

EEG Patterns of Unclear Significance & Nonepileptiform EEG Abnormalities: Highlights

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The difficulties of interpretation posed by these variants arise for several reasons. These patterns possess features that are similar in some ways to abnormal patterns. These variants can be challenging, and may commonly lead to misinterpretation. One important source of misinterpretation of these patterns may simply be that many of these patterns are uncommon¹. A significant number of clinicians who read EEGs in clinical practice may have only 2 to 3 months of closely supervised EEG training.

The chances of seeing a rare pattern are quite low during this short training. Without extensive preparation in the fundamentals of electroencephalography and extensive supervised experience, misinterpretation of a rare pattern would not be surprising.

Patterns of unclear significance

Encompasses a selection of electroencephalographic findings that may look abnormal, but usually are not. These variants² can be challenging, and may commonly lead to misinterpretation. These variants can be mainly grouped into two types:

Variant rhythms & variant transients, in combination or not with slow waves, and strongly associated with epilepsy.

When the non epileptiform EEG abnormalities are seen in an EEG record, they are not specific for an underlying etiology. EEG provides evidence of organic electrophysiological dysfunction and the patterns observed may orientate for the diagnostic possibilities

³The non-epileptiform EEG abnormalities may be characterized by several distinct patterns of normal expected for a given age and condition of the patient. They consist mainly of

- 1) Focal slow activity-It was first described by Walter⁴,
- 2) Regional or generalized bisynchronous slow activity⁵
- 3) Generalized asynchronous slow activity⁶
- 4) Focal attenuation⁷
- 5) Generalized attenuation / suppression⁸
- 6) Other abnormal activities (alpha, theta and spindles coma patterns, etc.).

One should be aware of these abnormalities, especially regarding its presence and clinical significance in the context of the neurological disorders.

Rhythms:

A few common features help distinguish these rhythms from definitely abnormal findings:

- 1) The variant rhythms are, for the most part, monomorphic. The term “monomorphic” simply means that each of the waves in the rhythm looks distinctly like the other waves in the rhythm. The waves have a characteristic shape, or “morphology,” that repeats.
- 2) Most of the variants have an arch-shaped appearance, although some of the normal rhythms may have notches in some or all of the waves.
- 3) Most importantly, the variant rhythms for the most part do not evolve. A particular challenge is the tendency toward subject bias.

Fast Alpha Variant/ Slow

Alpha Variant

The appearance is of a rhythm that is twice the frequency of the awake background; it is referred to as a “fast alpha variant”. A “slow alpha variant” has an apparent frequency that is about half that of the awake background. These harmonics will often have a notched appearance, as if many of the waves are simply being cut in half, but only partly so.

Slow and fast alpha variants are reactive to eye opening and closure

Fast alpha variant is easy to detect, since awake posterior background rhythms in the mid to upper beta range would be quite unusual.

True beta rhythms are most often seen in the frontal, central, and parietal regions. When a posterior background rhythm of 16, 18, or 20 is seen, it is easy to think of FAST ALPHA VARIANT.

However, slow alpha variant is more challenging, and may easily be misinterpreted as occipital slowing. This tendency to misread slow alpha variant as abnormal stems mostly from the fact that slowing is such an easily recognized abnormality, and all electroencephalographers look for signs of slowing. As a general helpful rule it is good to remember that any time one sees occipital slowing, one must think of slow alpha variant first, before calling the record abnormal. One should look to find a sample of normal awake background within the record. If the slowing is approximately half the frequency of the patient’s awake background, then the slowing is probably just a SLOW ALPHA VARIANT. An additional clue can be small notches in some of the “slow waves,”

Alpha Squeak⁹

Immediately on eye closure, the awake background is sometimes initially faster, and of lower voltage. Over approximately 0.5 to 2 seconds, the background slows, and increases in amplitude to become the normal awake background. This initial “speeding up” of the background immediately on eye closure is referred to as an “alpha squeak,” and is a normal finding.

FOCAL SLOW ACTIVITY: May be an indicative sign of focal cerebral dysfunction, especially in awake adults

Due to the result of a cortex differentiation from subcortical structures.

First described by Walter in 1936 who proposed the term delta waves for focal slow activity associated with tumours involving the cerebral hemispheres.

It is the most common phenomenon encountered in clinical EEG that is indicative of local structural lesion. FOCAL activity irregular in the delta frequency.

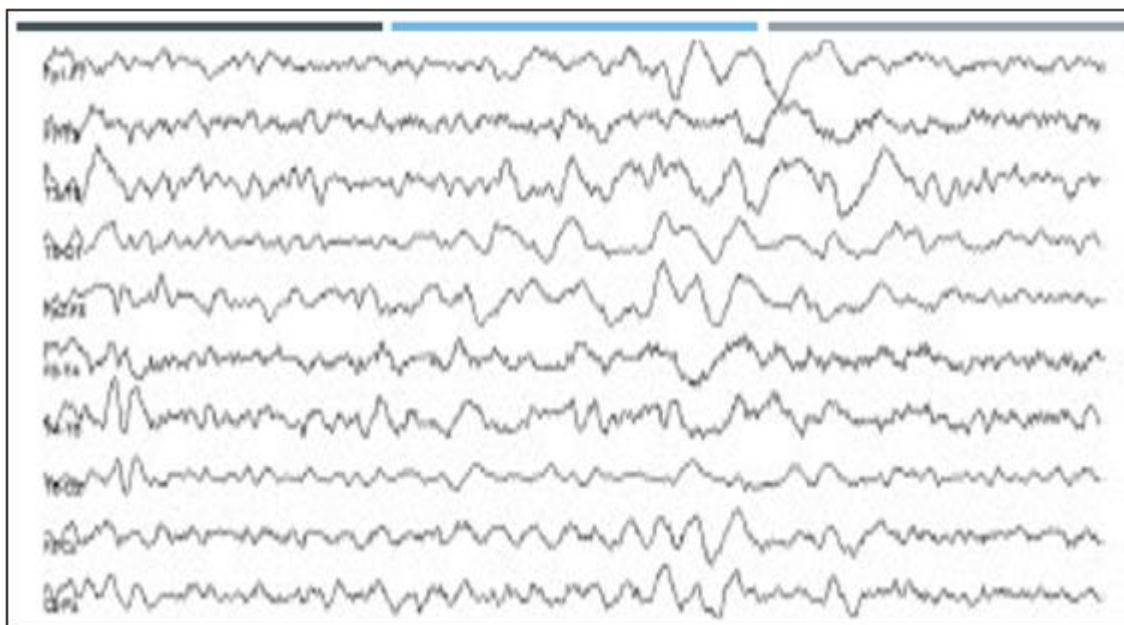
Due to a structural lesion or subcortical dysfunction

Focal slow activity is assessed with regard in amplitude, frequency, topography, persistence and reactivity

Reactivity is the most reliable indicator of dysfunction.

Persistence of focal abnormalities (continuous vs. intermittent) is a better indicator of degree of damage.

Continuous slow activity suggests a more severe brain damage-likelihood of increased mass effect, large lesion or deep hemispheric lesion.



- Gloor et al., investigated the location of structural pathology that produced localized, lateralized or generalized EEG slow activity. They found that cortical gray matter lesions alone did not produce slow activity, probably because the pure cortical lesions presumably destroy the neuronal generators located in the cortex, and localized lesions of subcortical white matter may cause irregular delta activity in the cortex overlying the lesion.
- Thalamic lesions generally produced focal or unilateral delta activity, but the slow activity varied in time of onset, amplitude and degree of focality
- Bilateral hypothalamic and bilateral mesencephalic lesions produced bilateral slow waves. The observations that cortical lesions failed to produce delta activity, but that interruptions of the afferent input to the cortex either in white matter, thalamus, hypothalamus or mesencephalon produced delta activity, suggest that some type of deafferentation of cortical neurons may be responsible for slow activity

Generalized or regional bisynchronous slow activity

Generalized or regional bisynchronous slow activity may be intermittent or continuous, and seems to be due to disordered circuits between the cortex and thalamus, although there has been some controversy about its genesis and significance

This type of abnormality can be found in conditions that affect both cortical as subcortical structures, as well as the presence of several toxic-metabolic encephalopathies, early stages of coma and deep midline lesions.

