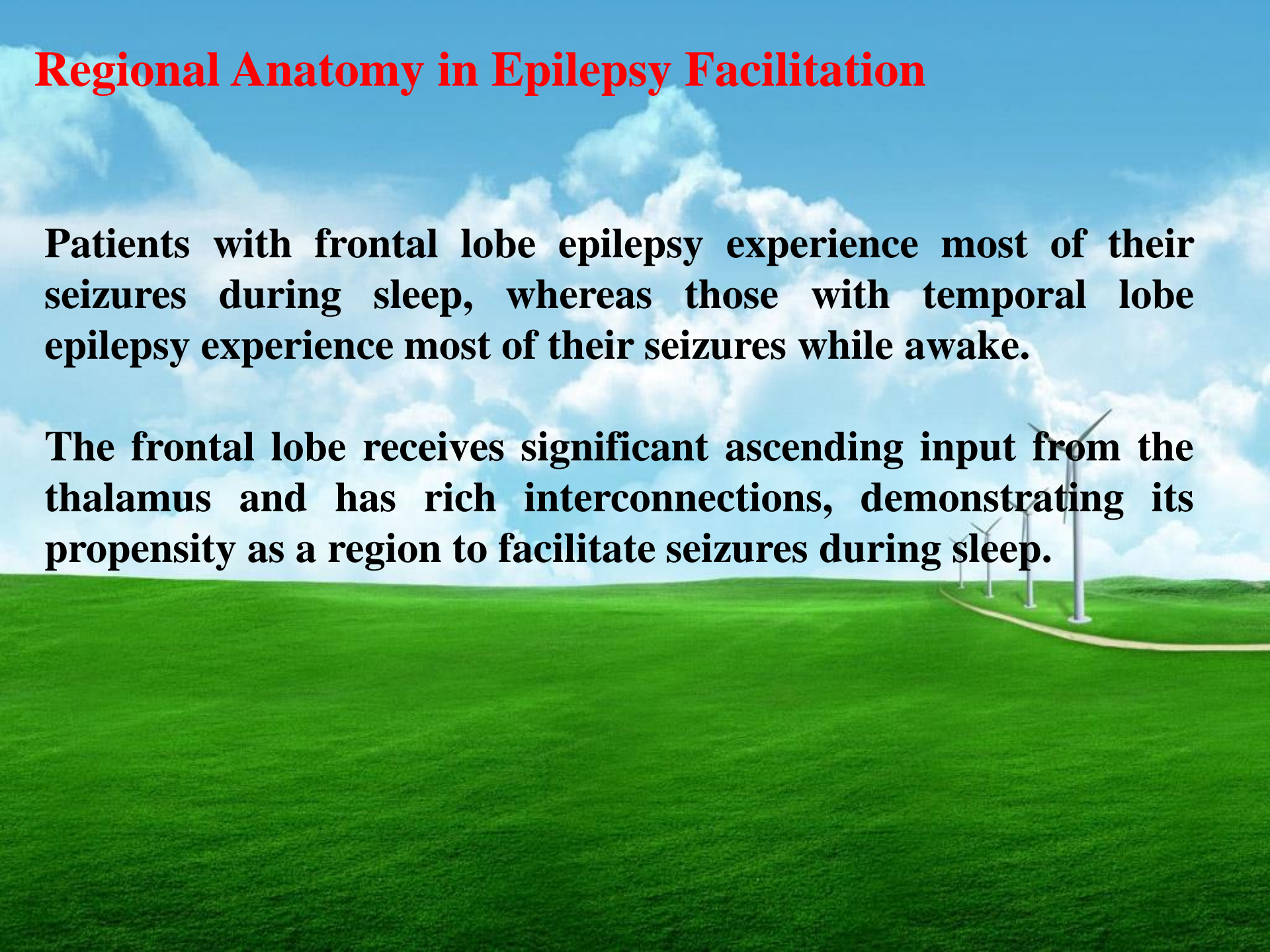
Seizures vs. State Epileptogenicity



Regional Anatomy in Epilepsy Facilitation

Patients with frontal lobe epilepsy experience most of their seizures during sleep, whereas those with temporal lobe epilepsy experience most of their seizures while awake.

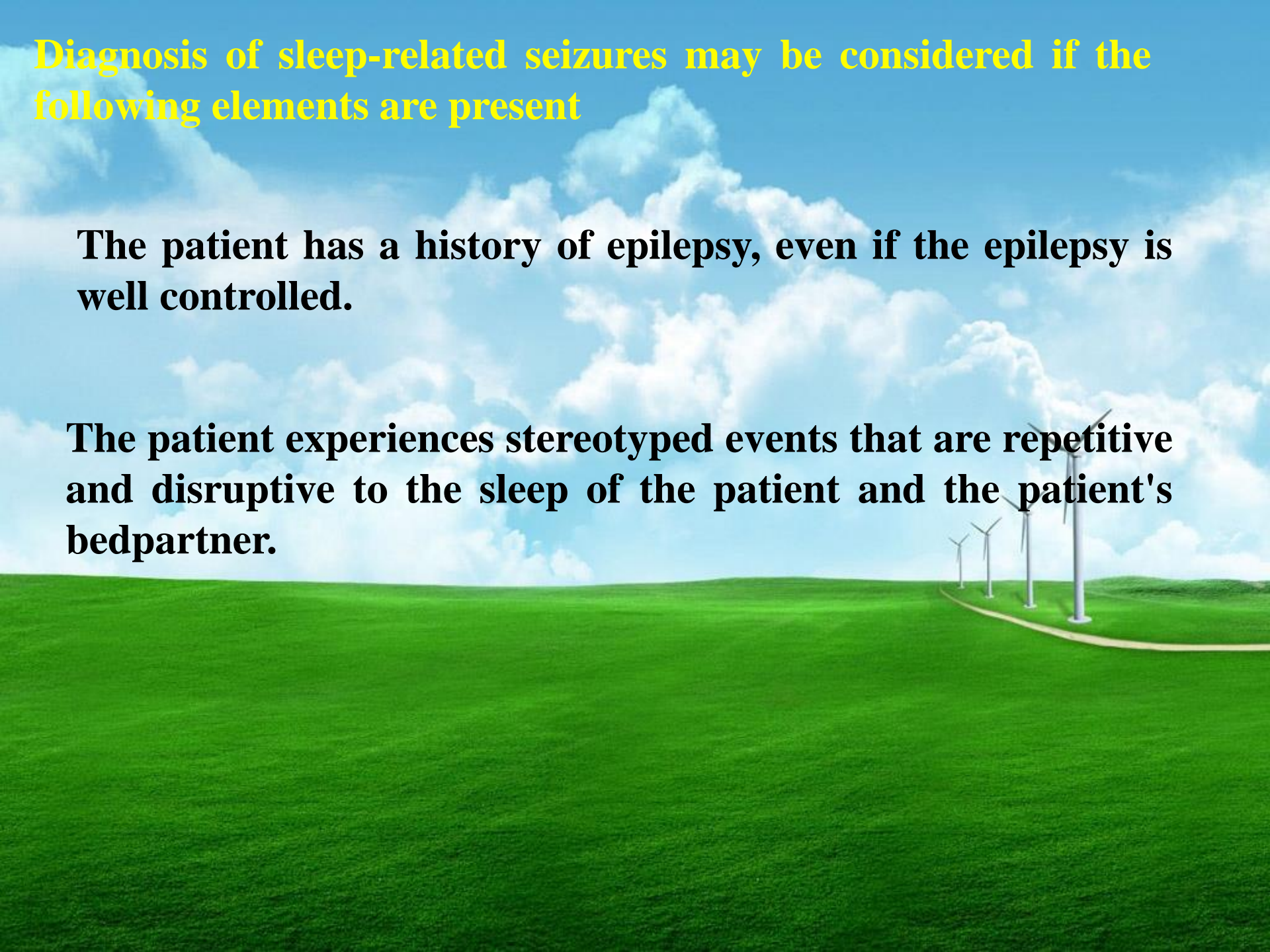
The frontal lobe receives significant ascending input from the thalamus and has rich interconnections, demonstrating its propensity as a region to facilitate seizures during sleep.



