Rev Mex Neuroci ahora en CONACyT

Vol. 19, issue. 4 (July-august 2018)

Revista Mexicana de Neurociencia

Publicación oficial de la Academia Mexicana de Neurología A.C.

Revista Mexicana de Neurociencia; 19,4 (2018):70-81

Órgano Oficial de Difusión de la AMN

Academia Mexicana de Neurología, A.C.

www.revmexneuroci.com / ISSN 1665-5044

Review

Liane Aguilar-Fabré,[1] René Francisco Rodríguez-Valdés.[1]

[1]*Clínica del Sistema Nervioso. Departamento de Investigación Biomédica. Facultad de Medicina. Universidad Autónoma de Querétaro. México.*

Periodic electroencephalographic patterns: a controversial and infrequent finding

Patrones electroencefalográficos periódicos: un hallazgo controversial e infrecuente

Abstract

Periodic electroencephalographic patterns are discharges usually epileptiform in appearance, which occur at regular intervals associated with acute brain injury such as cerebral vascular disease and encephalitis. They are commonly classified as periodic lateralized epileptiform discharges, periodic lateralized epileptiform discharges bilateral independent, generalized epileptiform discharges, triphasic waves and stimulus-induced rhythmic, periodic or ictal discharges. The aim of this study is to make a review of the periodic EEG patterns, emphasizing the importance of their recognition and clinical significance. The clinical significance of the periodic EEG patterns is uncertain, it is related to a variety of etiologies and suggest that these patterns are unequivocally epileptogenic in some cases and these patterns associated with poor prognosis. Their recognition and classification are important to establish an accurate correlation between clinical, neurological, laboratorial and neuroimaging data with the EEG results, which allow making adequate therapeutic benefit of critical patient behavior.

Keywords

Periodic electroencephalographic patterns, periodic epileptiform discharges, generalized epileptiform discharges, triphasic wave.

Resumen

Los patrones electroencefalográficos periódicos (PEP) son descargas con apariencia epileptiforme que aparecen a intervalos regulares asociadas a una lesión cerebral aguda como la enfermedad cerebro vascular y las encefalitis. Estas descargas se producen a intervalos regulares y se clasifican comúnmente como: descargas epileptiformes lateralizadas periódicas, descargas epileptiformes lateralizadas periódicas independientes bilaterales, descargas epileptiformes generalizadas, las ondas trifásicas y las descargas ictales o periódicas rítmicas inducidas por estímulos. El objetivo de este trabajo es hacer una revisión de los patrones electroencefalográficos periódicos, haciendo énfasis en la importancia de su reconocimiento y su relevancia clínica. La importancia clínica de los patrones periódicos en el electroencefalograma es incierta y está relacionada con diversas etiologías. Algunos autores sugieren que estos patrones son inequívocamente epileptogénicos y se asocian con pronósticos desfavorables. Su reconocimiento y clasificación es importante para establecer una correlación exacta entre los datos clínicos, neurológicos, de laboratorio y de neuroimagen con los resultados del electroencefalograma, lo cual permitiría establecer conductas terapéuticas adecuadas en beneficio del paciente crítico.

Palabras clave

Patrones electroencefalográficos periódicos, descargas epileptiformes periódicas, descargas epileptiformes generalizadas, ondas trifásicas.

Corresponding author:
Liane Aguilar Fabré
Nervous System Clinic Department of Biomedical Research
Autonomous University of Querétaro, School of Medicine
Calle Clavel No. 200, Col Prados de la Capilla; Querétaro, Qro. México. CP 76170
Phone: (442) 192-1200 ext: 6252
E-mail: aguilarfabre@yahoo.com

Introduction

Periodic electroencephalographic patterns (PEPs) are always an abnormal finding on the electroencephalogram (EEG). PEPs consist of discharges with diverse forms, which usually have an epileptiform appearance and occur with a regular frequency or at regular intervals intermittently.[1]

These patterns are usually classified as periodic lateralized epileptiform discharges (PLEDs), bilateral independent PLEDs (BIPLEDs), generalized periodic epileptiform discharges (GPEDs), triphasic waves, and stimulus-induced rhythmic, periodic, or ictal discharges (SIRPIDs).

The term PLEDs was introduced in 1964 by Chatrian et al.[2] although the phenomenon was described for the first time in 1952 by Echlin et al.[3] PLEDs are a relatively infrequent electroencephalographic pattern characterized by the presence of spike-and-wave complexes, or lateralized sharp waves or focal periodic or quasi-periodic present in most or all of the records.

The purpose of this review is to emphasize the importance and clinical significance of PEPs, considered controversial and infrequent. These patterns may be present in critically ill patients who need therapeutic interventions based on decision-making resulting from their interpretations, which is why we consider the approach of this topic of great importance.