Even when generalized projected, it usually predominates in a region.

In most cases, it occurs in a rhythmic and intermittent manner, and the most commonly and important types are frontal intermittent rhythmic delta activity (FIRDA), occipital intermittent rhythmic delta activity (OIRDA) and temporal intermittent rhythmic delta activity (TIRDA)

FIRDA¹⁰ frequency is around 2.5 to 3.0 Hz, initially attributed to deep midline lesions and posterior fossa

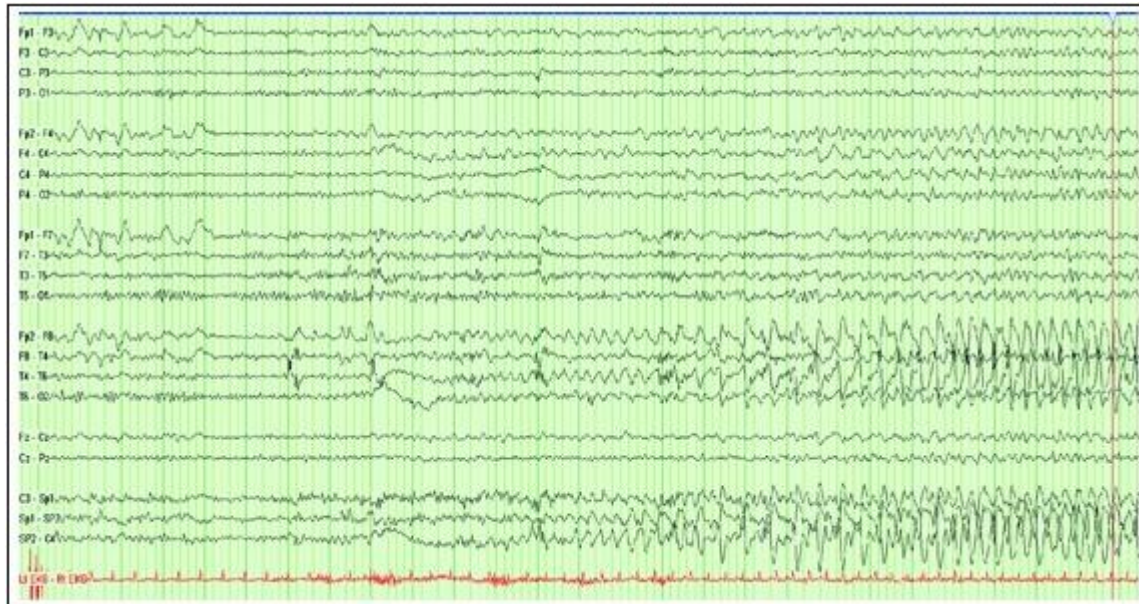
tumours, FIRDA and increased intracranial pressure, toxic-metabolic encephalopathies, early stages of coma, degenerative diseases

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

TIRDA¹¹

Defined as short bursts or trains of 3 seconds or more of repetitive, rhythmic, saw-toothed or sinusoidal 1.0 to 4.0 Hz activity of 50-100 µV in amplitude, predominantly over the anterior temporal region

It has a strong association with hippocampal atrophy and mesial temporal sclerosis in patients with temporal lobe epilepsy, but may infrequently occur in extratemporal epilepsy.

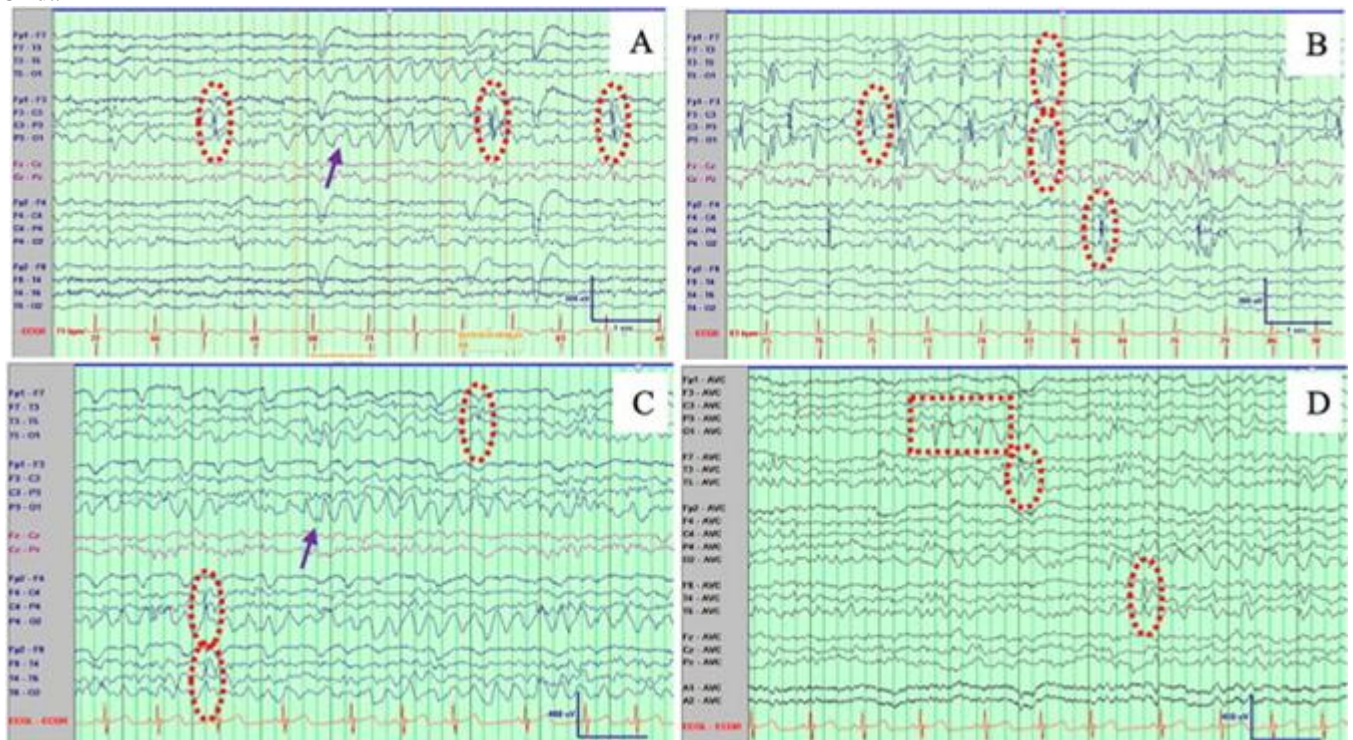


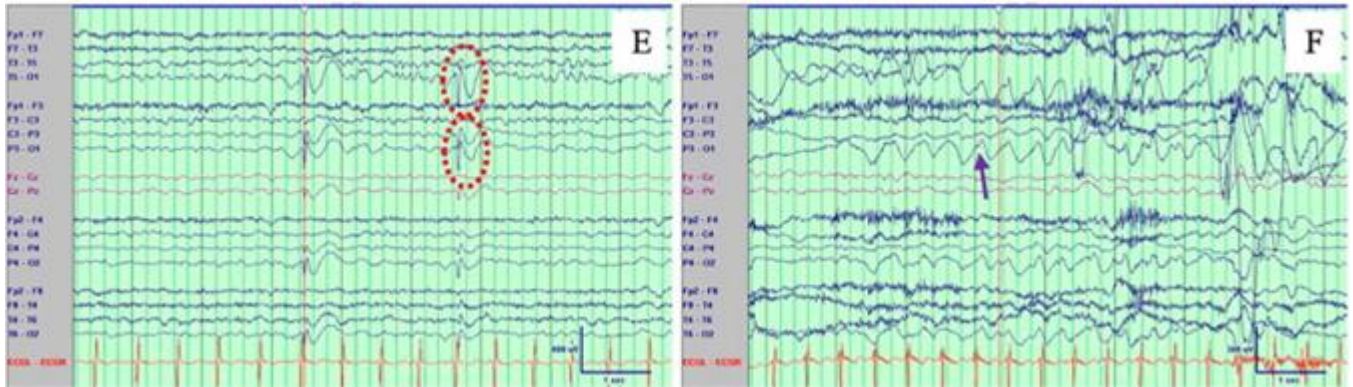
TIRDA

In patients with absence seizures and tonic-clonic seizures are more frequent in children with OIRDA, and its presence in children with typical absence seems to be a good prognostic factor.

