Normal electroencephalogram and benign variants

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ABSTRACT

Normal and variant EEG patterns often simulate abnormalities and their misinterpretation can lead to harmful misdiagnoses of epilepsy. Consequently, expertise in these phenomena is fundamental for patient safety. Mastery of normal features must include the Alpha rhythm, Mu rhythm, beta activity, slow activity, posterior slow waves of youth, Lambda waves, hyperventilation and photic responses, drowsy patterns, vertex waves, sleep spindles, K-complexes and positive occipital sharp transients of sleep. Features clearly outside of and unequivocally distinct from the abundantly wide range of normal are possibly abnormal. However, there are several distinctive benign variant patterns that resemble epileptiform abnormalities or seizure discharges, but which are not associated with epilepsy. Clinical diagnosis must exclude these insignificant patterns, which include small sharp spikes, 14 and 6 Hz positive bursts, 6 Hz "phantom" spike-and-wave, wicket waves, rhythmic temporal theta bursts of drowsiness, subclinical rhythmic electrographic discharges of adults, midline theta and frontal arousal rhythm. Proper normal and variant pattern recognition is precise and criteria-based rather than subjective. Because amplitude and sharpness per se are insufficient discriminators, analysis also considers frequency, distribution, morphology, polarity, state-dependence, reactivity and symmetry. This illustrated review summarizes multi-factorial criteria that identify some normal and benign variant phenomena.

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An incorrect electroencephalogram (EEG) report can mislead clinicians to a harmful misdiagnosis of epilepsy. One memorable case illustrates the potential consequences: A young woman who fainted was diagnosed epileptic after an EEG report of temporal sharp waves. Carbamazepine resulted in Stevens-Johnson syndrome, which was fatal. In the medico-legal proceedings, examination of the EEG disclosed only wicket waves. Reliably identifying the wide range of normal and variant EEG patterns requires substantial training. As an introduction for students and a summary for practitioners, this article reviews established criteria for identifying some of these patterns, illustrated with examples from the King Faisal Specialist Hospital & Research Center, Riyadh, Kingdom of Saudi Arabia (KFSH&RC) EEG laboratory. Recordings are displayed with 0.3 or 0.5 – 70 Hz filters and the patients’ age and gender are indicated in the top left corner. Interested readers are also referred to more comprehensive information available in recommended texts and atlases: Kellaway (1990), Westmoreland (1990), Niedermeyer (1993a and b) Blume and Kaibara (1999), Lüders and Noachtar (2000), and Blume et al (2002).

Normal patterns. The normal EEG is a tapestry of overlapping, waxing-and-waning rhythms generated by different cortical regions. Momentarily higher amplitude or isolated single waveform components of this "background" may suggest abnormality, but morphologically "fit in" with ongoing rhythms, thereby being normal. There are also intermittent transient waveforms that may simulate abnormality either alone or in random combination with sharp background components. All these normal features exhibit highly variable amplitude and commonly have sharp or spike-like contours. Therefore, amplitude and sharpness per se are insufficient to distinguish between normal and
abnormal. Rather, this distinction requires a more involved analysis also considering frequency, distribution, morphology, polarity, state-dependence, reactivity and symmetry. Multi-factorial criteria for some normal patterns follow.

**The Alpha rhythm.** Hans Berger first described human alpha frequency (8-13 Hz) EEG activity in the late 1920’s. "The" Alpha rhythm (Figure 1) is a specific example of an EEG pattern within this frequency band. It appears to be generated by "idling" occipital cortex in wakefulness and is recorded in the majority of normal subjects. **Criteria** (Kellaway, 1990; Niedermeyer, 1993a; Blume et al, 2002): An 8-13 Hz posterior (mainly occipital) rhythm of variable amplitude, regulation and persistence. Scalp field commonly extends to parieto-temporal regions and mastoids. Sometimes extends to central or even superior frontal (F4, F3) regions and ears. Frontopolar Alpha is reference (for example mastoid, common average) contamination or eye flutter artifact. Sinusoidal, notched or sharply contoured with a negative peak and rounded positive trough. Present in relaxed wakefulness with eyes closed, relative mental inactivity. Attenuates bilaterally with attention, especially visual (eye opening), mental effort. May persist into early drowsiness; disappears in sleep. Up to ≈ 50% asymmetry (usually but not always higher on the right). **Variations:** Fast: ≈ 2 x Alpha frequency intermingled with Alpha, same distribution and reactivity (Figure 2). Slow: ≈ 1/2 Alpha frequency intermingled with Alpha, same distribution and reactivity. Low amplitude (< 20 µV) to absent ("low-voltage fast" record).

