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Non-epileptiform EEG abnormalities

An overview

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ABSTRACT

More than 80 years after its introduction by Hans Berger, the electroencephalogram (EEG) remains as an important supplementary examination in the investigation of neurological disorders and gives valuable and accurate information about cerebral function. Abnormal EEG findings may include ictal patterns, interictal epileptiform activity and non-epileptiform abnormalities. The aim of this study is to make an overview on the main non-epileptiform EEG abnormalities, emphasizing the pathologic findings and the importance of their recognition, excluding periodic patterns and EEG physiologic changes. Scientific articles were selected from MEDLINE and PubMed database. The presence of non-epileptiform EEG abnormalities provide evidence of brain dysfunction that are not specific to a particular etiology and may be related to a number of disorders affecting the brain. Although these abnormalities are not specific, they can direct attention to the diagnostic possibilities and guide the best treatment choice.

Key words: abnormal EEG, non-epileptiform abnormalities, slow activity.

Anormalidades eletrencefalográficas não epileptiformes: uma visão geral

RESUMO

Mais de 80 anos após sua introdução por Hans Berger, o eletrencefalograma (EEG) permanece como importante exame complementar na investigação de transtornos neurológicas e fornece informações valiosas e precisas a respeito da função cerebral. Achados eletrencefalográficos anormais podem incluir padrões ictais, atividade epileptiforme interictal e anormalidades eletrencefalográficas não epileptiformes. O objetivo deste estudo é fazer uma revisão das principais anormalidades eletrencefalográficas não epileptiformes, enfatizando os achados patológicos e a importância de seu reconhecimento, excluindo padrões periódicos e alterações eletrencefalográficas fisiológicas. Foram selecionados artigos científicos por meio de pesquisa nas bases de dados MEDLINE e PubMed. A presença de anormalidades eletrencefalográficas não epileptiformes fornece evidências de disfunção cerebral, as quais não são específicas para uma etiologia particular e podem estar relacionadas a uma série de desordens que afetam o encéfalo. Embora essas anormalidades não sejam específicas, elas podem direcionar a atenção para as possibilidades diagnósticas e guiar a escolha do melhor tratamento.

Palavras-Chave: EEG anormal, anormalidades não epileptiformes, atividade lenta.

Since its introduction by Hans Berger, in 1929, the electroencephalogram (EEG) has been widely used in the diagnosis and monitoring of patients with neurological disorders, especially epilepsy. The possibility of detection of interictal epileptiform abnormalities, or even demonstrating ictal patterns, causes the EEG is not only able to provide support for the diagnosis of epilepsy, as well as support for the classification of the epileptic disorder and provide information prognostic

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in some cases[1]. However, the EEG is also useful in the investigation of other neurological disorders, in addition to epilepsy, or in cases of symptomatic epilepsy (secondary to a disorder-based). The demonstration of non-epileptiform EEG abnormalities in patients with altered mental status or level of consciousness, for example, can be especially useful in guiding decision making and the best treatment[2-4]. The EEG provides valuable information on the investigation of patients with signs of acute cerebral suffering, rapidly progressive dementia, toxic-metabolic encephalopathy, coma and brain death, among others[2-4]. It is worth remembering that the non-epileptiform EEG abnormalities may be present in patients with epilepsy[5-9]. Vanrumste et al.[5] demonstrated the presence of non-epileptiform activity arising from the same area as epileptiform activity in five children with focal epilepsy, using an automated technique. The presence of irregular slow waves, assumed as a non-epileptiform abnormality, was included as an interictal EEG diagnostic criteria for temporal lobe epilepsy proposed by The International League Against Epilepsy (ILAE), in 1981[10].

Although the EEG does not provide specificity to determine the etiology of the brain dysfunction, its sensitivity in different events can guide the diagnostic possibilities and makes the recognition of the non-epileptiform EEG abnormalities an important tool of knowledge. Considering this, allied to the fact of the most publications, especially in the form of scientific papers, gives greater emphasis on the epileptiform changes themselves, we believe that non-epileptiform EEG abnormalities are a relevant topic to review.