Search strategy and selection criteria

A PubMed/MEDLINE search was performed using the following terms and phrases (combining two with the Boolean operator "and"): periodic epileptiform discharges, electroencephalography, PLEDs, BIPLEDs, GPEDs, triphasic wave, clinical significance, etiologies.

The following limits were established: "only items with links to free full text, Humans, Meta-Analysis, Practice Guideline, Review, English, Spanish, published in the last 10 years."

On some occasions, publications cited in the articles initially selected were included, regardless of the year of publication, provided they presented current and important information for the development of the review. Only those articles in which the full text could be reviewed were used as references. Those that were considered to have important methodological deficiencies, that were not adequate to the specific topic, or which presented information that had been covered sufficiently in other texts that were considered of higher quality or more recently updated, were discarded.

Periodic Lateralized Epileptiform Discharges (PLEDs)

PLEDs are the most common PEP. These are spike-and-wave complexes followed by slow waves that repeat every 1 to 2 seconds. The periodic complexes are limited to a focal cerebral area (often a hemisphere).[2] PLEDs have a frequency of 0.2-0.3 Hz, are often biphasic, triphasic, or polyphasic, and are associated with a localized attenuation of base activity between discharges.[2,4,5] Periodicity is what characterizes this electroencephalographic pattern, generally varying less than 20% in the same subject but able to vary significantly between one patient and another.[6] In 1950, Cobb7 attributed the periodicity of the discharges to a disconnection between the cortex and subcortical structures caused by a white matter lesion. However, through experimental studies, Chatrian2 showed that any injury could be associated with PLEDs. The prevalence of PLEDs in the routine EEG varies between 0.1% and 1%.[2,6,8]

They are observed in the context of multifocal or diffuse brain lesions such as anoxia and they announce an unfavorable prognosis associated with a higher mortality. Approximately 80% to 90% of patients with PLEDs experience clinical seizure activity, mainly focal motor seizures. In 1991, Reiher et al.[9] described the "PLED plus" entity characterized by PLED mixed with polyspikes of high frequency and low voltage. These have a stronger correlation with the presence of clinical crises and status epilepticus. PLEDs are generally not considered an ictal pattern, although this has

been reported and remains a subject of ongoing debate.[10,11]

Bilateral independent periodic lateralized epileptiform discharges (BIPLEDs)

Bilateral PLEDs which occur independently (BIPLEDs) were recognized by Chatrian in 1964,2 and characterized by de la Paz and Brenner in 1981.[12]

BIPLEDs occur when PLEDs are viewed in both hemispheres independently and asynchronously. This pattern is less common than PLEDs and is highly associated with the occurrence of seizures in patients with acute diseases.[13] Unlike PLEDs, BIPLEDs can be presented as asynchronous complexes that usually differ in morphology, amplitude, frequency of repetition, and topography.[14] Some studies report an incidence of BIPLEDs of 4 to 22% in the ICU and a prevalence of 0.1 in the routine EEG.[2,6,15]

A study by Fitzpatrick6 in 21 patients with BIPLEDs showed a mortality of 52% and a study conducted by de la Paz[12] showed a mortality of 61%.

BIPLEDs are typically associated with acute structural injury with or without metabolic disorders.[12,13,16] The most common cause of BIPLEDs is anoxic encephalopathy and central nervous system infections, with a high incidence of coma.[12,16]

Generalized Periodic Epileptiform Discharges (GPEDs)

Periodic discharges are defined by a "repetition of a waveform with a relatively uniform morphology and duration, with a quantifiable interdischarge interval between consecutive waveforms, and recurrence of the waveform at nearly regular intervals," where waveforms are characterized by a duration of 0.5 s or less or limited to three phases.[17]

GPEDs occur in both hemispheres symmetrically, diffusely and synchronously.[4] GPEDs are classified taking into account the interval between short and long discharges. Diffuse periodic discharges with short intervals (periodic short-interval diffuse discharges, PSIDDs) are those whose duration of the interval is between 0.5 and 4 sec. They occur in hypoxic or hepatic encephalopathies, drug toxicity, and neurodegenerative diseases such as Jakob Creutzfeldt disease.[4] PSIDDs are associated with toxic-metabolic encephalopathies and anoxic brain damage and related to a fatal evolution and severe neurological sequelae, especially those associated with repetitive myoclonic jerks. Diffuse periodic discharges with long intervals (periodic long-interval diffuse discharges, PLIDDs) are those whose duration of the interval is between 4 and 30 sec.[18]

