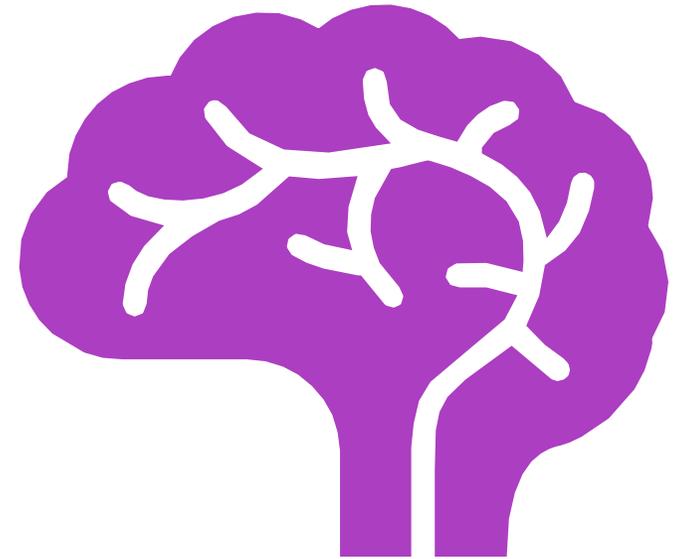


# EEG IN ENCEPHALOPATHY

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## MAIN CONTRIBUTION OF EEG IN SLOWING

- Providing objective measure of severity of encephalopathy
- Not specific to any particular etiology
- Help in guiding effectiveness of therapy
- Prognosis???

# EXPECTED FINDINGS:

Background  
slowing

Intermittent  
slowing

Continuous  
slowing



<b>Conditions such as toxic or metabolic abnormalities affect entire brain</b>	<b>EEG typically show generalized abnormalities.</b>
Supratentorial lesions	focal or diffuse dysfunction, (temporal lobe focal dysfunction or diffuse change in increased intracranial pressure)
Infratentorial lesion	suppression of reticular activation-affect consciousness-diffuse changes.

# EEG PATTERNS SEEN IN CEREBRAL DYSFUNCTION

**White matter disruption** -Higher amplitude, poly morphic slowing without paroxysmal synchronous activity

**Gray matter disruption** -tend to produce paroxysmal synchronous activity

Mechanism of these rhythms generation remain unstudied.

- Continuous slow activity suggests a more severe brain damage
- Intermittent slow activity usually indicates a small lesion and absence of mass effect.
- Reactive focal slow activity had evidence of less cerebral damage than did patients with non reactivity.

Field, amplitude and frequency of focal slow waves do not distinguish lesion size, density or mass effect.

Reactivity and persistence of focal abnormalities (continuous versus intermittent) were significantly better indicators of damage degree (Schaul et al)

# CLASSIFICATION

- 1) Focal slow activity--Regional or generalized (bisynchronous or asynchronous slow activity)
- 2) Attenuation: Focal attenuation; generalized attenuation
- 3) Suppression
- 4) other abnormal activities (alpha, theta and spindles coma patterns)

# FOCAL SLOW WAVE ACTIVITY

- May be indicative of focal cerebral dysfunction
- Result of cortex deafferentiation from subcortical structures
- We classify slow waves theta/delta

## PATHOLOGICAL SLOW WAVES

- Two types
  - THETA
  - DELTA

**Theta slowing seen in hypoxia, hypoglycemia, CVA, dementia, toxic and metabolic encephalopathy, drug toxicity**

**Delta slowing-  
Hallmark of mass lesions such as brain tumors, abscess and infarctions.**

# DELTA ACTIVITY

- First recognized and named by Walter
- Focal or Generalized
- Rhythmic or arrhythmic
- Continuous or intermittent
- Can be seen in localized, lateralized or diffuse encephalopathy
- Significant but non specific sign of cortical disturbance.
- Postulated to occur by disruption of blood supply, energy substrate and microenvironment, loss of synaptic input into cortical neurons.