Diagnosis of sleep-related seizures may be considered if the following elements are present

The patient has a history of epilepsy, even if the epilepsy is well controlled.

The patient experiences stereotyped events that are repetitive and disruptive to the sleep of the patient and the patient's bedpartner.



Episodes occur at any time of night (more commonly during NREM sleep than during REM sleep).

Similar events may occur during the day.

A trial of antiepileptic drugs produces a favorable response .



Nocturnal Epilepsy

It has long been known that seizures, both convulsive and nonconvulsive, often occur during sleep, especially in children.

Seizures may occur soon after the onset of sleep or at any time during the night, but mainly in stages 1 and 2 of NREM sleep or, rarely, in REM sleep.

They are also common during the first hour after awakening.

Deprivation of sleep may be conducive to a seizure.

Sleeping epileptic patients may attract attention to their seizures by a cry, violent motor activity, unusual but stereotyped actions, such as sitting up and crossing the arms over the chest, the adoption of a "fencing" posture, or labored breathing.

If the nocturnal seizure is unobserved, the only indication of it may be disheveled bedclothes, a few drops of blood on the pillow from a bitten tongue, wet bed linen from urinary incontinence, or sore muscle.

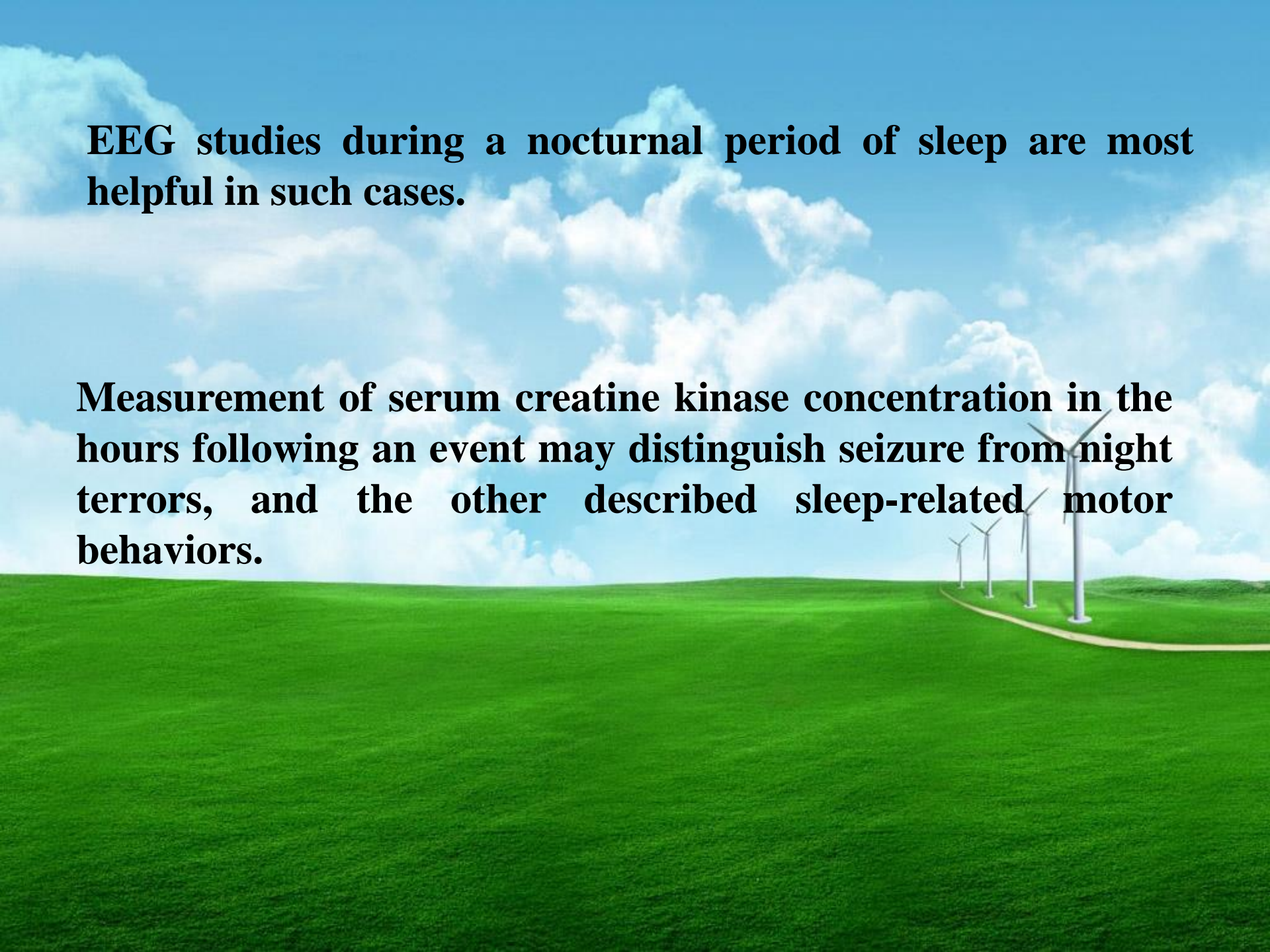
Rarely, a patient may die in an epileptic seizure during sleep, sometimes from smothering in the bed clothes or aspirating vomitus or for some obscure reason (possibly respiratory or cardiac dysrhythmia).

Epilepsy occasionally occurs in conjunction with night terrors and somnambulism; the question then arises whether the latter disorders represent postepileptic automatisms.

Usually no such relationship is established.

EEG studies during a nocturnal period of sleep are most helpful in such cases.

Measurement of serum creatine kinase concentration in the hours following an event may distinguish seizure from night terrors, and the other described sleep-related motor behaviors.



Nocturnal Frontal Lobe Epilepsy

One isolated seizure disorder is characterized by paroxysmal bursts of generalized choreoathetotic, ballistic, and dystonic movements occurring during NREM sleep.

