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REVIEW

# A reappraisal of the value of video-EEG recording in the emergency department

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**ABSTRACT**

**Introduction**: Some neurologic conditions that can quickly and with low costs be recognized, classified and treated thanks to the availability of an EEG recording in an emergency setting. However, although considered a cheap, not invasive, highly accurate diagnostic investigation, still today, an EEG recording in emergency, in real time during the event paroxysmal ictal phase, is not yet been become a routine.

**Areas covered**: This review will cover the role and utility of EEG recording in the emergency setting, both in emergency department and intensive care unit, in adult and pediatric age, in people admitted for status epilepticus (convulsive or non-convulsive), paroxysmal non-epileptic events, or other conditions/diseases presenting with mental status changes.

**Expert opinion**: The prompt recognition of some specific EEG-patterns can permit an immediate and appropriate therapeutic choice with the resolution of dramatic clinical pictures, which, if not recognized, sometimes could result in severe prognostic events with high mortality or neuropsychiatric disability. It is important in the next future, to improve the availability of these EEG digital continuous monitoring, which should be widely used in emergency settings, developing moreover tools and techniques permitting also review, analysis and EEG-reporting by experts who can work away from the hospital.

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Emergency EEG; video-EEG; continuous EEG; acute differential diagnosis; status epilepticus; nonconvulsive status epilepticus

## 1. Introduction

Despite the major technological improvements in neuroimaging and laboratory data observed in the last few decades, electroencephalogram (EEG) remains the hallmark tool for the evaluation of patients with epileptic seizures or paroxysmal non-epileptic events, change in mental status and coma, as well as the assessment of brain death.

The improvement in EEG recording in the last years is also due to the transition from analogue to technological digital instrumentation and even more recently to the ability to record simultaneous video (V-EEG) and to record for prolonged periods.

In fact, for many years, EEG meant 'routine' EEG, a brief (20–30 min) recording without video, and, even, in many Centers, without simultaneously polygraphic recording. Over the last 20 years, with technological (digital) progress, EEG has evolved in two major ways: the ability to record simultaneous video EEG/polygraphy, and the ability to record for prolonged periods. Thus, somewhat ironically, while the utility of routine EEG has decreased, the utility of prolonged EEG and V-EEG have increased and its use expanded rather dramatically [1].

Emergency EEG (eEEG) can be required: a) for immediate performance during non-business hours, performed within 3 hours from the initial request [2]; B) for excluding a non-convulsive status epilepticus [3]; c) for emergent basis and performed within one hour [4].

However, although EEG is generally considered a highly accurate and cost–effective diagnostic tool in the emergency department (ED), obtaining EEG as soon as possible is not yet been routine in many institutions. Nevertheless, recording a technically acceptable EEG in electrically hostile environments, such as an ED, remains challenging especially with uncooperative patients, and particularly in the pediatric age (i.e. high line noise in the recorded signals due to high ambient noise levels, long electrode wires, and relatively high electrode impedances and inter-electrode impedance differences) [5]; also, local access to neurological and EEG expertise, access to technical personnel and equipment, and local practice patterns limit the performance of EEG in ED.

Recognition and appropriate diagnosis of conditions such as seizures, non-epileptic seizures, change in mental status and brain death) in the ED are essential for immediate and future management and prognosis [6].

We aim to review the usefulness of eEEG and/or V-EEG in ED highlighting its role in differential diagnosis and predictive outcome. We evaluated EEG literatures in the emergency clinical setting, considering both EEG and V-EEG. Since, someone consider continuous EEG (cEEG) monitoring a continuum of eEEG, in this review we also try briefly to consider this aspect.

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**Article Highlights**

- EEG represents the hallmark tool for the evaluation of patients with epileptic seizures or other paroxysmal non-epileptic events, change in mental status, coma and assessment of brain death. An EEG recording during the event paroxysmal ictal phase, can identify some neurologic treatable conditions.
- Sometimes, the prompt recognition of specific EEG-patterns can permit an immediate and appropriate therapeutic choice with immediate resolution of dramatic specific clinical pictures reducing, thus, high mortality rate or neuropsychiatric disability.
- Although considered a cheap, not invasive, highly accurate diagnostic investigation, an EEG recording in emergency setting is not yet been become a routine investigation.
- Particularly an ictal EEG recording is mandatory to appropriately identify and treat the various possible status epilepticus, with crucial implications particularly in pediatric age.
- In the next future, we must develop tools and techniques permitting continuous EEG monitoring in emergency setting and ICU, and also analysis and tele-EEG-reporting by experts who can work away from the hospital, on demand.

We initially focus our attention on four questions that are frequently addressed in the literature and then we will discuss the individual clinical conditions in which eEEG is indicated.

### *1.1. When is indicated emergency EEG?*

The application of eEEG recording in ED is still mattered. The American College of Emergency Physicians (ACEP) recommends EEG in patients with suspect of non-convulsive status epilepticus or subtle convulsive status epilepticus [7]. However in 1996, a meeting of French experts established a set of guidelines supporting the usefulness of an eEEG in the emergency setting [8]. After this consensus meeting, no further international consensus with such extensive indications for an eEEG has been published. Nevertheless, the importance of an eEEG has been highlighted, both, in adults and pediatric populations with new-onset seizures and unexplained altered consciousness. EEG may be very helpful in predicting recurrences in patients with new-onset seizures and might also suggest that the yield of EEG increases with the closer proximity to the event eEEG further enabled the prompt recognition and treatment of patients with non-convulsive status epilepticus that would have gone undetected [6]

### *1.2. Technical difference between EEG monitoring*

Different types of electrodes are available: disk or cup electrodes are classically used, however subdermal needle electrodes can be applied rapidly and do not require scalp abrasion. Needles electrodes are not recommended for awake patients or prolonged recording, but are very useful because of their rapid positioning and for brief recording in some comatose patients.

The number of electrodes used in eEEG studies varies considerably. Usually the 10–20 system places 21 electrodes. Alternatives to this full montage have been described: – subhairline recording, which uses four bipolar derivations: left temporal (left anterior temporal electrode to left mastoid electrode), left frontal (left frontal electrode to left anterior temporal electrode), right frontal (right frontal electrode to right anterior temporal electrode) and right temporal (right frontal anterior temporal electrode to right mastoid electrode); – montage with eight recording electrodes uses four couples of electrodes: forehead FP1, FP2; central C3, C4; temporal T3, T4; and occipital O1, O2. The reference electrodes should be at the bilateral earlobe or mastoid levels. The grounding electrode should be placed at the midpoint of the frontal pole (FPz), and the common reference electrode should be placed at the median central point (Cz). However, in several cases, EEG recording is performed with a reduced number of electrodes, or in altered positions, in particular when surgical wounds, ventricular drains, or neuromonitoring devices limit the available surface of the skull. It is critical however to maintain the symmetry of the left and right side electrodes. In long-term EEG monitoring, is sometimes necessary to clean the skin or change the location of some electrodes to avoid scalp ulceration or infection [9].

### *1.3. There are any differences between children and adult patients?*

The indication for eEEG in children and adults are different [10]. In pediatrics, the most common indications for EEG are the evaluation of paroxysmal events, subclinical status epilepticus, investigation of a witnessed first episode concerning for seizures. The main reasons for ordering an eEEG in adults are suspected brain death, altered level of consciousness of various etiology, non-convulsive status epilepticus, subtle status epilepticus, and follow-up of convulsive status epilepticus, while evaluation of paroxysmal non-epileptic events, is not so common as in children [5,10].

Based on the available evidence, an EEG has been recommended in all non-provoked seizures in children. In this condition eEEG is more demanding and consuming than routine one. However an eEEG may be helpfull to avoid improper treatment as well as hospitalization in intensive care unit, with less emotional impact on parents too. Although an EEG obtained early after a seizure (before 24 hours) is more likely to detect epileptic abnormalities, unilateral slowing after a seizure can provide lateralizing evidence in patients with focal seizures. Yet, abnormalities such as postictal slowing can be transient and should be interpreted with caution in the context of presentation and time frame after a seizure [5,10]. In one series of 111 adults, the eEEG contributed to the diagnosis in 77.5% of the cases and led to a modification in treatment in 37.8% [11]. In another series of 449 adults who underwent an EEG within 16 hours of the initial paroxysmal event, an abnormal result was present in 71% of those admitted to the hospital and in 59.5% of those who were discharged from the ED, but the clinical relevance of these numbers is uncertain [12]. In children, the published data are limited but do suggest a utility for the eEEG. In a series of 32 children, eEEG findings were useful in the decision-making process in 94% of cases [13]. In a series of 56 children, within a mean follow-up period of 19.5 months, the risk of seizure recurrence was much higher in those with an abnormal eEEG, and it was estimated that the eEEG contributed to the diagnosis in 84% [6].