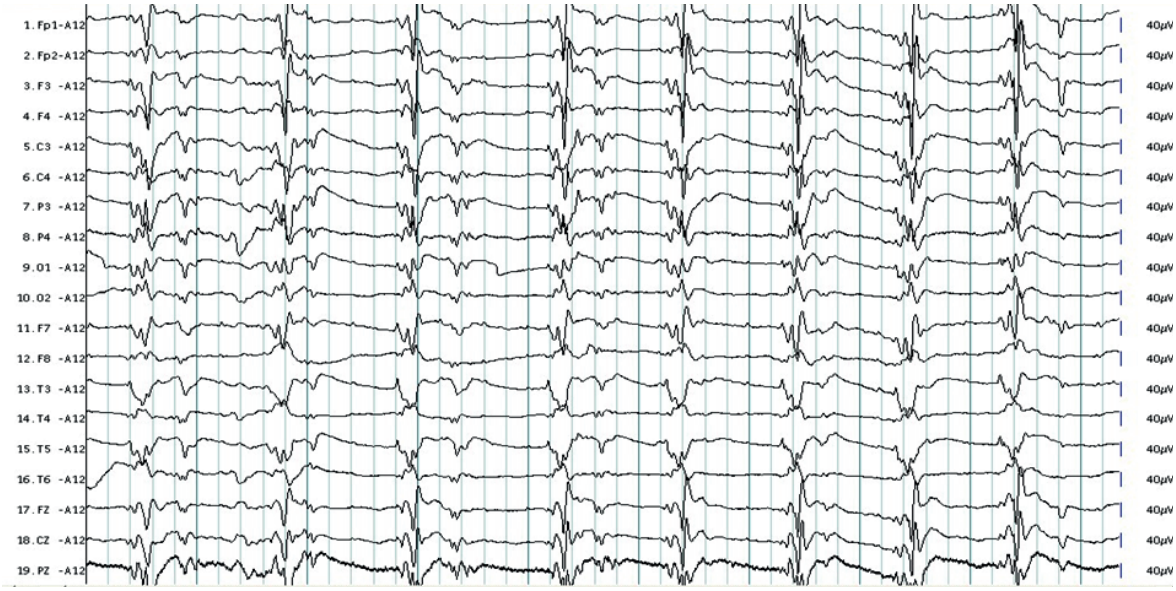
In a standard 20-minute EEG record, the incidence of GPEDs is approximately 1%,[19,20] occurring in approximately 20% of patients in a coma due to a severe postanoxic encephalopathy after cardiac arrest,[21-23] typically presenting in the first 12 to 48 hours after resuscitation.[24,25] Other causes are diffuse metabolic encephalopathy,[16,26] including encephalopathies associated with sepsis,[19] acute brain damage, and cerebrovascular accident.[23,27] An example of generalized periodic epileptiform discharges can be seen in Figure 1.

Triphasic waves

Generalized periodic discharges also include triphasic waves, a pattern initially described in 1950 by Foley.[28] This term was coined in 1955 by Bickford[29] in reference to their typical morphology characterized by three phases. They consist of periodic generalized acute waves or strongly contoured delta waves with a triphasic morphology (typically with a negative-positive-negative polarity, with a duration of each phase longer than the previous one), which are repeated between 1.0 and 3.0 Hz.

Triphasic waves are periodic and generalized, usually have a frontal predominance and do not always have an epileptiform appearance (a reason why they are often not included in the GPEDs category).[4,13] This pattern can occur in any toxic metabolic or structural encephalopathy, although the first descriptions were associated with hepatic

Figure 1. Generalized epileptiform discharges in a male 8-year-old patient with herpetic viral encephalitis.



encephalopathy.[26,30,31] Recent studies by Foreman[32] conclude that the triphasic wave is a clinically ambiguous electroencephalographic descriptor that is not reliable in the prediction of seizures or in the presence of toxic-metabolic encephalopathy. An example of triphasic waves can be seen in Figure 2.

Rhythmic ictal or periodic discharges induced by stimuli.

These EEG patterns were first described by Hirsch in 2004,[33] observing that by stimulating patients in a coma or stupor, EEG patterns of ictal appearance were obtained. These were referred to as stimulus-induced rhythmic, periodic, or ictal discharges (SIRPIDs). The SIRPIDs are considered periodic when it comes to recurrent epileptiform discharges at regular or almost regular intervals, with an identifiable inter-discharge interval. Some patients with clinical seizures have SIRPIDs, especially focal motor seizures, but this pattern is usually purely an electroencephalographic change that is not accompanied by obvious clinical manifestations. The pathophysiology, clinical, therapeutic, and prognosis of the SIRPIDs is not yet well defined.[13,33,34]