Sometimes the patient appears awake and has a fearful or astonished expression, or there are repetitive utterances and an appearance of distress, similar to what is seen in night terrors.



The attacks may begin at any age, affect both sexes, and are usually nonfamilial.

Two forms of this disorder have been recognized: in one, the attacks last 60 s or less; they may be diurnal as well as nocturnal; some patients in addition have epileptic seizures of the more usual type.

All respond to treatment with carbamazepine.

The studies of Tinuper and coworkers, using prolonged video-EEG monitoring, indicate that these brief attacks of nocturnal paroxysmal dystonia may actually be epileptic seizures of frontal lobe origin.



In a second and more rare type, the attacks are longer lasting (2 to 40 min).

Ictal and interictal EEGs during wakefulness and sleep are normal.

And these attacks do not respond to anticonvulsants of any type. Except for the lack of familial incidence and occurrence only during sleep, the disorder is very much the same as the "familial paroxysmal dystonic choreoathetosis" described by Lance.



Table 10.3-1. Characteristics of Nocturnal Frontal Lobe Epilepsy (NFLE) vs. Parasomnias

CLINICAL FEATURE	NFLE	PARASOMNIAS
Age at onset (years)	11.8 ± 6.3	Usually <10
Attacks/month (n)	36 ± 12	1–4
Clinical course	Increasing or stable	Decreasing/ disappearing
Movement semiology	Stereotypic	Polymorphic
Attack onset	Any time during the night	First third of the night
Attack distribution	2-NREM (65%)	3-4 NREM
Motor pattern	2-3 repetitive types of attacks	Absence of motor pattern
Duration of the attacks	Less than 1 min (excl. prolonged episodes)	Some minutes

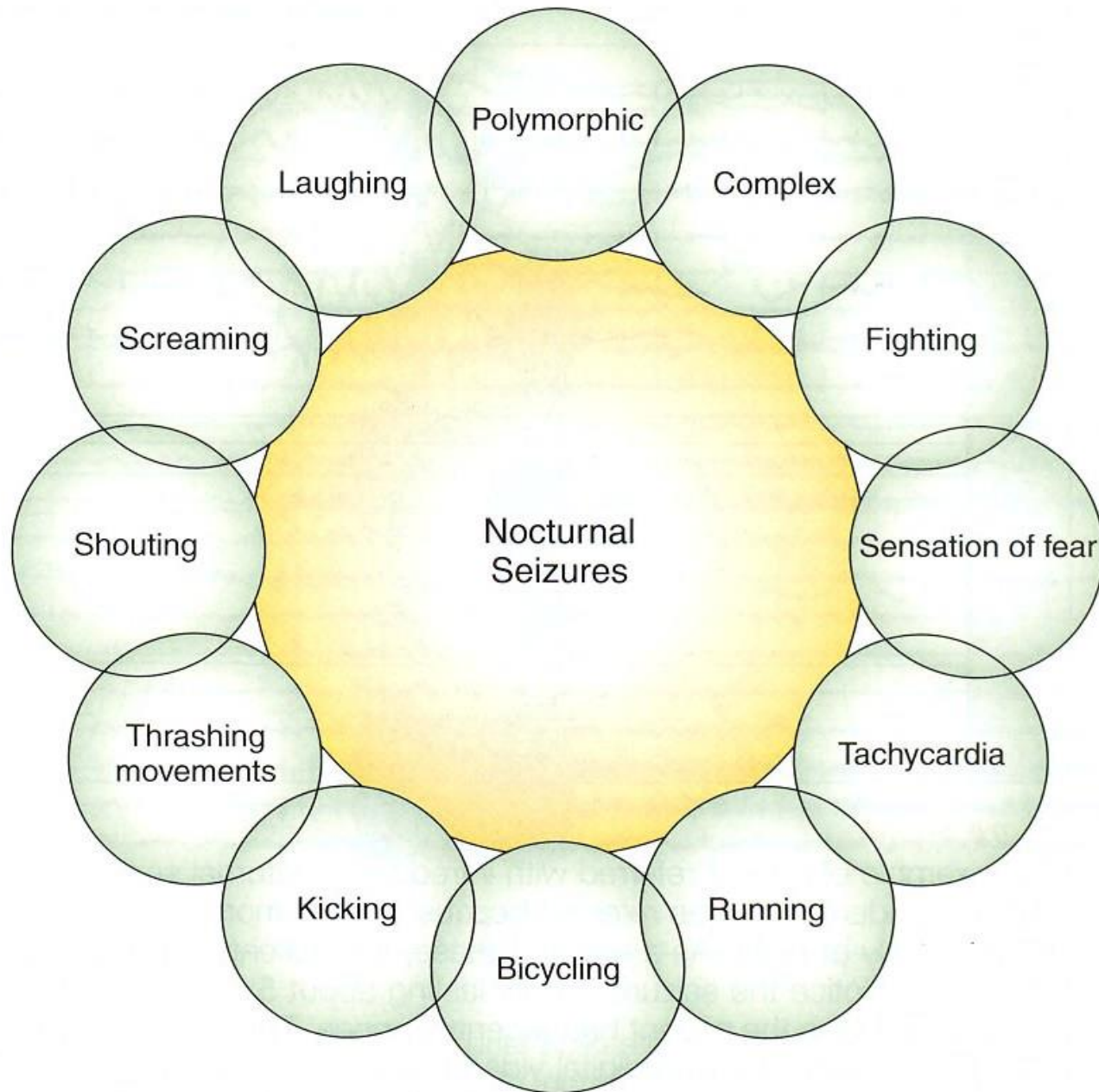
Table 10.3-2. Effects of Antiepileptic Drugs on Sleep

DRUG	EDS	INSOMNIA	SL	REM	SLOW WAVE	AWAKENINGS	OTHER
Gabapentin	++			▲		▼	▲Weight
Lamotrigine		+		▲	▲	▲	
Levetiracetam	+						
Oxycarbazepine	+						
Tiagabine	+++				▲		
Topiramate	++						▼Weight
Zonisamide	+	+					
Carbamazepine	+			▼			▲PLMs
Phenytoin	+	+	▼			▲	
Valproate	+		▲				▲Weight
Phenobarbital	+++					▼	▲OSA
Ethosuximide		+			▼		
Felbamate		++					▲Weight

Key: + Mild effect, ++ Moderate effect, +++ Severe effect, ▼ Decrease/Reduce, ▲ Increase

EDS, excessive daytime sleepiness; SL, sleep latency.