**Mu rhythm.** This rhythm (Figure 1) is also in the alpha frequency band and was described in 1952 by Henri Gastaut as "rythme rolandique en arceau", which aptly depicted its location and morphology. It appears to be generated by "idling" motor-sensory cortex in wakefulness but is inconspicuous or absent in the majority of normal subjects. **Criteria** (Kellaway, 1990; Niedermeyer; 1993a Blume et al, 2002): An alpha-frequency central or centro-parietal rhythm of variable amplitude and persistence. Sharp arciform morphology with a negative peak and rounded positive trough. Present in relaxed motionless wakefulness with eyes open or closed. Attenuates with movement planning or execution or with somatosensory stimuli. May be intermingled with similarly reactive beta frequencies (analogous to the fast Alpha variant). May persist into early drowsiness; disappears in sleep. Commonly shifts from side to side; up to 100% asymmetry can be normal.

**Beta activity.** Also first described by Hans Berger in the late 1920’s, beta activity (>13 Hz) is generated in all cortical regions, but especially the frontal lobes (Figure 3) and is almost always present in normal subjects. It can be misinterpreted when of normal distribution but relatively high amplitude or bursting. **Criteria** (Kellaway, 1990; Niedermeyer, 1993a; Blume et al, 2002): Frontal-dominant > 13 Hz rhythms of variable amplitude, < 20-30 µV in 99%. Should not be generalized and high amplitude (sedatives, some...
encephalopathies). Sinusoidal, sometimes sharply contoured, bursting. Present in wakefulness; may persist or increase in drowsiness and light sleep, subsides in deep sleep. No specific reactivity. Should not be consistently > 35% asymmetric or focal. Special type 1: Central beta - intermingled with Mu, same distribution and reactivity. Special type 2: Occipital beta reactive to eye opening (fast Alpha variant).

**Lambda waves.** Yves Gastaut described these transient waveforms in 1951 and demonstrated their dependence on exploratory saccadic eye movements (Figure 4). They are probably spontaneous visual evoked potentials elicited by shifts of the subject's gaze. Their incidence varies (up to about 50%) depending on laboratory environment and procedures (lighting, patterned ceiling tile, use of routine picture viewing). When prominent they can be mistaken for abnormal sharp waves. Criteria (Kellaway, 1990; Niedermeyer, 1993a; Blume et al, 2002): Transient predominantly occipital potentials occurring singly or in irregular trains. Field may extend to parietal and posterior temporal regions. Diphasic or triphasic with a predominantly positive sharp peak of 75-200 ms duration. Variable amplitude may exceed 50 µV, especially in children (Figure 4). No prominent after-following slow-waves. Occur exclusively during visual exploration of a brightly illuminated object or room. Time-locked (=100 ms peak latency) to saccadic eye movements. May persist during photic stimulation if visual exploration continues (Figure 4). Disappear with blank card viewing, darkness, eye closure, drowsiness and sleep. Bisynchronous but may be asymmetric. Oculographic monitoring assists identification (Figure 4).

**Slow activity.** Walter first described slow activity (below 8 Hz) in brain tumor patients and excessive slow activity is a hallmark of cerebral dysfunction. However slow activity is almost always encountered in normal records, especially in children or the elderly and during drowsiness, sleep and hyperventilation at any age. Normal slow activity may randomly combine with sharp background components to simulate abnormal epileptiform complexes. Criteria (Kellaway, 1990; Niedermeyer, 1993a; Blume and Kaibara, 1999; Blume et al, 2002): Delta: below 4 Hz, Theta: 4 to below 8 Hz. Minimal in awake resting adults but scattered theta normal at any age. Criteria for the normal quantity and distribution vary with state and age (see hyperventilation, drowsiness and ontogenesis, below).

**Hyperventilation responses.** Hyperventilation (HV) can activate abnormalities particularly generalized spike-and-wave complexes. However, potentially misleading normal EEG changes are common (Figure 5). The majority of subjects show some HV changes, but occasionally there is no effect. Criteria (Kellaway, 1990; Blume and Kaibara, 1999; Blume et al, 2002): Generalized slow activity intrusion. May be irregular or rhythmic, continuous or paroxysmal. Includes intermittent rhythmic delta activity of frontal (FIRDA, in adults) or occipital predominance (OIRDA, in children, see Figure 5). Sharp contours are common, but should not be frankly epileptiform. Builds up during HV, fades with variable duration post-HV. Shifting asymmetries are common, but should not be consistently regional.