The literature currently reinforces the belief that the availability of EEG and V-EEG recording in ED is useful, albeit for different reasons comparing adult and developmental age.

### 1.4. When is a video-EEG monitoring indicated?

When eEEG is not conclusive, a V-EEG monitoring should be considered, especially in Intensive Care Unit (ICU), to allow a differential diagnosis between epileptic and psychogenic (non-epileptic) seizures, to support the classification of the electro-clinical syndromes, to take therapeutic decisions [14].

The use of prolonged V-EEG recordings ICU setting has markedly expanded, mostly for the diagnosis and management of status epilepticus, especially non-convulsive or ‘subtle’ status epilepticus. As is true in non-ICU setting, the longer the better, with reasonable limits. The consensus is that 24–48 h of monitoring has the best yield [1] (See Table 1).

## 2. Clinical indication for emergency EEG, emergency video-EEG and subsequent continuous video-EEG monitoring

### 2.1. EEG ‘as soon as possible’ after the first seizure

About 8–10% of the population will experience a seizure during their lifetime and about 2–3% of them go to develop epilepsy [15]. EEG remains an essential diagnostic tool in the evaluation of seizure disorders. The clinical guidelines from the American Academy of Neurology for both children [16] and adults [17] recommend that an EEG be obtained after a single unprovoked seizure. Indeed, EEG abnormalities may be useful in identifying epileptogenic foci, structural abnormalities, and/or electrographic patterns associated with specific epilepsy syndromes. However, it is the potential predictive value of EEG that can have the most prominent role in the evaluation of a single unprovoked seizure. The literature suggests that EEG epileptiform abnormalities, after new-onset seizure predict seizure recurrence. The risk of seizure recurrence is greatest within the first 1–2 years (21–45%) after a single unprovoked seizure in adults [18]. Similarly, in children, the risk is also greatest within the first 1–2 years (14–65%) after a single seizure. Diagnosis is important because epilepsy is a treatable condition, with 47% of patients achieving seizure remission on their first anticonvulsant therapy. It is, however, important to know that 2.2% of individuals without epilepsy have been recorded epileptiform discharge on EEG [19]. Patients presenting with new-onset seizure who do not completely recovery within 30–60 minutes after the end of seizure should be considered for hospital admission and continuous EEG-monitoring [15]. However as previously discussed routine cEEG monitoring is possible only in few hospital, so is important to perform almost Standard EEG as soon as possible. Standard EEG after a new–onset seizure is helpful to determinate the likely seizure type (focal or generalized) and to determinate the risk of recurrence after the first event. Patients with new-onset seizure have 29% of possibility to have epileptiform abnormalities on their first EEG [17] while there does appear higher if EEG is performed within 24–48 hours of new onset seizure [20,21]. King et al. [22] showed that earlier EEG has a higher rate of finding epilepsy (51%) than delayed EEG performance (34%).

Despite is not clear the optimal duration of EEG, performing a 3 hours Video EEG monitoring, showed that two-thirds of epileptiform discharges were detected within 30 minutes, while a 3 hours monitoring is a reasonable option when a routine EEG fails to detect epileptiform discharges. The latency to the first epileptiform discharge was shorter for generalized epilepsy than in patients with focal epilepsy [23].

Repeat EEG studies after an initial normal EEG often include sleep deprivation and or prolonged polysomnographic recording of sleep stages [24] because particularly in some idiopathic epilepsy, the sleep stages can induce/increase both clinical and EEG epileptic manifestations. In a Dutch study, neurologist indicated that 48% physicians requested a sleep deprivated EEG if the initial EEG and brain MRI were normal [25]. Routine sleep deprivation-EEG in a pediatric population increase the yeld of identifying epileptiform discharges from 56 to 67% [26].

Table 1. Definition of EEG terminologies. See text for more explanation.

| | |
|---|---|
| Emergency EEG (eEEG) | eEEG has been variably defined as: EEG requested for immediate performance during non-business hours, performed within 3 hours from request; EEG done toexclude various types of ‘non-convulsive status epilepticus’; EEG requested on an emergent basis and performed within one hour |
| Video-EEG (VEEG) | VEEG refers to EEG recorded for a more or less prolonged period with simultaneous video recording of the clinical manifestations |
| Continuous EEG (cEEG) | cEEG is a gold standard tool in ICU to monitoring NCSE and subclinical seizures. Almost 24 hours of continuous EEG monitoring is an appropriate screen for non-convulsive seizures in comatose and non-comatose patients. It may also help improve outcomes in patients in whom NCSE or subclinical seizures are not coexistent with other serious comorbidities like anoxic brain injury etc.<br>Preferelly, cEEG should be associated with video (VcEEG), if possible |

### 2.2. EEG in disorders of consciousness

Altered mental status, a nonspecific manifestation of brain dysfunction with variable etiology, is a common presentation in the ED [27].

The neurophysiologist does commonly encounter patients with encephalopathy/delirium (altered consciousness with impaired cognition, usually with sleep–wake cycle alteration and lethargy) or coma (an eyes-closed state of unresponsiveness) in the hospital setting.

EEG may have a great value in altered mental status patients, particularly with normal neuroimaging findings [28], although, EEG in the disorder of consciousness is highly sensitive but show low specificity. While neuroimaging such as Computerized Tomography (CT) or Magnetic Resonance Imaging (MRI) provide anatomical data, EEG is the only readily available test that provides information about the functional status of the brain. Assessing the background frequency of the

EEG as well as the presence or absence of other features (reactivity, periodic discharges such as triphasic waves).

EEG can provide insight into the patient's underlying condition, increasing or decreasing the probability of specific altered mental status etiology altered mental status etiologies, including toxic metabolic, encephalopathy, hepatic or uremic encephalopathy, encephalitis, and various types of status epilepticus including absence status, focal status, and prolonged generalized convulsive status [29], and in some cases may provide prognostic information. Also, EEG can differentiate psychogenic from organic etiologies of altered mental status patients, and aid in determining whether the pathology is focal or diffuse [11]. Although EEG slowing is a nonspecific manifestation of cerebral dysfunction, its magnitude correlates with the functional severity of a clinical encephalopathy, and its distribution can help distinguish between diffuse, focal, or multi-focal pathologies. For instance, while transient diffuse slowing is a common finding after concussion, focal slowing after head injury indicates cerebral contusion even in the absence of focal deficits on neurological examination or focal abnormalities on head CT scan [30].

### 2.3. EEG in status epilepticus

In 2015, the International League Against Epilepsy (ILAE) task force defined status epilepticus (SE) as 'a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation mechanisms, which lead to abnormally prolonged seizures' and proposed a new classification [31]. From neurophysiological point of view, is important to distinguish motor clinical seizure from subtle or non-convulsive seizure to determinate the usefulness of eEEG particularly V-EEG, both in adults and pediatrics, considering the peculiarities age-related.

The two main semiological forms are convulsive SE and non-convulsive SE (NCSE).

### 2.4. EEG in convulsive status epilepticus

Convulsive SE is a medical emergency in which intervention times are critical, they must be early as it is associated with significant morbidity and mortality. Although EEG monitoring is not considered essential in the acute diagnosis and treatment of convulsive SE, its use should be promptly considered to correctly identify a NCSE that may persist even after the pharmacological control of a generalized convulsive SE. In fact, 'subtle' or NCSE should be considered in all patients that remain comatose after the termination of the convulsive seizure, whenever paralytics condition render the neurologic examination impossible, and whenever the patient is placed in a pharmacologically induced comatose state. This consideration is important in that up to 48% of patients in one series continued to have electrical seizures (subtle SE) on EEG monitoring during the 24-hour period after treatment for generalized convulsive SE, despite having no clinical signs of ongoing convulsions [32].

Specific EEG patterns recorded after control of convulsions were shown to be significantly correlated with prognosis. In the study by Jaityl et al. [33], the presence of periodic lateralizing epileptiform discharges (PLEDs) was a functional predictor of a high mortality rate, whereas EEG normalization after convulsive SE was correlated with a good outcome. Therefore, this indicates that EEG monitoring after clinical control of generalized convulsion SE serves as a prognostic indicator, and clinical evidence argues for its availability in emergency settings [34].