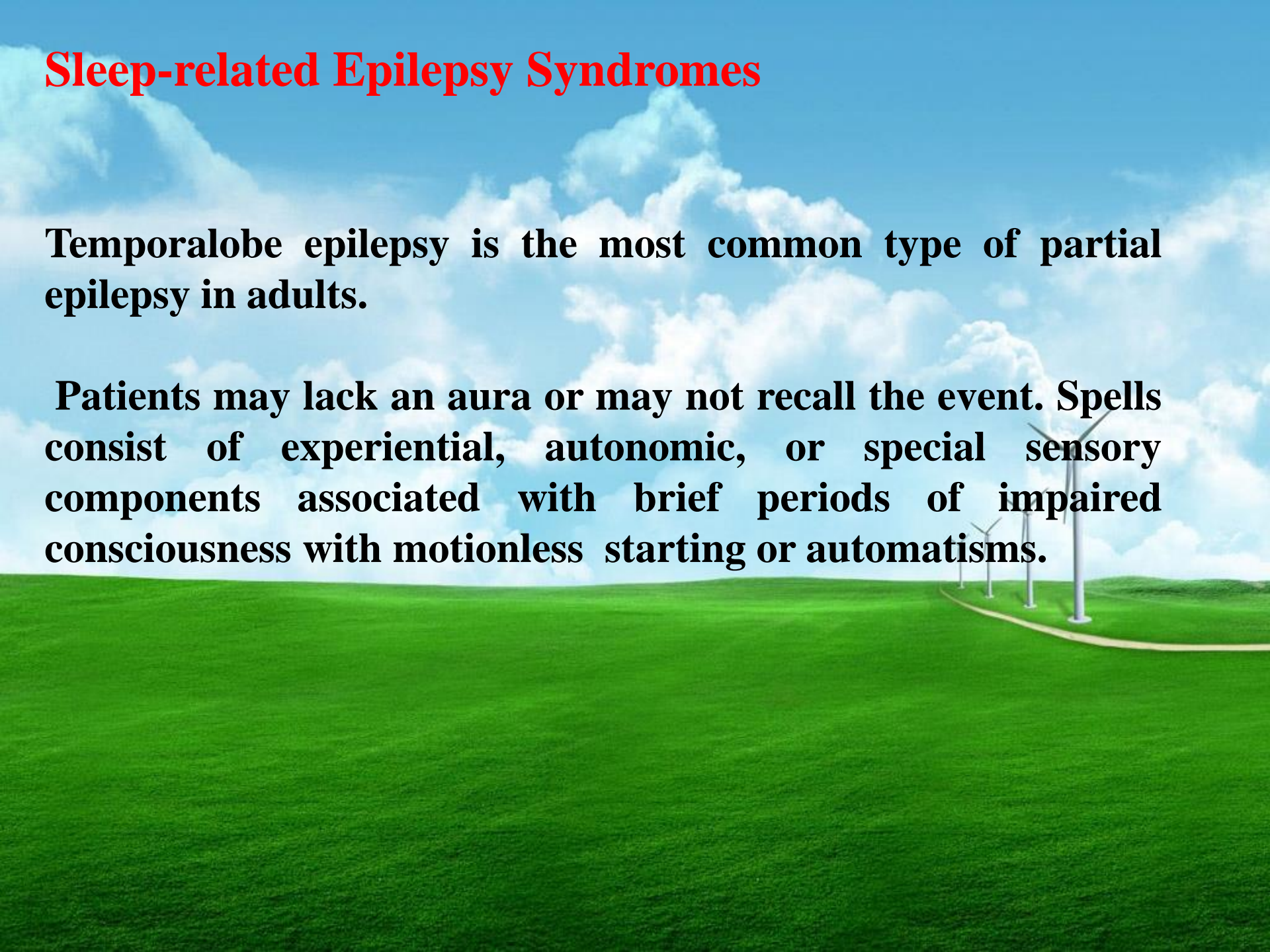
Antiepileptic drugs have variable effects on sleep variables.



Sleep-related Epilepsy Syndromes

Temporallobe epilepsy is the most common type of partial epilepsy in adults.

Patients may lack an aura or may not recall the event. Spells consist of experiential, autonomic, or special sensory components associated with brief periods of impaired consciousness with motionless staring or automatisms.



BENIGN EPILEPSY OF CHILDHOOD WITH CENTROTEMPORAL SPIKES

Benign epilepsy with centrotemporal, or Rolandic, spikes is the most common form of partial epilepsy in children .

In 70% to 80% of cases, seizures are confined to sleep.

Spells are characterized by paresthesias in one half of the face, sometimes involving the tongue and lips, followed by clonic jerks involving the face, tongue, lips, larynx, and pharynx.



The electroencephalogram (EEG) reveals characteristic high-amplitude interictal spikes followed by slow waves in the mid-temporal and central spikes, or sharp waves particularly during sleep.



NOCTURNAL PAROXYSMAL DYSTONIA

Nocturnal paroxysmal dystonia is a form of frontal lobe epilepsy that consists of a sudden arousal associated with a complex sequence of movements, repeated dystopia, or a dyskinetic (ballistic or choreoathetotic) pattern .

Patients may also move their legs and arms with cycling or kicking movements.

Consciousness is often preserved.

Carbamazepine is the agent of choice and provides an excellent response.

