Epileptic Disord 2005; 7 (1): 37-41

Periodic lateralized epileptiform discharges (PLEDs) as the sole electrographic correlate of a complex partial seizure

Gagandeep Singh, Mary-Anne Wright, Josemir W. Sander, Matthew C. Walker

Department of Clinical & Experimental Epilepsy, Institute of Neurology, London, United Kingdom

Received August 20, 2004; Accepted December 15, 2004

ABSTRACT – We describe a patient in whom the only electrographic manifestation of a complex partial seizure recorded by video-EEG telemetry was periodic lateralized epileptiform discharges (PLEDs) with a left anterior temporal emphasis. The abnormality, which persisted throughout the whole recorded seizure, lends support to the claim that PLEDs can be an ictal phenomenon.

Key words: complex partial seizure, periodic lateralized epileptiform discharges, electroencephalographic seizure

Correspondence:
M.-C. Walker
Department of Clinical & Experimental Epilepsy
Institute of Neurology
Queen Square
London WC1N 3BG
United Kingdom

Periodic lateralized epileptiform discharges (PLEDs) are sharp transients, such as spikes or sharp waves, which repeat in a periodic or semiperiodic fashion, having regional or lateralized distribution. Since their first description, there has been continued debate concerning their nature (Chatrian *et al.* 1964, Bozkurt *et al.* 2002, Brenner 2002). Typically, they are noted upon EEG in the setting of an acute neurological insult with impairment of consciousness, and are commonly associated with seizures in 50-70% cases (Garcia-Morales *et al.* 2002). In most cases, PLEDs however, either presage seizures or follow episodes of status epilepticus. Descriptions of ictal PLEDs, i.e., those that are temporally concurrent with clinical seizures are scarce (Terzano *et al.* 1986, Assal *et al.* 2001). We report their occurrence in a prolonged complex partial seizure recorded by surface EEG during prolonged, telemetric monitoring for presurgical evaluation.

Case report

An 18-year-old woman was admitted to the telemetry unit of the National Hospital for Neurology and Neurosurgery, London, for presurgical evaluation. She had her first seizure at the age of 10 years, when she became encephalopathic following a prodrome comprising fever, malaise and lymphadenopathy. Encephalitis was suspected and she was admitted to a different hospital, where she underwent MRI that revealed non-specific, left temporal lobe abnormalities. Cerebrospinal fluid examination revealed no abnormality. She was dis-

charged from hospital within a week having fully recovered. A few months later, she developed her habitual seizures, which persist to the present day. Her seizures have been frequent since onset, currently on a weekly basis and have proved resistant to several antiepileptic drugs. She was referred for consideration of epilepsy surgery. On admission, her neurological examination was unremarkable. Neuropsychological assessment showed marked impairments of verbal and non-verbal memory. MRI showed clear-cut, left hippocampal sclerosis with no other abnormality.

She underwent 95 hours of video telemetry recording with surface EEG without drug reduction. The interictal EEG revealed background activity of 9-10 Hz, intermixed with theta and delta activity, and multifocal, epileptiform sharp wave discharges, although the latter were most frequent over the left anterior temporal region.

A single, habitual seizure was recorded. This began with an aura of a non-specific, cephalic sensation followed initially by behavioral arrest, and one minute later by oral automatisms and bilateral eyelid blinking. After 30 seconds there was posturing of the right upper limb, followed by right lower limb automatisms and then left upper limb automatisms. She was then positioned by the telemetry staff, and following this remained unresponsive and uncooperative, while demonstrating intermittent shivering movements mainly of the left upper limb for 24 minutes after clinical seizure onset at which time she was noted to be responsive but aphasic upon testing.

With seizure onset, there was no change in the EEG *(figure 1)*. The first EEG change occurred 50 seconds after clinical onset, and consisted of left hemisphere PLEDs that were of maximal amplitude over the anterior and mid-temporal electrodes *(figure 2)*. These spread transiently to the right hemisphere and were then obscured by muscle artifacts, but persisted largely at a frequency of 0.6-0.7 Hz for 20 minutes, at which time they became increasingly irregular and finally disappeared nearly 30 minutes after clinical onset.