Another important challenge for the ED neurology physician is the diagnosis of focal convulsive SE without impairment of consciousness, defined as a pathological condition localized to a discrete area of the cerebral cortex. In this situation, EEG monitoring must diagnose subtle status epilepticus by correlating the onset and persistence of localized ictal discharges and neuroradiological investigations can be crucial to identify symptomatic etiologies.

### 2.5. EEG in non-convulsive status epilepticus (NCSE)

It is reported that NCSE may be present in more than one third of patients with persistent, unexplained acute alterations of consciousness [35]. Typical NCSE is Absence Status Epilepticus in Idiopathic Generalized Epilepsy (Figure 1).

Therefore, eEEG is mandatory to perform diagnosis of NCSE particularly for clinical difficulty of identifying behavioral changes in the absence of a clear tonic and/or clonic seizure activity. Apart from the wide range of behavioral manifestations occurring in NCSE that justify the need for routine EEG availability, NCSE may also include various ictal morphologies that are difficult to interpret in emergency settings [36]. For this reason studies done in the ED and the ICU have reported significant delays in the diagnosis of NCSE, especially when subtle alterations were attributed to other etiologies [37–39]. To exclude this condition, a consensus statement of the American Clinical Neurophysiology Society recommend use of cEEG [40], both in adults and children, after seizures, if impaired consciousness persists after initial treatment; in cases of unexplained alteration of mental status without known acute brain injury; when specific EEG patterns are recognized on routine recording, such as, generalized periodic epileptic discharges (GPEDs), PLEDs, or (bilateral independent periodic epileptic discharges (BIPEDs); when neuromuscular blocking drugs are used in high risk patients.

Further, 8–48% of these patients with convulsive SE will have also NCSE diagnosed by EEG [32,41]. The mortality of NCSE, which can follow the treatment of generalized convulsive SE, can exceed 30% if the seizure lasts more than one hour [42].

Nevertheless, approximately 2% of EDs in the US have EEG technicians available to obtain tracings and neurophysiologists to interpret EEG 24 hours a day seven days a week, and studies have shown that it takes three hours on average to obtain and interpret an EEG in the ED [40]. Earlier recognition of NCSE, may save lives and costs by diagnosing a previously unrecognized cause of a patient's altered mental status and/or by avoiding overtreatment of presumed seizures [43].

After the introduction of V-EEG monitoring there has been increased identification of NCSE in lethargic or comatose patients in ICU. EEG appears mandatory for the diagnosis of


Figure 1. Absence status epilepticus.

NCSE relies largely on EEG findings. because the clinical signs (if any) are most often subtle, non specific and pleomorphic.

### 2.6. EEG/video-recording in pediatric age has a crucial role in differentiating two types of 'non convulsive status epilepticus' (NCSE)

*A) Autonomic Status Epilepticus (Panayiotopoulos Syndrome or 'autonomic epilepsy')*

*B) Confusional Status Epilepticus:*

*1) with focal confusional NCSE (with more or less significant impaired awareness)*

*2) with generalized confusional NCSE (absence epilepsy)*

Each of them presents peculiar and different classification difficulties, to justify as 'useful or even mandatory' carrying out long-term V-EEG recording for different reasons. In each of these two types, timely recognition and correct classification presents greater 'prognostic' (mainly in focal or generalized confusional NCSE) or crucial 'diagnostic' (mainly in autonomic epilepsy) relevance, to avoid aggressive approach in 'autonomic epilepsy' and, vice versa, on the other side, to identify and promptly stop the 'confusional status epilepticus', taking into account the potential long-term consequences, whose extent has not yet been elucidated by strong scientific evidences. In addition, although a clear-cut differentiation between 'focal and generalized confusional NCSE', on a clinical or even EEG-clinical grounds, is difficult and sometimes arbitrary, an effort should always be made for differential diagnosis because classification, prognosis and therapeutic choices can be very different.

Additionally, all forms, 'Autonomic (Panayiotopoulos Syndrome) and Confusional (focal or generalized)' NCSE, are peculiar of children or show many considerable different aspects from the 'picture presentation' of the non-convulsive status epilepticus in Adults (see the previous paragraph): from the EEG, clinical, epidemiological, etio-physio-pathogenetic and prognostics points of view; some forms, such as for Panayiotopoulos Syndrome, are exclusively present in developmental age [44]. See Figure 2 for typical Panayiotopoulos seizure.

(A) *EEG in autonomic seizures and autonomic status epilepticus in pediatric age: Panayiotopolous syndrome, a peculiar but common, benign, pediatric, epileptic condition.*

Panayiotopoulos syndrome usually occurs in pediatric population, during sleep, between 1 and 14 years, but it peaks in early childhood (between 3 and 6 years) with no apparent sex or race predilection. Autonomic seizures and autonomic SE are the hallmarks of the disease [45]. Autonomic seizures in Panayiotopoulos syndrome consist of episodes of altered autonomic function with emesis as the predominant symptom [46]. Other autonomic manifestations can be pallor (or, less often, flushing or cyanosis), mydriasis (or, less often, miosis), cardiorespiratory and thermoregulatory alterations, incontinence of urine and/or feces, hypersalivation, and modifications of intestinal motility, rarely ictal syncope or cardiorespiratory arrest are described [47]. The autonomic SE consists of these prolonged isolated or prevailing subdominant autonomic seizures, lasting more than 30 min or shorter epileptic seizures, without complete recovery between the seizures (lasting more than 30 min), characterized by manifestations of the autonomic nervous system associated with loss of consciousness [48,49]. Panayiotopoulos syndrome is frequent in pediatric age, the fever is often a trigger and autonomic SE, or secondary generalized tonic-clonic status epilepticus may rarely represent the onset of this syndrome requiring, sometimes, intensive care evaluation [45].

EEG studies, usually, describe predominant abnormalities in the posterior regions [45], although EEG recording can show, focal or diffuse bilateral spreading in extra-occipital regions, or, even, an onset in central, temporal or anterior regions. The first description of the Syndrome [50] defined this form as benign occipital epilepsy (early-onset benign childhood occipital epilepsy, or Panayiotopoulos syndrome), precisely because of the prevalence of EEG discharges in the occipital regions. Subsequently it was then clarified that the predominant and most characteristic element of this epilepsy are its autonomic manifestations, rather than the location of the anomalies, which

