

Disclosures.

The author has no conflict of interest to disclose.

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Author response**To the editor,**

We read with great interest the comments of Onder regarding our recent case report (Guldiken *et al.*, 2015). He addressed that the cause-effect relationship of seizures and asystole has remained unclear in some previously published case reports where asystole preceded the onset of ictal activity on scalp EEG (Irsel Tezer and Saygi, 2011; Howell and Blumhardt, 1989). A primary cardiac pathology has been discussed as a probable cause of asystole in these patients. Ictal EEG seizure patterns on scalp EEG may frequently occur after clinical seizure onset. In some of these patients, invasive EEG evaluation may demonstrate earlier seizure onset relative to surface evaluations if the invasive electrodes are placed in the seizure onset zone.

In our patient, however, the asystole was beyond any doubt secondary to the epileptic seizure activity. She did not have any cardiac disorder. She had had a left temporal lobe epilepsy due to cavernoma and we could demonstrate that the ictal EEG seizure pattern in the left temporal lobe preceded the asystole by 30 seconds. Thus, the asystole in our patient resulted from the temporal lobe seizure activity and her recurrent falls were due to syncope-associated atonia, rather than an expression of secondary seizure generalization. □

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