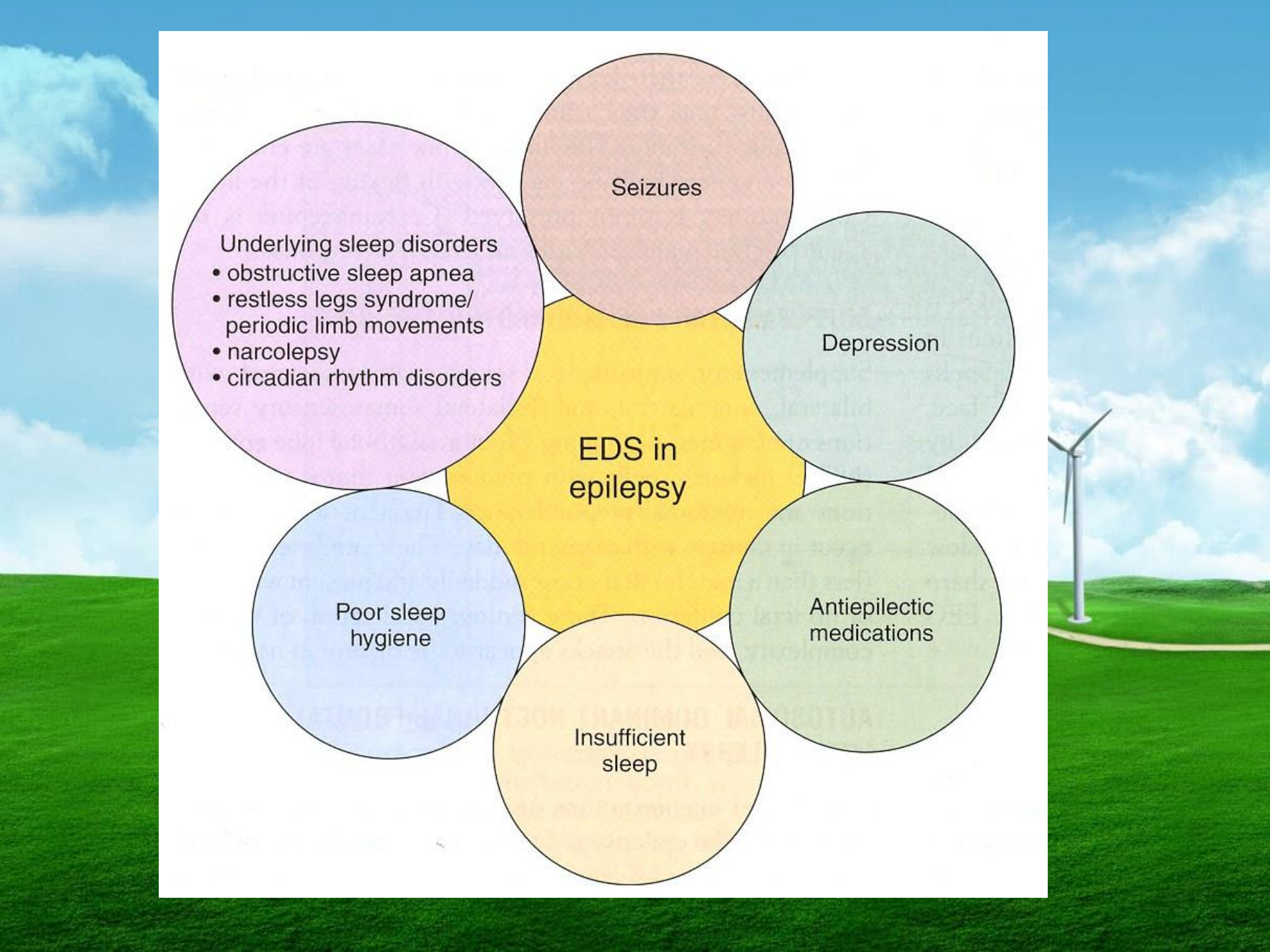
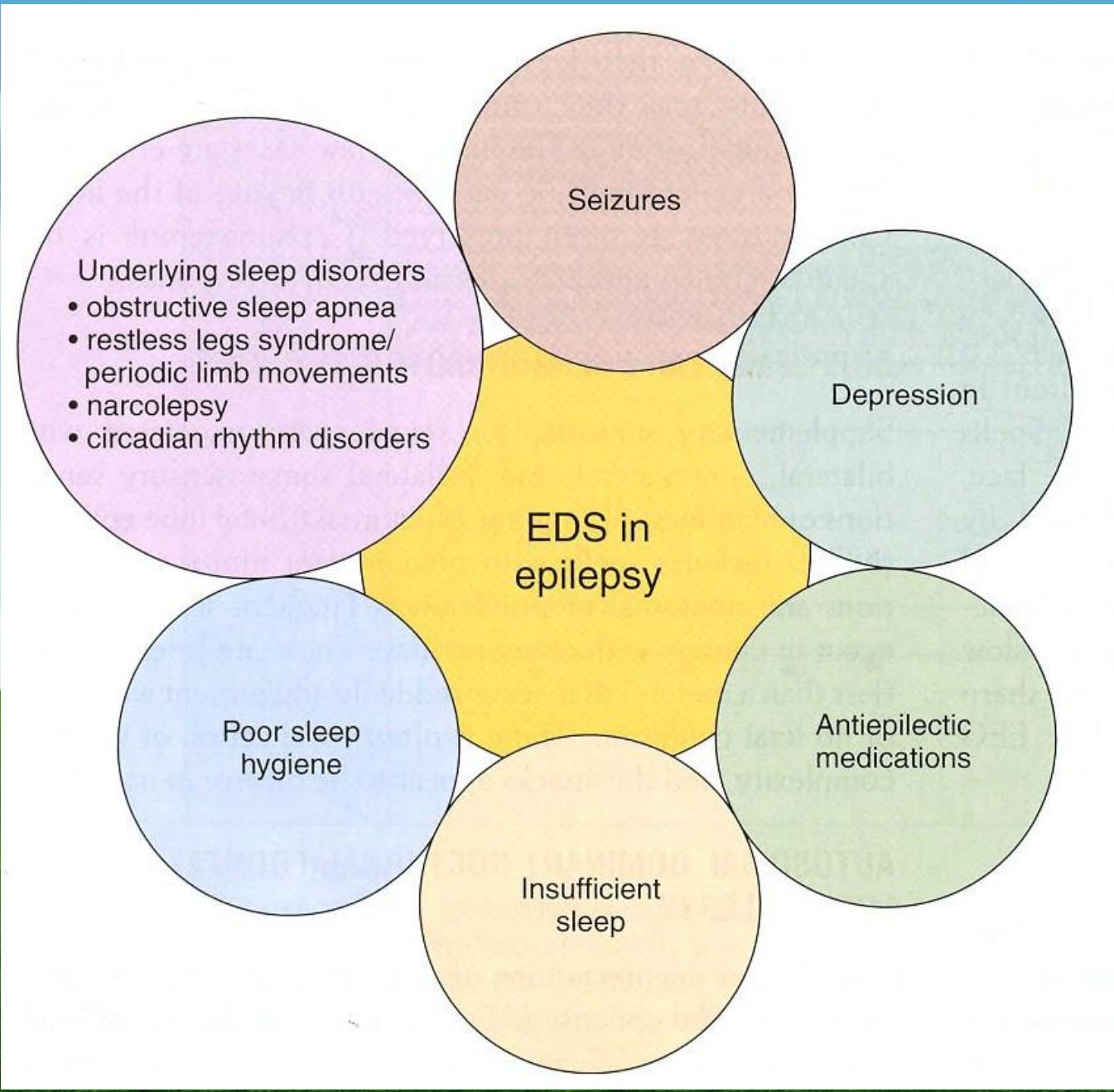
Obstructive Sleep Apnea and Epilepsy

Sleep apnea may coexist with epilepsy.

Several potential mechanisms of action have been proposed as to the etiology of seizure facilitation in obstructive sleep apnea (OSA).