**Photic stimulation responses.** Stroboscopic flash stimulation over a range of frequencies may activate abnormalities, particularly generalized spike-and-wave complexes. However, normal responses representing flash visual evoked potentials can have epileptiform features (Figure 6). Occipital responses are present in the majority of subjects but may be absent. Criteria (Lüders and Noachtar, 2000; Blume et al, 2002): Occipital responses time-locked to the stimulus with a short (=100 ms) delay. May include harmonics (=2 x flash frequency) and sub-harmonics (=1/2 flash frequency). Epileptiform responses are considered normal if limited to the occipital region, but should not become generalized, outlast or arise outside the stimulus. Asymmetry of driving response amplitude or frequency is common (Figure 6).

**Drowsy patterns.** Electroencephalogram background interpretation considers the alert state. Intermittent eye opening and arithmetic questioning are 2 techniques to maintain alertness. Drowsiness often intrudes insidiously and may produce manifold normal EEG changes. Prominent drowsy patterns are frequent in children and Santamaria and Chiappa (1987) importantly documented numerous potentially misleading drowsy patterns in healthy adults (Figure 7). Furthermore, many benign epileptiform variant patterns emerge in this state. Consequently, the electroencephalographer must be vigilant for signs of drowsiness, which may not be noticed by the recording technologist. Criteria (Santamaria and Chiappa, 1987; Kellaway, 1990; Niedermeyer, 1993b; Klass, 1995; Blume and Kaibara, 1999; Blume et al, 2002): Cessation of blinking and arithmetic questioning are 2 techniques to maintain alertness. Intermittent eye opening and arithmetic questioning are 2 techniques to maintain alertness. Intermittent eye opening and arithmetic questioning are 2 techniques to maintain alertness.

**Sleep patterns.** Sleep can activate epileptiform abnormalities. However, sleep transients that occur in all normal subjects are sharply contoured and benign epileptiform variant patterns are common in light sleep. Both can be mistaken for abnormality. When very sharp sleep transients are difficult to differentiate from...
Figure 4 - Lambda waves. The occipital sharp positive potentials look like abnormal sharp waves. However, they are time-locked to saccadic eye movement with eyes open even during photic stimulation and disappear with eye closure, so are Lambda waves. The clinical diagnosis was suspected cerebral palsy with no seizures. HEOG and VEOG, horizontal and vertical electro-oculogram.

Figure 5 - Hyperventilation response: linked-ear (A12) reference. The posterior emphasis of physiologic intermittent rhythmic delta activity is common in children, as is the presence of some generalized irregular slow activity at rest. As a general rule, only consistently regional or unequivocally epileptiform features should be considered as potential abnormalities during hyperventilation.

Figure 6 - Asymmetric sharp photic driving responses. The clinical diagnosis was headache and sleepiness.

Figure 7 - Drowsiness. Slow eye movement artifacts at F8 and F7 indicate drowsiness. On the left, hypnagogic hypersynchrony occurs in a child. On the right, an irregular asymmetric drowsy burst occurs in an adult with a clinical diagnosis of migraine and no seizures. Tiny 5 Hz spike components suggest a “phantom” spike-and-wave variant. Similar patterns without the spike components are common.

Figure 8 - Very sharp vertex waves. The patient had a seizure during acute renal and pulmonary failure that had resolved 2 weeks before the EEG without prior or subsequent seizures. Enhanced MRI of the brain was normal. These vertex waves fit established normal criteria and do not suggest an underlying seizure disorder.

Figure 9 - Shifting vertex wave asymmetry. One is very sharp with an F4 maximum but still fits normal criteria and does not clarify diagnosis of the patient’s suspected seizure.
abnormal midline or parasagittal spikes (Figures 8 & 9) one should favor the side of caution.

**Vertex transients** (Vertex waves, K-complexes). Criteria (Kellaway, 1990; Niedermeyer, 1993b; Blume and Kaibara, 1999, Blume et al, 2002): Sharp transients of variable amplitude arising singly or in rhythmic trains. Usually maximal near the vertex ("V-wave") or frontal midline ("F-wave"). Broad scalp field extending bilaterally to parasagittal and even temporal regions. Usually predominately negative but may be positive or biphasic. May have a prominent following slow wave with superimposed sleep spindle (K-complex). May be very high amplitude and very sharp, especially in children (Figure 8). Exclusive to stage I (V-wave) or stage II (K-complex) and deeper non-REM sleep. Can be evoked by auditory stimuli, such as tapping a pen. Shifting asymmetries with a right or left central or frontal maximum are common but should not be consistently asymmetric (Figure 9).