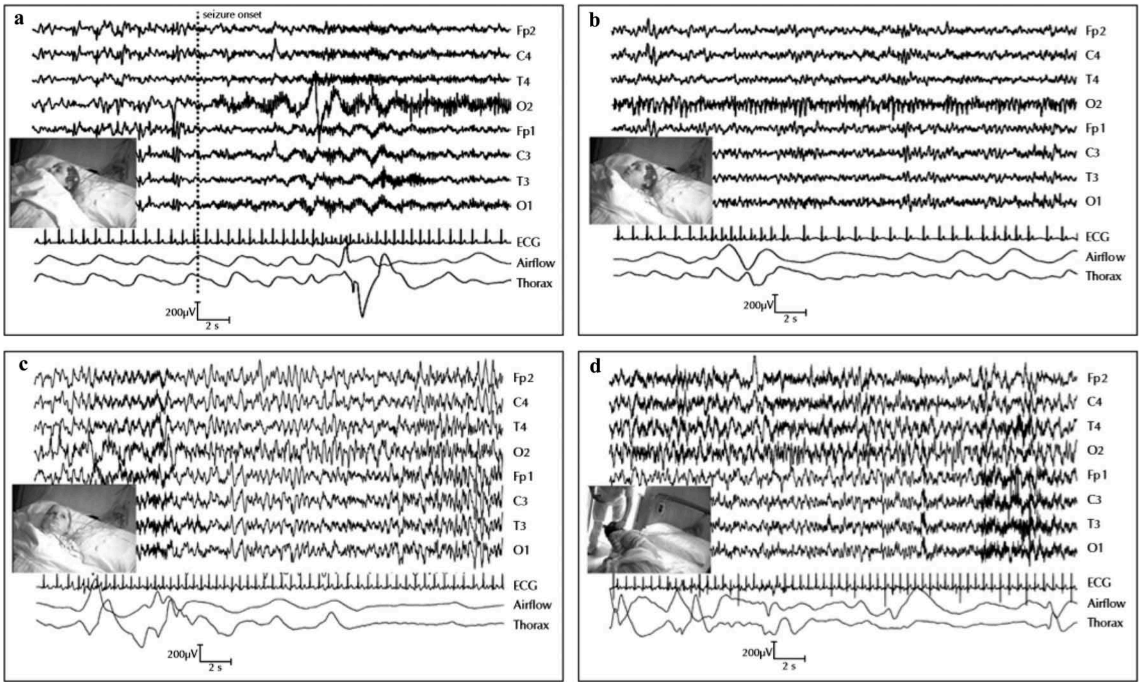


**Figure 2.** Video-EEG study of the seizure in Panayiotopoulos syndrome.

(a) Seizure onset with a run of fast spikes(at approx.7 Hz),mainly involving the right occipital region; a clear increase in heart rate is evident to get her with irregular breathing. (b) Spike-and -wave activity at 3 Hz,initially located almost exclusively in the right occipital region; heart rate irregularities correlate with irregular breathing.(c) Tonic conjugate deviation of the eyes to the left, accompanied by high-amplitude slow waves in the theta and delta range intermixed with more rapid activity and increased heart rate.(d) Same type of EEG activity and heart rate during vomiting.

does not show any specificity (stressing the multifocality and the volatility of the interictal spikes and the predominance of the autonomic symptoms and signs 'with or without' occipital symptoms); in fact it is now currently classified as 'Autonomic Epilepsy' [46].

The reason why, in this review, the PS is considered an epilepsy of interest in ED, it can be ascribed precisely to its high frequency in developmental age, and to its benign evolution, so that the correct classification in children with Panayiotopoulos syndrome in ED it allows us to avoid misdiagnosis and aggressive and inappropriate interventions on such children who can show confounding clinical pictures, in terms of the more frequent or challenging differential diagnosis (encephalitis, syncope, migraine, cyclic vomiting syndrome, motion sickness, sleep disorder, gastroenteritis, or gastroesophageal reflux) [45,47]. This is an idiopathic, benign, self-limiting, age-related epilepsy, which in most cases does not require drug therapy.

(B) *'Confusional Status Epilepticus (CSE)' in pediatric age* :
(1) *with focal NCSE*

The focal NCSE is rarely reported in children, and it occurs mainly associated with temporal lobe epilepsy or with other lesional epilepsies. In the focal NCSE cases, the ictal semiology can be represented by speech or behavioral arrest, psychotic, cognitive, emotional, stereotypical automatisms or sensory manifestations and even atonic attacks and erratic myoclonias, with or without associated autonomic ictal semiology, which, on the other hand, (if present) is clearly different from the 'autonomic manifestations' typically present in 'autonomic epilepsy' or 'Panayiotopoulos Syndrome'; in fact, in focal NCSE, if present, the autonomic ictal manifestations are not isolated, nor prolonged ictal manifestation at the onset, neither prominent and prevalent, compared with the other associated ictal symptoms/signs; the EEG anomalies in focal NCSE are even more different than the clinical ones (compared with the EEG anomalies in autonomic epilepsy), showing, in focal NCSE, fast EEG discharges over one or both temporal lobes and bilateral slow-wave activity, often intermixed with low-voltage fast activity. The interictal EEG in focal NCSE frequently shows unilateral frontocentral focus of polymorphic spikes and slow waves, which are clearly different from the cluster of EEG anomalies which can be observed in idiopathic epilepsies such as the 'autonomic epilepsy'.

These ictal manifestations can be associated with more or less significant impaired awareness. The availability of an ictal V-EEG recording and a careful and detailed neurologic exam, can sometimes be crucial to recognize other possible 'subtle', subclinical, associated ictal/interictal manifestations.

This form of epilepsy is much more rare and not so benign compared with the 'Autonomic Epilepsy', and it usually occurs in the course of temporal lobe or other lesional/cryptogenic epilepsies and the availability of high resolution neuro-radiological investigations can be decisive for diagnostic purposes. Identify cryptogenic/lesional epilepsies can be also crucial to start and choose the most appropriate drug therapy.

(4) *with generalized NCSE (Absence Status Epilepsy)*

Generalized non convulsive status epilepticus is characterized by variable degrees of clouding of mental processes, from simple slowing of ideation to complete unconsciousness associated with bilateral discharges of spike-wave-complexes. It has been variably termed as 'absence status' or 'spike-wave stupor' or 'petit mal status'. In 70% of cases 'absence status epilepticus' occurs before the age of 18 years, and most take place during the first decade. Absence status epilepticus can be in a minority of children the first manifestation at the onset of epilepsy (See Figure 1).

Considered that the impairment of consciousness can vary from barely noticeable (which permit the automatic execution of the acts of everyday life, or even the persistence of higher cognitive functions), to a severe impairment of consciousness, it results clear the importance of the EEG recording availability in ED in order to stop these conditions that rarely may be marked only by subtle behavioral alterations that last for weeks or months without even necessarily being recognized.

It is even more evident how important it is to diagnose this type of epilepsy since they are idiopathic generalized forms, which require drug therapy but, in most cases, showing an age-related, drug-responsive, benign prognosis.

### 2.7. EEG in paroxysmal non-epileptic manifestation

The incidence of psychogenic non-epileptic seizures is high, between 1.4 and 4 per 100,000 [51]. A major subcategory of these patients presents to ED with pseudo-status epilepticus, an entity that puts patients at a high risk of iatrogenic harms comprised of unnecessary intravenous medications and ventilatory support for airway protection [34].

The hallmark of EEG evaluation in non-epileptic manifestation is about normal background activity during paroxysmal episode. Then is important also to identify all clinical signs and symptoms during this clinical manifestation to differentiate itself from other possible different episodes during his life.

Vital signs are measurements routinely monitored at the site of a medical emergency by medical professionals and health care providers include body temperature, heart rate, respiration rate, and blood pressure. In recent years, several investigators have focused on studying changes of heart rate and other vital signs before, during, and after the seizures [52,53]. Ictal vital signs extremely fluctuate and thus analysis of their changes can predict morbidity and mortality in patients with epileptic seizures and may help in differentiation between it and recurrent psychogenic non-epileptic attacks or other paroxysmal events that are frequently misdiagnosed as epilepsy [54]. Usefulness of V-EEG monitoring became a fundamental step in identifying epileptic seizures, verifying its type, and to diagnose others spells that mimic epilepsy.

Despite the established role of prolonged V-EEG in diagnosing non-epileptic seizure, identification of suggestive features is still important for further diagnostic monitoring. Documenting a negative interictal EEG in the ED might enhance clinical suspicion and thereby preclude the need for inpatient monitoring. Thus, in many respects, the 'certification' of a negative EEG might increase the diagnostic yield of other clinical/historical features in an acute setting where access to V-EEG is restricted.

It is worth noting that certain types of epileptic seizures, such as frontal lobe seizures, may be mistakenly diagnosed as psychogenic [34].

### 2.8. EEG in encephalitis

EEG should be performed routinely as part of the diagnostic work-up of encephalitis because may provide supportive information. Although nonspecific background slowing in most cases, EEG is a sensitive marker for brain dysfunction and is abnormal in 87% to 96% of children with encephalitis. Cases of Herpes simplex encephalitis often have a temporal focus and may show PLEDs, both proper and plus types (see Figures 3 and 4). PLEDs can also be seen in other disease as well; however, this is neither a sensitive nor a specific finding for Herpes Simplex Encephalitis. EEG may also be used to monitor for seizure activity or to differentiate encephalitis from non-

