

Correct Diagnosis of Psychogenic Nonepileptic Seizures

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We were most surprised to read the recent article in the electronic version of your journal, “A Proposed Etiology of Psychogenic Nonepileptic Seizures” by Dr. CA Carlson. This piece appears to call into question a large body of research into the etiology of psychogenic nonepileptic seizures (PNES),¹ and proposes that PNES may in fact be undiagnosed epileptic seizures. However, there is a clear pathophysiological difference between these entities: epileptic seizures are caused by abnormal cortical electrical discharges, and PNES are not.² The fact that diagnostic difficulties undoubtedly exist cannot be used to support a claim for a proposed epileptic etiology of PNES. The practical difficulties with the diagnosis of PNES have been addressed by a multi-disciplinary Task Force formed under the auspices of the International League Against Epilepsy in 2011, comprising epileptologists, neuropsychiatrists, and neuropsychologists. This working party has produced a report representing international expert opinion that can be considered as an evidence-based guideline on minimum diagnostic requirements for PNES,² which Dr. Carlson cites.

The video-electroencephalographic (EEG) recording of habitual events, interpreted by practitioners with sufficient expertise, is indeed the gold standard for diagnosis of PNES. However, the author of the current article appears to have misinterpreted the levels of diagnostic certainty that are detailed in the ILAE report by LaFrance et al²: these are hierarchically organized into four categories (“possible,” “probable,” “clinically established,” and “documented”), based on the quantity and quality of evidence available, and, crucially,

the level of experience of the diagnostician. These different levels of certainty do not call into question the validity of the diagnosis itself. The highest level (“documented”) requires video-EEG recording of a habitual episode, viewed by a clinician experienced in diagnosing seizure disorders. Even when epileptic seizures occur without clearly visible scalp EEG discharge, as may occur when deep or buried cortex is involved or when EEG systems with few surface electrodes are used, and even when these manifest in the form of complex behavioral changes, the clinical pattern provides definite clues for the experienced diagnostician, because of anatomical and functional correlations in the brain.³ Such interpretations in the absence of scalp EEG changes are not simply a matter of opinion. They are based on several decades of experience with invasive EEG recordings, which will invariably show an association of epileptic seizures and epileptic discharges if EEG is sampled from relevant areas in the brain. Spread of epileptic activity will occur following anatomically and physiologically determined patterns. This is completely different in PNES—although PNES also tend to occur in recognizable patterns with specific semiological clusters,⁴ facilitating diagnoses on clinical grounds. In PNES, visible or subjective seizure manifestations do not follow the same anatomical or physiological rules of spread of epileptic discharges observed in epileptic seizures. Thus, PNES is not a diagnosis of “exclusion” but rather a positive one, and negative EEG alone is never sufficient to eliminate an epileptic cause for seizures. Therefore, the majority of misdiagnoses (seizures labeled as PNES that subsequently prove to have an

epileptic cause, or the reverse) occur because the proposed diagnostic framework has not been respected, rather because of any conceptual etiological error.

The incidences of “misdiagnosis” cited by Dr. Carlson in patients undergoing depth electrode intracerebral EEG exploration⁵ were in fact patients with both epilepsy and PNES.² With the exception of rare diagnostic errors based on a serious misinterpretation of noninvasive diagnostic data, intracerebral EEG exploration is only ever performed in definite cases of epilepsy in the context of presurgical evaluation, not for diagnostic differentiation between epileptic seizures and PNES. The fact that approximately 10% of patients with PNES have comorbid epilepsy is well recognized and readily accommodated by a biopsychosocial understanding of PNES.⁶

Unfortunately, diagnostic delay in correctly establishing a diagnosis of PNES is all too common⁷ and outcomes may indeed be poor,⁸ as Dr. Carlson states. Patient management is often very complex, and clear information with straightforward communication is an essential aspect for the patient, their family, and for other clinicians. Indeed, the effects of misinformation about the nature of seizures, often sustained over many years and associated with mismanagement (e.g., unnecessary use of antiepileptic drugs), contribute both to the psychological difficulties for an individual patient with PNES and the physical risks of inappropriate management, which may be serious and even life-threatening.⁹ Withdrawal of inappropriately prescribed antiepileptic drug treatment does not lead to an increase in PNES and may improve outcome if done at the time the diagnosis is explained to patients.¹⁰ It therefore seems most irresponsible to suggest that many cases of PNES may in fact be epileptic in nature without more robust evidence to support this statement. We do not know Dr. Carlson’s level of experience in diagnosis of seizure disorders but are very concerned about her misunderstanding or misrepresentation of the evidence about PNES.

The author states in the last paragraph “Clinicians and PNES researchers need to revisit diagnostic practice.” This goal was precisely why the ILAE Task Force on PNES was formed, and much progress has already been made; future

improvements require increasing numbers of specialist personnel trained in diagnosis and management of PNES, to reduce the confusion produced by non-specialist opinion.

Conflict of Interest

None declared.

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