When the EEG shows unilateral FIRDA or OIRDA, it generally indicates a focal than diffuse disturbance

Oirda





When it is due to a focal lesion and presented as bilateral and symmetric, this lesion classically is near the third ventricle, diencephalic or mesencephalic midline, brainstem or cerebellum^{15, 26} fast-growing tumors, such as glioblastoma multiforme or metastatic brain tumors, are associated with focal slow activity occurring in the delta frequency

Slow-growing tumors, such as meningiomas, are usually associated with focal slow activity that occurs more frequently in the theta range

TIPDA

Some epileptic focus can produce irregular focal slowing, associated or not with structural lesions, as well as postictal slowing after a focal seizure

A non-rhythmic, temporal intermittent polymorphic delta activity (TIPDA) may occur interictally in patients with temporal and extratemporal epilepsy with an equal frequency of 19% and, when lateralized, is an excellent indicator of the side of the epileptogenic focus.

Generalized asynchronous slow activity

Consists of frequencies less than 4.0 Hz is highly nonspecific and its presence usually indicates encephalopathy and is always abnormal in awake adults.

Some possibilities include degenerative processes, encephalitis, extensive multifocal vascular diseases and toxic-metabolic encephalopathies.

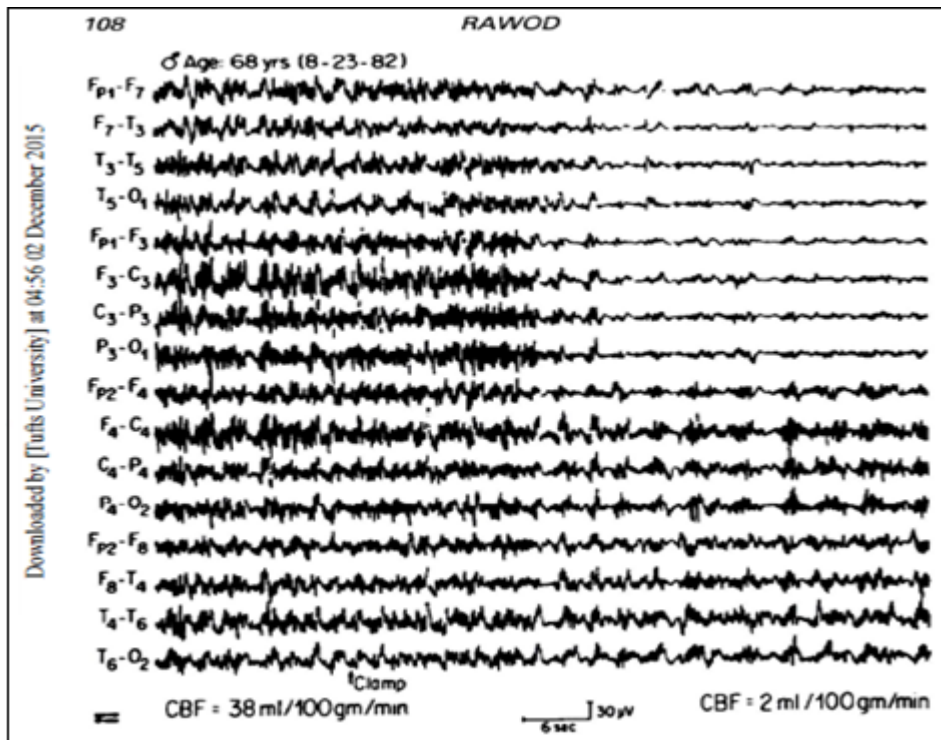
It should be considered an abnormal activity when the pattern is inconsistent with age and stages of sleep.

Focal attenuation

Attenuation indicates reduced amplitude of one type of activity that occurs at certain frequency, or of the entire EEG activity. Attenuation generally indicates focal cortical lesion or reversible cortical dysfunction (post-ictal state, for example), but may be related to the presence of a collection between the cortex and recording electrode (like an hematoma or subdural empyema) or a tumor (a dural based tumor, such as a meningioma, for example), leading to an increased distance between the cortex and the recording electrode. Other common causes include cerebral ischemia, post-ictal states (arising from a crisis of focal onset), swelling of the scalp and subdural collections.

RADWOD¹¹ (regional attenuation without delta)

EEG in acute ischemic stroke can reveal a distinctive EEG pattern called regional attenuation without delta (RADWOD)



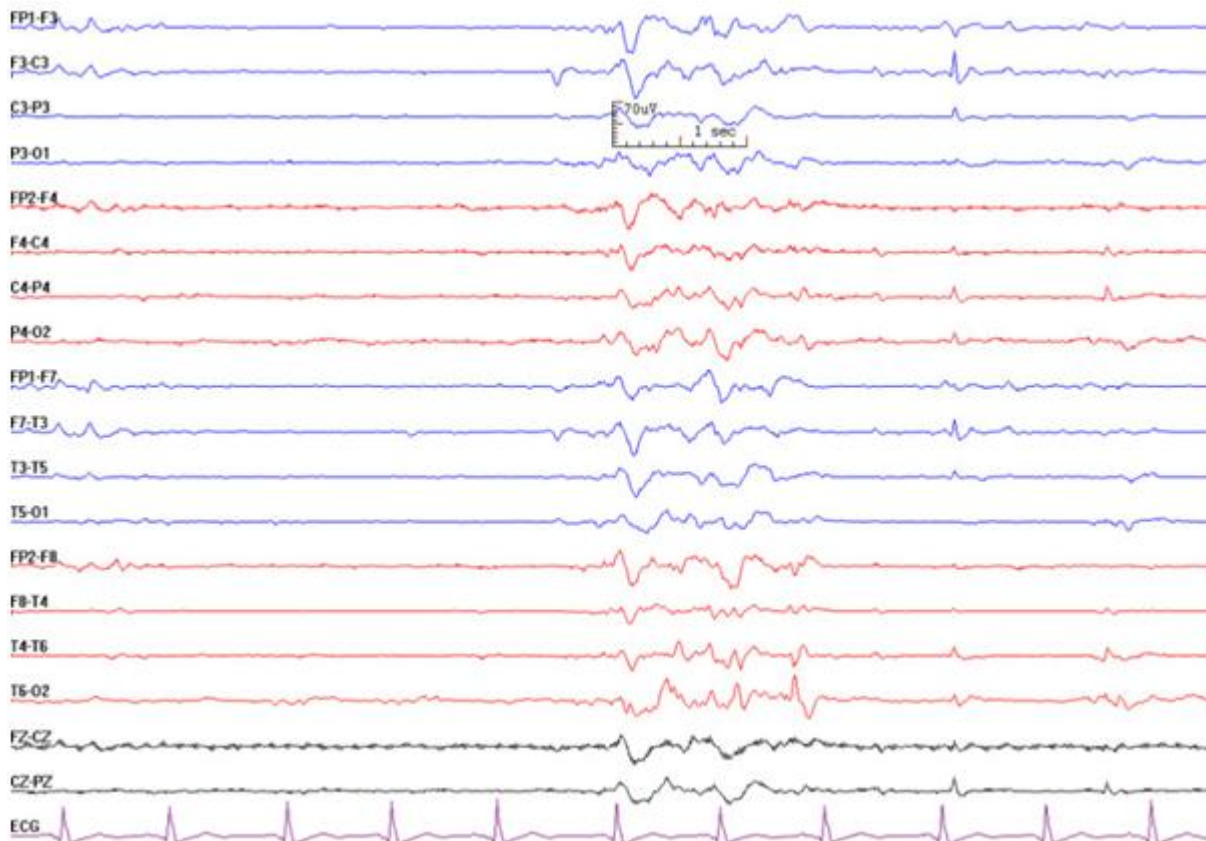
This finding suggested that patients with RADWOD may be candidates for early intervention for cerebral edema due to acute ischemic stroke, but they are unlikely to benefit from thrombolysis