Etiology

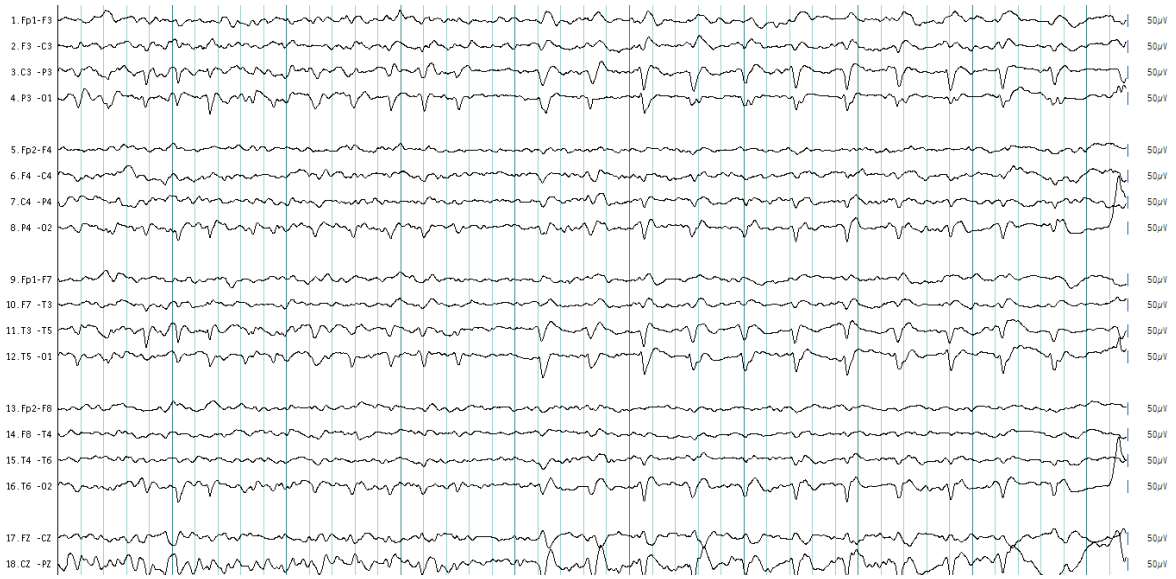
The PLEDs suggest an acute brain dysfunction of diverse etiology or a unilateral brain injury, usually of a destructive nature. This pattern has been described in patients with cerebrovascular accidents, rapidly growing brain tumors such as glioblastoma multiforme, brain abscesses, viral encephalitis, Jakob Creutzfeldt disease, bruising, and less frequently in patients with demyelinating diseases, anoxia, primary epilepsy, and migraines, among others.[13,35-39]

In cases of cerebral infarction, PLEDs are obtained in the area adjacent to the infarction. This area is partially affected by the disease but is still capable of generating electrical activity. Some authors emphasize the importance of the presence of structural brain injury associated with metabolic disorders in the production of PLEDs.[13,40,41]

BIPLEDs and GPEDs are associated with diffuse or multifocal brain damage as occurs in anoxia and announce an unfavorable prognosis with high mortality.[4,6,12,42]

A study conducted by Orta[16] reported that approximately 45% of patients with periodic

Figure 2. Triphasic waves in a male 65-year-old patient with hepatic encephalopathy and a consciousness disorder.



epileptiform discharges presented evidence of an associated acute etiology, particularly patients with BIPLEDs and GPEDs. On the other hand, there was a statistical association between acute etiology and a higher probability of death only in those patients who had PLEDs. However, patients with BIPLED and GPED had a higher mortality (29% -39% vs 24%) than the PLED group.

Some authors have studied the association of GPEDs with structural damage. Structural changes have been observed in approximately 78% of patients with GPEDs,[19,20] most have found a predominance of subcortical lesions typically in subcortical gray matter either alone[19,27] or in combination with cortical lesions.19 The absence of structural lesions in magnetic resonance imaging has been reported in 20-25% of patients with GPEDs.[8,19]

Evolution

PLEDs are usually associated with acute-subacute presentation of the underlying disease process.[40] In patients with chronic brain diseases, PLEDs are usually observed during an acute process. The PLEDs are transient and resolve spontaneously between two to three weeks, the discharges tend to decrease in amplitude and distance until they disappear.[13,30,43]

Chronic PLEDs have been reported in patients with chronic epilepsy, during alcohol withdrawal or in chronic toxic metabolic syndrome.[8,14,16,43] Many authors consider PLEDs the reflection of acute brain damage associated or not with seizures depending on many factors, such as individual propensity, the existence of underlying processes and the coexistence of metabolic disorders. The evolution is more related to the age of the patient and the etiology than to the specific periodic pattern.[8,43,44]

Gurer45 reported that 7% of the 71 adults studied exhibited chronic PLEDs and that chronic lesions were found in 35% of these patients. Fitzpatrick6 showed a similar incidence of 10% attributable to the presence of a cortical dysplasia or a severe distant brain injury, all had partial seizures. Fushimi[46] described a patient who presented bilateral PLEDs for more than six months and stated that there were no notable symptoms except for a slight deterioration of memory. Da Silva and Bartolucci[47] reported that the PLEDs disappeared before four days in most of their patients and that this pattern was replaced by the

appearance of a slow base activity, delta rhythms, voltage suppression periods, and focal paroxysmal activity.

Ictal vs. interictal pattern

Garzón[43] performed a prospective study in 55 patients, with a total of 62 epileptic status and 254 ictal/postictal EEG records and analyzed the relationship between PLEDs and status epilepticus.