# CLASSIFICATION WITH ETIOLOGIES

- Diffuse encephalopathy
- Specific encephalopathy
- Degenerative causes

# EEG GRADING WITH SEVERITY

1

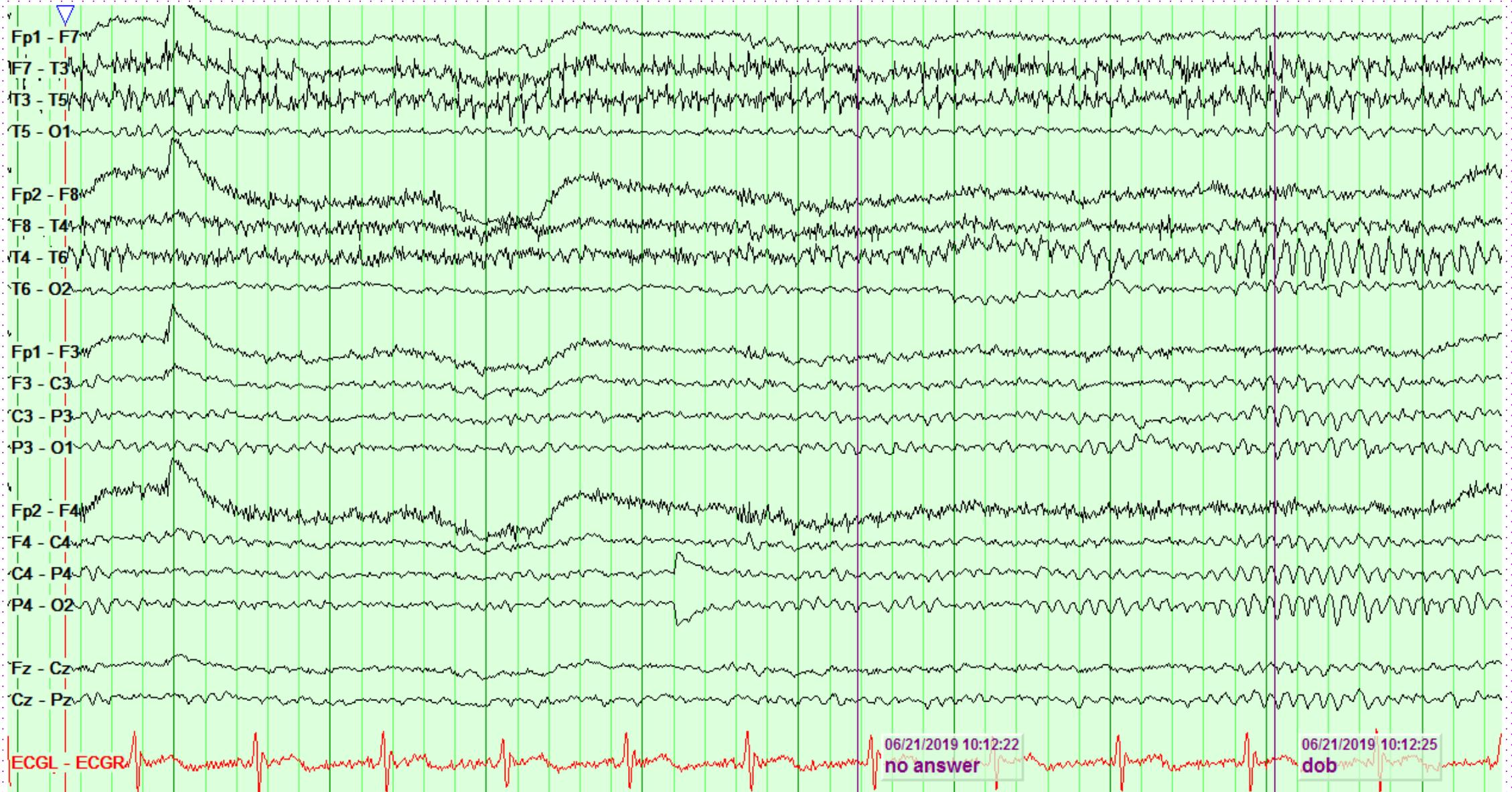
Mild- non specific  
background slowing

2

Moderate-admixture  
of theta/delta  
background

3

Severe-Delta  
background



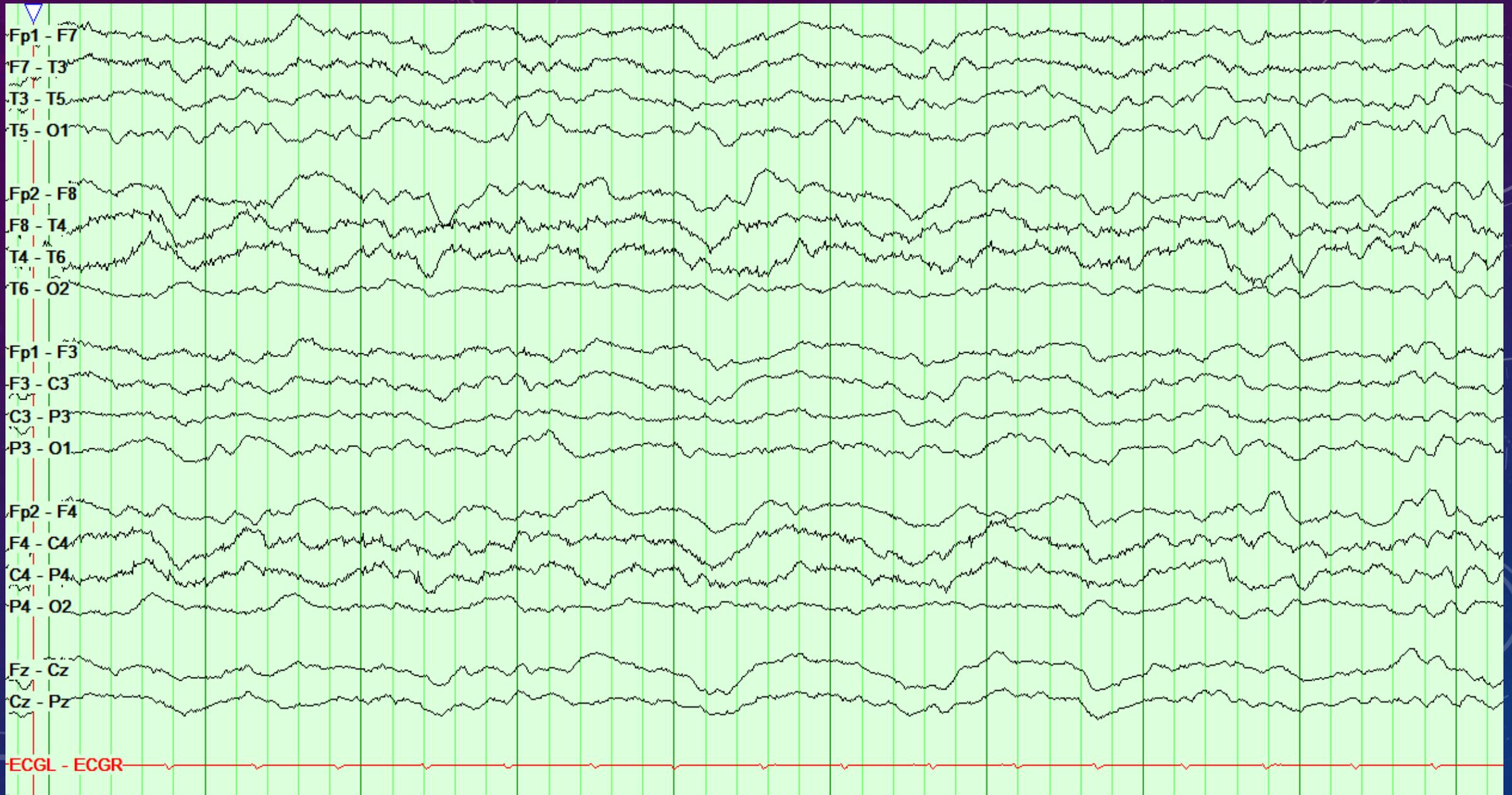




GENERALIZED SLOWING (THETA/DELTA)



GENERALIZED SLOWING (THETA/DELTA)



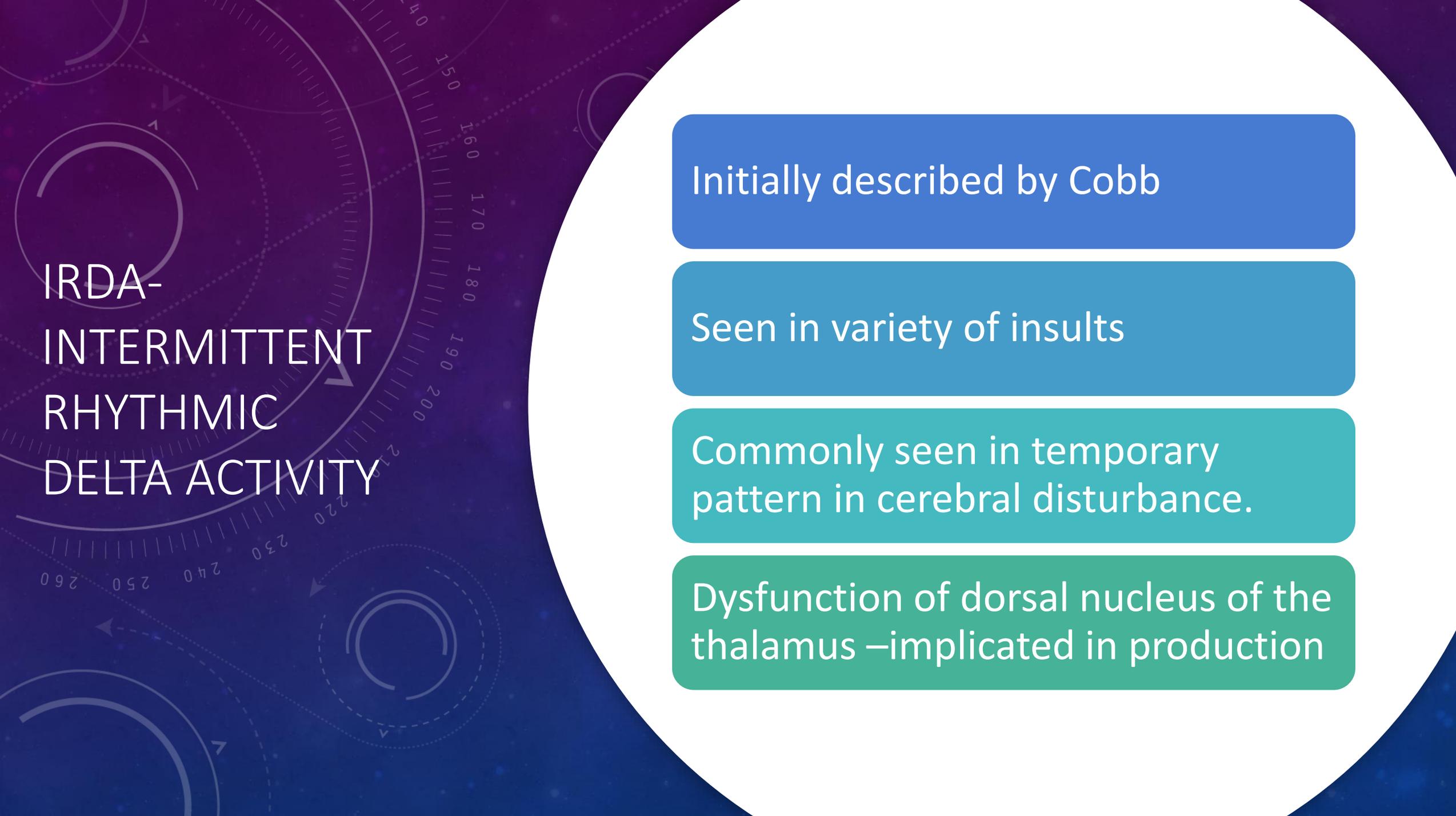
GENERALIZED SLOWING (DELTA)



SEVERE GENERALIZED SLOWING



Hyperventilation

The background features a dark blue gradient with intricate circular patterns. These include concentric circles, dashed lines, and radial tick marks, some of which are labeled with numbers such as 140, 150, 160, 170, 180, 190, 200, 210, 220, 230, 240, 250, and 260. The overall aesthetic is technical and scientific.

# IRDA- INTERMITTENT RHYTHMIC DELTA ACTIVITY

Initially described by Cobb

Seen in variety of insults

Commonly seen in temporary pattern in cerebral disturbance.

Dysfunction of dorsal nucleus of the thalamus –implicated in production

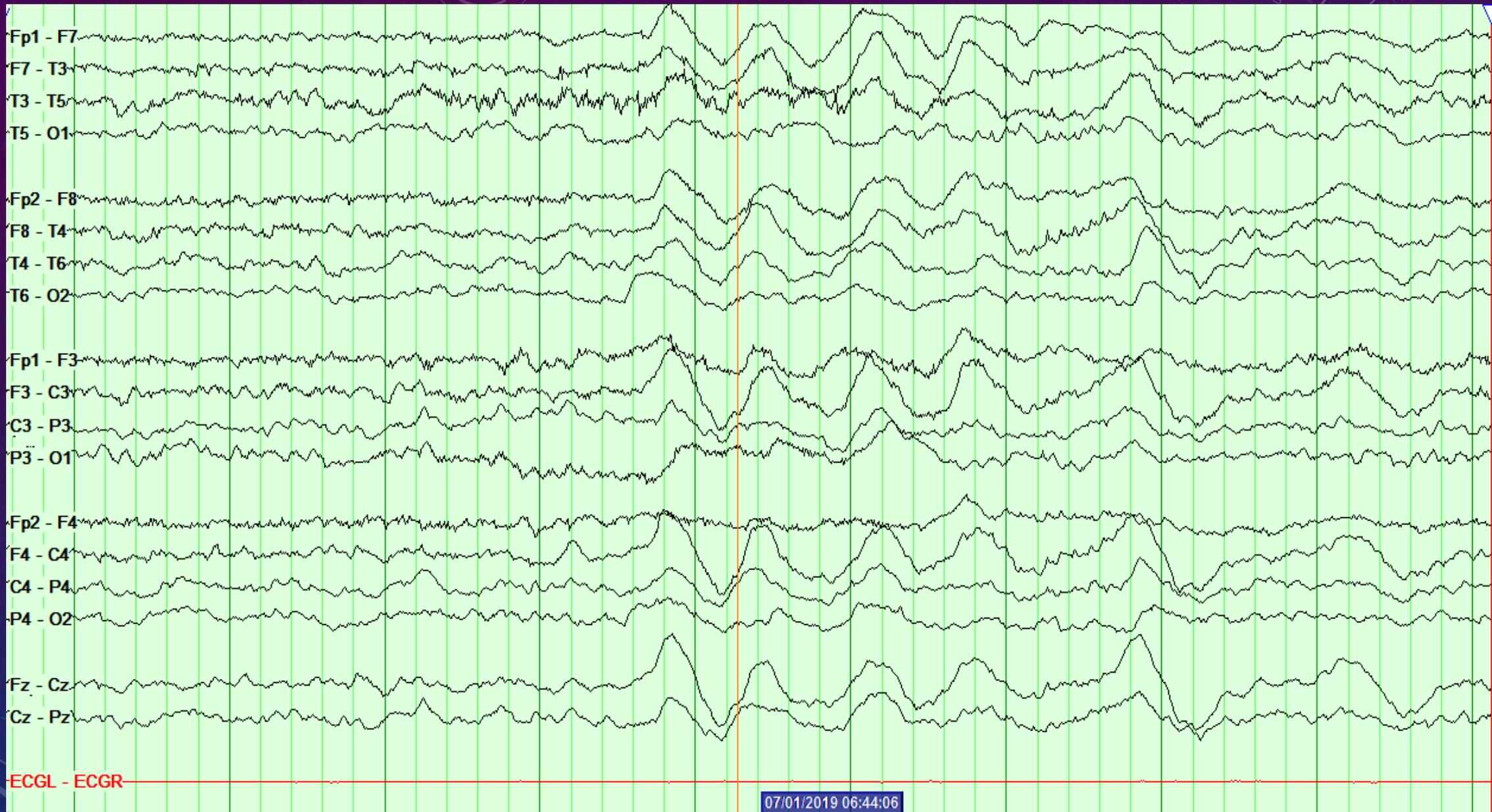
## OTHER FEATURES:

Anterior predominance –FIRDA (named by Van der Drift and Magnus)

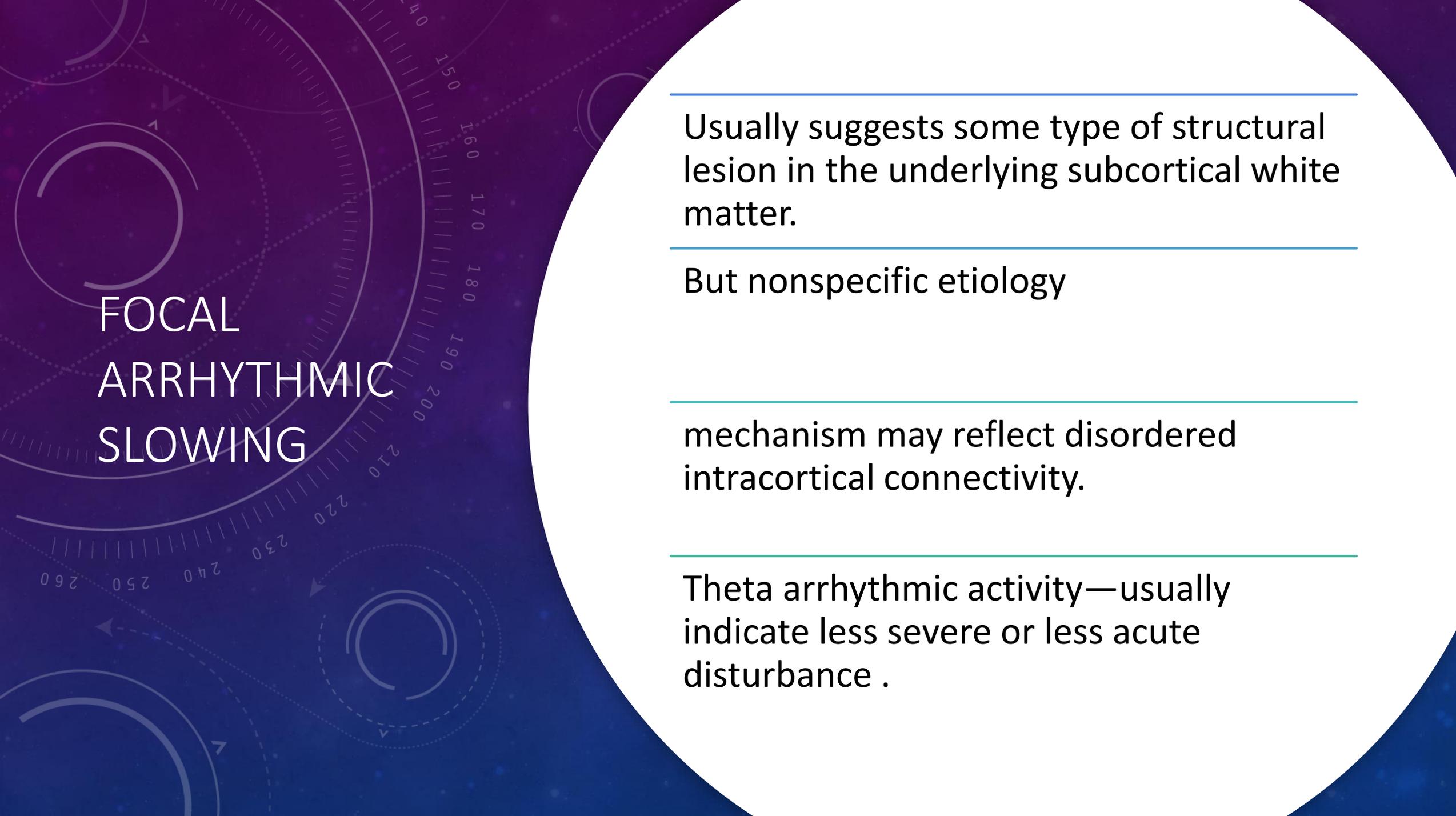
Can be diffuse, asymmetrical, lateralized or even unilateral.

Onset abrupt

Hyperventilation and hypnagogic hypersynchrony in children should be differentiated



GRDA

The background features a dark blue to purple gradient with several overlapping circular patterns. Some are solid lines, others are dashed. Numbers like 140, 150, 160, 170, 180, 190, 200, 210, 220, 230, 240, 250, and 260 are scattered around the circles, suggesting a scale or measurement. The text 'FOCAL ARRHYTHMIC SLOWING' is centered in white, bold, uppercase letters.

# FOCAL ARRHYTHMIC SLOWING

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Usually suggests some type of structural lesion in the underlying subcortical white matter.

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But nonspecific etiology

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mechanism may reflect disordered intracortical connectivity.

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Theta arrhythmic activity—usually indicate less severe or less acute disturbance .

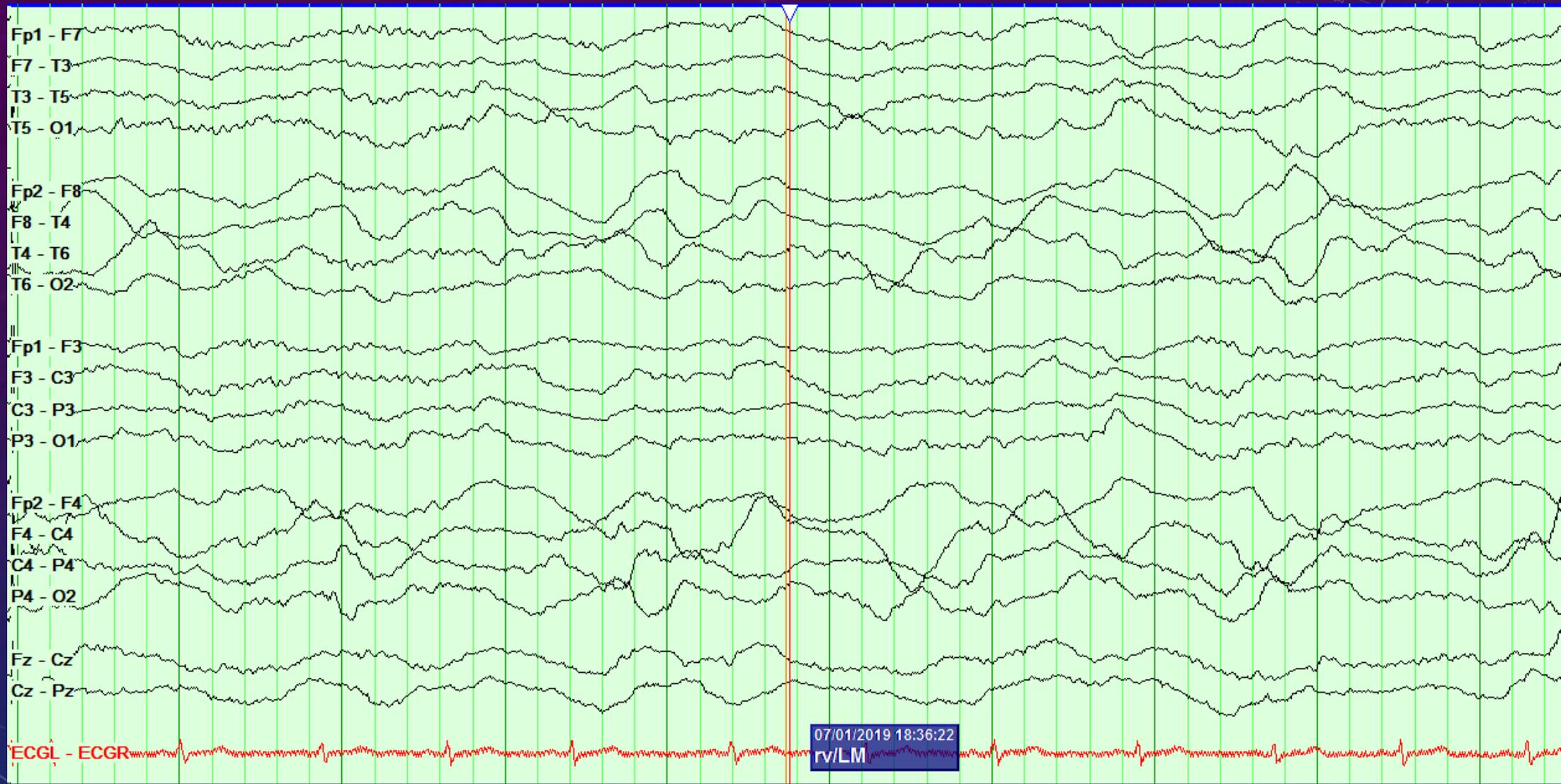
Abscesses, ischemic strokes, tumors, contusions may produce this pattern.