We searched MEDLINE and PubMed data base with the key words: abnormal EEG patterns, coma and EEG, epileptiform EEG, FIRDA, OIRDA, TIRDA and slowing EEG. We selected 41 articles of interest, from 1936 to 2011, and two relevant book chapters, onwards and then hand searched these for earlier publications, focusing on those that were related to the main non-epileptiform abnormalities, its significance, clinical correlation and importance of their recognition. Twenty nine of the selected articles were directly related to non-epileptiform EEG abnormalities. Were excluded from the search articles related to EEG changes due to periodic patterns, because their nature is still unclear (if epileptogenic or not, in some cases), and normal or physiologic variants.

Concept and definitons

Abnormal EEG findings include ictal patterns (observed during an epileptic ictal event), interictal epileptiform activity and non-epileptiform abnormalities[11]. In a carefully screened population of young and middle age asymptomatic adults, Jabbari et al.[12] found an incidence of epileptiform activity, photoparoxysmal response or excessive slowing less than 1%. Unlike the interictal epileptiform activities, characterized by the presence of spikes and sharp waves, in combination or not with slow waves, and strongly associated with epilepsy, the non-epileptiform EEG abnormalities may be characterized by several distinct patterns of normal expected for a given age and condition of the patient (if awake or asleep, for example)[11,13]. They consist mainly of[11,13]: focal slow activity; regional or generalized bisynchronous slow activity; generalized asynchronous slow activity; focal attenuation; generalized attenuation / supression; 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.

The non-epileptiform EEG abnormalities in the context of the neurological disorders

When the non-epileptiform abnormalities are seen on an EEG record, they are not specific for an underlying etiology[11,13]. In these situations, however, the EEG provides evidence of organic electrophysiological dysfunction and the patterns observed may orientate for the diagnostic possibilities.

The most important types of the non-epileptiform EEG abnormalities are described below:

Focal slow activity – Focal slow activity may be an indicative sign of focal cerebral dysfunction, especially in awake adults, and it seems to be the result of a cortex deafferentation from subcortical structures[11,13]. It was first described by Walter[14], in 1936, who proposed the term "delta waves" for focal slow activity associated with tumors involving cerebral hemispheres. It is the most common phenomenon encountered in clinical EEG that is indicative of a localized structural lesion[11]. Slow activity is classified according to frequency in theta activity (ranging from 4.0 to 7.9 Hertz (Hz) or cycles per second), and in delta activity (around 0.5 to 3.9 Hz)[13]. In some cases, focal slow activity may presents as focal and irregular slow activity in the delta frequency, receiving the designation of polymorphic delta activity (PDA) (Fig 1), which is usually due to a structural lesion or a subcortical dysfunction (although the anatomic correlation is not always exact)[11]. Focal slow activity is assessed with regard in amplitude, frequency, topography, persistence and reactivity (that is the most reliable indicator of dysfunction degree)[11]. In an EEG and computed tomography scan correlation study, Schaul et al.[15] showed that field, amplitude and frequency of focal slow waves do not distinguish lesion size, density or mass effect, but reactivity and persistence of focal abnormalities (continuous versus intermittent) were significantly better indicators of damage degree. Continuous slow activity suggests a

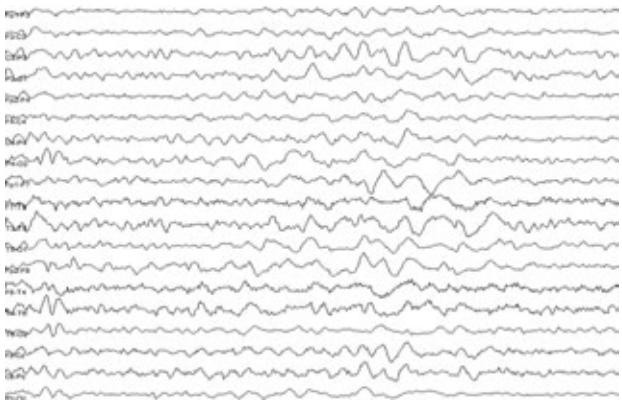

Fig 1. A polymorphic delta activity (PDA) may be seen predominantly over the left temporal and parietal regions, with widespread, in the EEG of a 72-year-old man presenting with altered mental status. The imaging studies revealed an extensive subacute ischemic area correlated topographically with this EEG change.