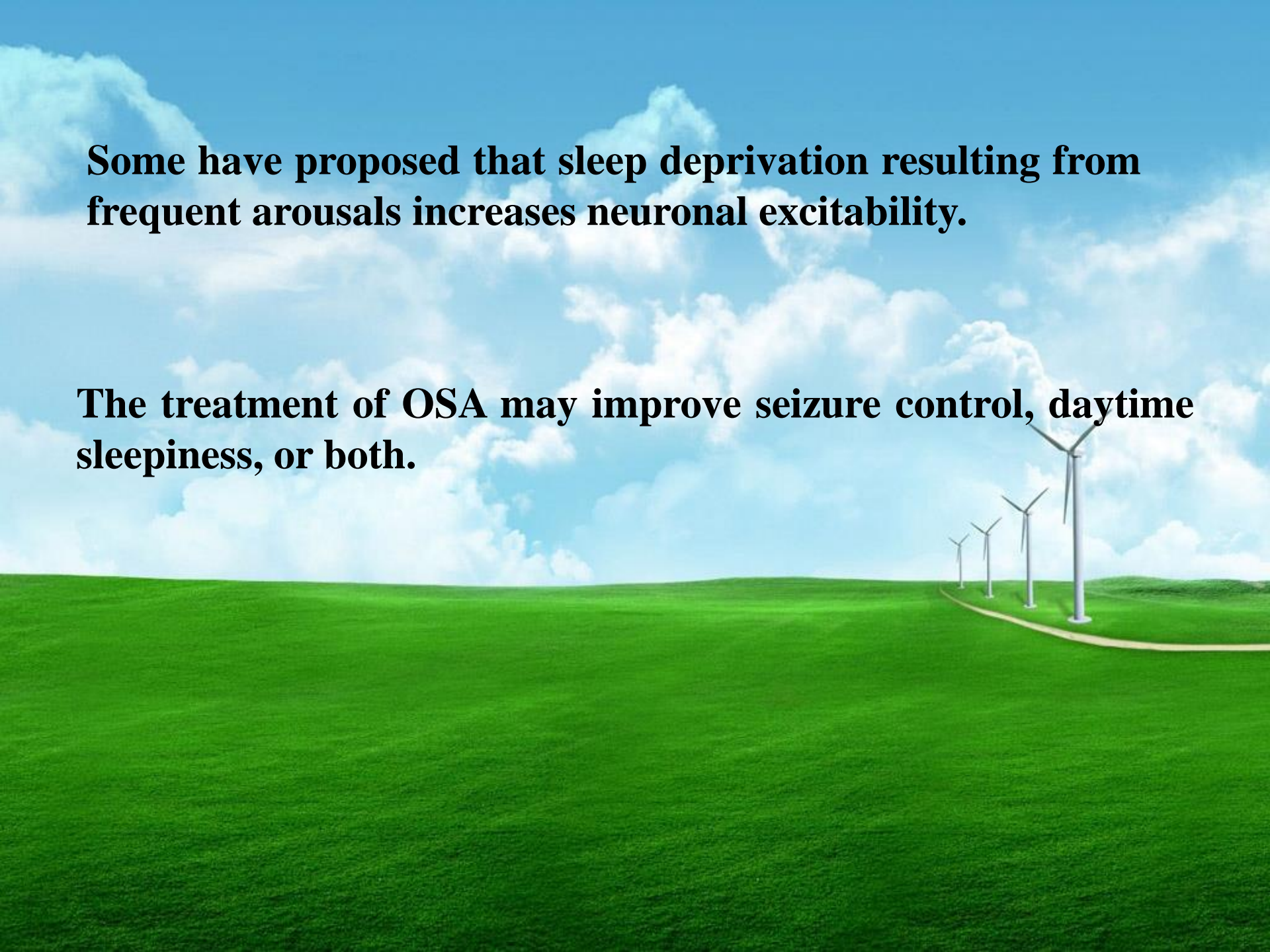
Prolonged asystole, which may occur in OSA, may be associated with seizures.





Some have proposed that sleep deprivation resulting from frequent arousals increases neuronal excitability.

The treatment of OSA may improve seizure control, daytime sleepiness, or both.



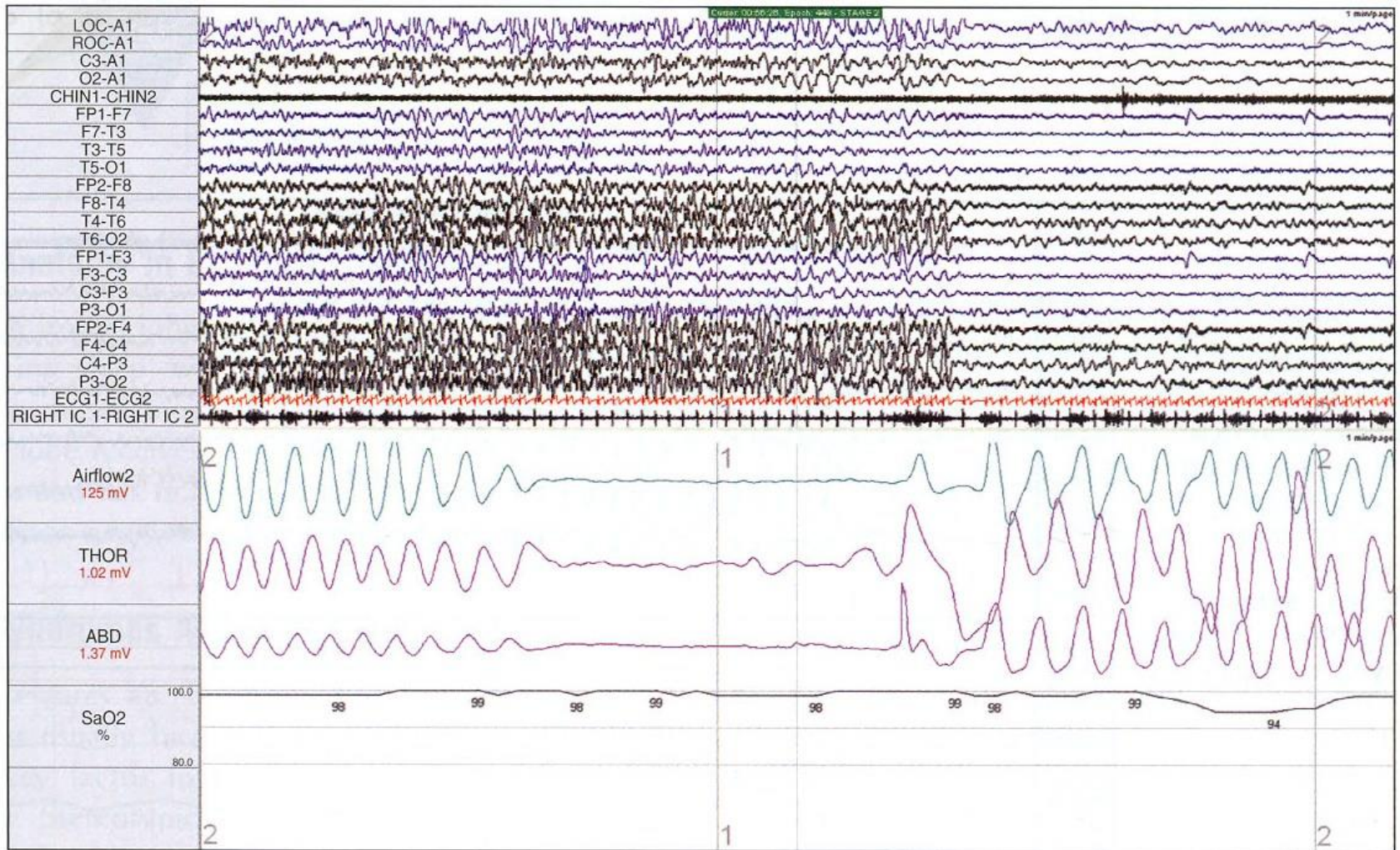


Figure 10.3-6.

PSG example of right-sided seizure resulting in central apnea. In this example the seizure was originally missed because the seizure activity was only on the right side (notice the even-numbered electrodes) and the scoring montage did not include them. Such a seizure montage is useful in cases of unexplained central apneas (*black, right; blue, left*). (PSG courtesy M. Mahowald.)