Even transient functional disturbances, such as migraine and the postictal state, can be responsible.

Thus follow-up EEGs looking for evolution or resolution of any focal slowing is valuable.

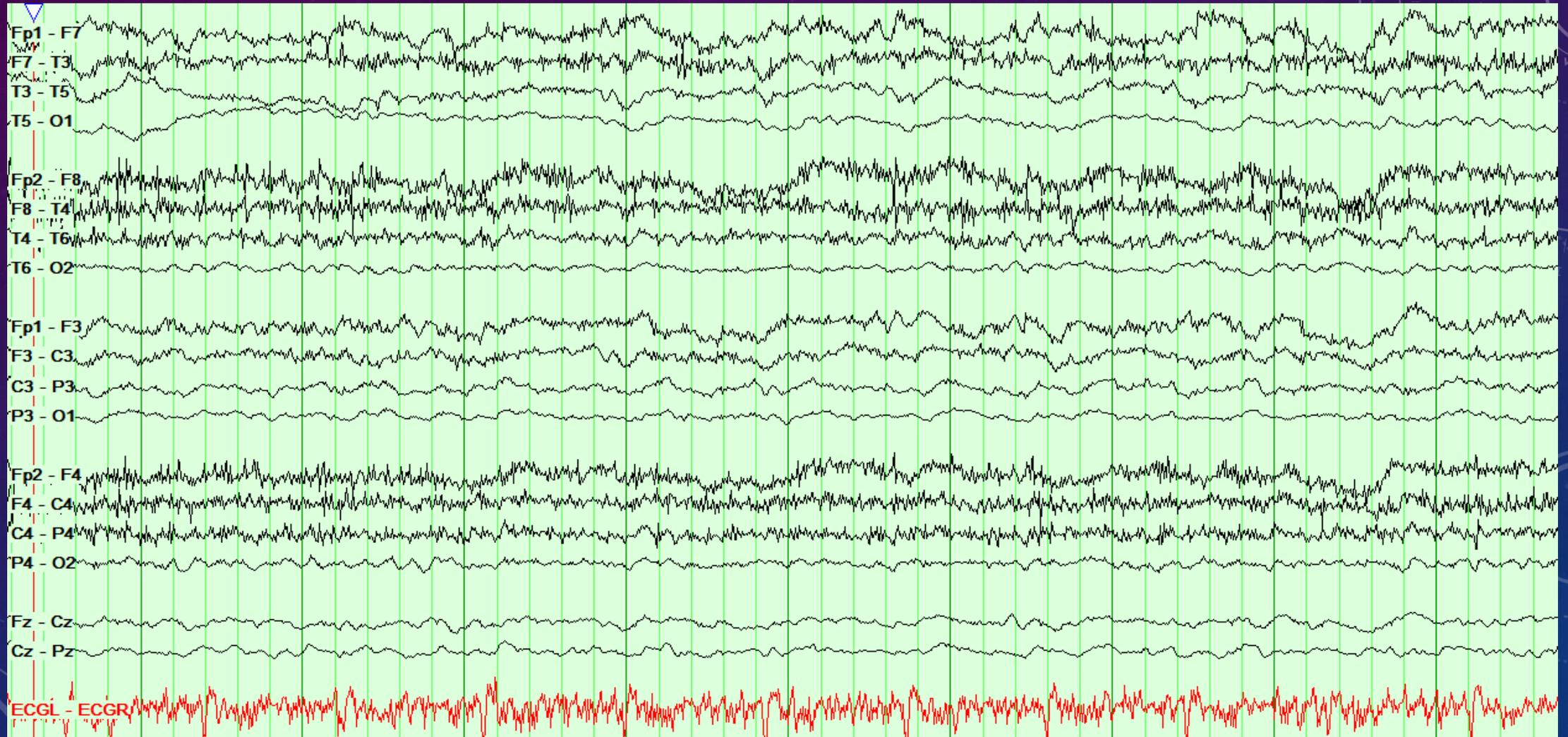


CONTINUOUS RIGHT TEMPORAL SLOWING



FOCAL RIGHT TEMPORAL SLOWING

# CONTINUOUS FOCAL DELTA SLOWING



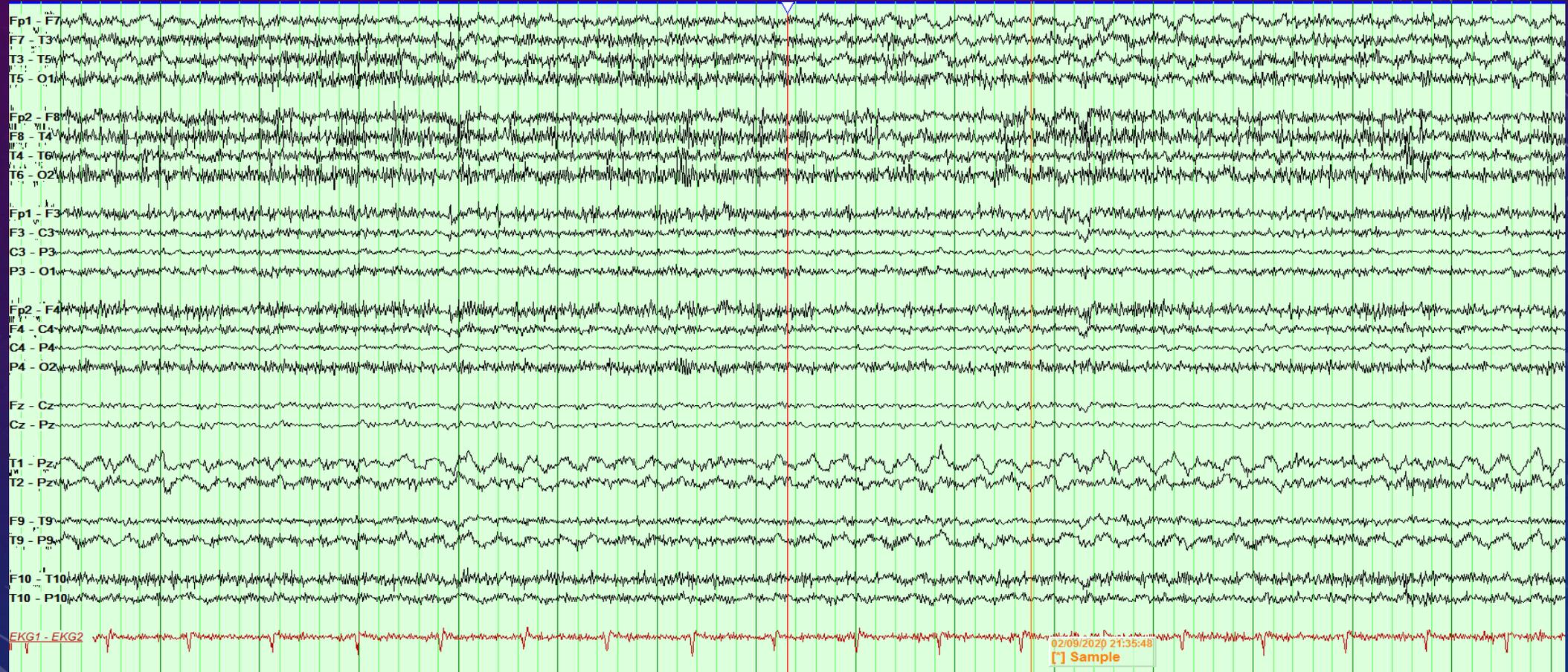
# FOCAL RHYTHMIC SLOWING

By contrast more commonly associated with underlying gray matter lesions.

Focal voltage attenuation or epileptiform abnormalities seen rather than focal slowing.

When the focal slowing shows exceptional rhythmicity or frequency evolution- seizure focus is also suggested.

Location is significant. For example, if observed in the temporal regions, an underlying epileptogenic focus is more likely.