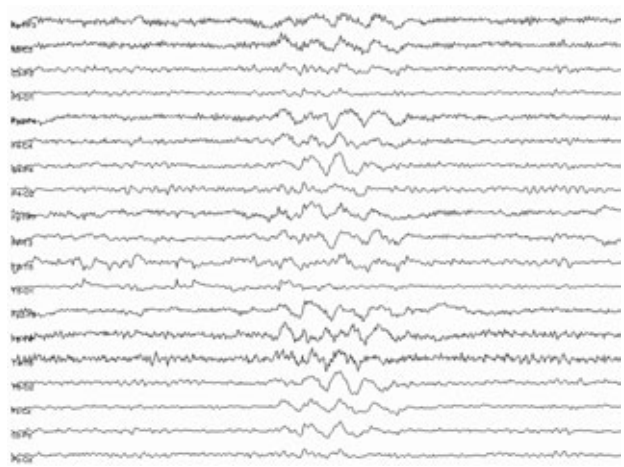

Fig 2. Frontal intermittent rhythmic delta activity (FIRDA). The slow activity has generalized bisynchronous projection, predominantly in the bilateral frontal regions.

more severe brain damage (likelihood of increased mass effect, large lesion or deep hemispheric lesion), whereas intermittent slow activity usually indicates a small lesion and absence of mass effect[15]. Patients with reactive focal slow activity had evidence of less cerebral damage than did patients with non reactivity[15].

Gloor et al.[16], 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[16]. Bilateral hypothalamic and bilateral mesencephalic lesions produced bilateral slow waves[16]. 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 deafferenttion of cortical neurons may be responsible for slow activity[15,16]. Studies suggested that thalamic deafferentation from the cortex rather than cortical deafferentation from below may be the slow wave mechanism[11,15,16].

In general, fast-growing tumors, such as glioblastoma multiforme or metastatic brain tumors, are associated with focal slow activity occurring in the delta frequency[16]. Slow-growing tumors, such as meningiomas, are usually associated with focal slow activity that occurs more frequently in the theta range. Epileptiform discharges can coexist with focal slow activity produced by brain tumors, especially slow growth (the least destruction of the brain parenchyma with slow growth tumors increases the possibility of detection of epileptiform discharges)[16].

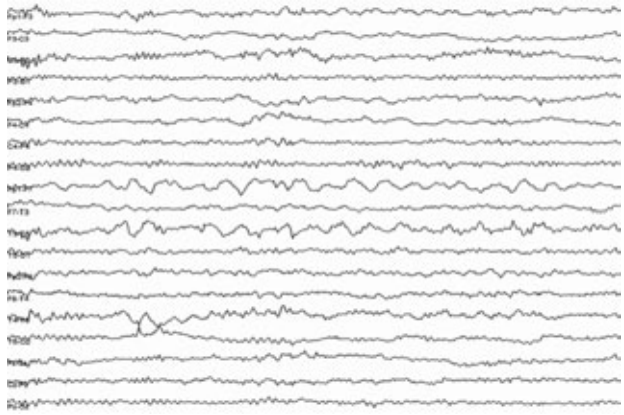

Fig 3. Temporal intermittent rhythmic delta activity (TIRDA) over the left temporal region in a 33-year-old man, who performed a routine EEG for investigation of focal onset seizures.

Some epileptic focus can produce irregular focal slowing, associated or not with structural lesions, as well as postictal slowing after a focal seizure[6]. 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[6].

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[11,13]. 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[11,13]. Some authors, however, has been described that this pattern is generally related to disease states affecting neurons at cortical and subcortical levels, and they are not specific to deep midline pathology or increased intracranial pressure[11,17]. 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) (Fig 2), occipital intermittent rhythmic delta activity (OIRDA) and temporal intermittent rhythmic delta activity (TIRDA) (Fig 3).