This researcher showed that although PLEDs were not always associated with seizures and status epilepticus, it could be unequivocally an ictal pattern. An increase in focal glucose metabolism has been shown to be associated with PLEDs, reinforcing their probable epileptogenic nature.[48]

Other investigators[13,30] consider the PLEDs an interictal change or an unstable ictal-interictal continuum; although their pathophysiology is unknown, they could indicate an ictal pattern in some cases.

Studies have reported that PLEDs are generally associated with the state of clouding in 95% of patients, focal seizures and neurological signs in 80%, and a continuous partial epilepsy in 30% of patients.[2,39] A study conducted by Snodgrass[42] reports clinical seizures or status epilepticus in the course of the disease in 90% of patients: 50% presented a partial motor status epilepticus, 22% partial motor seizures, 6% continuous partial epilepsy, 6% isolated generalized seizures, and 8% generalized epileptic state.

Seizures that occur in patients with PLEDs may be partial, generalized sensorimotor seizures, complex partial epileptic status, and continuous partial epilepsy.[2,49-51]

PLEDs plus and BIPLEDs plus have stronger correlations with clinical crises and status epilepticus.[6,9] Foreman[23] demonstrated a strong association between non-convulsive status epilepticus and GPEDs while the presence of triphasic waves has a low association with the development of epileptic seizures.[32]

Baykan[52] reported that the extension of PLEDs is also important with respect to the association with seizures. Their results showed that PLEDs that appeared prominent on one side but with a slight contralateral spread had a stronger relationship with status epilepticus or with frequent recent seizures than when PLEDs presented a very strict location.

The pathophysiological processes underlying the PLDEs are still controversial. Some studies have reported hypermetabolism and hyperfunction in the PLED centers studied through PET and SPECT respectively.[48,53] Hypermetabolism is a condition usually associated with ictal patterns, PLEDs, in this case, represent a partial epileptic state.[48] Assal[54] came to a similar conclusion, that hyperperfusion was probably related to partial epileptic status. Other authors through studies conducted with SPECT and IMR concluded that this pattern was probably ictal.[55-57] The increase in cerebral focal blood flow and metabolism found during PLEDs is in contrast to the hypometabolism usually seen during interictal discharges. Finally, Singh[58] concluded that the pattern was ictal and that it should be considered as an epileptic state due to the presence of a continuous pattern, it would also be a continuous partial epilepsy. Many authors consider it a non-convulsive status epilepticus, a subtle status, or a partial status epilepticus.[58,59]

Nei60 reported that PLEDs are the only characteristic of the EEG related to a poor prognosis in epileptic status regardless of the etiology. Snodgrass[42] found that most EEGs with PLEDs were obtained within the first four days with convulsive activity or epileptic status and postulated that the phenomenon of PLEDs in the EEG could be considered as the final stage of epileptic status.

PLEDs can be considered as an electroencephalographic activity closely associated with recent seizures, which is a manifestation of an increase in neuronal excitability caused by various etiologies.[52] A study by Orta[16] reports that the absence of seizures at the beginning (acute etiology) was associated with death.

PLEDs should be verified in patients in intensive care units who do not recover their usual alert level. If there is severe brain disease in the terminal phase, then medication with antiepileptic drugs should not be considered.

Some authors have reported that antiepileptic medication has been effective. Terzano[61] reported that carbamazepine (CBZ) was effective, concluding that acute PLEDs can represent a non-convulsive status epilepticus. In this same study, the authors observed that patients did not respond adequately to mental tests when the frequency of discharge of PLEDs was 2/sec, 25% responded correctly when the frequency was 0.5/sec, and significantly 80% responded correctly when there were no PLEDs. Corda62 also used CBZ to normalize the "electroclinical state." Medication with barbiturates and phenytoin has not been very helpful, however, good responses to treatment with sodium valproate and felbamate have been reported, resulting in the abolition of PLEDs.[63,64]

In certain medical conditions, specific medications not considered antiepileptic drugs can be effective. For example, in a patient with herpes simplex encephalitis, acyclovir was administered and the PLEDs disappeared.[65] In a case of meningoencephalitis, corticosteroid therapy was dramatically effective.[66] In the case of PLEDs associated with multiple sclerosis, intravenous steroids are needed for complete recovery, although standard antiepileptic drugs are partially effective.[67]

Conclusions

The clinical importance of PEPs remains uncertain and controversial. PEPs are seen in a wide range of etiologies and they are electrographically heterogeneous. Therefore, patients should be carefully investigated for infectious, toxic-metabolic, and/or intracranial lesions, and a non-convulsive status epilepticus should be considered. Its recognition is important to try to establish an exact correlation between the clinical, neurological, laboratory, and neuroimaging data with the results of the EEG and to guide the therapeutic decisions.