Generalized attenuation / suppression attenuation suggest cortical generalized injury or transitory dysfunction 13. However, an attenuated EEG in adults may be a normal

variant if the pattern is constituted by a reactive generalized beta activity, less than 20.0 μV in amplitude.

Suppression corresponds to a state worse than attenuation, and it indicates complete or nearly complete disappearance of electroencephalographic activity.

Burst-suppression¹²



The term burst-suppression refers to the presence of brain activity bursts of variables amplitude, duration and form, followed by a marked depression of the activity, which occur on a cyclical basis.

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In the burst portion of this pattern, sharp waves and spikes are usually present. In the suppression period, or interburst interval, there is absent or severely attenuated activity of delta and theta frequencies. Severe cerebral damage in

postan-oxic encephalopathy, under the effect of anesthetic drugs or drug-induced coma, period following the generalized tonic-clonic *status epilepticus*, in which the prognosis is better.

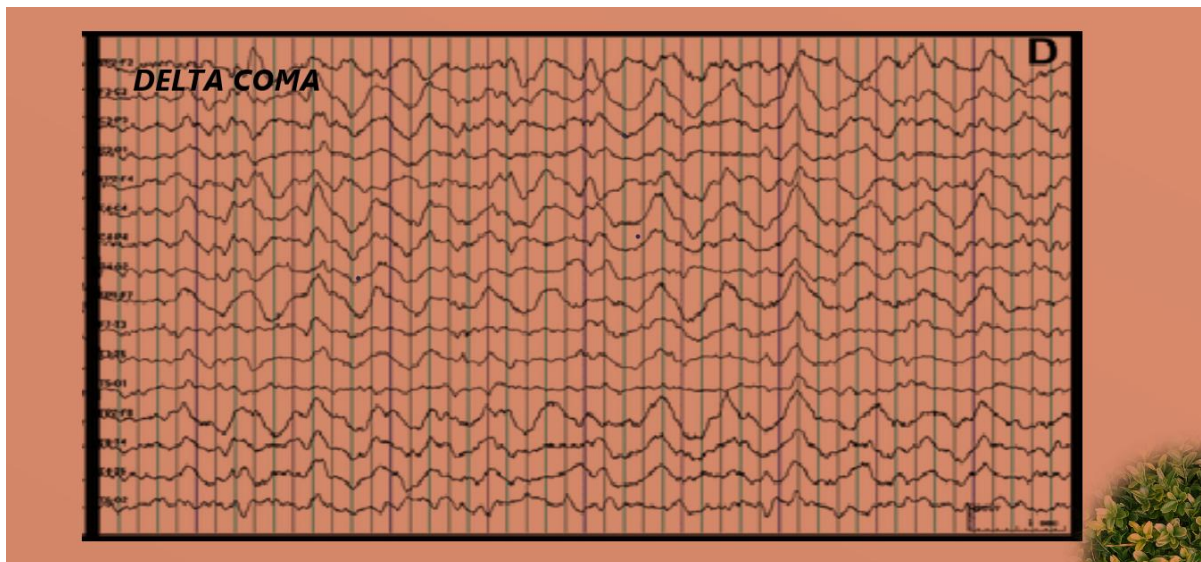
The EEG patterns in coma states are not specific with regard to the etiology or prognosis¹⁷. However, it is known that the prognosis is worse if the etiology is hypoxic-ischemic encephalopathy¹⁸. The presence of reactivity to passive eye opening, auditory or nociceptive stimuli is early indicative of more favorable prognosis, especially in comatose patients after a severe head injury¹⁹

BETA COMA



ALPHA COMA





THETA COMA



The alpha coma¹⁴ is defined as the appearance of EEG activity predominating in the alpha frequency band (8.0 to 13.0 Hz) in unconscious or comatose patients.

In patients after cardiac arrest and postanoxic coma, who had a poor outcome. It is usually have two main EEG patterns, one of this being associated with a generalized brain dysfunction, in which the alpha activity tends to have a widespread distribution, sometimes with frontal predominance, and not reactive to stimulation. This pattern is typically associated with hypoxic-ischemic encephalopathy.

In another one pattern, the alpha activity tends to have a posterior predominance and is often reactive to a passive eye opening and closing. This pattern is generally associated with pontine lesions.

THETA COMA

Variant of alpha coma, emphasizing its association with a poor prognosis. The designation of theta coma pattern is used when an activity widespread, persistent and non-

reactive in the theta frequency is present in the EEG of a comatose patient.

ALPHA-THETA COMA: The transition from alpha to theta coma, or vice-versa, and the coexistence of both patterns in some patients with postanoxic coma, hypothesized a common pathophysiologic mechanism.

SPINDLES COMA:

EEG shows activity resembling sleep spindles in unconscious or co-matose patients. It is generally characterized by spindles in 9.0 to 14 Hz range, often with vertex sharp waves and K-complexes. These spindles are, however, much more diffuse in distribution than normal sleep spindles

Spindles coma was first described in 1953, by Jasper et al., in a patient with neoplasia involving the midbrain near the third ventricle. It was subsequently found with several pathologic conditions, such as head injury, ischemic and hemorrhagic strokes, encephalopathy