As its name indicates, FIRDA is a rhythmic activity in the delta frequency, which occurs in a bilateral and synchronous manner with frontal predominance. Generally, its frequency is around 2.5 to 3.0 Hz[13]. It was originally described by Cobb[18], in 1945, and was initially attributed to deep midline lesions and posterior fossa tumors[18-20]. In early descriptions, some authors hypothesized a relationship with FIRDA and increased intracranial pressure[21,22]. Later studies, however, contended that such etiologic factors comprise only a small percentage of all conditions associated with FIRDA[15,17,23-28]. These studies correlated the presence of FIRDA with a large number of disorders, including toxic-metabolic encephalopathies, early stages of coma, degenerative diseases and other conditions, affecting both cortical and subcortical structures[15,17,23-28]. Schaul et al.[15] studied 42 EEG records with FIRDA and found that this finding was non specific and had no correlation with increased intracranial pressure. In a large study of 301 patients with FIRDA, Rowan et al.[26] described an association with brain tumors in 35%, cerebrovascular disease in 19% and epilepsy of unknown cause in 13%. Accolla et al.[23] investigated the clinical correlates of FIRDA in 36 patients and compare them with a control group of 80 subjects. Epilepsy was more frequently in the control group and structural brain lesions and encephalopathy were independently associated with the occurrence of FIRDA. They concluded that FIRDA appears more common than previously reported, it is associated with a high range of intracranial lesions (especially if asymmetric) and encephalopathies and its occurrence should prompt investigation for these conditions. FIRDA is found in a number of conditions and its origin and pathophysiology are still unclear[23-26]. Additional diligent diagnostic investigation is recommended when FIRDA appears on an EEG of abnormal or otherwise normal subjects[27,28].

Rhythmic delta activity that occurs in children usually presents predominantly posterior projection (especially occipital), which corresponds to OIRDA. This feature was first described by Cobb in patients with absence seizures[18]. The presence of OIRDA in children is due to causes similar to those of FIRDA in adults[13]. Lesions in the occipital lobe with involvement of the posterior lateral ventricle may lead to the emergence of OIRDA as a projected rhythm[13]. However, OIRDA seems related to the presence of epilepsy, especially generalized, in children[7,9]. Generalized 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[7]. In 15 to 30% of children with absence epilepsy, the interictal EEG may also contain OIRDA at 3.0 to 4.0 Hz, which has a high amplitude, without sharp waves[9]. Dysfunctions probably located in the brainstem and diencephalon, or in their non specific projections, would explain the coexistence of spike and wave activity and OIRDA in children with typical absence seizures[7].

When the EEG shows unilateral FIRDA or OIRDA, it generally indicates a focal than diffuse disturbance, and sometimes its occurrence is contralateral to the pathology[9,23,24]. 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, brainsteim or cerebellum[15,26].

Intermittent rhythmic delta activity occurring well localized over the temporal region (temporal intermittent rhythmic delta acitivity - TIRDA) is indicative of potential epileptogenicity of the ipsilateral temporal lobe[8,29]. It was first described by Reiher et al.[8], in 1989, and it was 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[8,24,29]. 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[6]. So, it has more specificity than FIRDA and OIRDA. Its encounter in these patients has the same meaning of interictal epileptiform activity, particularly during sleep, although it is not classified as an epileptiform activity[13,24]. Sometimes, interictal epileptiform activity and TIRDA can coexist in the same patient[8]. Gennaro et al.[29] studied 129 patients affected by drug-resistant partial epilepsy and found in 52 (40.3%) significant correlations between TIRDA and mesial and mesio-lateral temporal lobe epilepsy, mesial temporal sclerosis, interictal epileptiform discharge localized over the anterior temporal regions and 5.0 to 9.0 Hz temporal ictal discharges. The presence of TIRDA may also be related to structural changes affecting the lateral ventricle, corresponding to a projected or a distance rhythm over the temporal lobe[13].

Generalized asynchronous slow activity – Generalized asynchronous slow activity consisting of fre-

quencies less than 4.0 Hz is highly nonspecific and has a broad differential diagnosis[11]. Its presence usually indicates encephalopathy and is always abnormal in awake adults[11,13,30]. Some possibilities include degenerative processes, encephalitis, extensive multifocal vascular diseases and toxic-metabolic encephalothies[11,13,30]. Assessing generalized asynchronous slow activity requires analyzing frequency, amplitude, distribution, persistence and reactivity, searching an associated finding that may indicate an underlying lateralized or localized disorder[30]. In a study of diffuse encephalopathies correlating EEG findings with sites of histopathology, Gloor et al.[16] observed continuous diffuse PDA in patients with disease processes extensively involving hemispheric white matter or white and gray matter.

It is worth remembering that the correct interpretation of generalized asynchronous slow activity takes into account the age and condition of the patient, given that widespread slow activity may be normally present in drowsiness and sleep in all ages and in awake children, depending on the age[13,30]. It should be considered an abnormal activity when the pattern is inconsistent with age and stages of sleep[13,30].