Conflicts of interest

The authors declare that there are no potential conflicts of interest to report regarding this scientific report.

Funding sources

The authors have not declared any source of funding for this scientific report.

References

1. Andraus MEC, Andraus CF, Alves-Leon AV. Periodic EEG patterns: importance of their recognition and clinical significance. *Arq Neuropsiquitr.* 2012;70:145-151.
2. Chatrian GE, Shaw GM, Leffman H. The significance of periodic lateralized epileptiform discharges: an electrographic clinical and pathological study. *Electroencephalogr Clin Neurophysiol.* 1964;17: 177-193.
3. Echlin FA, Arnett V, Zoll J. Paroxysmal high voltage discharges from isolated and partially isolated human and animal cerebral cortex. *Electroencephalogr Clin Neurophysiol.* 1952;4:147-164.
4. Brenner RP, Schaul N. Periodic EEG patterns: classification, clinical correlation, and pathophysiology. *J Clin Neurophysiol.* 1990;7(2):249-267.
5. Téllez-Zenteno JF, Pillai SN, Hill MD, Pillay N. Chronic PLEDs with transitional rhythmic discharges (PLEDs-plus) in remote stroke. *Epileptic Disord.* 2007; 9(2):164-169.
6. Fitzpatrick W, Lowry N. PLEDs: clinical correlates. Can J Neurol Sci. 2007;34(4): 443-450.
7. Cobb W, Hill D. Electroencephalogram in subacute progressive encephalitis. *Brain.* 1950;73:392-404.
8. García-Morales I, Gracia MT, Galán-Dávila L, *et al*. Periodic lateralized epileptiform discharges: etiology, clinical aspects, seizures, and evolution in 130 patients. *J Clin Neurophysiol.* 2002;19:172–7.
9. Reiher J, Rivest J, Grand'Mason F. Periodic lateralized epileptiform discharges with transitional rhythmic discharges. *Electroencephalogr Clin Neurophysiol.* 1991;78:12-17.
10. Pohlmann-Eden B, Hoch DB, Cochius JL, *et al*. Periodic lateralized epileptiform discharges: a critical review. *J Clin Neurophysiol.* 1996; 13:519-530.
11. Pohlmann-Eden B, Hoch OB, Cochius JI, *et al*. Stroke and epilepsy: critical review of the literature. Part I: Epidemiology and risk factors. *Cerebrovasc Dis.* 1996;6:332-338.
12. de la Paz D, Brenner RP. Bilateral independent periodic lateralized epileptiform discharges. *Arch Neurol.* 1981;38: 713-715.
13. Chong DJ, Hirsch LJ. Which EEG patterns warrant treatment in the critically ill? Reviewing the evidence for treatment of periodic epileptiform discharges and related patterns. *J Clin Neurophysiol.* 2005; 22:79-91.
14. Brenner RP. EEG in convulsive and nonconvulsive status epilepticus. *J Clin Neurophysiol.* 2004;21:319-331.
15. Gross DW, Wiebe S, Blume WT. The periodicity of lateralized epileptiform discharges. *Clin Neurophysiol.* 1999;110: 1516-1520
16. Orta DSJ, Chiappa KH, Quiroz AZ, Costello DJ, Cole AJ. Prognostic implications of periodic epileptiform discharges. *Arch Neurol.* 2009;66: 985-991.
17. Hirsch LJ, LaRoche SM, Gaspard N, Gerard E, Svoronos A, Herman ST *et al.* American Clinical Neurophysiology Society´s Standarized Critical Care EEG Terminology: 2012 version. *J. Clin Neurophysiol.* 2013;30(1):1-27.
18. Andraus MEC, Andraus CF, Alves-Leon SV. Periodic EEG patterns: importance of their recognition and clinical significance. *Arq Neuropsiquiatr.* 2012;70 (2):145-151.
19. Yemisci M, Gurer G, Saygi S, Ciger A. Generalized periodic epileptiform discharges: clinical features, neuroradiological evaluation and prognosis in 37 adult patients. *Seizure.* 2003;12(7):465–72.
20. San-Juan OD, Chiappa KH, Costello DJ, Cole AJ. Periodic epileptiform discharges in hypoxic encephalopathy: BiPLEDs and GPEDs as a poor prognosis for survival. *Seizure.* 2009;18:365–8.
21. Rossetti AO, Logroscino G, Liaudet L, Ruffieux C, Ribordy V, Schaller MD, *et al*. Status epilepticus: an independent outcome predictor after cerebral anoxia. *Neurology.* 2007;69(3):255–60.
22. Tjepkema-Cloostermans M, Hofmeijer J, Trof R, Blans M, Beishuizen A, van Putten MJAM. EEG predicts outcome in patients with postanoxic coma during mild therapeutic hypothermia. *Crit Care Med.* 2015;43(1):159–67.
23. Foreman B, Claassen J, Abou Khaled K, Jirsch J, Alschuler DM, Wittman J, *et al*. Generalized periodic discharges in the critically ill: a case–control study of 200 patients. *Neurology.* 2012;79(19):1951–60.
24. Rittenberger JC, Popescu A, Brenner RP, Guyette FX, Callaway CW. Frequency and timing of nonconvulsive status epilepticus in comatose post-cardiac arrest subjects treated with hypothermia. *Neurocrit Care.* 2012;16: 114–22.