SMALL SHARP SPIKES (SSS),

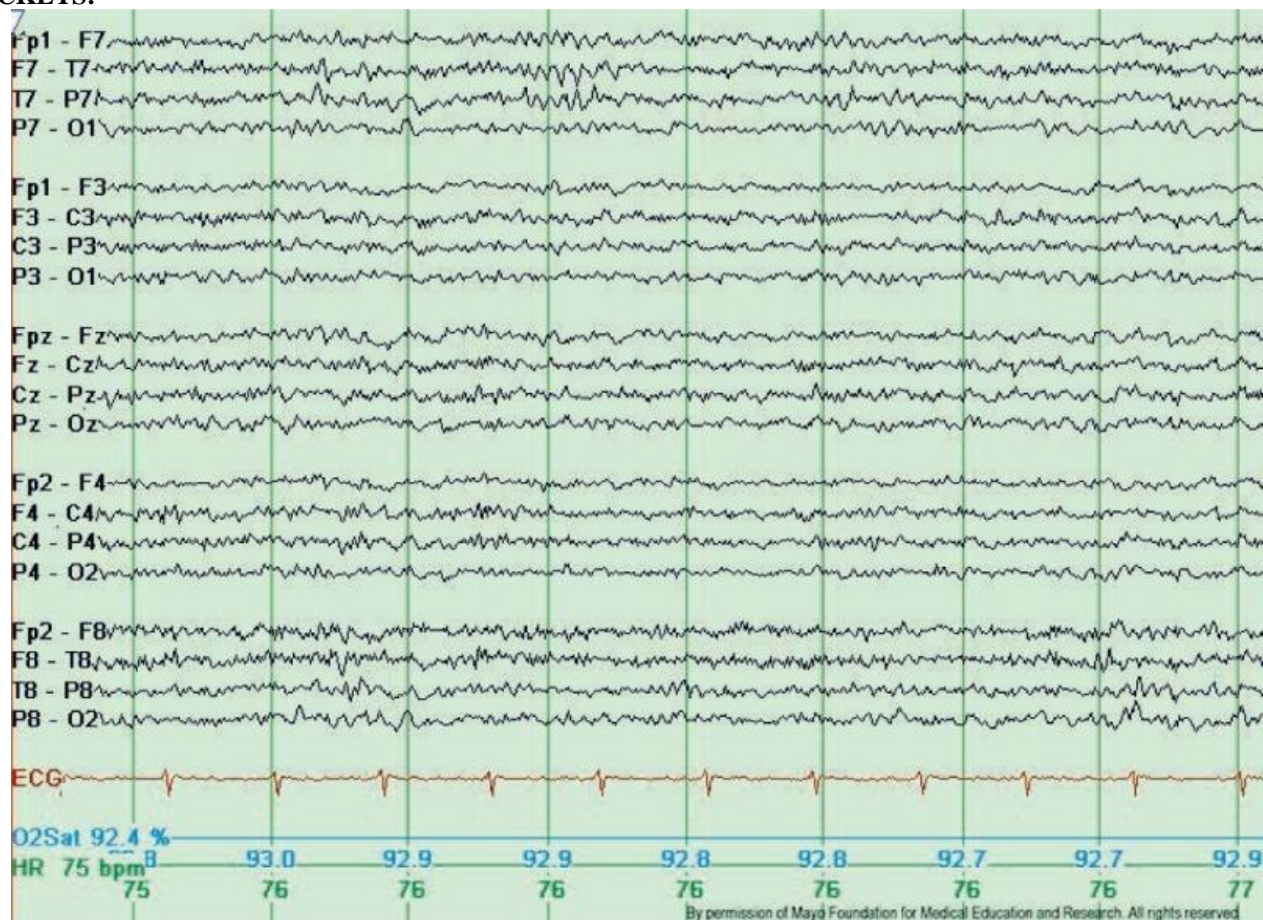
The term originally described by Gibbs and Gibbs, are also known as benign epileptiform transients of sleep (BETS) or benign sporadic sleep spikes (BSSS).

These are short in duration and low in amplitude. Although they are usually less than 50 msec and less than 50 V, duration can be slightly longer and amplitude can be slightly higher depending on the recording circumstances and montage used. The shape is rather simple and consists of mono-or diphasic spike with steep descending arm.

An after going slow wave is not prominent and usually is lower in amplitude than the spike component. Background activity at the region of BSSS is not disrupted. These discharges are best seen during drowsiness and light sleep (stage I and II) and are seen in anterior and mid temporal

orear electrodes. Although they have been reported in children and adolescents, BSSS are mainly seen in adults. Accepted as a variant of normal EEG activity. However, over the years, several reports tried to find a connection between BSSS and seizures or certain psychiatric conditions. BSSS were thought to be a sign of epileptogenicity. There is a higher incidence of BSSS in patients with seizures particularly in patients of younger age BSSS and certain psychiatric conditions. They are very common EEG findings seen with up to 25% incidence in the normal population. Although several studies reported increased frequency of BSSS in certain psychiatric conditions and seizures because of their high incidence in normal population, we believe this peculiar EEG activity is of unknown clinical significance with no statistical relationship to a certain disease.

WICKETS:



Wickets are archiform waveforms first described as wicket spikes by Reihner and Lebel in 1977. They are simple monophasic waveforms with surface negativity. Initial descriptions revealed that wicket spikes are found in both awake and sleep EEGs. Because of the intermixed background activities during the awake state, they are better recognized during sleep. They are best seen during initial stages of sleep but also reported during REM sleep.

Their location remains unchanged during different stages of sleep. Wickets can occur as isolated "spikes" or they may come in runs. If they occur in runs, the usual frequency is between 6 and 11 Hz. The amplitude of a wicket spike may range between 60 and 210 V and is maximally expressed

over the temporal regions. Although they shift sides, often one side is more dominant than the other.

Wickets are usually seen in adults. They are almost exclusively reported in adults 30 years or older; however, cases as young as 20 years of age have also been reported.

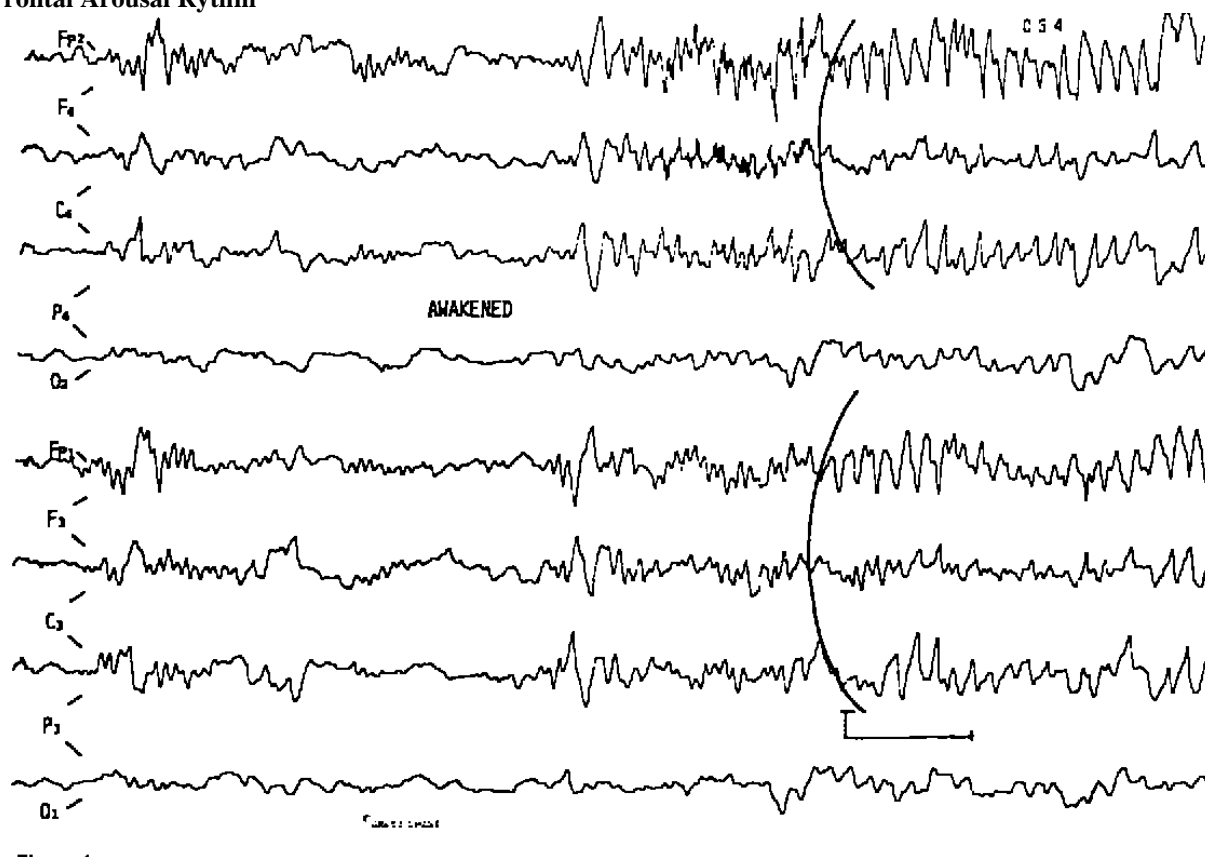
Wickets are reported to be an uncommon EEG pattern. Their incidence in large EEG series has been reported to be less than 1%, ranging between 0.03% and 0.96%. In adults 30 years and older this incidence may rise up to 2.9%. Wickets are considered to be a normal variant. In Reihner and Lebel's original description, wickets occurred four times more in patients with no history of seizures than in those with a An

experienced electroencephalographer can differentiate wickets from temporal sharp waves easily by the lack of after going slow waves and the absence of distortion of the background activities. However, one can also over-read or misinterpret wickets as epileptiform sharp waves, especially if they occur in isolation. Clues mentioned above, as well as observation of runs of wicket spikes somewhere else in the same EEG, should alert the reader that this is a non-epileptiform variant

FRONTAL AROUSAL RHYTHM (FAR)

This unique EEG pattern was first described by White and Tharp in 1974 although the pattern was first recognized in 1969. In their original report, the authors described 8 cases out of 4780 EEGs reviewed over a 4-year period. This group of eight children consisted of four males and four females with a mean age of 6.6 years, ranging from 2 to 14 years.