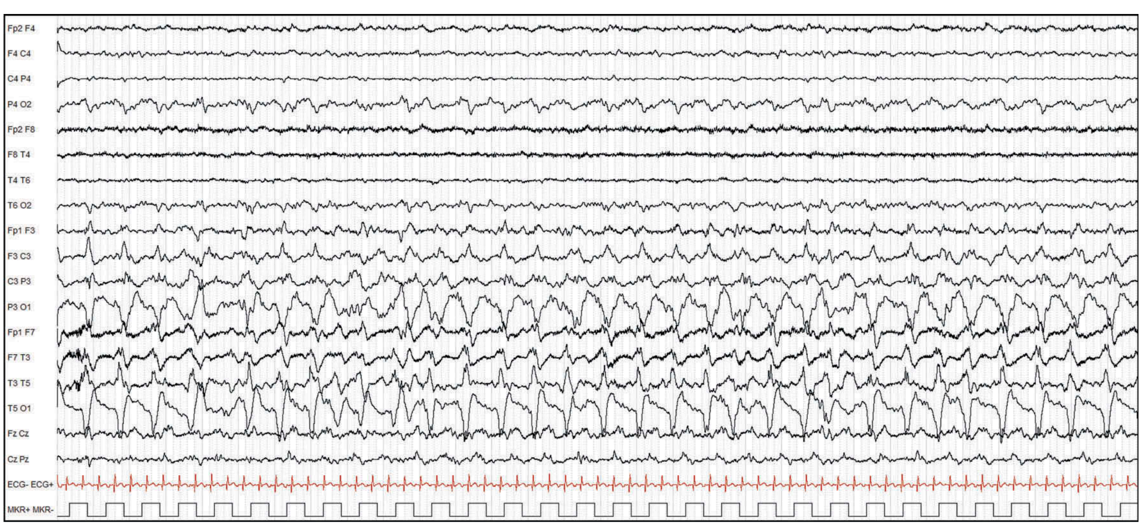


Figure 3. Periodic Lateralized Epileptiform Discharges (PLEDs proper). PLEDs-proper, in which the periodicity of the discharges is relatively stable, the discharges are simply configured and uniform, and there are no associated rhythmic discharges.

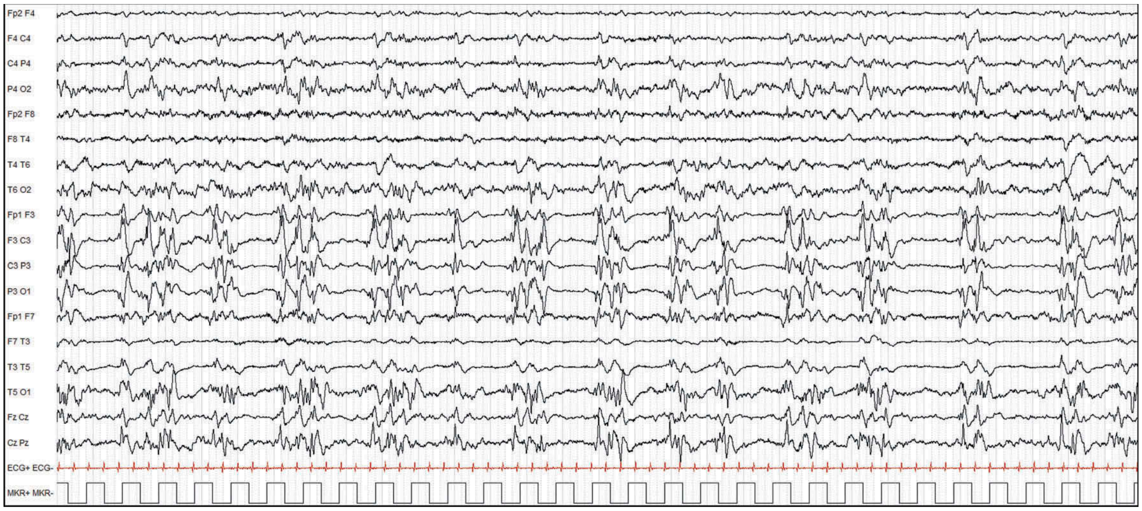


**Figure 4.** Periodic Lateralized Epileptiform Discharges (PLEDs plus). PLEDs-plus, in which the periodicity of the discharges is variable and there is associated low amplitude rhythmic activity with the discharges.

convulsive status epilepticus or 'subtle' seizures, in patients who are confused, obtunded, or comatose [55].

The EEG may show extreme delta brushes, a finding which suggests the presence of underlying anti-NMDA receptor encephalitis and temporal slowing and epileptic discharges in limbic encephalitis [56]. In a recent study EEG was helpful in prognosticating the outcome of children with acute encephalitis [57].

In a population of adult patients with acute encephalitis, electrographic seizures were detected in 41% of patients, and 28% met criteria for status epilepticus. Seizures were associated with lower serum sodium levels, a higher frequency of cortical imaging abnormalities, and a number of EEG abnormalities including PLEDs, focal slowing, and generalized delta activity and low or suppressed voltage [58]. Nearly half of the patients diagnosed with limbic encephalitis have experienced seizures. Epileptic seizures and facio-brachial dystonia can precede the appearance of cognitive problems by several months [59,60] in autoimmune encephalitis.

Subacute sclerosing panencephalitis (SSPE) is a slowly progressive brain disorder caused by mutant measles virus. SSPE affects younger age groups, characterized by cognitive decline, periodic myoclonus, gait abnormalities, vision loss, and ultimately to a vegetative state. EEG shows characteristic periodic discharges (see Figure 5).

EEG abnormalities are observed almost constantly in NMDA-R encephalitis, but they are usually nonspecific (focal or diffuse polymorphic slow-waves). However, some particular patterns that could help to suggest the diagnosis have been recently described: generalized rhythmic delta activity (GRDA); excessive beta frequency activity; and their co-occurrence that gives rises to, a peculiar pattern named 'extreme delta brush', which is considered highly specific of the disease [61]. In limbic encephalitis, the EEG may show generalized slow-wave activity even in the absence of onconeural antibodies but with hyperintensities in bilateral mesial temporal structures [62]. In atypical cases in which MRI can be normal, or

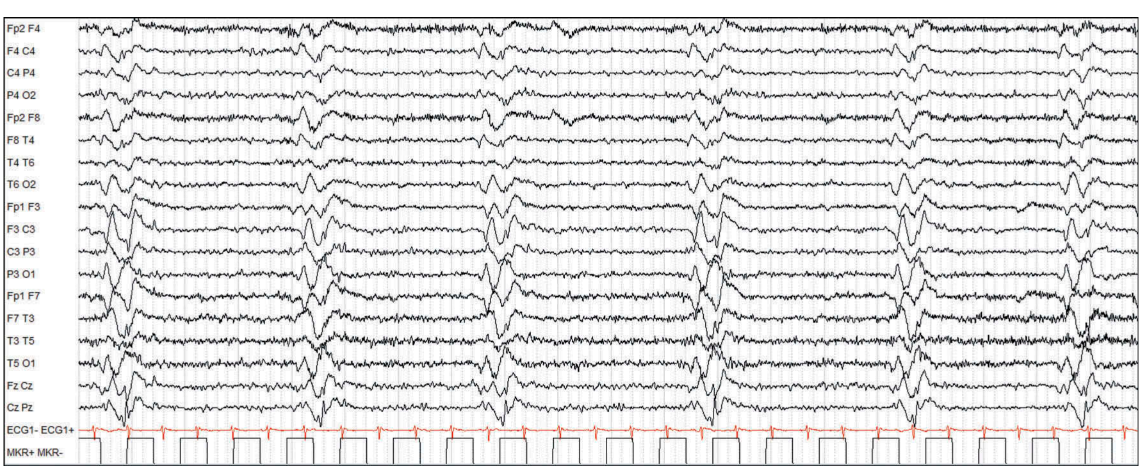


**Figure 5.** Periodic EEG complexes consist of generalized and synchronous bursts of sharp-slow wave discharges occurring every 3 seconds in patient with Subacute Sclerosing Panencephalitis (SSPE).

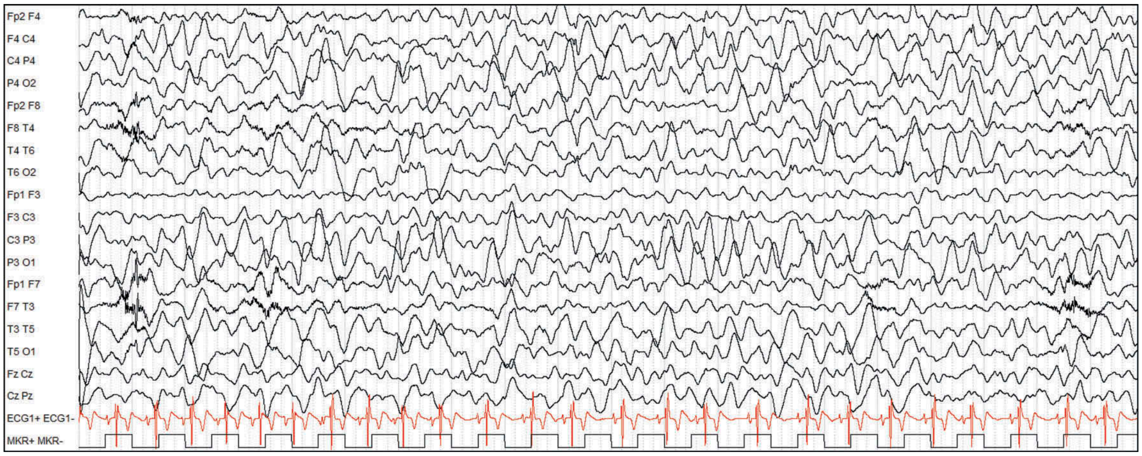


Figure 6. Diffuse slow activity in hepatic encephalopathy.

alternately in which it does show limbic changes but with atypical clinical features, the EEG can help guide the physician.