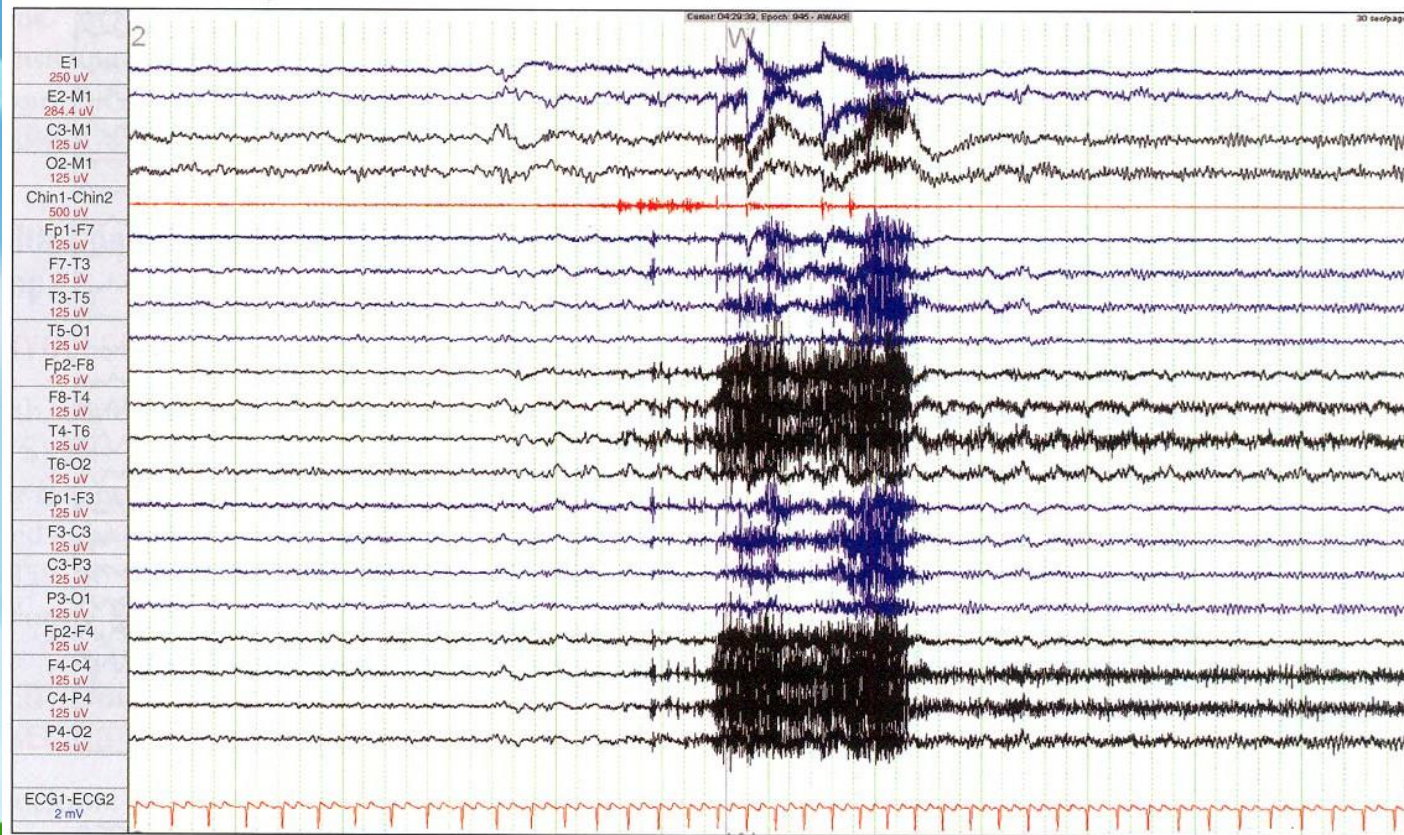


Figure 10.3-7.

PSG example of temporal seizure. In this example there are brief spikes in the right temporal region (note the abnormalities in T4-T6 and T6-O2) that begin just before the increased activity in the chin electrodes. This is followed by arousal. On the conventional sleep scoring montage, this would appear to be a simple, nonspecific arousal. The EEG montage reveals periodic spikes from the right mid-temporal region culminating in an arousal followed by residual postictal slowing over the same region. This “arousal” was actually the sole clinical manifestation of a focal temporal lobe epileptic discharge. (These may occur hundreds of times a night, resulting in frequent arousals [sleep fragmentation] presenting as excessive daytime sleepiness.) For this reason, it is prudent to employ a full seizure montage in all patients with a history of seizures and the complaint of excessive daytime sleepiness. If these arousals are associated with extremity movements, an erroneous diagnosis of periodic leg movements could be made (*black, right; blue, left*). (PSG courtesy M. Mahowald.)

Figure 10.3-4.

PSG example of nocturnal frontal lobe epilepsy. This is a representative PSG sample from a 35-year-old woman with a history of bizarre nocturnal spells in the middle of the night. The episodes are usually brief, less than a minute. This PSG shows spike and slow-wave complexes more prominent centrally during stage N2 sleep. (PSG courtesy M. H. Kryger.)

