

Repetitive focal seizures evolving into ictal asystole: a case report with 18 seizures during video-EEG monitoring

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Case description

- 63-year-old otherwise healthy male
- Since 1980, episodes started with a metallic taste and epigastric aura, sometimes progressing to déja-vu and subsequent loss of consciousness with oromandibular automatisms
- Diagnosed with epilepsy in 2008 after a first bilateral tonic-clonic seizure.
- EEG showed spikes in the right mid-temporal region and MRI showed mesial temporal sclerosis and parietal cortical dysplasia, both on the right side.
- In 2019, admitted for video-EEG monitoring to determine epilepsy surgery eligibility due to five to six monthly focal seizures despite polytherapy.
- Upon admittance, he was treated with lacosamide 450 mg, eslicarbazepine acetate
 1600 mg and topiramate 200 mg per day



EMU

- Admitted for four days under video-EEG monitoring
- Gradual tapering of ASM
- 18 focal seizures, of which the 17th and 18th seizures were longest and lasted 97 and 113 seconds

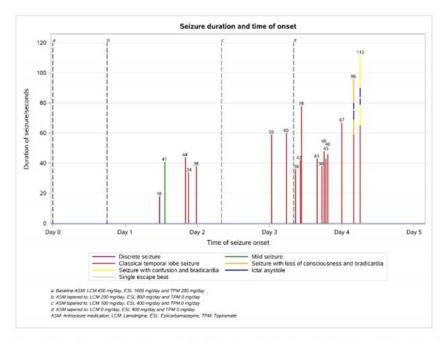


Figure 2 Time of onset and duration in seconds for all 18 seizures. The baseline antiepileptic drugs (AED) and time for every tapering are marked with dotted lines. The frequency and duration of the seizures increased as AED's were tapered During the last two seizures episodes of ictal asystole were observed.



- Towards the end of the 17th and 18th seizures the ECG showed bradycardia progressing to asystolic periods lasting 10.3 and 10.0 seconds.
- Epileptiform activity terminated during asystole in both seizures.

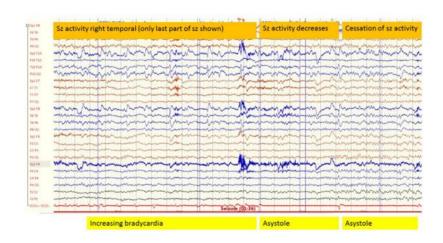


Figure 1 EEG and ECG by the end of seizure (sz) no. 17. Ictal activity over the right temporal region is seen. ECG shows bradycardia followed by asystole lasting 10.3 (5.2 + 5.1) seconds. When asystole begins, ictal activity decreases and ceases shortly thereafter.



Discussion

- The increased seizure frequency, and indeed the increased duration of the two last seizures, possibly allowing for propagation to areas critical to autonomic control, might explain why this patient without a history of cardiac disease suddenly had seizures with IA.
- Some authors have therefore speculated that IA serves as a mechanaism to ensure seizure termination. This requires further investigation but may be impossible to answer conclusively given how rarely SUDEP occurs in monitored patients.
- Our observation of asystole co-occurring with the end of electrographical seizure activity might raise the question of whether pacemaker insertion could prolong seizure activity

