

Gaze Deviation from Contralateral Pseudoperiodic Lateralized Epileptiform Discharges (PLEDs)

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Summary: Pseudoperiodic lateralized epileptiform discharges (PLEDs) usually produce “negative” neurologic findings. This contrasts with seizures which typically induce cortical activation with “positive” clinical manifestations. Gaze preference may arise from ipsilateral frontal eye fields (FEFs) damage because of the unopposed action of an intact contralateral FEF. Epileptic nystagmus (EN) and gaze deviation (GD) can also occur with focal temporo-parieto-occipital or hemispheric seizures in awake or obtunded patients.

A patient with old right frontal and parieto-temporal cerebral infarctions manifested leftward gaze preference and devia-

tion (without nystagmus) while alert and talking. Digitized EEG demonstrated PLEDs at ~1 Hz over the right fronto-central region, without electrographic seizures.

This report illustrates that PLEDs without seizures may excite frontal regions proximate to the FEFs to produce contraversive gaze preference in an awake patient, and discusses putative mechanisms. Gaze deviation, in this case, was the principal clinical feature of PLEDs. **Key Words:** Periodic lateralized epileptiform discharges—PLEDs—Gaze deviation—Epileptic nystagmus—Awake.

Epileptic gaze deviation and nystagmus occur with ictal excitation at the temporoparietooccipital junction in awake patients (1,2), whereas gaze deviation with seizure foci in the frontal eye fields (FEFs) usually impair consciousness. Conversely, gaze preference and deviation (GD) are seen with destruction of the ipsilateral FEFs, arising from the unopposed physiologic action of the intact contralateral FEF (3). Pseudoperiodic lateralized epileptiform discharges (PLEDs) are regarded as excitatory epileptiform discharges but not seizures, although they may have positive clinical features such as contralateral limb jerking or myoclonus or, conversely, negative features such as paresis, aphasia, or diminished level of consciousness (4). This report describes an awake patient with PLEDs but without seizures who had gaze preference contralateral to the PLEDs focus. The case illustrates that a sole clinical manifestation of PLEDs without seizures may represent an excitatory effect of PLEDs on the FEFs, inducing gaze deviation.

A 69-year-old man with hypertension, intracranial hematoma and schizophrenia was admitted with progressive confusion and confirmed, new-onset conjugate leftward eye deviation. He was able to answer questions.

Head computed tomography (CT) scan showed old right frontal and parietotemporal cerebral infarctions with encephalomalacia. An EEG was requested for investigation of new-onset leftward GD. EEG was recorded by using a digitized, Biologic CEEG-Graph machine with electrodes placed at the standard international 10–20 system sites; low-frequency filter, 1 Hz, and a high-frequency filter, 70 Hz. Eye movements were examined by a bedside observer and recorded on video-EEG recording. The direction of the patient’s gaze, eye deviation, and correlation to EEG, as well as responsiveness to verbal commands, were ascertained. While alert and talking, the patient was noted to have a conjugate GD to an extreme, leftward position. When asked to move his eyes to the examiner or in any direction, he answered, “There’s nothing to look at.” Because of schizophrenia, the patient was reluctant to follow commands, and cooperation was intermittent. He would not enter into other meaningful conversation but would withdraw appropriately. If requested, the patient could volitionally move his eyes conjugately to the right during the PLEDs. No nystagmus was noted. Intact oculocephalic reflexes were noted to the right. Leftward gaze was often noted shortly after passive opening of his eyelids. No changes in level of consciousness or speech fluency were noted at different times during video-EEG.

GD and PLEDs resolved over the next 3-week period, and at follow-up, the patient had a full range of eye

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FIG. 1. The EEG shows pseudoperiodic lateralized epileptiform discharges every 1.0 to 1.2 s over the parasagittal derivations (phase reversing at C-4), overlying the right frontal eye field. Concurrent leftward gaze deviation and preference were present.

movements, clear sensorium, followed commands, and had an EEG that showed resolution of PLEDs.

The EEG during hospital admission, day 2, showed polymorphic multiphasic sharp slow-wave discharges every 1 to 1.2 s, with phase-reversals on the right at C4 (Fig. 1). A lower-voltage “volume” conduction of the slow-wave component of the epileptiform complex activity to the temporal derivations was seen. Eye-movement artifact at FP1-F3 was noted in some instances, beginning within 100 ms of discharge, bilaterally, but on others was not synchronized with the PLEDs. Throughout a 20-min recording, no electrographic seizures or significant change in PLED discharge frequency were noted. The eyes were conjugately deviated to the left.

GD with or without epileptic nystagmus (EN) is an infrequent manifestation of ictal activity. EN and GD have been described with seizures (1,2,5), but GD has not been reported with PLEDs alone. The usual clinical correlates of PLEDs include diminished levels of consciousness and cognition, aphasia, lateralized limb weakness, and amaurosis (6,7). “Positive” phenomena thought to reflect the

irritative nature of PLEDs include time-locked contralateral limb jerks that may involve any focal or regional contralateral body parts (4). Gaze preference and deviation will occur ipsilateral to a cortical infarction, representing the effect of unopposed intact contralateral FEFs which drive the eyes conjugately and unopposed toward the inactivated FEF (3). Conversely, epileptic excitation from seizures may drive the eyes contraversively from the ictal focus by activation of the FEFs, which are inadequately opposed by normal contralateral FEF activity (2).

GD can be engendered by EN with contraversive quick phases due to epileptic activation of cortical saccade regions, resulting in contraversive quick phases driving the eyes to the contralateral eccentric position (3). In the absence of a deficit in gaze-holding abilities such as with antiepileptic drugs, the eyes will tend to maintain this position (1,2). The pathway involved in activation of the cortical saccade region, at least in monkeys, involves activation of Brodmann areas 6, 7, or 8 lying in the frontal or intraparietal sulcal regions. Impulses then project to

the superior colliculus and from there to the pontine gaze center (8,9).

With PLEDs, however, epileptic excitation occurred in this awake patient at ~ 1 Hz (2). Other studies have demonstrated that GD with EN in awake patients largely involves a higher discharge frequency (> 10 Hz) located at the temporoparietooccipital (TPO) junction contralateral to the direction of epileptic gaze (2,10). In obtunded patients, GD and EN are believed to arise from a gaze-holding defect permitting slow-phase movements back toward the midline (2). In such patients, an epileptic focus at the TPO junction is not necessary. With a slower discharge frequency, the FEF intraparietal saccade pathways or pursuit areas may be stimulated intermittently so as not to impair consciousness and to allow volitional return of the eyes to the midline or even contralaterally, producing a gaze preference. In effect, with a low-frequency discharge, the eyes also are under some volitional control, a point demonstrated when the patient was asked to look about. Finally, an alternate mechanism is that PLEDs induce inhibition of the right hemisphere pursuit areas, enabling the left pursuit areas to prevail, resulting in contralateral GD.

This case demonstrates that epileptic excitation of frontal regions by PLEDs may produce contraversive gaze preference without significant impairment of consciousness. This finding may be the principal clinically apparent feature of PLEDs.

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