The Frontal Arousal Rhythm



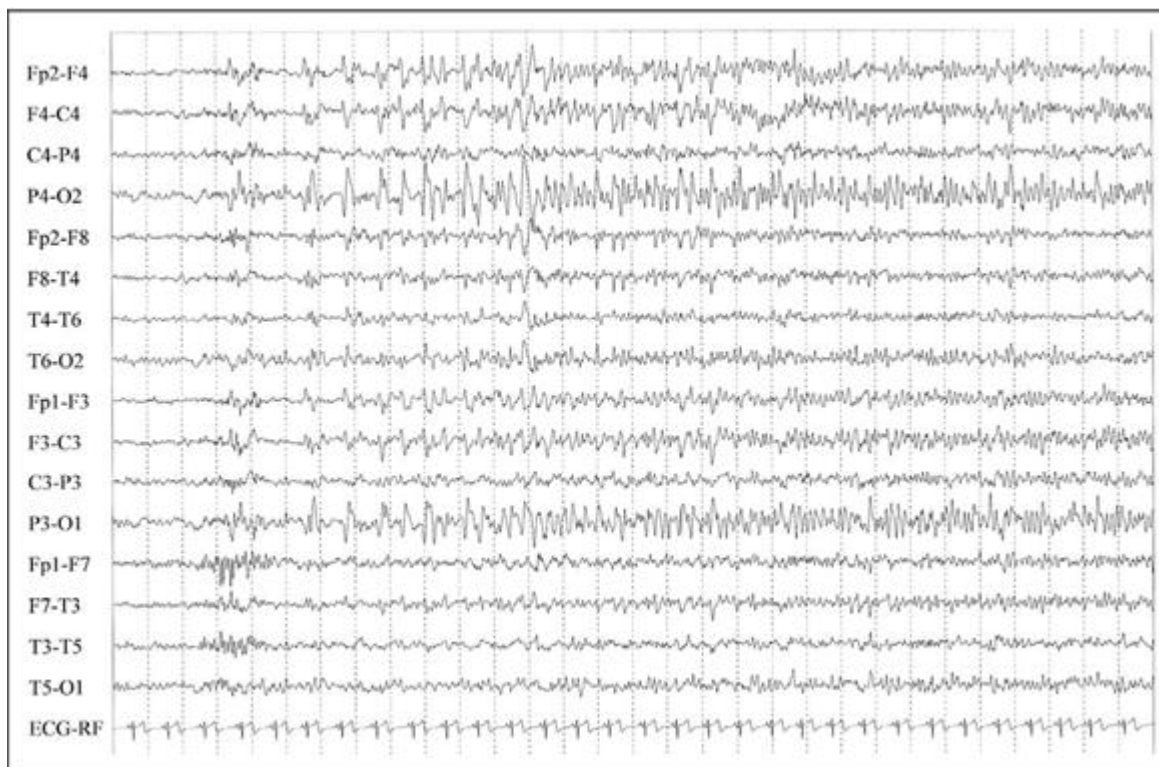
While FAR was the only atypical pattern in 58% of these patients, nearly half (42%) also showed other abnormalities, including focal and generalized epileptiform activities. Clinically, 70% of the children had a seizure disorder, and 56% had both seizure and cognitive/behavior disorders. Only 6% of the reported patients had neither seizure nor cognitive/behavior disorders. However, the incidence of cognitive/behavior disorders was not statistically different from their control group whose EEG did not show a FAR pattern. Their conclusion was that FAR is related to seizures. To support this view, a recent case report described a 6-year-old boy with mental retardation and possible seizure disorder. A typical FAR pattern was seen during few episodes associated with eye fluttering followed by chewing, increased inspiration, and upper lip quivering. These

Their initial diagnoses were mild cerebral dysfunction and/or seizure disorder. Representative EEG samples showed normal awake and sleep patterns. However, during arousal from stage II sleep, a 6.5-to-8.5-Hz rhythm was seen over the frontal regions involving mainly F3 and F4 electrodes with no or little spread to adjacent regions. The overall amplitude of this activity was between 30 and 150 V, with duration of up to 13 seconds. This pattern was originally described in children with evidence of minimal cerebral dysfunction and seizures but authors noted that the incidence in a normal population was unknown at the time of the publication. Twenty-five years after the initial description of this rare and unusual EEG pattern, Hughes and Daaboul discussed their findings in 50 cases. The incidence of FAR was 0.22% in their series of 22,500 patients seen over an 8-year period. The characteristics of the pattern were similar to those described before.

episodes were all recorded during arousal from sleep and there were no clinical changes seen when the patient was awakened without the FAR pattern present. Few illustrative reports are available in the literature. The incidence of FAR in general population is unknown. The general consensus is that FAR is more of a non-specific EEG pattern with unknown clinical significance. However, the reported case series as well as demonstration of FAR as an ictal pattern in a reported case warrants careful review for other interictal activity, and detailed questioning for possible seizure-like activity.

SREDA (Subclinical Rhythmic**Electrographic Discharge in Adults**

SREDA is a fairly rare pattern that can be easily misinterpreted.



It is perhaps the most challenging pattern among those that are considered normal variants. First described in 1961 by Naquet et al., this pattern was originally thought to be associated with hypoxia. Westmoreland and Klass described 65 patients who had 142 EEG recordings, and provided the term “Subclinical Rhythmic EEG Discharge in Adults.” In this series, SREDA was seen in patients aged 42 to 80, with an average age of 61 (The pattern is seen mostly during relaxed awake or drowsy states, although SREDA can be seen during sleep). The typical appearance of SREDA is a diffuse, sharply contoured, theta rhythm that is usually maximal in the temporal and parietal regions. While SREDA is usually bilateral, it may be asymmetric. An interesting feature of SREDA is that it often occurs in long runs, such as 15 seconds to a minute or more. The appearance on visual inspection is of a monomorphic rhythm. However, a more recent detailed digital study using frequency spectral analysis and Laplacian montages suggests that SREDA is “composed of a complex mixture of multiple rapidly shifting frequencies predominantly in the theta range, which show poor spatial and temporal resolution”

This particular pattern has a distinctive feature that is different from other non-epileptic variants and that makes it especially difficult to recognize and easy to misdiagnose. The SREDA pattern may initially *evolve*. SREDA may begin with a single sharp transient or a series of sharp transients, initially at delta frequencies, and then increasing to theta, before becoming a well-defined 4-to 7-Hz frequency range. A smaller number of cases remain in the 2-to 4-Hz frequency range. After the initial period in which the rhythm

is established, however, SREDA does not continue to evolve, despite the fact that SREDA may be seen in prolonged runs of 20 seconds to several minutes. The rhythm ends abruptly in about half of patients, and in the other cases, the SREDA gradually dissipates and merges with ongoing normal background rhythms. Unlike seizures, SREDA is not followed by “postictal” slowing, and no clinical changes accompany SREDA. While this pattern may easily be mistaken for an ictal pattern, evidence suggests that this pattern has no clear association with seizures. In a large series, there was not only a low incidence of clinical history of seizures or subsequent development of seizures, but also a “uniform lack of any clinical accompaniment during the bursts, even with the involvement of both hemispheres and persistence of several minutes”. One study of ictal single photon emission computer tomography demonstrated no significant changes in uptake during the bursts, further suggesting that the pattern is not ictal.