# FOCAL SLOWING



# FOCAL ATTENUATION

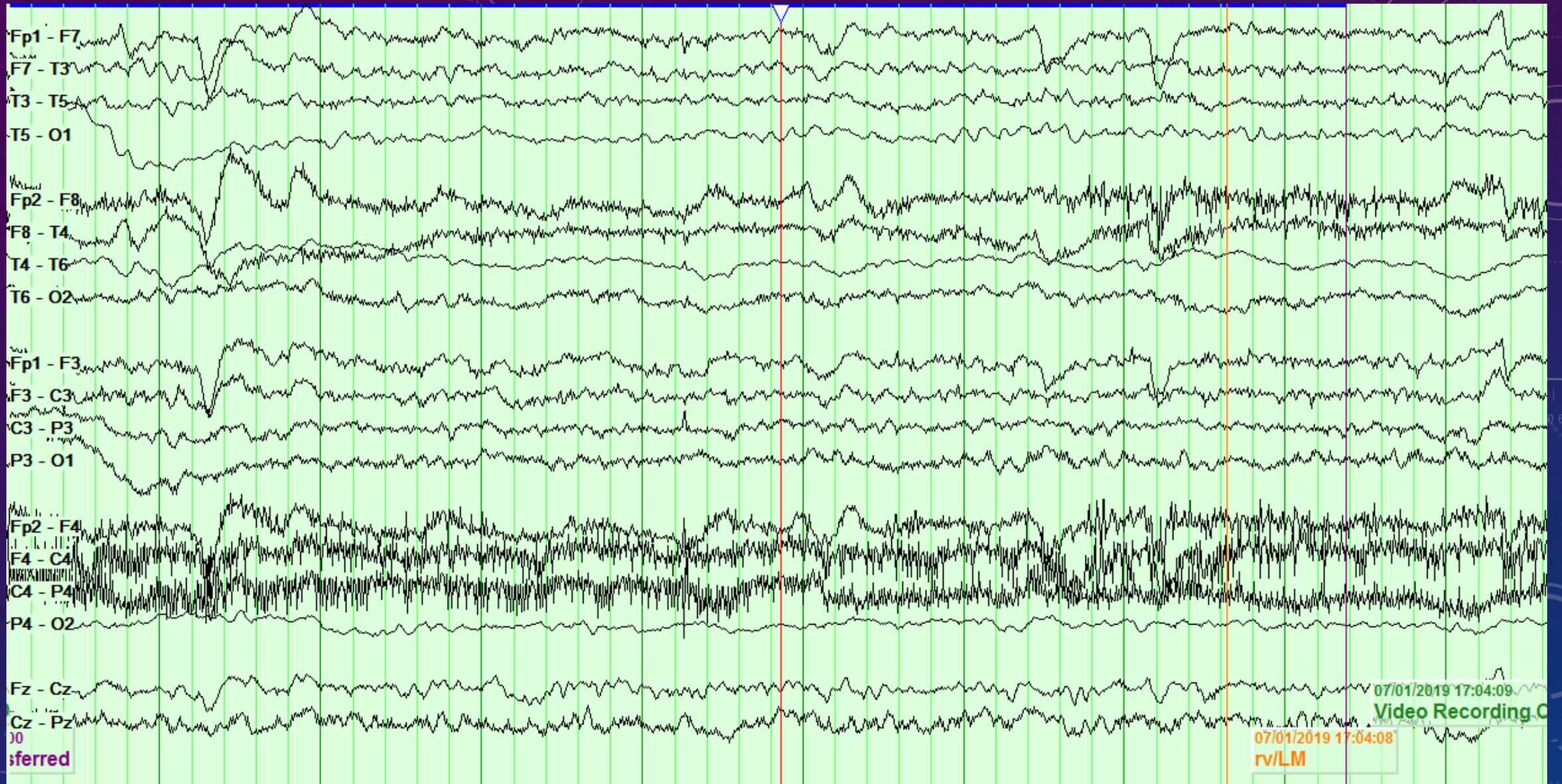
Reflect focal reduction  
in electrical activity

Seen in

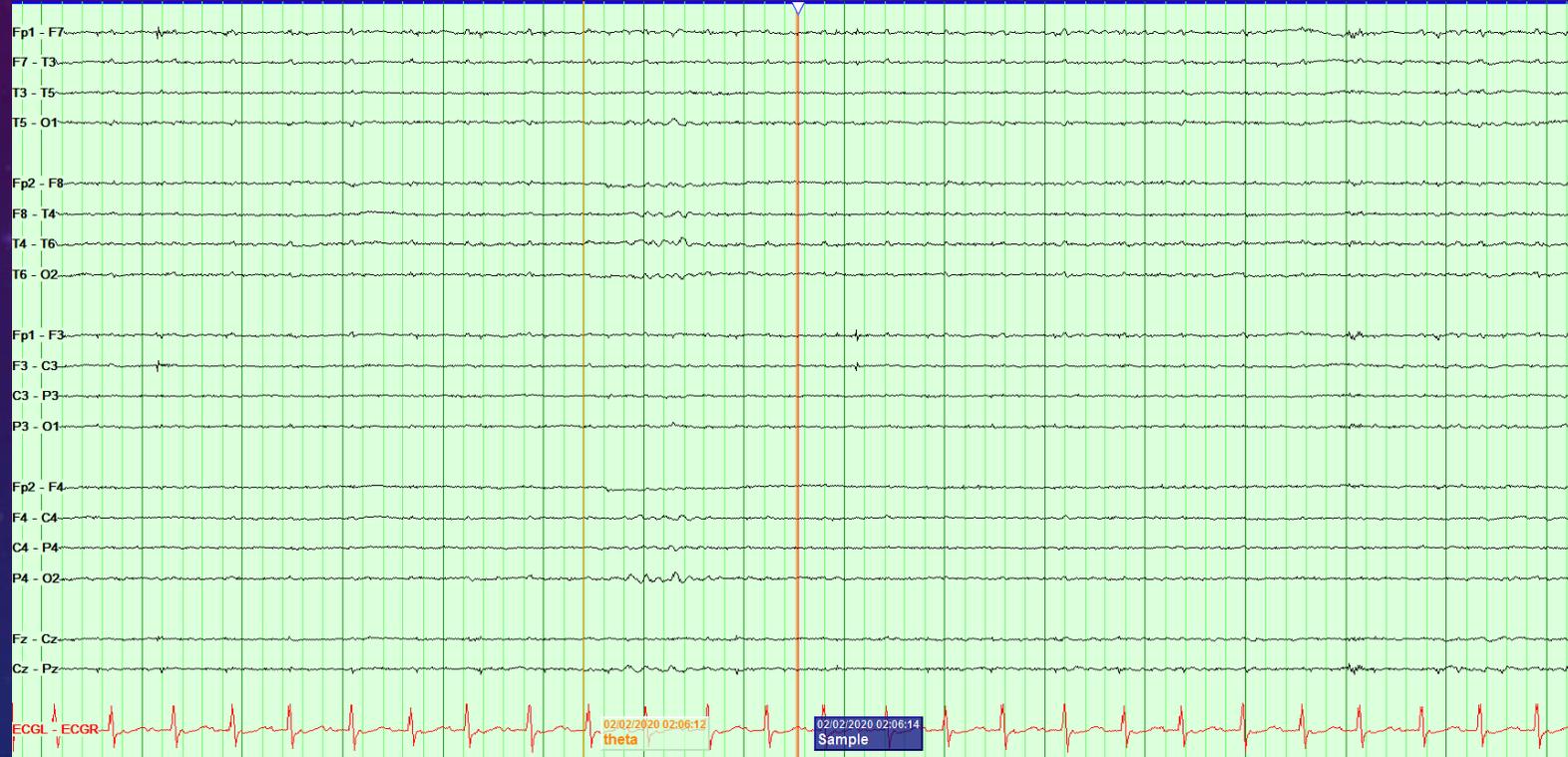
May reflect increased  
distance between  
between cortex and  
recording electrode (ex  
subdural hematoma)