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[13]. Other common causes include cerebral ischemia, postictal states (arising from a crisis of focal onset), swelling of the scalp and subdural collections[13]. Scheneider et al.[31], suggested that the use of Emergency EEG in acute ischemic stroke can reveal a distinctive EEG pattern called regional attenuation without delta (RADWOD), that adds value to the selection of patients for thrombolytic and cerebral edema treatment. 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 – Generalized attenuation may 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[13].

Suppression corresponds to a state worse than attenuation, and it indicates complete or nearly complete disappearance of electroencephalographic activity[13].

Generalized attenuation and suppression can occur by[13]: reduced synchronicity of the cortical activity; reduction of the cortical activity; excess of fluid or tissue over the cerebral cortex.

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[11]. Cortex deafferation from thalamic projections is tought to be the underlying pathophysiologic explanation[11,32]. In the burst portion of this pattern, sharp waves and spikes are usually present[32]. In the suppression period, or interburst interval, there is absent or severely attenuated activity of delta and theta frequencies[32]. It is most often found in patients who suffered severe cerebral damage in postanoxic encephalopathy, under the effect of anesthetic drugs or drug-induced coma[32,33]. Hypnotic sedative drugs, as barbiturates, may disconnect the cortex from the subcortical structures[11,32,33]. Steriade et al.[33], studied burst-supression induced by various anesthetics agents in adult cats, and showed that 95% of cortical neurons become electrically silent during flat EEG epochs. Hyperpolarization of cortical neurons preceded EEG flattening. The hyperpolarization is due to increased K+ conductance which in turn is secondary to increased GABAergic inhibition at cortical synapses that leads to functional disconnection from thalamic input[11]. However, 30 to 40% of thalamic cells continue firing, due to their intrinsic pacemaker properties at modest levels of hyperpolarization[11]. Volleys from these thalamocortical neurons account for the cyclic EEG wave burts[11].

Other causes of burst-supression patterns include the period following the generalized tonic-clonic *status epilepticus*, in which the prognosis is better than when shown by default in subclinical or non-convulsive *status epilepticus*[13]. Sometimes, it can be observed in some complex partial non-convulsive *status epilepticus* as diffuse slowing alternating with periods of attenuation or suppression without evident focal abnormalities[34].

Other abnormal activity (alpha, theta and spindles coma patterns) – The EEG patterns in coma states are not specific with regard to the etiology or prognosis[35]. However, it is known that the prognosis is worse if the etiology is hypoxic-ischemic encephalopathy[35]. 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 injury[36,37].

The alpha coma 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[36,38]. The first descriptions appeared between the 1960 and 1970 decades, in patients after cardiac arrest and postanoxic

coma, who had a poor outcome[35,39]. 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[13,35]. This pattern is typically associated with hypoxic-ischemic encephalopathy. In the another one pattern, the alpha activity tends to have a posterior predominance and is often reactive to a passive eye opening and closing[35]. This pattern is generally associated with pontine lesions[35].

The theta coma was first described by Synec et al.[40], in 1984, as a 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[40]. Its meaning is the same as the pattern of alpha coma and, in some records, the two standards can coexist (alpha-theta coma)[35]. 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[35]. The pathogenesis of alpha, theta or alpha-theta coma is still unknown and the value of these patterns in predicting outcome remains controversial[35].

The term spindles coma is used when the EEG shows activity resembling sleep spindles in unconscious or comatose patients[41]. It is generally characterized by spindles in 9.0 to 14 Hz range, often with vertex sharp waves and K-complexes[41]. These spindles are, however, much more diffuse in distribution than normal sleep spindles[41]. Spindles coma was first described in 1953, by Jasper et al.[42], 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, and others[43]. The pathophysiological mechanism of spindles coma is presumed to be the preservation of intact pontine raphe nuclei and thalamocortical circuits subserving sleep spindles activity in an intact cerebrum, and also, of impairment of ascending reticular activating pathways at the midbrain level that maintains consciousness[43] . So, when this pattern is present in a comatose patient, they may be a sign of better prognosis than similar coma without spindles (alpha or theta), because their presence indicates the sparing of normal thalamocortical physiologic pathways[43].

A normal EEG is incompatible with coma. In these cases, one should consider the possibilities of a psychogenic origin of coma or a locked-in state. In the latter, the patient has inability to move the muscles of the face and limbs, but his alertness is preserved.

In conclusion, the non-epileptiform EEG abnormalities provide evidence of brain dysfunction, which may 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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