**Positive occipital sharp transients of sleep (POSTS). Criteria** (Kellaway, 1990; Niedermeyer, 1993b; Blume and Kaibara, 1999, Blume et al, 2002): Predominantly occipital sharp positive transients occurring singly or in irregular trains. Scalp field often extends to parietal and posterior temporal regions. Variable amplitude, sometimes very sharp; no following slow-wave. Resemble Lambda waves ("Lambdoid waves"). Occur exclusively in non-REM sleep. No specific reactivity. Bisynchronous but may be asymmetric (Figure 10).

**EEG ontogenesis.** There are age-related developmental (ontogenic) changes in normal EEG patterns. Blume's Atlas of Pediatric Electroencephalography (1999) and Klass's "Electroencephalography of the elderly" (1995) are excellent sources of information about EEG maturation and senescence. An overview is presented here: Dominant background frequency: Reactive occipital rhythms (equivalent to the adult Alpha rhythm, but below alpha frequency initially) begin at about 4 Hz around 3-4 months of age, then rapidly accelerate through childhood and may again decline slightly in the elderly. A guideline for minimum normal dominant frequency (Lüders and Noachtar, 2000): Age 1 - 5 Hz; Age 4 - 6 Hz; Age 5 - 7 Hz; Age 8 - 8 Hz; Above - 8 Hz. Slow activity (Kellaway, 1990; Klass, 1995; Blume and Kaibara, 1999; Blume et al, 2002): Dominates in infancy and young children. Variable gradual decline through childhood and adolescence. Shifting asymmetry is common, but should not be consistently regional. Interpret generalized or posterior slow activity cautiously in children. Minimal in resting awake adults but scattered theta normal at any age. Some intermittent frontal and temporal slow activity may reappear in senescence. Posterior slow waves of youth (Kellaway, 1990; Blume and Kaibara, 1999): Transient occipital or posterior temporal delta waves. Intermingled with the Alpha rhythm and often asymmetric. Present in wakefulness with eyes closed and disappear with eye opening (Figure 11). Prominent in children but may persist into the third (or even fourth) decade.

**Benign variant patterns.** Electroencephalogram patterns outside of and unequivocally distinct from the wide range of normal features (which are more extensive than those described above) are potentially abnormal but first a number of distinctive but benign variant patterns must be excluded. Most of these were considered abnormalities with epileptogenic or other pathological significance when first described. When they are found in clinically suspected or definite epilepsy it is tempting to suppose that they might have significance. However, controlled clinical studies have not demonstrated an association with seizures. Therefore, they must be recognized and discarded from epilepsy diagnosis or classification. Some are transient epileptiform patterns that resemble spikes or sharp waves. Others are rhythmic patterns resembling seizure discharges. Established multi-factorial criteria for these distinctive variant phenomena follow.

**Small sharp spikes (SSS).** Gibbs and Gibbs (1952a) originally described SSS (Figures 12 & 13) and noted their occurrence in up to 10% of controls but considered some SSS to be epileptogenic. Small sharp spikes are now considered a benign feature that also arise in normal people (Westmoreland, 1990). "Benign sporadic sleep spikes" and "benign epileptiform transients of sleep" are synonyms. Criteria (Westmoreland, 1990; Blume et al, 2002): Sporadic single spikes of usually < 50 µV (but may be larger). Broad sloping field with a fronto-temporal maximum and extension across the midline (Figure 12). Display best with long inter-electrode distance referential montages (for example ear reference). Brief (< 50 ms) and biphasic with a steep negative ascent and steeper declination (Figure 13). Minimal same-polarity following slow-wave or opposite-polarity background "dip". May have an oblique inter-hemispheric dipole. Predominate in drowsiness and light sleep. Bilateral and independent if enough recording is obtained. Most common in, but not limited to adults and elderly.