The degree of EEG abnormalities has a positive correlation with the hospitalized stay time, and ICU stay time. An extreme delta brush pattern in 30% of 23 patients with anti-NMDA receptors encephalitis, associated with a more prolonged recovery is previously described [63]. The rates of consciousness and movement disorder, and coma increased significantly with the degree of EEG abnormalities [64].

### 2.9. EEG in encephalopathy

Encephalopathy is an acute/subacute consciousness disorder which may result from a variety of causes, including metabolic (see Figure 6), toxic, infective or structural brain dysfunction [65]. Despite the poor specificity of slow wave EEG, is well known that selective cortical damage is associated to slowing of the resting background in the theta frequency, while subcortical white matter damage produces polymorphic delta activity [66].

While neuroimaging study can provide information about structure alteration of the brain, EEG can provide brain function in patients with disorder of consciousness [67].

The sensibility of EEG is demonstrated from functional imaging studies that showed decrease of cerebral blood flow in patients with encephalopathy of different etiologies [68]. Furthermore, other studies demonstrated that blood-brain barrier damage, altered neuro-transmissions and inflammatory cytokines release may also play a role in EEG abnormalities [68–70].

EEG may be helpful to clinician to classify background activity, focal o diffuse alteration, reactivity and presence or absence of other typical features (triphasic waves, intermittent rhythmic slow activity, periodic discharges), to determinate the etiologies of encephalopathy or coma and to provide prognostic information [71]. See Figure 7 for diffuse brain injury and Generalized Periodic Epileptiform Discharges (GPEDs)

From prognostic point of view Sutter et al. [72,73] found that mixed delta-theta background activity or triphasic waves had a significantly lower mean GCS than did patients with

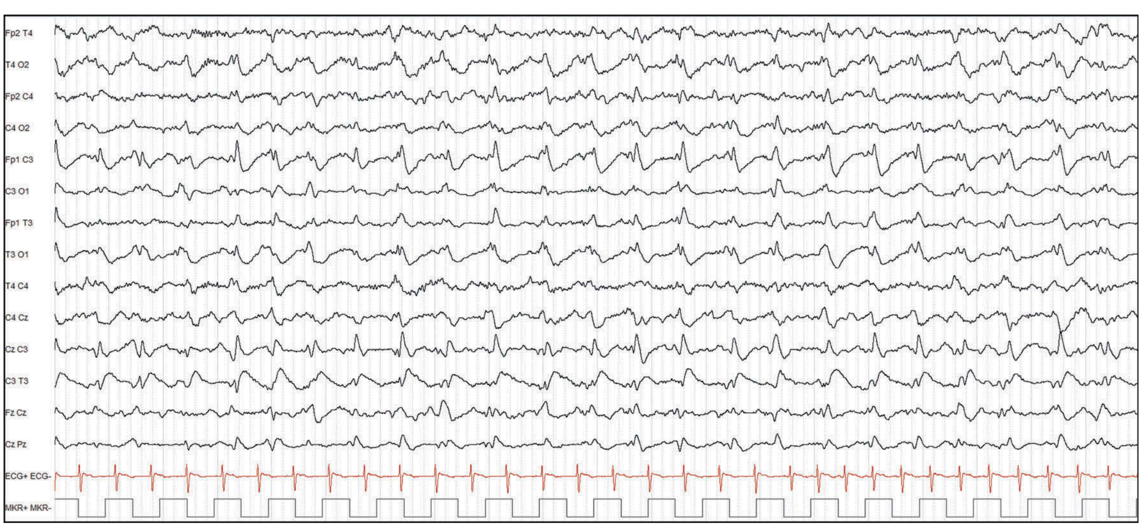

Figure 7. Generalized Periodic Epileptiform Discharges (GPEDs).

Frontal Intermittent Rhythmic Delta Activity (FIRDA) or theta background. Although FIRDA can be associated with encephalopathy, this pattern is usually recognized as nonspecific EEG findings [74]

However, in this section it is useful to discuss some peculiar EEG features in detail.

*A) Change background EEG frequency*

Beta activity on EEG in patients with disorder of consciousness may be secondary to hyperthyroidism, drug toxicity (benzodiazepine and phenobarbital) alcohol withdrawal, stimulants such as amphetamines and cocaine, as well as some antidepressants [65,75,76]. Other clinical condition, ranging from mild encephalopathy due to metabolic disorder to mild cognitive impairment, may induce however a Slow Posterior Dominant Rhytm [76]. With worsening of metabolic derangements, the EEG can progress to diffuse background slowing.

(E) *Change of EEG activity*

Theta slowing of background activity is seen in mild metabolic or infective encephalopathy and can worsening to FIRDA or diffuse delta slowing with more severe forms of septic associated encephalopathy [70]. Elevated theta and decreased alpha activity are described in patients with Creutzfeldt–Jakob disease [77]. Theta-delta activity was associated with an unfavorable outcome, especially in intracranial hemorrhage [65]. Slower delta activity is seen with more severe manifestations of encephalopathy. Patients with polymorphic delta activity have large areas of subcortical white matter dysfunction [66] or severe metabolic or infectious derangements [70]. Delta EEG may be also associated with posterior reversible encephalopathy.

(F) *Rhytmic EEG pattern*

Recent studies have identified FIRDA in a number of more chronic, structural conditions, hyperglycemia and other metabolic derangements, cortico-basilar degeneration and progressive supranuclear palsy, and Creutzfeldt–Jacob disease [76,78–80]. FIRDA was usually found on faster background activity and patients were less likely to be admitted to the ICU than were other patients often having a favorable outcome [65,72].

(G) *Periodic discharges*

Triphasic waves (TWs) are typical feature of some encephalopathy and were most commonly found on a theta-range background [72,73]. TWs are hypothesized to represent an abnormality in the activity of the underlying thalamo-cortical network. They are well described in hepatic encephalopathy but are now known to arise from a variety of etiologies, including antibiotics, intoxications, brainstem infarction, steroid-responsive encephalopathies, white matter lesions, and anoxic injury, multi-organ failure. TWs have a dose-dependent relationship with higher levels of urea and ammonia and are also associated with a higher risk of in-hospital mortality [72,73].

Encephalopathic patients often have periodic or rhythmic discharges other than TWs, which may be thought of as lying on the 'ictal–interictal continuum'. The nature of periodic pattern can provide some insight into the etiology of the patient's brain injury: PLEDs usually correspond to structural focal lesion [71]. However, metabolic process such as hyperglycemia may also rarely trigger PLEDs [76]. Bilateral independent periodic epileptic discharges (BIPEDs) are commonly reported in infections or anoxic injury, as well as stroke, tumors, and metabolic disorders [81,82]. Generalized periodic epileptic discharges (GPEDs) are also commonly seen in toxic-metabolic states or sepsis [81,83–85]. Generalized rhythmic delta activity (GRDA) is frequently seen in brain tumors and cerebrovascular disease [65,86]. Lateralized rhythmic delta activity (LRDA) is most often found in patients with a focal cortical injury on the side of the LRDA [87]. All the lateralized or focal periodic patterns are highly associated with seizures, and one of these patterns on a routine EEG in a patient with encephalopathy may prompt more prolonged monitoring to assess for subclinical seizure activity [81,85,87].

(H) *Sleep features*

The presence of sleep architecture on EEG seems to be an indicator of less severe brain injury and dysfunction. In a study of encephalopathy of various etiologies, K-complexes were independently associated with a good outcome in patients without focal brain lesions [73]. Patients with sleep elements (spindles, vertex waves, K-complexes) on EEG had a better outcome after brain injury [88]. Sleep spindles on EEG have also been associated with 1-month awakening after coma [89].