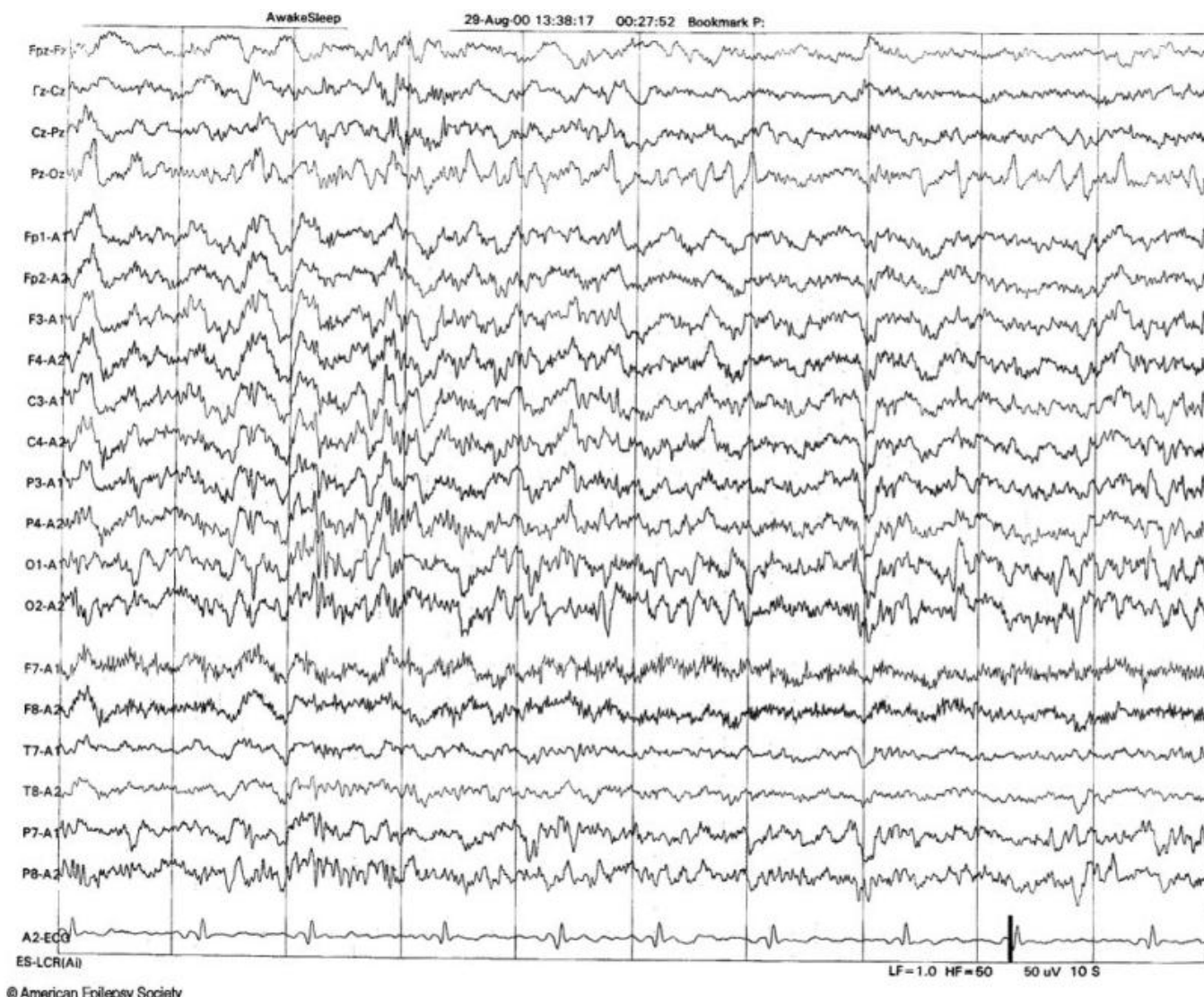
14-AND 6-HZ POSITIVE BURSTS

Like many other variants, this unique EEG phenomenon was also described by Gibbs and Gibbs. Although it was previously called 14-and 6-Hz positive spikes, the current preferred term is 14-and 6-Hz positive bursts. Lombroso et al. also proposed the name “ctenoids” due to their appearance and complex morphology resembling a comb (ktenos in Greek translates to “comb”).

The typical bursts last between 0.5 and 1 second and usually occur during light sleep or drowsiness. However, a few cases with occurrence in REM sleep have also been

reported. The bursts can be unilateral or bilateral but usually show a shifting predominance if enough bursts are recorded in the same patient. They are usually small in amplitude; therefore, referential or long-distance electrode montages reveal better waveforms. Although the laterality of these bursts can appear to shift, the maximum amplitude is

expressed over the posterior temporal head regions. There seems to be strong age predilection. This activity initially starts to appear during early childhood and reaches its peak during adolescence. The coexistence of 14- and 6-Hz positive bursts and 6-Hz spike and wave bursts was also reported.



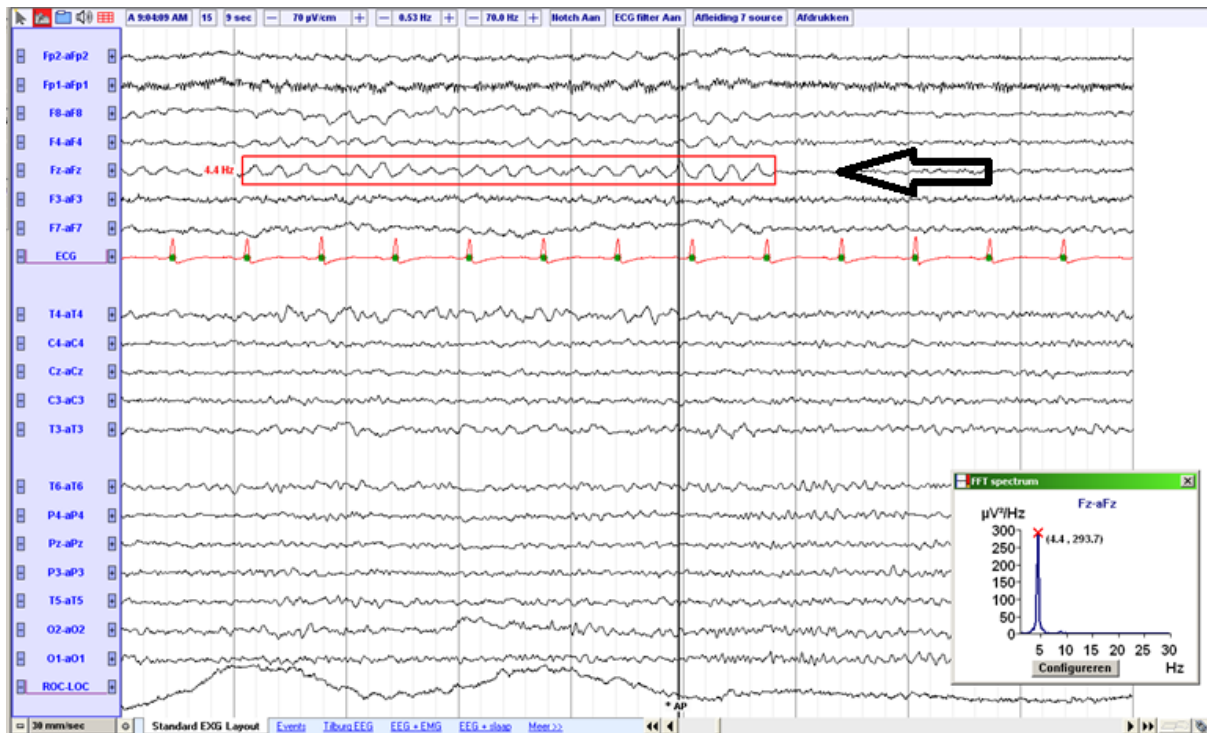
Silverman hypothesized that a maturational sequence might be responsible for this co-occurrence and that most of the 6-Hz spike and wave bursts are part of the 14- and 6-Hz positive bursts. The 14- and 6-Hz positive bursts are still seen during adulthood but the incidence decreases with advanced age. Lombroso et al. reported highest incidence of 58% in the literature in teenage boys between the ages of 13 and 16 years. In adults, the incidence is much lower. An incidence as low as 4% was seen in a group of psychiatric patients, whereas a study of EEG in asymptomatic normal adults revealed a rate around 12%. In fact, the latter claimed that 14- and 6-Hz positive bursts were the most common pattern of uncertain significance in their series of 100 asymptomatic adults.