**14 and 6 Hz positive bursts.** Gibbs and Gibbs (1952b) described this pattern (Figures 14 & 15) as 14 and 6 Hz positive spikes and a manifestation of a fabricated condition: thalamic or hypothalamic epilepsy. This mistake was based on attributing the presenting symptoms (some of which had autonomic aspects) to the presumed abnormality. Now this pattern is known to be normal and unassociated with epilepsy (Westmoreland, 1990). The currently preferred term "bursts" de-emphasizes the connotation of epileptiform abnormality conveyed by the original term. Criteria (Westmoreland, 1990; Blume et al, 2002): Short (usually < 3 seconds) rhythmic bursts of arciform or spike-like positive waves. Frequency of =14 or 6-7 Hz or both mixed together. Posterior quadrant (usually posterior temporal) maximum and broad field. Display best with long inter-electrode distance montages. Isolated single positive
Figure 10 - Positive occipital sharp transients of sleep (POSTS).

Figure 11 - Posterior slow waves of youth. The combination with sharp Alpha waves (underlined) mimics occipital epileptiform complexes and could be misinterpreted as supporting a diagnosis of occipital epilepsy. However, both attenuate normally with eye opening, while the abnormal left temporal intermittent slow activity (bracket) due to this patient’s temporal lobe epilepsy persists.

Figure 12 - Small sharp spikes in light sleep. Note the broad sloping potential field extending across the midline and bilateral independent occurrence. The clinical diagnosis was mitral stenosis with syncope.

Figure 13 - Small sharp spikes – morphological detail. Note the very steep declinations.

Figure 14 - Six Hz positive burst. Note the right posterior quadrant maximum and broad field.

Figure 15 - Fourteen & 6 Hz positive bursts (underlined) during light sleep; contralateral ear reference. The clinical diagnosis was suspected seizures, but the EEG finding cannot clarify the diagnosis.

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Figure 19 - Subclinical rhythmic electrographic discharge of adults (SREDA). Segments B and C are continuous from A. The discharge begins (A) with repetitive theta waves in the left and then right parietal region independently, then settles into a sustained biparietal 5-6 Hz rhythm (B) which fades out (C) after 50 seconds. Note the undisturbed intermittent Alpha rhythm in segment A. The patient was awake without clinical signs or symptoms. The clinical diagnosis was excessive daytime somnolence.

Figure 18 - Rhythmic temporal theta bursts of drowsiness. The brief bursts in B resemble wicket waves but have a 5-6 Hz frequency. In C the pattern is also ambiguous because it arises in wakefulness and is 7-8 Hz, but is quite prolonged for wicket waves. On the left of C, 2 individual waves have briefly higher amplitude but “fit in” with the more sustained pattern on the right. Patient A had syncope. Patients B and C had undiagnosed episodes of loss of consciousness. These EEG patterns do not clarify diagnosis.

Figure 17 - Wicket waves. A and B show typical wicket wave clusters, combined with a random left temporal slow wave in A. In C an isolated wicket wave (left panel) "fits in" with the shape and duration of the waveforms in a typical cluster in another recording segment (right panel). Patient A had "peculiar spells" diagnosed as epileptic after an EEG report of temporal sharp waves at another hospital, but was not diagnosed epileptic by an experienced King Faisal Specialist Hospital & Research Centre epileptologist. Patients B and C had remote undiagnosed episodes of loss of consciousness. Wicket waves do not clarify the diagnoses of these patients.

Figure 16 - Six Hz "phantom" spike-and-wave, linked-ear (A12) reference. The clinical diagnosis was psychiatric disturbance with no seizures.
waves may arise. Predominate in drowsiness and light sleep. Usually bilateral and independent. Most common in, but not limited to teenagers.

**6 Hz "phantom" spike-and-wave.** Gibbs and Gibbs (1952b) described this pattern (Figures 7 & 16) as epileptogenic. Hughes (1980) found it to be epileptogenic if frontal dominant in wakefulness but not if occipital dominant in drowsiness. Currently most authorities consider it benign (Westmoreland, 1990).

When difficulty distinguishing it from more significant spike-and-wave fragments occurs it is best to state so explicitly and err on the side of caution. The term "phantom" conveys the typical evanescent miniature spike-and-wave morphology.

**Criteria** (Westmoreland, 1990; Blume et al, 2002):
- Brief (usually < 1s) 5-7 Hz spike-and-wave complexes.
- Occipital, generalized or frontal dominant.
- The spike is usually smaller than the associated theta waves and is sometimes difficult to see.
- Can resemble 6 Hz positive bursts and both patterns may occur in the same recording. Predominates in drowsiness and light sleep. Bisynchronous but may be asymmetric. Most common in, but not limited to adolescents and adults.

