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LETTER TO THE EDITOR

Stimulus aborted periodic epileptiform discharges: A case description and possible relationship with stimulus induced discharges

KEYWORDS
SIRPID (stimulus-induced rhythmic, periodic, or ictal discharges);
Arousal;
Cortico-thalamic circuits;
Periodic EEG abnormalities

An EEG pattern is considered periodic when it consists of discharges recurring at regular or nearly regular interval with an identifiable interdischarge time. If the pattern becomes continuous with no separation between individual discharges, it is considered rhythmic rather than periodic. Ictal discharges are defined as any rhythmic discharges or spike-wave pattern with definite evolution in frequency, location, or morphology. ''Epileptiform'', a term commonly used in the EEG literature, refers to sharply contoured discharges not necessarily related to seizures (Hirsch et al., 2004). In periodic epileptiform discharges (PEDs) repetitive sharp waves, spikes, or sharply contoured waves are recorded without clear evolution in frequency and location; the abnormalities must recur at regular or nearly regular intervals with an identifiable periodicity. Frequent periodic EEG patterns are classified as:

- PLEDs (periodic lateralized epileptiform discharges)
- GPEDs (generalized PEDs)
- BiPLEDs (PLEDs occurring bilaterally, but independently and asynchronously)
- Triphasic waves.

When PEDs are consistently induced by alerting stimuli such as auditory stimuli, sternal rub or other patient-care activities the pattern is defined as SIRPIDs (stimulus-induced rhythmic, periodic, or ictal discharges). Some patients have SIRPIDs that manifest as clinical seizures, particularly focal motor seizures, but this pattern is usually a purely electrographic change, with no obvious clinical manifestations (Hirsch et al., 2005).

Our report describes a patient with GPEDs which were consistently ''abolished'' by alerting stimuli: this EEG pattern could be considered as the reverse of stimulus-induced discharges or SIRPIDs.

A 70-year-old woman was admitted to the neurology service for fatigue, anxiety and malaise followed by lethargy, disorientation and fever. A neurological examination revealed neck stiffness and disturbed consciousness. On the following day, the patient developed stupor. T1-weighted MRI showed hypointense signal change within the posterior horns of the lateral right ventricle. T2-weighted MRI showed multiple, tiny, hyperintense foci within the basal ganglia with blurring of the bi-frontal and bi-occipital gray/white matter junction. An examination of the CSF revealed 1374 leukocytes/mm^3 accompanied by high protein (648 mg/dL) and low glucose levels (4 mg/dL). CSF cultures evidenced Streptococcus Pneumoniae sensitive to penicillin. Following the diagnosis of pneumococcal meningoencephalitis she was started on 2 g of meropenem three times daily, 2 g of ceftriaxone twice daily, and 4 mg of dexamethasone four times daily. At admission, the EEG showed generalized theta slowing and runs of epileptiform discharges characterized by diffuse spike-wave complex with maximum amplitude (150—200 μV) over the right centroparietal channels occurring every 1—2 s: this pattern was considered corresponding to the pattern defined as GPEDs (Fig. 1). Rhythmic or ictal discharges, and triphasic waves were not recorded. The GPEDs consistently disappeared for 2—7 s after nociceptive and acoustic stimulations were applied (Fig. 2). Because of the EEG findings, levetiracetam 3000 mg daily was empirically started as seizure prophylaxis. This stimulus abolished GPED pattern persisted during the first ten days of treatment and was absent at two weeks from the onset; generalized periodic delta—theta with sporadic alpha activity was recorded at 3 weeks and arrhythmic widespread delta—theta on alpha dominant activity was observed 3 and 4 months after the acute meningitis. Treatment was continued for 30 days and gradually tapered with remission of her symptoms. After discharge, patient was noted to have gait apraxia and residual bradiphrenia with mild cognitive deficits. We interpreted the suppression of GPEDs induced by stimuli as a sign of reactivity of the EEG rhythms and thus as a possibly favorable sign. The relatively favorable outcome of the patient, who survived