Discussion

PLEDs have been recognized as a distinct EEG phenomenon for 40 years, yet their relationship to ictal activity remains controversial (Chatrian *et al.* 1964, Bozkurt *et al.* 2002, Brenner 2002, Garcia-Morales *et al.* 2002). On one hand, an ictal basis has been supported by SPECT studies that have demonstrated regional hyperperfusion during the occurrence of PLEDs that convert to hypoperfusion once they resolve (Young *et al.* 1977). On the other, PLEDs are not usually seen in ictal EEGs of seizures recorded in presurgical monitoring units or intensive care units. It is not clear from some of the larger studies describing PLEDs, whether they preceded, followed or were concurrent with seizures in patients with acute neurological illnesses or status epilepticus. Few authors consider them to be part of an ictal-interictal continuum.

There is limited literature on PLEDs occurring as ictal electrographic phenomena. Terzano *et al.* described the occurrence of PLEDs during recurrent, prolonged, confusional episodes in elderly individuals (Terzano *et al.* 1986). Administration of benzodiazepines acutely, led to both clinical and electrographic resolution, and chronic administration of carbamazepine prevented further recurrences. Responsiveness of both PLEDs and clinical abnormalities to anticonvulsants provided evidence of their ictal nature, but it is also likely that such a relationship is an age-related phenomenon restricted to the elderly. Other authors have described subtle motor phenomena such as eyelid closure in association with the periodic EEG discharges, and have proposed that such cases represent non-convulsive status epilepticus (Young *et al.* 1977). Lee *et al.* (2000) reported the concurrent occurrence of PLEDs in the hemisphere contralateral to the limb with epilepsia partialis continua in one patient with Creutzfeldt-Jacob disease (Lee *et al.* 2000). However, the temporal correlation between limb jerks and electrographic seizures was not clearly established. Finally, transient negative clinical phenomena including aphasia, homonymous hemianopia and agnosia have been associated concurrently with PLEDs (Primavera *et al.* 1996). The ictal basis of these isolated negative phenomena remains debatable in as much as focal parenchymal brain lesions of vascular or inflammatory nature have not been rigorously ruled out. Although such neurological episodes may represent seizures, one has to bear in mind the rarity with which seizures present as negative phenomena.

The present case report is the first and, to our knowledge, the only case of the occurrence of ictal PLEDs in a clinically overt, complex partial seizure recorded during routine pre-surgical telemetric monitoring. They were, however, not the initial EEG manifestation, but occurred late in the seizure.

Most accounts of PLEDs have been in association with destructive brain lesions in individuals not previously known to have epilepsy. Interictal PLEDs of lasting duration have however, been described rarely in chronic epilepsies (Westmoreland *et al.* 1986). PLEDs in the present case differ in being restricted to a clinical seizure.

The origin of PLEDs is a matter of controversy. While some authors propose a cortical origin, others believe that PLEDs occur because of isolation of portions of the cerebral cortex due to subcortical inhibition. Furthermore, their functional nature is indicated by the demonstration that PLEDs could be remotely located from sites of brain lesions. We suggest that ictal PLEDs in the present case represent functional disconnection of a region of the cortex by subcortical structures or afferents as a result of a propagated ictal discharge.

In conclusion, a case of prolonged, complex partial seizures with ictal PLEDs is described. This electrographic