Several reports were published in the literature correlating the existing of this pattern with certain symptoms and illnesses. Wide variety of psychiatric conditions and

neurovegetative symptoms were subjected to research. Epilepsy was also thought to be more frequent in patients with 14- and 6-Hz positive bursts. However, most of the patients with behavioural symptoms and organic CNS impairment were children or adolescents, which is the age of peak incidence for this phenomenon. One study suggests that a majority of patients with seizures and 14- and 6-Hz positive spikes also showed other epileptiform abnormalities in their EEGs.

This controversial pattern seems to be an age-related phenomenon. Although there are arguments regarding a pathological significance, the reported incidence of up to 58% in normal teenagers and 12% in healthy adults makes this relatively hard to prove.

CIGANEK RYTHM



SIX-HZ Spike and Wave Bursts

Six-Hz spike and wave bursts were first described in 1950 as “wave and spike phantom”. The first collected cases revealed a unique EEG pattern of 1-second bursts of 5-to 6-Hz spike and waves. The average amplitude of the discharge was low, around 25 V or less, with the spike component less than 30-msec duration. This pattern can be seen in both children and adults, with amplitude usually higher over the fronto-central regions. These bursts were mostly Subclinical electrographic discharges of adults (SREDA).

Part III

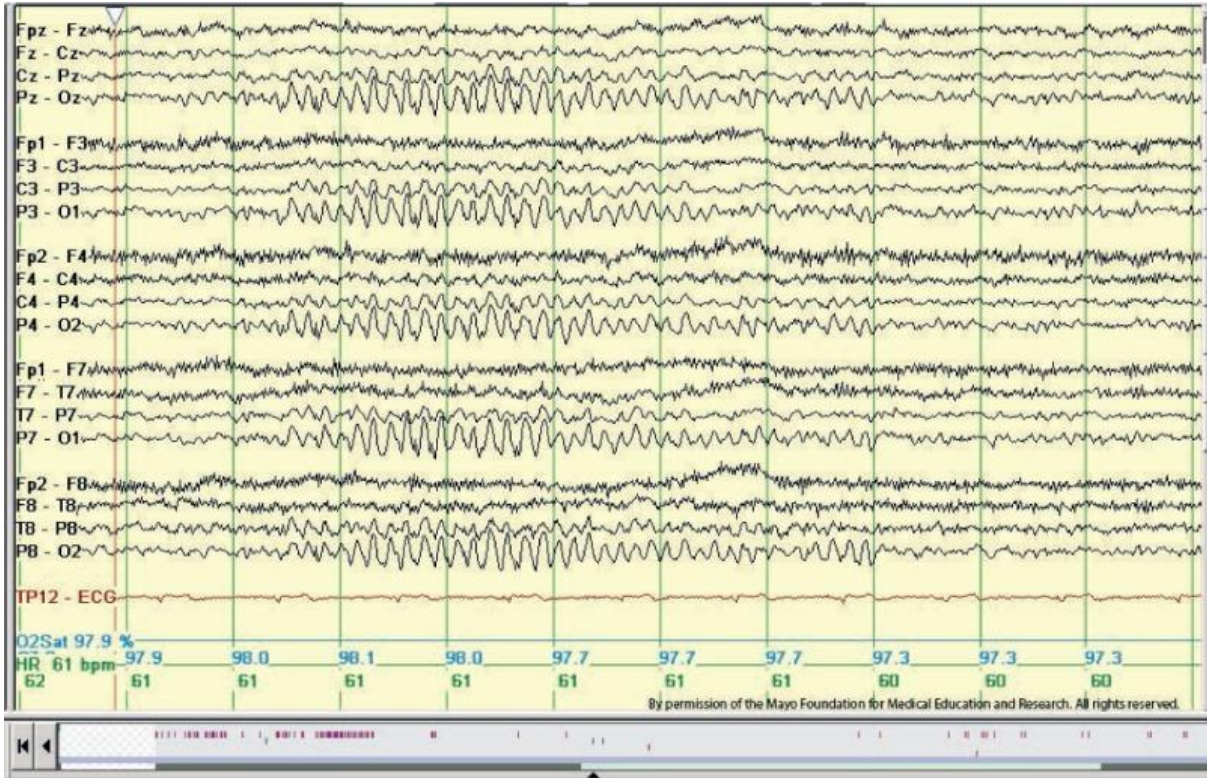
Clinical EEG: Generally seen during drowsiness and light sleep but they were also seen during full wakefulness. Bursts are usually diffuse but may show anterior or posterior predominance. Marshall suggested the comparison linkage that measures the potential difference between analogous scalp areas for better viewing rather than traditional bipolar or referential montages.

Average incidence of 6-Hz spike and wave bursts is between 1% and 2.5% of all EEG recordings.

Several authors tried to link this controversial pattern to specific complaints, ranging from simple fainting to vegetative or dysautonomic pathologies, and from

psychiatric disturbances to seizures. However, occurrence in normal people and absence of clinical symptoms during the bursts make it difficult to label these discharges as a specific indicator of any certain disease. As mentioned earlier, Silverman suggested that there is relationship as a maturational sequence and waveform spectrum between 6-Hz spike and wave bursts and 14-and 6-Hz positive bursts. He claimed that most of the 6-Hz spike and wave bursts are part of the 14-and 6-Hz positive bursts and transition between these two types may lead to occurrence of both patterns in the same person.

On investigating more than 60, 000 EEGs over 30 years, Hughes proposed two distinct subtypes of this pattern. The features of these two variants were summarized by acronyms WHAM (Wake, High amplitude, Anterior location, Male gender) and FOLD (Female gender, Occipital location, Low amplitude, Drowsiness). Hughes indicated that these two variants are more extreme forms, and also suggested that the more components of the WHAM form that exist in a single person, the more likely is the association with seizures. Current consensus is that this EEG pattern is of unclear clinical significance; however, high amplitude of the spikes, slower rate than 6 Hz, and persistence of the discharges in deeper stages of sleep are more likely associated with seizures phantom spikes



Conclusion

The non-epileptiform EEG abnormalities provide evidence of brain dysfunction, be focal or generalized. Many pathological processes can lead to their appearance, which, when properly analyzed, could help the diagnosis. The EEG record should be compared with the medical history,

physical examination, laboratory tests and neuroimaging studies. Data obtained by the literature show that, although these EEG abnormalities are not specific to a particular disorder, they can direct attention to the diagnostic possibilities, indicate additional investigation and guide the treatment choice.



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