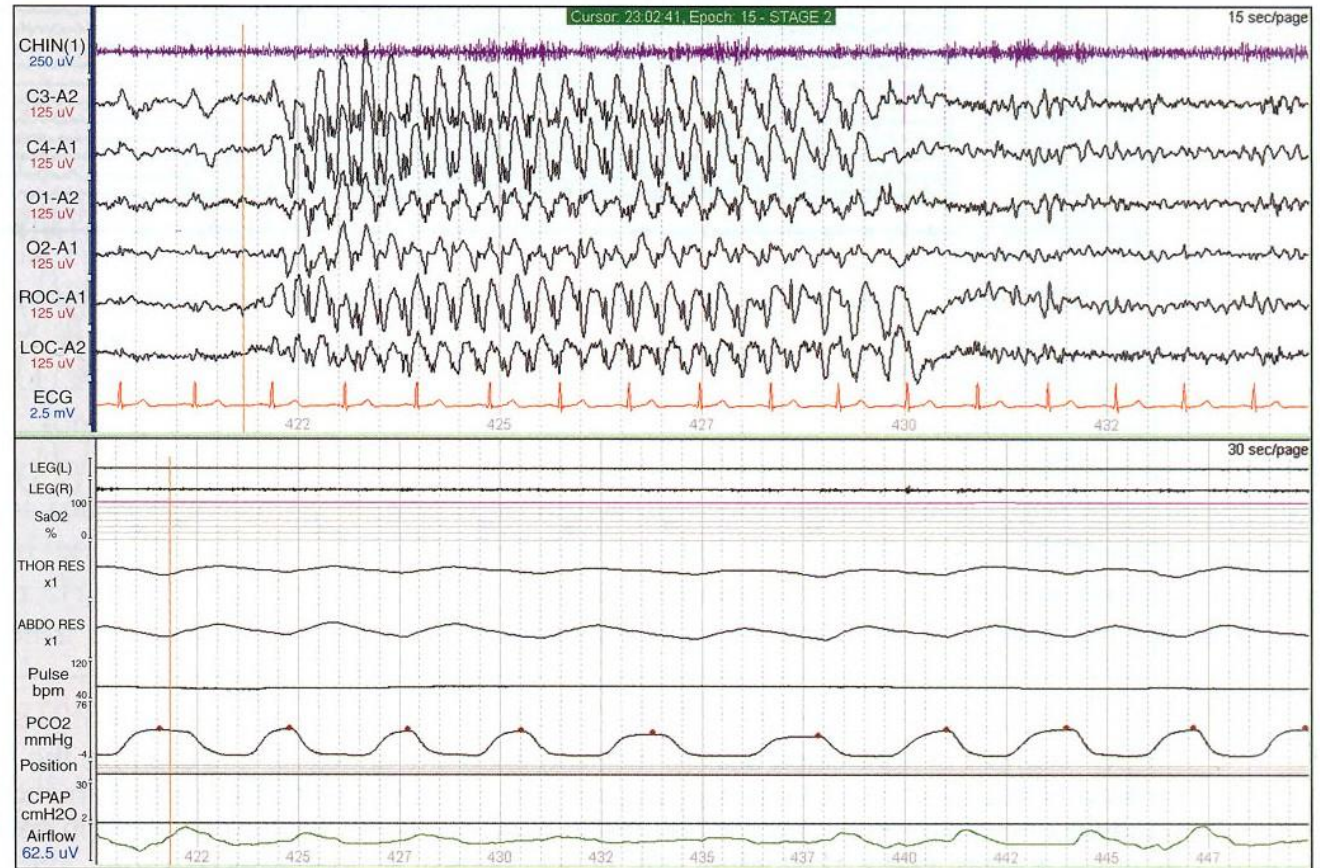




Figure 10.3-5.

PSG example of patient referred with infrequent nocturnal seizures. This example is from a 20-year-old student who was referred because of a 5-month history of infrequent seizures that occurred only at night. As a result of these, the patient had dislocated his shoulder and bitten his tongue. Notice the seizure activity lasting about 5 seconds in the middle of the epoch, during which time the patient had a central apnea. There were only three similar findings during the night. On synchronized digital video there was absolutely no movement visible. The sleep study may be entirely normal in patients with infrequent seizures. (PSG courtesy M. H. Kryger.)

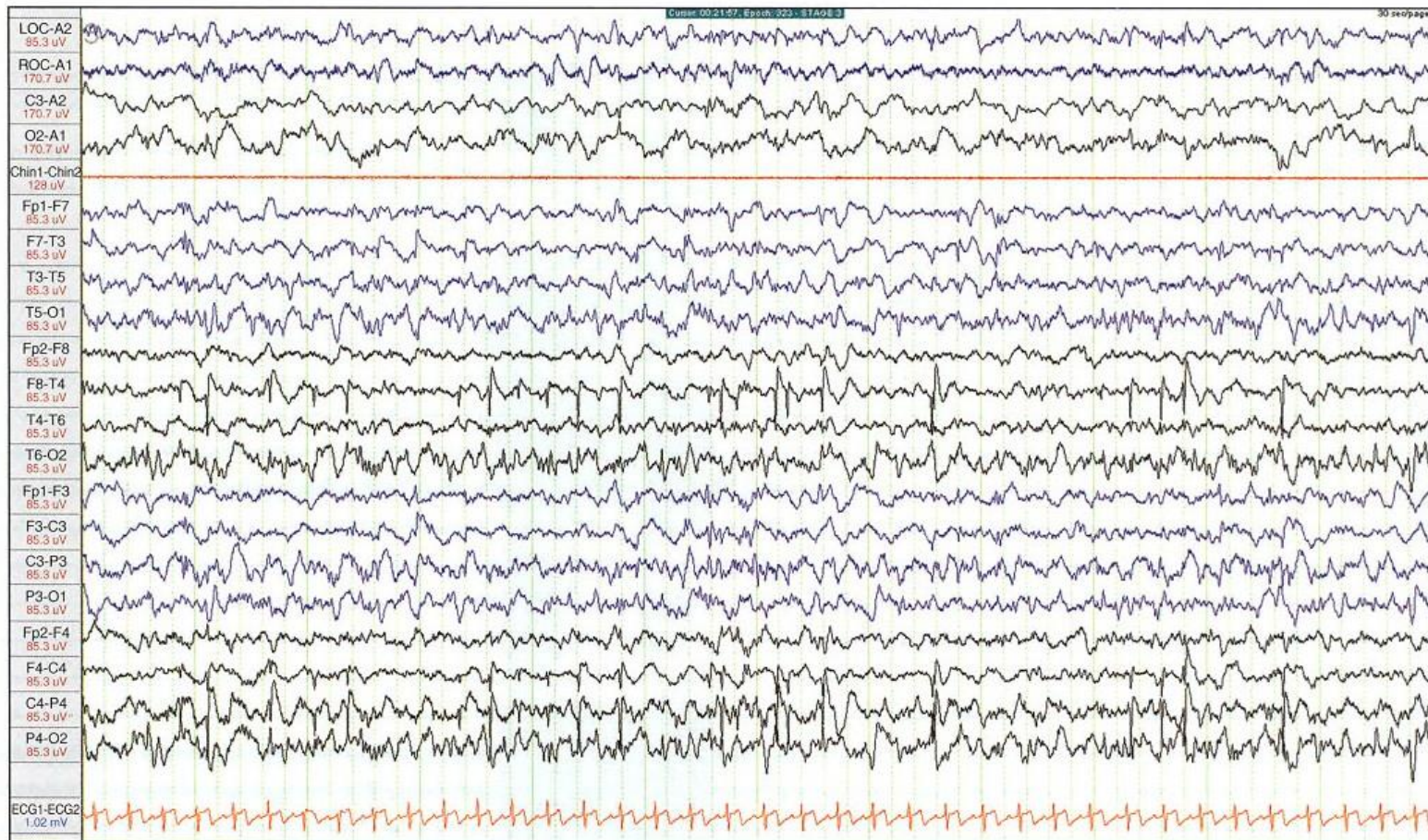


Figure 10.3-8.

PSG example of Rolandic spikes. In this example the spikes are right sided (note their presence primarily in even-numbered electrode pairs). These spikes are characteristic of Rolandic spikes (seen in benign Rolandic epilepsy). They are most prominent over the central and mid-temporal region and are often present only during NREM sleep, when they may become very active. Clinically, there may be twitching of the mouth on the contralateral side with or without drooling. Occasionally these usually trivial seizures will generalize. The prognosis is generally very good, with the natural history of spontaneous resolution over time, hence the term *benign Rolandic epilepsy* (black, right; blue, left). (PSG courtesy M. Mahowald.)



**Thanks For Your
Attention**