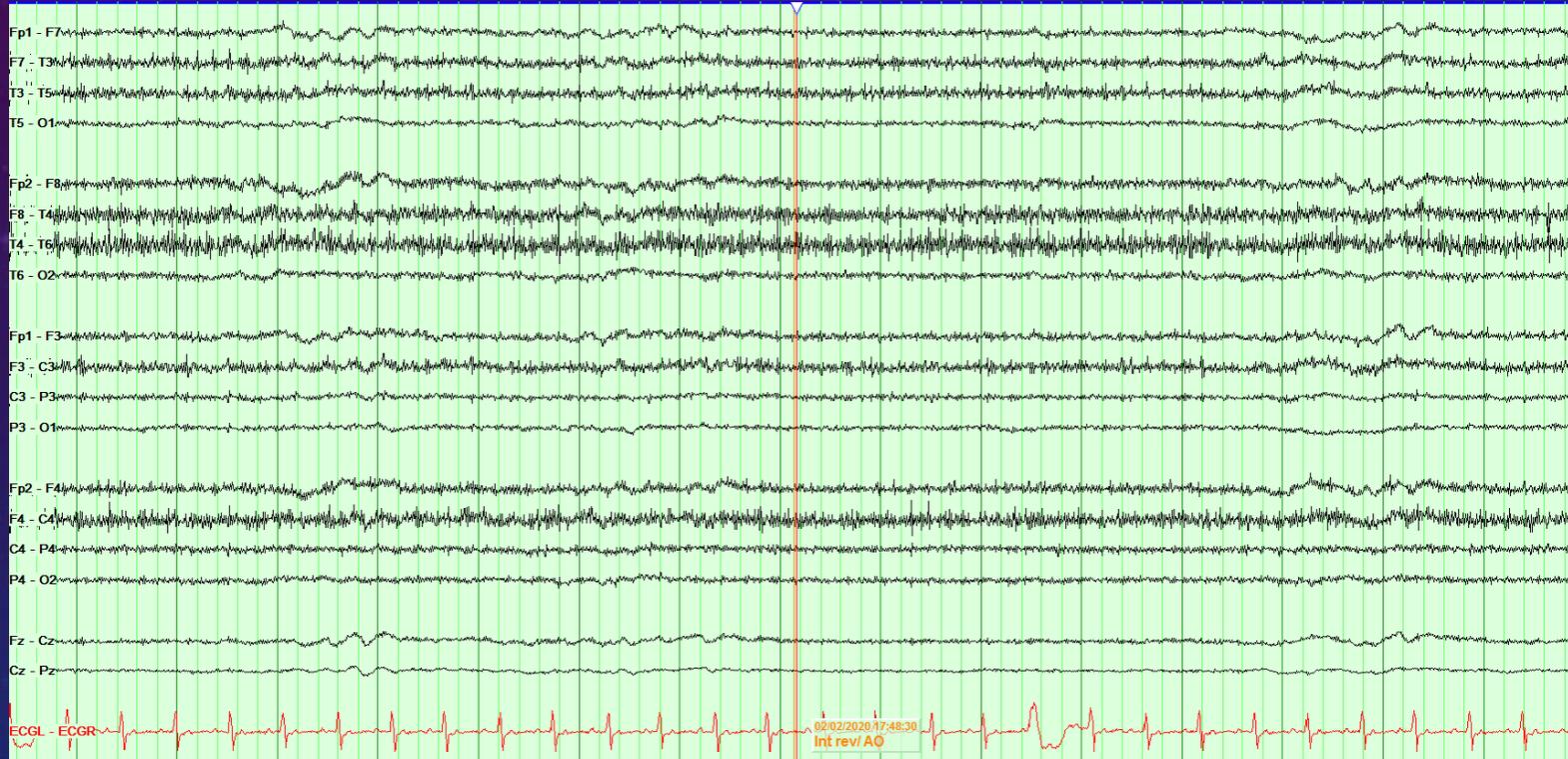
a) structural cortical  
damage

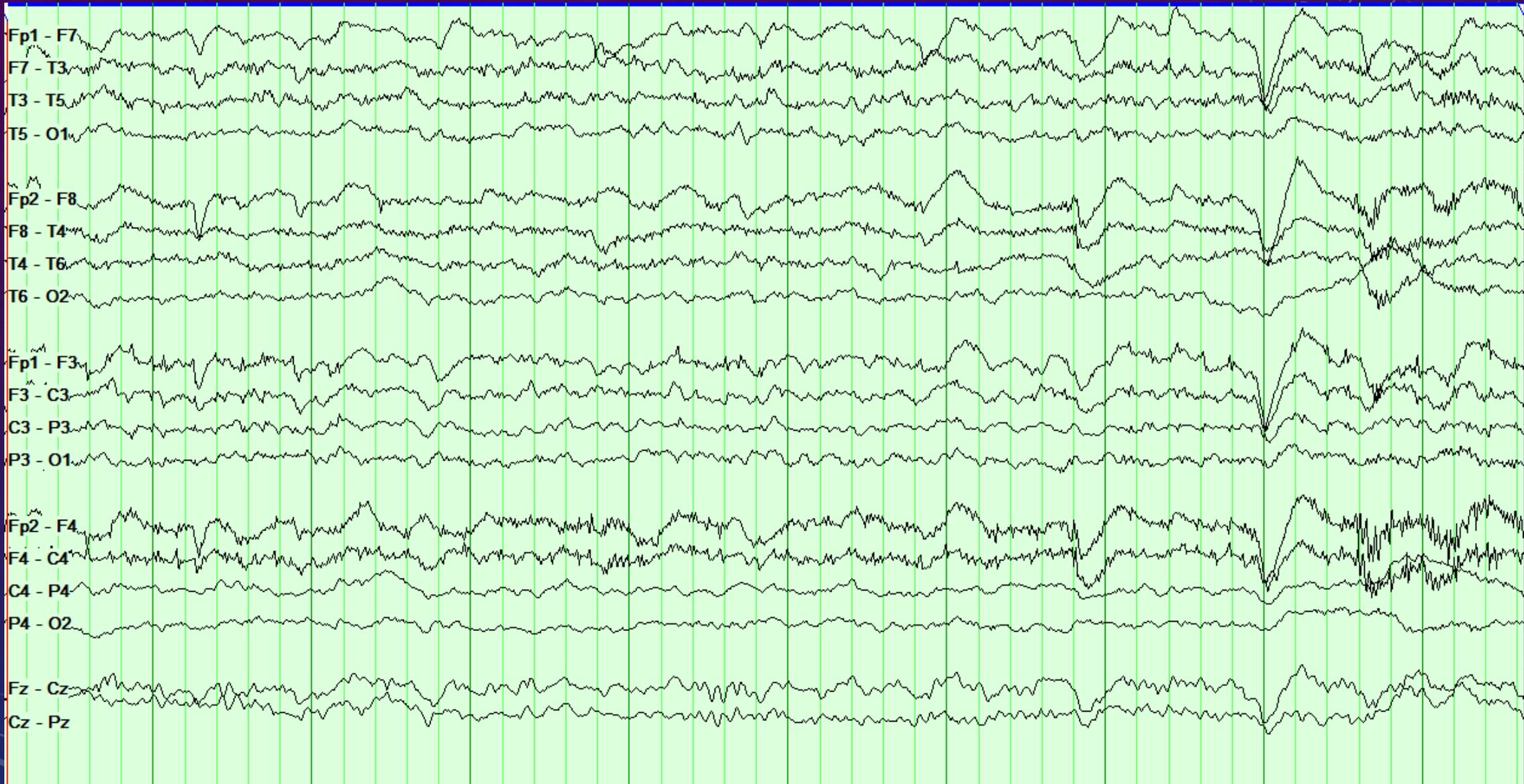
b) disorder of cortical  
function (ex: cortical  
ischemia, postictal  
attenuation)



ATTENUATED RHYTHMS RIGHT TEMPORAL









GENERALIZED DISCHARGES, TRIPHASIC MORPHOLOGY





## TRIPHASIC WAVE

Periodic repeating waveforms

First described by Foley et al with hepatic encephalopathy

Name coined by Bickford and Butt

Prominent surface positive, preceded and followed by smaller amplitude negative waves

Metabolic abnormalities and structurally diffuse white matter lesions specific pattern.

## OTHER FEATURES

Typically medium- to high-voltage slow waves occurring at a frequency of 1 to 2.5 Hz.

Bilaterally symmetric, bisynchronous fashion. Fronto-occipital delay

Wax and wane

Triphasic waves usually show a phase lag of 25 to 140 ms across the anterior–posterior axis.

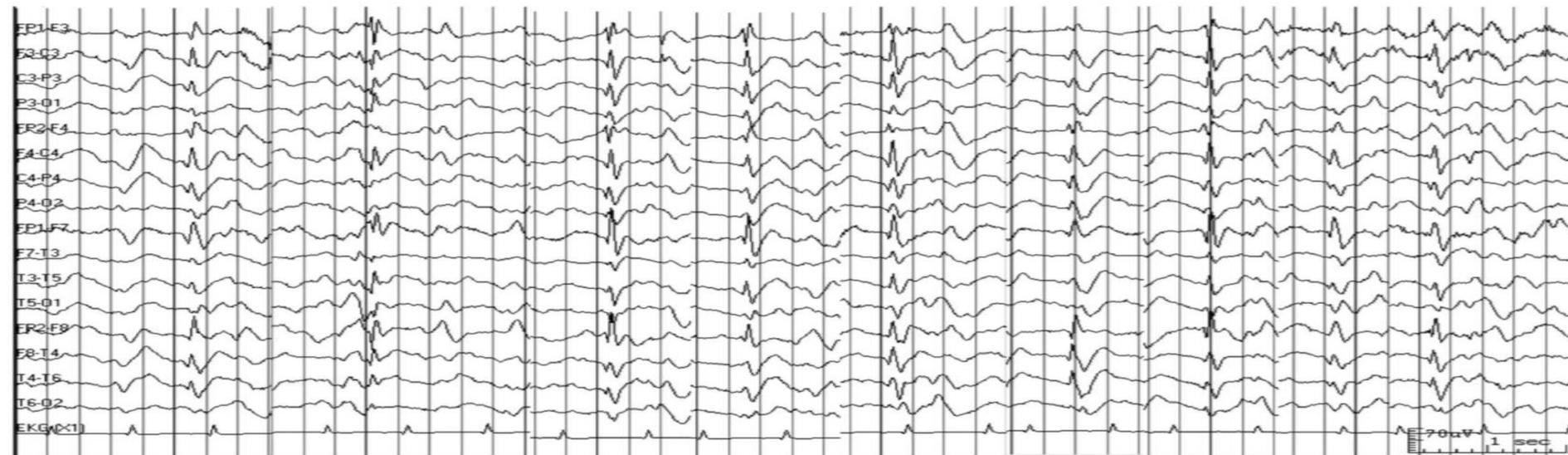
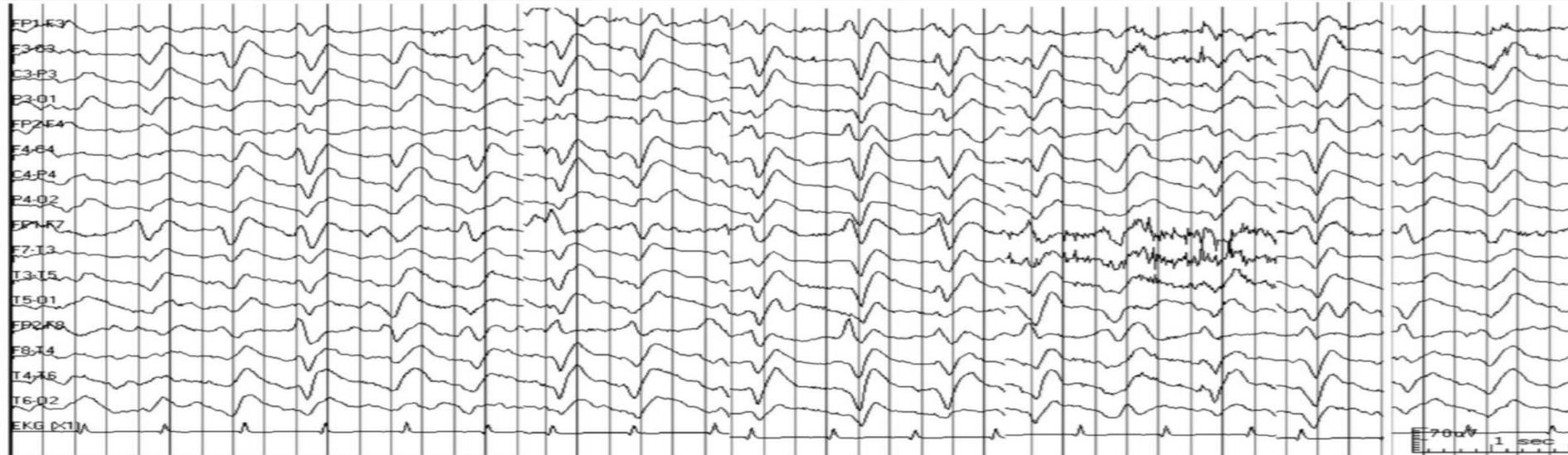
# ELECTROPHYSIOLOGICAL BASIS