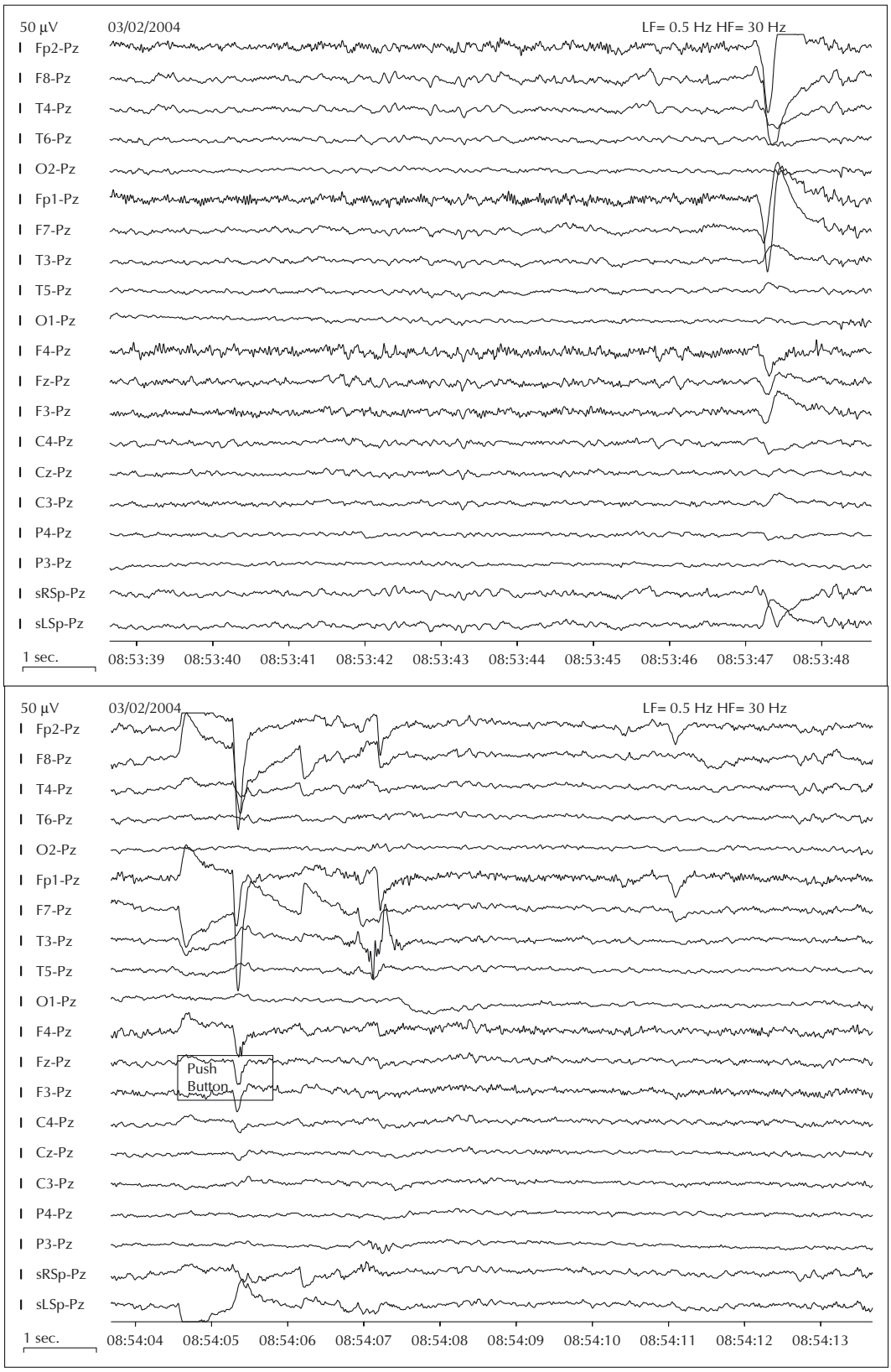

Figure 1. a: EEG prior to seizure onset. **b**: EEG at onset of seizure.