25. Ruijter BJ, Van Putten MJAM, Hofmeijer J. Generalized epileptiform discharges in postanoxic encephalopathy: Quantitative characterization in relation to outcome. *Epilepsia.* 2015; 56 (11):1845-54.
26. Husain AM, Mebust KA, Radtke RA. Generalized periodic epileptiform discharges: etiologies, relationship to status epilepticus, and prognosis. *J Clin Neurophysiol.* 1999;16(1):51–8.
27. Nicolai J, van Putten MJ, Tavy DL. BIPLEDs in akinetic mutism caused by bilateral anterior cerebral artery infarction. *Clin Neurophysiol.* 2001;112(9):1726–8.
28. Foley JM, Watson CW, Adams RD. Significance of the electroencephalographic changes in hepatic coma. *Trans Am Neurol Assoc.* 1950;51:161-165. 53.
29. Bickford RG, Butt HR. Hepatic coma: the electroencephalography pattern. *J Clin Invest.* 1955;34:790-799.
30. Hirsch LJ, Brenner RP. Periodic discharges and other controversial EEG patterns. In: Hirsch LJ, Brenner RP (Eds). *Atlas of EEG in critical care.* Chichester: Wiley-Blackwell, 2010: 129-160.
31. Brigo F, Storti M. Triphasic waves. *Am J Eletroneurodiagnostic Technol.* 2011;51:16-25.
32. Foreman B, Mahulikar A, Tadi P, Claassen J, Szaflarski J, Halford J, Dean B, *et al*. Generalized periodic discharges and ¨triphasic waves¨: A blinded evaluation of inter-rater agreement and clinical significance. *Clinical Neurophysiology.* 2016;127: 1073-1080.
33. Hirsch LJ, Claassen J, Mayer SA, Emerson RG. Stimulus-induced rhythmic, periodic, or ictal discharges (SIRPIDs): a common EEG phenomenon in the critically ill. *Epilepsia.* 2004;45:109-123.
34. Hirsch LJ, Brenner RP, Drislane FW, *et al*. The ACNS subcommittee on research terminology for continuous EEG monitoring: proposed standardized terminology for rhythmic and periodic EEG patterns encountered in critically ill patients. *J Clin Neurophysiol.* 2005;22:128-135.
35. Jacome DE. Periodic EEG patterns in cerebral fat embolism. *Clin Electroencephalogr.* 1983;14:27-34.
36. Au WJ, Gabor AJ, Vijayan N, Markand ON. Periodic lateralized epileptiform complexes (PLEDs) in Creutzfeldt-Jakob disease. *Neurology.* 1980;30:611-617.
37. Pohlmann-Eden B, Hoch DB, Cochius JI, Chiappa KH. Periodic lateralized epileptiform discharges: a critical review. *J Clin Neurophysiol.* 1996;13:519-530.
38. de los Reyes EC, McJunkin JE, Glauser TA, Tomsho M, O'Neal J. Periodic lateralized epileptiform discharges in La Crosse encephalitis, a worrisome subgroup : clinical presentation, electroencephalogram (EEG) patterns, and long-term neurologic outcome. *J Child Neurol.* 2008;23:167-172.
39. Gandelman-Marton R, Rabey JM, Flechter S. Periodic lateralized epileptiform discharges multiple sclerosis: a case report. *J Clin Neurophysiol.* 2003;20(2):117-121.
40. Neufeld MY, Vishnevskaya S, Treves TA, *et al*. Periodic lateralized epileptiform discharges (PLEDs) following stroke are associated with metabolic abnormalities. *Electroencephalogr Clin Neurophysiol.* 1997;102:295-298.
41. Chu NS. Periodic lateralized epileptiform discharges with preexisting focal brain lesions. Role of alcohol withdrawal and anoxic encephalopathy. *Arch Neurol.* 1980;37:551-554.
42. Snodgrass SM, Tsuburaya K, Ajmone-Marsan C. Clinical significance of periodic lateralized epileptiform discharges: relationship with status epilepticus. *J Clin Neurophysiol.* 1989;6:159-172.
43. Garzón E, Fernández RM, Sakamoto AC. Serial EEG during human status epilepticus: evidence for PLED as an ictal pattern. *Neurology.* 2001;57:1175-1183.
44. Silva AB, Bertolucci PHF. Descargas periódicas lateralizadas II. Aspectos evolutivos. Arq Neuropsiquiatr 1988; 46:10-15.
45. 45. Gurer G, Yemisci M, Saygi S, *et al*. Structural lesions in periodic lateralized epileptiform discharges (PLEDs). *Clin EEG Neurosci.* 2004;35:88–93.
46. Fushimi M, Matsubuchi N, Sekine A, *et al*. Benign bilateral independent periodic lateralized epileptiform discharges. Acta Neurol Scand 2003;108:55–9.
47. DaSilva AB, Bertolucci PH. Periodic lateralized epileptiform discharges: II. Developmental aspects. *Arq Neuropsiquiatr.* 1988;46:10–5.
48. Handfort A, Cheng JT, Mandelkern MA, Treiman DM. Markedly increased mesiotemporal lobe metabolism in a case with PLEDs: further evidence that PLEDs are a manifestation of partial status epilepticus. *Epilepsia.* 1994;35:876-881.
49. Markand, O. N. and Daly, D. D. Pseudoperiodic lateralized paroxysmal discharges in electroencephalogram. *Neurology.* 1971; 21: 975–981.
50. Schwartz, M. S., Prior, P. F. and Scott, D. F. The occurrence and evolution in the EEG of the lateralized periodic phenomenon. *Brain.* 1973; 96: 613–622.
51. Schrader, P. L. and Singh, H. Seizure disorders following periodic lateralized epileptiform discharges. *Epilepsia.* 1980; 21: 647–653.