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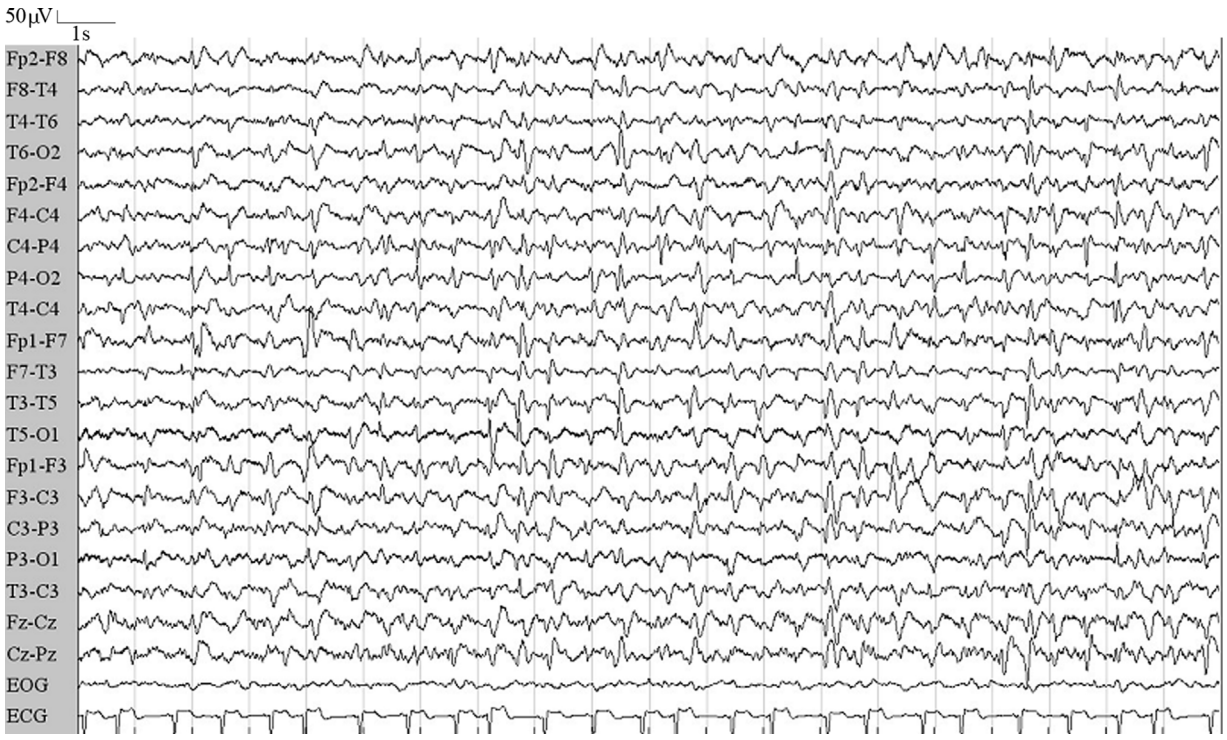

Figure 1 Generalized theta slowing and periodic runs of epileptiform discharges (GPEDs) characterized by diffuse spike-wave complex with maximum amplitude (150—200uV) over the right centro-parietal channels and occurring every 1—2 s.

from severe meningitis although with residual neurological deficits, could be cautiously considered as supportive of our interpretation.

Our finding shows that a pattern of stimulus abolished periodic epileptiform discharges can be observed in a case of acute encephalopathy: this pattern might be considered as the reverse of the documented SIRPID pattern (Hirsch et al., 2004).

Despite these apparently different EEG patterns, with opposite reactivity to stimuli, similar mechanisms might underline the two phenomena. PEDs are thought to represent an increment in cortical ''irritability'' (Hirsch et al., 2004), they are observed in metabolic disorders (Neufeld et al., 1997) and can rarely behave like seizures responding to benzodiazepines (Kaplan and Duckworth, 2011), occurring as ictal manifestations (Handforth et al., 1994), or in terminal phases of status epilepticus (Pohlmann-Eden et al., 1996). Gloor (1968) and Gross et al. (1998) suggest that PEDs represent an abnormal response of the cortex and thalamocortical neurons to rhythmic burst firing generated by the reticular thalamic nucleus, suggesting a dysfunction of cortical-subcortical circuits in the pathophysiology of periodic patterns.

The effects of arousal and sleep on periodic EEG patterns have been studied mostly in triphasic waves, which are commonly associated with hepatic encephalopathy, renal failure and anoxic injury. Triphasic waves can be increased or reduced during waking and sleep (Baldy-Moulinier et al., 1981; Egido et al., 1996; Gross et al., 1998). Noxious or auditory stimulation increased triphasic waves (Boulanger et al., 2006) while had no effect on concomitant epileptiform patterns suggesting that the response to stimulation could be helpful to distinguish different conditions with periodic discharges. Arousal induced increments of infantile spasms and of clonic activity in epilepsia partialis continua were also described (Froscher, 1991). Stimulations can induce bursts of epileptiform activity in postanoxic encephalopathy (Van Cott et al., 1996).

The occurrence of SIRPIDs probably involves arousal neuronal circuits including the midbrain reticular formation, and reciprocal connections between cortex and thalamus (Steriade, 2000).

We hypothesized that, similarly to SIRPIDs, the pathophysiology of stimulus abolished periodic epileptiform discharges probably involves dysregulation of thalamocortical and other cortical-subcortical projections in a brain with hyperexcitable cortex. Stimulation of various modalities may provide an afferent input into the thalamus via sensory or arousal pathways. The sensory input can de-activate or activate abnormal cortical circuits abolishing or inducing periodic epileptiform discharges. In our patient only periodic discharges have been demonstrated to be aborted by stimulation: therefore a possible acronym SARPIDs (Stimulus-Abolished Rhythmic, Periodic, or Ictal Discharges), in comparison with the documented SIRPIDs acronym, will not be fully justified until a series of patients is studied showing that ''rhythmic'' and ''ictal'' discharges are aborted in the same way.