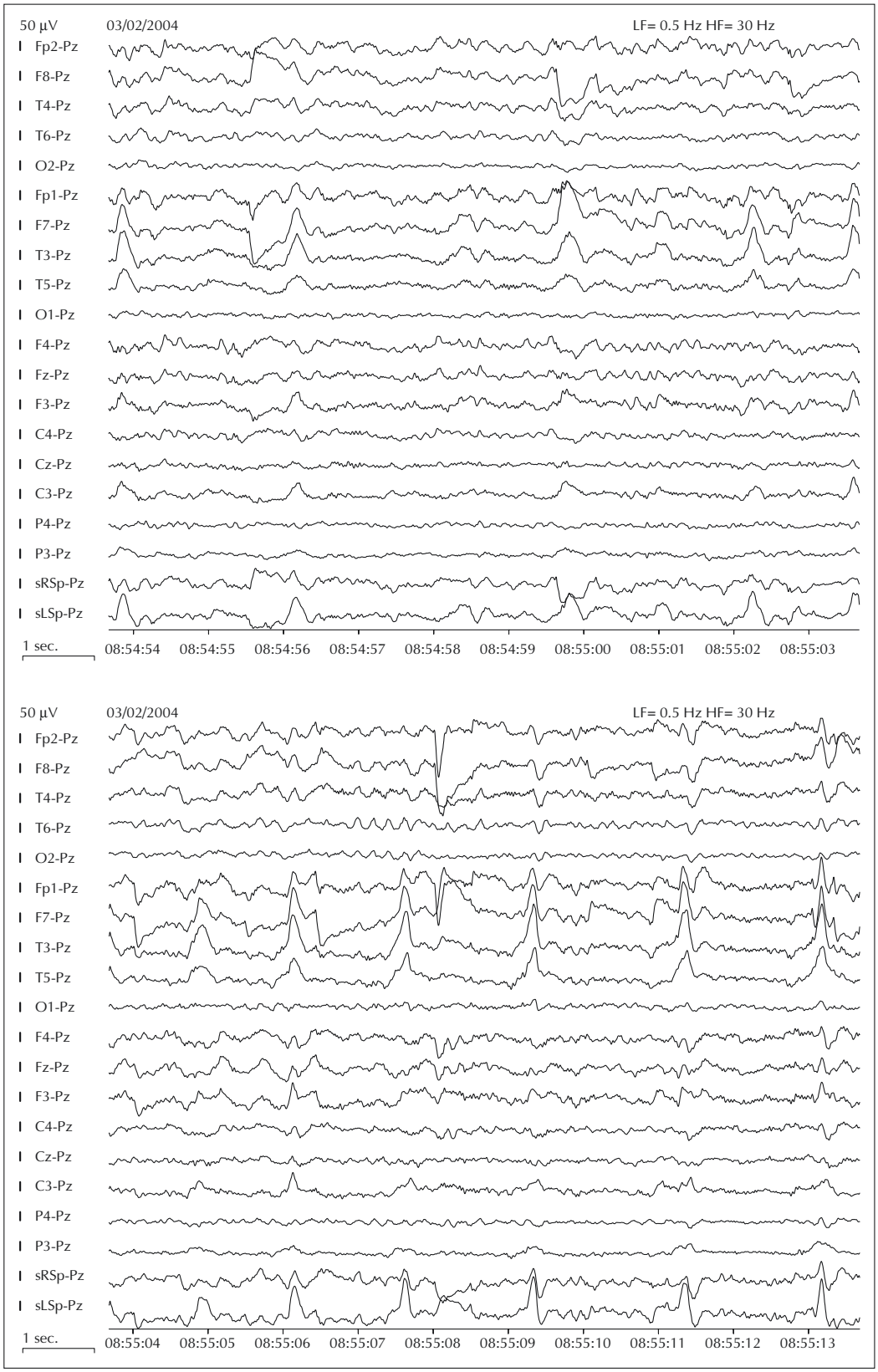

Figure 2. First definite EEG change noted 50 seconds after clinical seizure onset.

finding is exceedingly rare. Nevertheless, PLEDs may be added to the list of ictal patterns known to accompany seizures. Meanwhile, the nosological debate on PLEDs – ictal or interictal – continues. □

References

Assal F, Papazyan JP, Slosman DO, *et al*. SPECT in periodic lateralized epileptiform discharges (PLEDs): a form of partial status epilepticus. *Seizure* 2001; 10: 260-4.

Bozkurt MF, Saygi S, Erbas B. SPECT in a patient with postictal PLEDs: is hyperperfusion evidence of electrical seizure? *Clin Electroencephalogr* 2002; 33(4): 171-3.

Brenner RP. Is it status? *Epilepsia* 2002; 43(suppl.3): 103-13.

Chatrian GE, Show CM, Leffman H. The significance of periodic lateralized epileptiform discharges in EEG: an electrographic, clinical and pathological study. *Electroencephalogr Clin Neurophysiol* 1964; 17: 177-93.

Garcia-Morales I, Garcia MT, Galan-Davila L, *et al*. Periodic lateralized epileptiform discharges: etiology, clinical aspects, seizures and evolution in 130 patients. *J Clin Neurophysiol* 2002; 19(2): 172-7.

Lee K, Haight E, Olejniczak P. Epilepsia partialis continua in Creutzfeldt-Jacob disease. *Acta Neurol Scand* 2000; 102: 398-402.

Primavera A, Gianelli MV, Bandini F. Aphasic status epilepticus in multiple sclerosis. *Eur Neurol* 1996; 36: 374-7.

Terzano MG, Parrino L, Mazzucchi A, *et al*. Confusional states with periodic lateralized epileptiform discharges: a peculiar epileptic syndrome in the elderly. *Epilepsia* 1986; 27: 446-57.

Westmoreland BF, Klass DW, Scarbrough FW. Chronic periodic lateralized epileptiform discharges. *Arch Neurol* 1986; 43: 494-6.

Young GB, Brown JD, Bolton CF, *et al*. Periodic lateralized epileptiform discharges and nystagmus retractorius. *Ann Neurol* 1977; 2: 61-2.