**Wicket waves.** Reiher and Lebel (1977) described this pattern (Figure 17) as wicket spikes and showed it to be benign but its resemblance to temporal sharp waves makes it a common source of misinterpretation. The currently preferred term "waves" de-emphasizes the connotation of epileptiform abnormality conveyed by the original term. **Criteria** (Reiher, 1977; Westmoreland, 1990; Blume et al, 2002):
- Clusters or trains of mid- or anterior temporal 6-11 Hz negative sharp arciform waves.
- Isolated wicket waves morphologically "fit in" to wicket clusters in other recording segments.
- No after-following slow wave or background disruption (beware combination with random slow activity, see Figure 17a).
- Arise during wakefulness, drowsiness and light sleep.

Unilateral, bilateral independent or bisynchronous. Most common in, but not limited to subjects over age 30.

**Rhythmic temporal theta bursts of drowsiness (psychomotor variant pattern).** Gibbs and Gibbs (1952b) originally described this pattern (Figure 18) as "psychomotor variant" because of its resemblance to psychomotor (temporal lobe) seizure patterns. They considered it an abnormality sometimes associated with epilepsy. It is now known to be benign and can arise in healthy subjects. It can be difficult to clearly distinguish from wicket waves because the criteria overlap somewhat in terms of frequency, polarity, state and distribution (Figure 18b & c). "Rhythmic mid-temporal theta discharges" (RMTD) is another synonym. **Criteria** (Westmoreland, 1990; Blume et al, 2002):
- Rhythmic mid- or anterior temporal 5-7 Hz negative polarity waveforms.
- Sinusoidal, notched or sharply contoured.
- Variable duration, may be quite prolonged (for example one minute). Waxes and wanes, but does not evolve in frequency and amplitude like a seizure pattern.
- Arises predominantly in drowsiness, but may be seen in relaxed wakefulness. No concurrent clinical signs or symptoms.
- Unilateral, bilateral independent or bisynchronous.

**Subclinical electrographic discharge of adults (SREDA).** Naquet originally reported this striking pattern (Figure 19) and Westmoreland and Klass (1981) subsequently showed it to be a non-specific phenomenon with no association to any particular neurologic condition or epilepsy and coined the term SREDA. It is easily misinterpreted because of its rarity and resemblance to seizure patterns. **Criteria** (Westmoreland and Klass, 1981; Westmoreland, 1990; Westmoreland and Klass, 1997; O’Brien et al, 1998):
- Rhythmic sharp 5-7 Hz activity lasting several seconds to over one minute. Usually bilateral parietal or posterior temporal maximum but may be asymmetric.
- Duration 20 seconds to a few minutes. May begin abruptly, or with a series of
individual sharp theta waves. Ongoing background activity (e.g., Alpha rhythm, Mu) undisturbed. Predominantly arises in resting wakefulness or during hyperventilation. No concurrent clinical signs or symptoms. Unusual variants include predominantly delta frequency, notched waveforms, more frontal or focal location and occurrence in sleep. Usually above age 50 but recently reported in 3 children (Nagarajan et al., 2001).

Midline theta. Ciganek first described this rare pattern (Figure 20) and thought it was a manifestation of temporal lobe epilepsy (Westmoreland, 1990). Westmoreland and Klass (1986) subsequently showed it to be a non-specific irregularity of uncertain significance seen in a variety of neurologic disorders but without any specific association to epilepsy. This pattern is not truly "benign" in the sense of occurring also in healthy individuals, but cannot clarify epilepsy diagnosis or classification. Criteria (Westmoreland and Klass, 1986; Westmoreland, 1990): Waxing-and-waning 5-7 Hz rhythmic activity. Maximal at the frontal or central midline with variable adjacent spread. Sinusoidal, arciform, sharp or spike-like morphology. Does not evolve in frequency like a seizure pattern. Predominantly in wakefulness and drowsiness (I have also seen it continue into sleep). Variable reactivity to alerting, eye opening or movement. No concurrent clinical signs or symptoms.

Frontal arousal rhythm (FAR). White and Tharp originally described FAR (Figure 21) and thought it was an abnormality seen in children with minimal brain dysfunction but it is currently considered a non-specific EEG finding of uncertain significance (Westmoreland, 1990). Its rhythmic and sometimes sharp characteristics can easily be mistaken for a seizure pattern. Criteria (Westmoreland, 1990): Trains of bilateral frontal 7-10 Hz rhythmic activity lasting up to about 20 seconds. May be sharp or notched. Occurs during arousal from sleep or drowsiness. Subsides with full wakefulness. No concurrent clinical signs or symptoms.

Further Reading