### 2.10. EEG in coma

Coma is a deep state of prolonged unconsciousness in which a person cannot be awakened, which arises when cortical and brainstem pathways are injured. Patients with coma have longer intensive care unit stays than do patients without coma, with more complications and higher mortality [71].

EEG is useful in distinguishing coma from other states of unresponsiveness and has also been used to prognosticate survival [90]. The level of coma may be associated with slowing of EEG frequency: primarily delta-range EEG in 50% of patients with GCS of 4. With increasing GCS, patients had faster EEG frequencies, and with GCS of 7 or higher, the majority (80%) had a theta-range EEG, while none had a delta-range EEG [91]. EEG amplitude is also related to the severity of coma: with more severe metabolic etiologies, the EEG amplitude increases as frequency decreases [84]. However, in comatose or minimally conscious patients undergoing intensive rehabilitation, lower EEG amplitude predicts a worse recovery [92].

EEG patterns are a dynamic finding; any given patient may progress through several different patterns within a recording [93]. One study showed that in 10% of post-cardiac arrest patients, an initially favorable EEG had later deterioration (i.e. Burst-Suppression pattern, see Figure 8), associated with poor outcome, which is thought to be related to delayed neuronal damage [94].


Figure 8. Burst-Suppression (BS).

In comatose patients, EEG reactivity to stimulation is also good prognostic indicator, with reactivity 88.9% specific for predicting a good outcome in one study of 50 comatose patients [95].

(A) *Typical EEG pattern*

Beta coma is seen in some drug intoxications such as sedatives and benzodiazepines. Depending on the depth of coma, the background activity may be reactive or non-reactive. Alpha coma was classified by Husain et al. [96] into three main etiologies: post-anoxic injury, toxic encephalopathies, and alpha coma following a brainstem stroke (i.e. locked-in state). In most cases, outcomes are very poor with little meaningful recovery, particularly if reactivity is absent [36,96,97]. However, if a reactive alpha pattern is present following a drug overdose, the prognosis is good with over 90% recovering [90].

The patient with theta coma are usually clinically stuporous. In these patients if EEG is not reactive outcome is often poor [98]. Similar to alpha coma, a slower, diffuse, unreactive theta coma pattern can also occur after hypoxic–ischemic injury, with a poor prognosis [99]. Alpha coma may transition to theta coma, and vice versa [93].

Delta coma may be induced by a large disruption of the subcortical white matter. Traumatic brain injury, metabolic abnormalities, stroke, and hypoxia are additional common causes [90]. Patients with delta coma activity who survive to long-term rehabilitation placement have poorer recovery than do patients with faster frequency EEG patterns [92].

Intermittent rhythmic delta activity, which is usually frontally predominant, is a nonspecific pattern that may be seen in a variety of causes, including metabolic encephalopathies, increased third ventricle pressure, hemispheric lesions, or widespread dysfunction of the cortical and subcortical structures [66,96]. Burst suppression or Suppression burst pattern is commonly seen following severe hypoxic–ischemic injury, though it may evolve or transition later to alpha or theta coma, and then to electro-cerebral inactivity [93]. Burst suppression may also be seen in late stages of severe sepsis-associated encephalopathy; it portends a poor prognosis in this setting also, with high mortality [70]. When burst suppression is a result of drug overdose, good recovery is possible [100].

Spindle coma consists of a slow background with frequent superimposed spindle shaped bursts of 10–14 Hz activity. Other features of sleep architecture such as vertex waves or K-complexes may be present and may be induced by external stimulation [90,101]. Midbrain injuries are commonly reported causes of spindle coma [90,101], but cases following seizure, intoxication, and hypoxic–ischemic injury are also reported. Patients with post-traumatic spindle coma often have a good recovery.

(B) *Electrocerebral inactivity*

Electrocerebral inactivity, consisting of isoelectric (<2 μV), non-reactive EEG recording or electrocerebral silence, is an indicator of severe diffuse brain dysfunction. When EEG meets these criteria and there is a clear etiology without reversible conditions (i.e. sedating medications, hypotension, hypoxia, hypothermia), the EEG is consistent with 'brain death'. Patients with electrocerebral inactivity on EEG all have irreversible coma, with all patients dying or remaining in a persistent vegetative state [96].

### 2.11. EEG in traumatic brain injury

Standard EEG may help to assess the severity of trauma shortly after the event (from 10 min to 1 h), but it is not helpful in assessing subsequent or chronic clinical symptoms. Furthermore, within 24 hours EEG is often normal and any focal abnormality tends to disappear within months or few years; abnormal EEG does not predict clinical impairment and EEG do not always correlate with neurological findings. The most common EEG patterns found after a mild Traumatic Brain Injury (mTBI) are: attenuated posterior-alpha and focal irregular slow wave activity with a preponderance of theta waves in the temporal region; however, minor alpha asymmetry is of dubious diagnostic value [5]. While Liguori et al. [102], showed evidence that the EEG could be correlated with the CT scan findings in mTBI patients, Oster [103] showed that EEG study is not indicated in these patients and the examination is unrevealing and misleading and may lead to unnecessary diagnostic procedures.

In moderate to severe TBI, with deterioration of clinical conditions, loss of consciousness, drowsiness, amnesia, prolonged headache and clinical evidence of basal or non-frontal skull fracture that have been identified as risk factors for a negative prognosis, the performance of an urgent EEG is mandatory [5].

Moreover, it has been demonstrated that in severe TBI EEG correlates well with the depth of post-traumatic coma. Despite a wild variety of abnormal EEG patterns, including increased slow activity, amplitude suppression in greater injuries, typical sleep features, epileptic spikes, periodic lateralized epileptiform discharges, and triphasic waves, none is pathognomonic of trauma per se [5].

The above consequences of severe TBI could be delayed in emerging and be unnoticed by early cerebral imaging, thus justifying the need for a continuous neurophysiological monitor, for which EEG is appropriate [34].

Epileptic seizures are a common complication of moderate to severe TBI, with 4–11% of patients developing clinically apparent seizures within the first week after injury [104]. Between 52% and 100% of the seizures detected may be subclinical or associated with minimal clinical changes. Standard EEG often don't recognize seizures particularly subtle or without clinical manifestations (NCSE) [105].

In the last years however a potential indication for the use of EEG monitoring in ICU is progressively increase not only for detecting epileptiform activity in a predisposed brain, but also for monitoring impending neuropathological consequences.

The European Society of Intensive Care Medicine recommends the use of cEEG monitoring in all patients with a GCS<8, and the Neurocritical Care Society recommends cEEG monitoring for TBI patients with altered mental status or intracranial hemorrhage [106,107].

Despite incidence of isolated or recurrent epileptic seizures, patients with moderate to severe TBI also have a high incidence of status epilepticus which is associated with significant morbidity and mortality. Status epilepticus may be present in 6.6–8% of all patients with moderate to severe TBI on cEEG monitoring [108,109]. The presence of status epilepticus is associated with worse mortality. In one study of 91 patients with severe TBI on cEEG monitoring, all patients with status epilepticus died, whereas patients without status epilepticus only had a 24% associated mortality [108].

### 2.12. EEG in ischemic stroke

Stroke is the third most common cause of death and the leading cause of acquired neurologic handicap in adult [110]. EEG is very sensitive to changes in neuronal function resulting from ischemia [111]. In conditions of mild to moderate ischemia, EEG shows changes in rhythmic activities. A decrease in alpha and beta activities is followed by an increase in the delta band, depending on the severity and duration of ischemia [112]. During ischemic stroke, excitotoxicity triggers a number of events that can further contribute to tissue death. Such events include peri-infarct depolarizations and spreading depolarization within the ischemic penumbra [113].

Acute stroke causes 22%–30% of adult status epilepticus cases [114]. Long-term cumulative risk of post-stroke epilepsy varies from 3% to 30% [115] and the risk of developing epilepsy remains high up to 10 years after stroke [116].

cEEG monitoring studies show that most early seizures occurred within the first 24 h [109]. Some studies have attempted to delineate interictal EEG findings able to predict a major risk of developing seizures or epilepsy and PLEDs are significantly more frequent in patients with early-onset seizures [117]. The involvement of the parieto-temporal junction, supramarginal gyrus, and superior temporal gyrus enhances the risk of developing epilepsy [118]. Early seizures are more frequently partial seizures. Late seizures are more frequently generalized or secondarily generalized [117].