52. Baykan B, Kinay D, Gökyigit A, Gürses C. Periodic lateralized epileptiform discharges:association with seizures. *Seizure.* 2000;9:402-406.
53. Lee, B. I. and Schauwecker, D. S. Regional cerebral perfusion in PLEDs: A case report. *Epilepsia.* 1988; 29: 607–611.
54. Assal F, Papazyan JP, Slosman DO, *et al*. SPECT in periodic lateralized epileptiform discharges (PLEDs): a form of partial status epilepticus? *Seizure.* 2001;10:260–5.
55. Funakawa I, Katoh H, Hara K, *et al*. A case of herpes simplex encephalitis with periodic lateralized epileptiform discharges. *Rinsho Shinkeigaku.* 1991;31: 669–73.
56. Ali II, Pirzada NA, Vaughn BV. Periodic lateralized epileptiform discharges after complex partial status epilepticus associated with increased focal cerebral blood flow. *J Clin Neurophysiol.* 2001;18:565–9.
57. Ergün EL, Salanci BV, Erbas B¸ Saygi S. SPECT in periodic lateralized epileptiform discharges (PLEDs): A case report on PLEDs. *Annals of Nuclear Medicine.* 2006; 20: 227–231.
58. Singh G, Wright MA, Sander JW, *et al*. Periodic lateralized epileptiform discharges (PLEDs) as the sole electrographic correlate of a complex partial seizure. *Epileptic Disord.* 2005;7:37–41.
59. Young GB, Blume WT. Periodic lateralized frontal epileptiform discharges with ipsilateral epilepsia partialis continua. *Epilepsia.* 2007;48:597–8.
60. Nei, M., Lee, J. M., Shanker, V. L. and Sperling, M. R. The EEG and prognosis in status epilepticus. *Epilepsia.* 1999; 40: 157–163.
61. Terzano MG, Parrino L, Mazzucchi A, *et al*. Confusional states with periodic lateralized epileptiform discharges (PLEDs): a peculiar epileptic syndrome in the elderly. *Epilepsia.* 1986;27:446–57
62. Corda D, Rosati G, Deiana GA, *et al*. "Erratic" complex partial status epilepticus as a presenting feature of MELAS. *Epilepsy Behav.* 2006;8:655–8.
63. Hughes JR, Fuller T. The use of felbamate in patients with periodic lateralized epileptiform discharges (PLEDs). *Clin Electroencephalogr.* 1995;26:180–3.
64. Rejdak K, Papuc E, Dropko P, *et al*. Acute stroke-elicited epilepsia partialis continua responsive to intravenous sodium valproate. *Neurol Neurochir Pol.* 2008;42: 157–60.
65. Shimada K, Koh CS, Hashimoto T, *et al*. A case of aseptic meningoencephalitis with recurrent abnormal behavior, status epilepticus and aphthous stomatitis, improved by acyclovir. *Rinsho Shinkeigaku.* 1989;29:367–70.
66. Nakamura A, Hashimoto T, Matsuda M, *et al*. Non-herpetic fulminant meningoencephalitis with periodic lateralized epileptiform discharges. *J Clin Neurosci.* 2002;9:190–2.
67. Maingueneau F, Honnorat J, Isnard J, *et al*. Partial non-convulsive status epilepsy in multiple sclerosis. *Neurophysiol Clin.* 1999;29:463–72.

Revista Mexicana de Neurociencia, 2018; 19(4): 70-81
www.revmexneuroci.com