- increase in astrocytic pH with hyperammonemia → induces calcium-dependent release of glutamate, N-methyl-D-aspartate acetylation, and excitotoxicity.
- decrease chronic astrocytic glutamate supplies, inactivate glutamate transportation, and hence decrease the postsynaptic glutamate receptor numbers on astrocytes and neurons.
- Increase in cerebral inhibition and increased gamma-aminobutyric acid tone. The increased gamma-aminobutyric acid-A receptor activation increases postsynaptic neuron inhibition (Ahboucha and Butterworth, 2004).
- These acute changes are associated with astrocytic and cytotoxic edema (Shawcross and Jalan, 2005), which may affect the subcortical cerebral white matter .

- The EEG slowing has been thought to reflect the level of hyperammonemia and to correlate with the degree of liver disease
- The authors postulated that the primary disturbance occurred at a thalamic (subcortical) level through thalamocortical relays (Bickford and Butt, 1955).
- TW not seen in younger children and do not occur in animal models

## THE DISTINCTIONS BETWEEN THE TW AND THE EPILEPTIC DISCHARGE INCLUDE

- more acute narrow-angled, briefer, and frontopolar emphasis for generalized spike waves .
- lack of response to stimulation with GSW discharges.
- conversely, longer duration, frontocentral, stimulus-altered blunter TWs



## OBSERVED IN VARIOUS DISORDERS:

uremia,  
hyperthyroidism,  
hypercalcemia,  
hypoglycemia,  
hyponatremia, and lithium intoxication.

Alzheimer's disease and other dementias; prion diseases;  
structural pathologies, such as stroke and subdural hematoma;  
cerebral carcinomatosis can also demonstrate this pattern.

# PROGNOSIS

- Patients with TWs had mortalities ranging from 30% to 100% depending on whether the cause was renal, hepatic, or anoxic encephalopathy (Karnaze and Bickford, 1984)

# ATYPICAL TRIPHASIC WAVES

Largely represent “TW look-alikes” such as with drug toxicities (e.g., lithium, baclofen, and cefepime) or in forms of nonconvulsive status epilepticus.

Atypical forms not related to metabolic problems or white matter disease

Might be asymmetric,

Have a more sharply contoured phase I and steeply declining phase II, be more diffusely and sporadically distributed

less affected by arousal

# HEPATIC ENCEPHALOPATHIES

- Mild form-EEG shows BG slow and progressive increase of gen slow
- Moderate form-triphasic waves typically seen
- Frank coma-high amplitude slowing/decrease or absent triphasic wave
- EEG may be useful in assessing severity of hepatic encephalopathies well as effectiveness of therapy

# RENAL ENCEPHALOPATHIES

- Uremic encephalopathy
- Seizures usually GTC
- EEG slowing parallel deterioration of mental status
- Abnormalities include Triphasic, photomyoclonic, photoparoxysmal response and increased slowing with HV
- IRDA seen in dialysis disequilibrium syndrome (brain swelling due to lag of osmolar shifts)

# TOXIC ENCEPHALOPATHIES

Drug intoxication common cause of coma in ER setting  
EEG parallels the degree of mental alteration.

Sedative intoxication-may show beta

Severe cases with burst suppression and ECI

In general, drug intoxication has relatively good prognosis no matter how severe the EEG looks.

# ANOXIC ENCEPHALOPATHIES

- Variety of pattern can be seen
- Range from generalized slowing to alpha, theta coma, periodic patterns, burst suppression and ECI
- GPDs- represent severe disruption of cortical and subcortical structure.

# ENDOCRINE ENCEPHALOPATHIES

<b>Hyperthyroidism</b>	Slight acceleration of alpha Enhancement of fast activity Augmentation of rlandic mu rhythms. Burst of epileptiform discharge and seizure can occur	Thyrotoxicosis and Hashimotos encephalopathy diffuse slowing FIRDA, sharp waves, Occ triphasic waves seen
Hypothyroidism	Low amplitude and diffuse slow	Seizure in hypothyroidism associated with prolonged post- ictal recovery
Hypocalcemia	Both generalized and focal seizure, epileptiform discharges seen	Mechanism: Hypocalcemia increase Na <sup>+</sup> conductance, membrane depolarization and repetitive neuronal firing
Hypercalcemia	Diffuse slowing, sometime triphasic waves	
Adrenal gland abnormalities	Insufficiency (Addison's disease) produce slowing	Hyperfunction (Cushing syndrome) –isolated reports of enhanced seizure susceptibility with ACTH doses
Pituitary gland abnormalities	Diffuse slowing	

# TEMPORAL SLOWING OF ELDERLY

Seen in person over the age of 60 years

Focal theta/delta common over temporal region, especially the left.

Enhanced by drowsiness

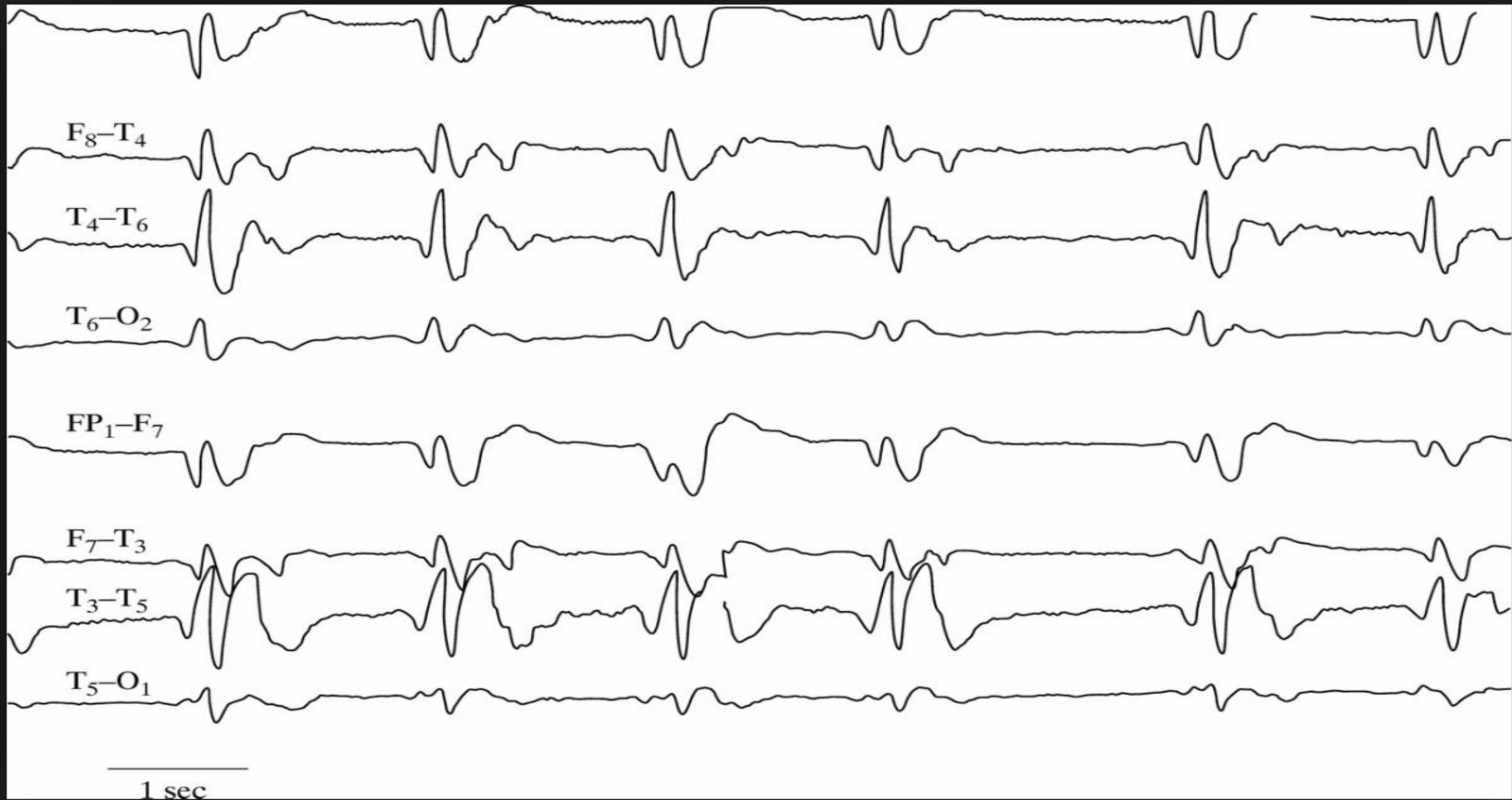
Termed as benign and interpreted conservatively “when they temporal in location, theta frequency range, rhythmic or arrhythmic but occurs mainly in drowsiness”.