In conclusion, in stroke patients, cEEG is indicated in the case of unexplained alteration of consciousness and should be performed in the case of PLEDs.

## 3. Emergency EEG in ICU

EEG is one of the simplest ways to investigate cerebral activity in ICU, easily recorded at the bedside and sensitive to changes in both brain structure and function. In the last years cEEG monitoring in critically ill patients has increased and it was recommended by international guidelines with well-defined indications [9]. There was a > 10-fold increase in cEEG use from 2004 to 2013 [119].

However, despite cVEEG was associated with lower in-hospital mortality, it was used for only 0.3% of the critically ill population. While administrative claims analysis supports the utility of cV-EEG for critically ill patients, findings suggest variable benefit by diagnosis, and investigation with greater clinical detail is warranted [120].

Within the ICU setting, cV-EEG is most useful in the evaluation of subclinical or subtle clinical seizures. cV-EEG allows the implementation of management goals and therapeutic targets, such as seizure cessation or specific EEG pattern such as burst suppression, may precede and predict neurologic outcome. It is also useful in identification of dynamic lesion such as secondary ischemia. cEEG can help in the assessment of altered mental status and rarely contributes to establishing a specific etiology to an unclear presentation. Nevertheless, clinical indications for cVEEG are often not homogeneous in literature, and it still represents an underused tool. Traditional 30–60 min EEG recordings identify non-convulsive seizures in only 45–58% of patients in whom seizures are eventually recorded. To exclude this condition, a consensus statement of the American Clinical Neurophysiology Society recommends use of cVEEG after seizures, if impaired consciousness persists after initial treatment [40].

About 80–95% of patients with non-convulsive seizures can be identified within 24–48 h [9]. Recording for at least 24 h is recommended, but in some cases shorter or longer periods may be necessary. In ICU patients admitted for seizures, DeLorenzo et al. [32], using cEEG monitoring, reported that non convulsive seizures were observed in 48% of cases, and NCSE in 14% of cases.

Claassen et al. [121] also identified non convulsive seizures with cEEG monitoring in 25% of 570 critical care patients; in a subgroup of 110 patients who had seizures before monitoring, non-convulsive seizures were observed on cEEG in 43% of the cases. In one prospective study, 75% of diagnoses of NCSE or non-convulsive seizures on cEEG were not preceded by clinical seizures. In these cases, symptoms may be subtle or absent [9] as well as in coma, associated with NCSE in over 10% of patients [122]. Moreover, cEEG is mandatory when specific EEG patterns are recognized on routine recording [123] such as GPEDs, PLEDs, or BIPEDs. Several conditions have been reported to be correlated with NCSE, with incidences of up to 30% in subarachnoid hemorrhage, head injury, intracerebral hemorrhage, CNS infections. The incidence may be even higher in post-anoxic encephalopathy, ranging from 10 to 60%. When neuromuscular blocking drugs are used cEEG should be initiated as soon as possible, as higher morbidity and mortality has been observed, and early treatments are likely to be more effective [124].

Finally, EEG data in ICU are easily contaminated by signals of non-neural origin. Artifact independent components can be often identified via visual inspection but are often ambiguous and even experts may disagree about how to categorize a particular component [125]. Recently, the application of methods to automatically select artifactual components for rejection, and implementation of selection algorithms looks particularly promising [126,127]. However, no automated method can accurately isolate artifacts without supervision, although this may improve objectivity and reproducibility in reporting pre-processing procedures. Continuous EEG monitoring in the ICU is different from a planned EEG due to the rather urgent nature of the indications, and it requires a close collaboration between neurophysiology teams and intensive care teams. It is preferable to work within a tele-EEG network, so that the neurophysiologist has the possibility to give an interpretation on call, and EEG recordings can be sent to them for remote interpretation. Tele-EEG procedures and related technical guidelines can be considered a complete medical act that needs to be carried out with the same quality requirements as a local one in terms of indications, formulation of the medical request and medical interpretation. As it regards for the related human resources, materials, tele-EEG network and medical fees of the physician interpreting the EEG recording, it must adheres to the same quality requirements and should follow all rules and guidelines of good medical practices. The financial model of this organization must be detailed in a convention between all parties involved (physicians, management of the healthcare structure, and the company providing the tele-EEG service) which, on the other hand, must respect all rules for safety and confidentiality, and ensure the traceability and storing of all requests and reports [128–130].

## 4. Conclusions

Although considered a cheap, not invasive, highly accurate diagnostic investigation, still today, obtaining an EEG recording in ED and or ICU, as soon as possible, it is not yet been routine, in many institutions. Nevertheless, Video-EEG and eEEG recording in ED and or ICU has been demonstrated to be a fundamental diagnostic tool in the both, Adult and Pediatric Age, due to its high predictive value for identification of neurological conditions/diseases requiring both, further diagnostic investigations and hospitalizations, or, conversely, a prompt, crucial, specific, and appropriate therapeutic choice, followed by an immediate resolution of, sometimes, dramatic clinical pictures; in some of these patients, an EEG recording can identify suggestive or, more rarely, pathognomonic EEG pattern, allowing a specific disease diagnosis.

On the basis of a literature revision, we can suggest expanding the use of eEEG and Video-EEG recording in ED and, obviously even more in ICU, for neurological patients not only affected by possible status epilepticus (convulsive or non-convulsive) and paroxysmal non-epileptic events, but also in many other conditions/diseases presenting with neurological signs and symptoms associated with major traumatic brain injuries, cerebral infections, acute cerebrovascular accidents, change in mental status, coma, and, even, rarely, for persistent headache not responsive to therapeutic drugs administrations.

## 5. Expert opinion

EEG recording remains the hallmark tool for investigations of patients with epileptic seizures or paroxysmal non-epileptic events, change in mental status and coma, as well as assessment of brain death. The transition from analogue EEG recording to technological digital instrumentation and, even more recently, to the synchronous recording of video and EEG (V-EEG) for prolonged periods of time, has led, in many centers, to the possibility of using in emergency departments, an efficient and effective diagnostic tool that can improve and speed up the timely diagnosis, on the one hand, of immediately reversible conditions/diseases and, on the other hand, to the identification of conditions/diseases that require hospitalization in ICU and surveillance and prolonged treatments and more in-depth investigations. These technological digital improvements, over the last 20 years, have expanded rather dramatically the requests for immediate performance of eEEG, in many neurological conditions, for adults and children admitted in ED and ICU, although, vice versa, the scarce availability of equipment and technical experts in this field, limit the performance of EEG in these settings.

Another important aspect is represented by the electrically hostile environments which is a challenging question, particularly in pediatric age; however, given the importance of the need for a correct emergency diagnosis to reduce mortality and health costs, efforts must be multiplied to arrive at a correct and timely diagnosis of some neurological conditions, whereas a prolonged EEG registration can provide clear and crucial information for timely diagnosis and therapy adequate and decisive. In fact, the recognition and appropriate diagnosis of conditions such as seizures, pseudo-seizures, change in mental status of different etio-pathogenetic origins and brain death in the ED are essential for immediate and future management and prognosis.

Starting from this perspective, our review was aimed to evaluate all EEG literature in Adult and Pediatric emergency clinical setting (both, in ED and ICU), considering EEG, Video-EEG and cEEG, and highlighting their role in differential diagnosis and predictive prognosis.

Focusing our attention mainly on three aspects (when, why and age-related peculiarities for requiring eEEG/V-EEG in ED) we discuss the main and relevant clinical conditions in which eEEG should be considered; in addition, it should be specified, that the decision to initiate an EEG recording, the frequency of review, and communication of results to ED and/or ICU caregivers/

physicians can be different according to local resources, monitoring indications, EEG findings, and the patient's clinical status.

The effort we have put into writing this literature review is justified by the need to make a prompt recognition of some severe neurological diseases and their differential diagnosis by using of awake/sleep standard-EEG, eEEG and/or Video-poligraphic/EEG in EDs/ICUs, in acute, brief or long-term monitoring, in emergency settings. Although, the use of these techniques, and mainly the timing of their performances, have been still widely debated, this review highlights the importance of emergency EEGs and video-EEGs in clinical practice for the recognition of different neurological diseases and a related correct diagnostic and therapeutic follow-up, as well as, to evaluate the cost–benefit balance of their standard management in acute and emergency rooms.

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