# SUBACUTE SCLEROSING PANENCEPHALITIS

EEG findings one of the most characteristic and specific.

Clinically has 4 stages

High voltage repetitive polyspike and sharp and slow wave complexes.



# PRION DISEASE-CJD

:Early in the disease EEG can be normal or show non specific slowing

As the diseases progress, typical periodic complex appear.

Diagnosis of CJD-periodic complexes have a specificity of 67% and sensitivity of 90%

Myoclonic jerks frequently but not always time locked to these periodic discharges.

# AIDS AND HIV INFECTION

- EEGs in HIV symptomatic infection and AIDs usually show non specific abnormalities
- Epileptiform discharges are rare.
- Epileptic seizure occur less than 5% but in advanced stage can increase to 10%

## TAKE HOME POINTS

Frequency of slowing and its persistence are two important characteristics.

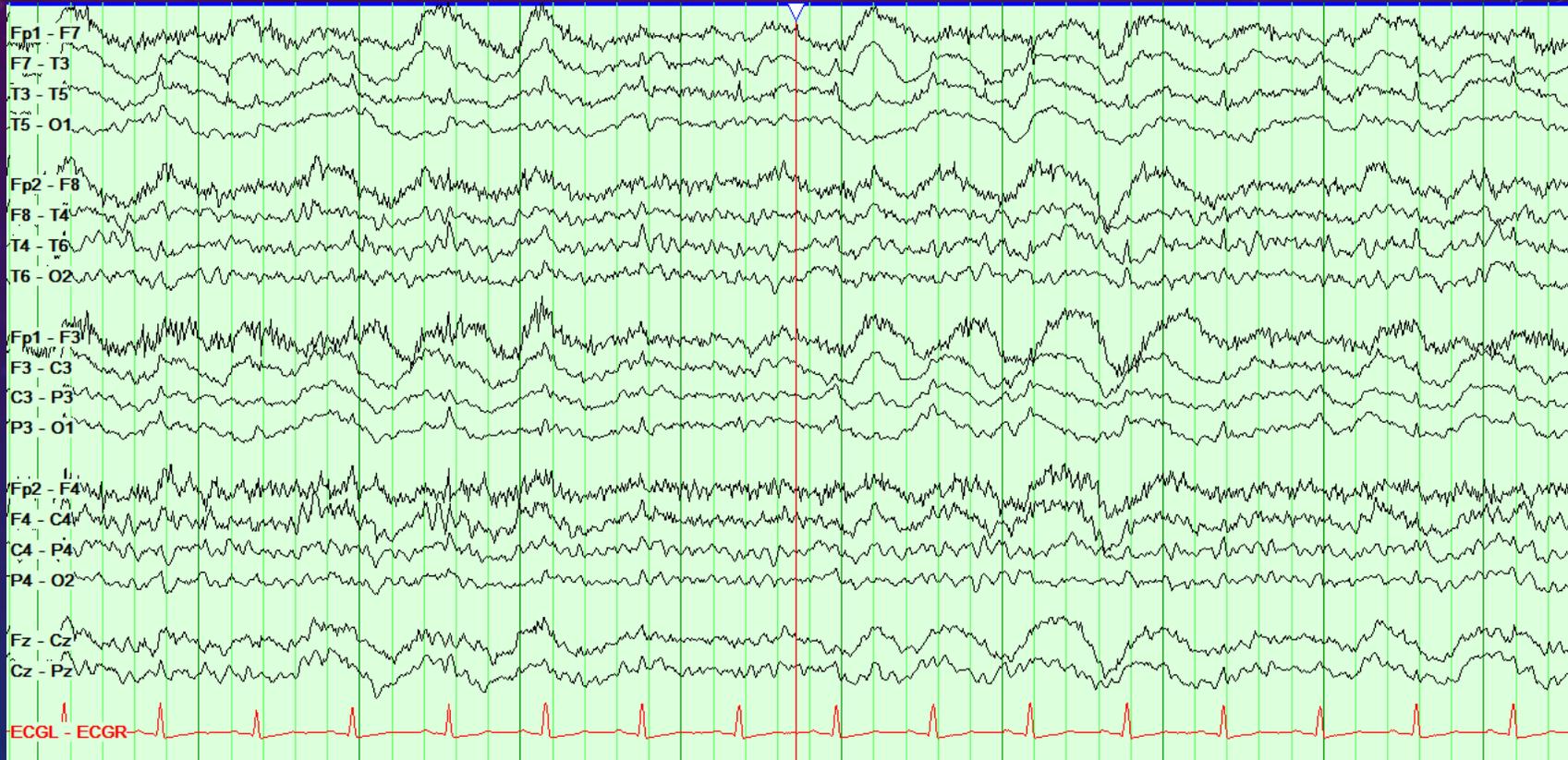
Focal, arrhythmic polymorphic slowing usually implies a focal subcortical white matter lesion.

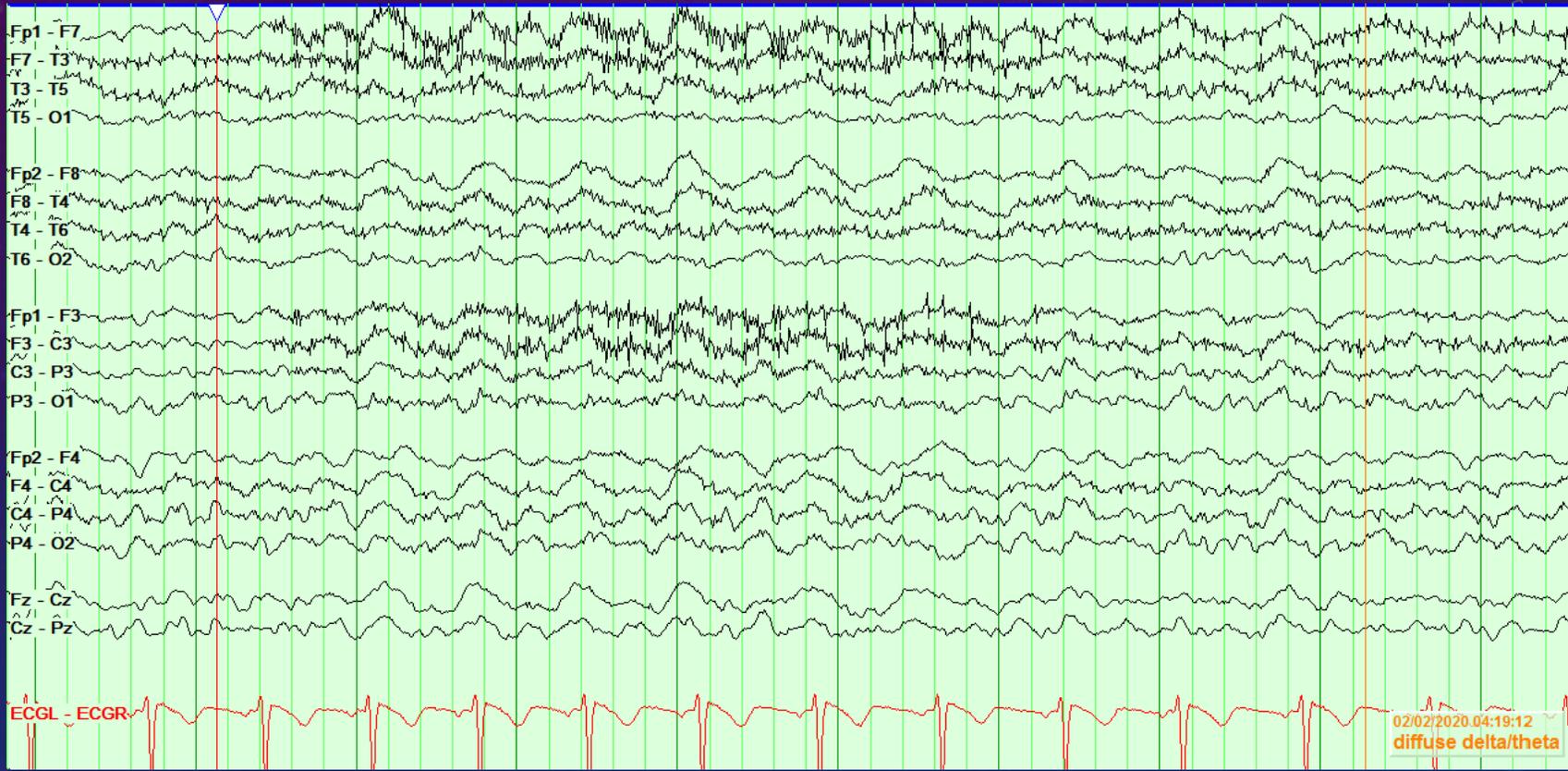
Focal, rhythmic monomorphic slowing usually suggests a lesion of the underlying gray abnormality.

IRDA usually signifies an acute or subacute process/  
diffuse slow waves are subcortical in format with characteristic

triphasic morphology. mainly occur in metabolic encephalopathies, usually in hepatic insufficiency, but are not exclu







- Studies suggested that thalamic deafferentation from the cortex rather than cortical deafferentation from below may be the slow wave mechanism

# GENERALIZED ASYNCHRONOUS SLOW ACTIVITY —

- Generalized asynchronous slow activity consisting of frequencies less than 4.0 Hz is highly nonspecific and has a broad differential diagnosis