



## Clinical letter

## Ictal vomiting; A dominant hemisphere phenomenon as demonstrated by intracranial depth electrode seizure mapping

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## 1. Introduction

Ictal vomiting can be a rare clinical manifestation of seizures. It is thought to originate in the anterior part of the temporal or insula lobes. It has been associated with the non-dominant hemisphere and has been considered a lateralising sign [1]. A few cases have lateralised seizure onset to the dominant hemisphere [2]. We describe a case of ictal vomiting where intracranial electroencephalography (EEG) has demonstrated seizure lateralisation to the dominant hemisphere and localisation of ictal vomiting to the dominant insula lobe.

We discuss one patient's clinical course and intracranial depth electrode conclusions. The epileptogenic zone was substantiated by extensive investigation with neuroimaging, scalp EEG and intracranial EEG and neuropsychometry.

We report the history for this thirty three year old right handed man whose seizures onset at nineteen years. Initially he describes having two generalised convulsions. He has no memory of events. It was reported that he had convulsions of all four limbs lasting up to two minutes each. He has no recollection of a preceding warning. He was treated with anti-epileptic medications and now only has generalised seizures if he omits these.

Currently his semiology consists of a warning of a 'strange' feeling in his abdomen. This lasts for three seconds and then he loses awareness. His wife reports that he will retch, often repeatedly. He may repeat words, especially profanities, after which he may fiddle, often with his right hand. He may then laugh or hum. The event typically lasts three minutes after which he will regain awareness but has post-ictal confusion. He has this seizure weekly despite taking Eslicarbazepine, Lacosamide and Sodium valproate.

Historically he had several minor head injuries at sixteen years without loss of consciousness and not requiring hospitalisation. There were no reported complications at birth. He had meningitis at eighteen months with possible seizures and probable regression in developmental thereafter. The exact details are unclear. He rarely drinks alcohol however he took illicit drugs including cocaine and cannabis as a teenager. He finished mainstream school at sixteen years and then undertook general maintenance work. He lives with his wife and two children.

Investigations were carried out under the Epilepsy team at Kings College Hospital, London. An epilepsy series 1.5 T MRI brain scan showed left hippocampal sclerosis, but also extensive encephalomalacic changes on the left. Those anteriorly were thought to be post-traumatic, however there were also abnormalities posteriorly possibly related to the history of meningitis.

Scalp EEG showed left temporal sharp waves inter-ictally. Runs of slow waves were seen over the fronto-temporal regions bilaterally.

Scalp video EEG telemetry captured five of the patient's habitual seizures which were characterised by clinical onset with vomiting, impaired awareness and motor automatisms. The EEG changes were thought to be more consistent with a right anterior temporal seizure onset.

Neuropsychometry was consistent with left hemispheric involvement in someone who is left hemisphere dominant, likely to be the case given his right handedness.

Considering his scalp telemetry suggested a right hemispheric seizure onset and his brain MRI suggested left hippocampal sclerosis and he had bilateral inter-ictal temporal abnormalities, it was decided he should undergo intracranial depth electrode mapping of both

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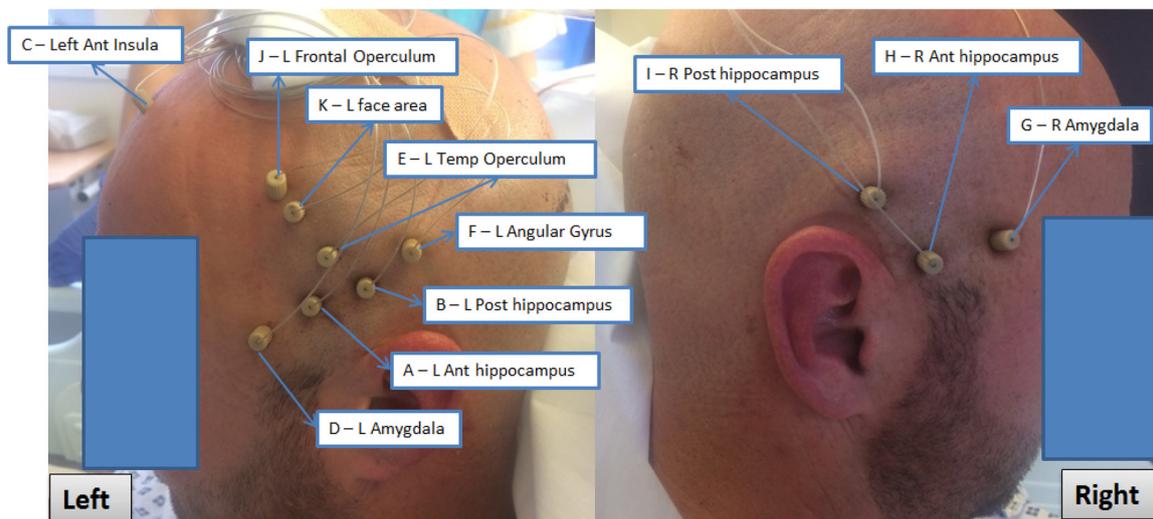


Fig. 1. Implantation of 10 Dixi® depth electrodes with a ‘temporal plus’ implantation covering the left amygdala and hippocampus, lateral temporal lobe, insula, face motor area and right amygdala and hippocampus.

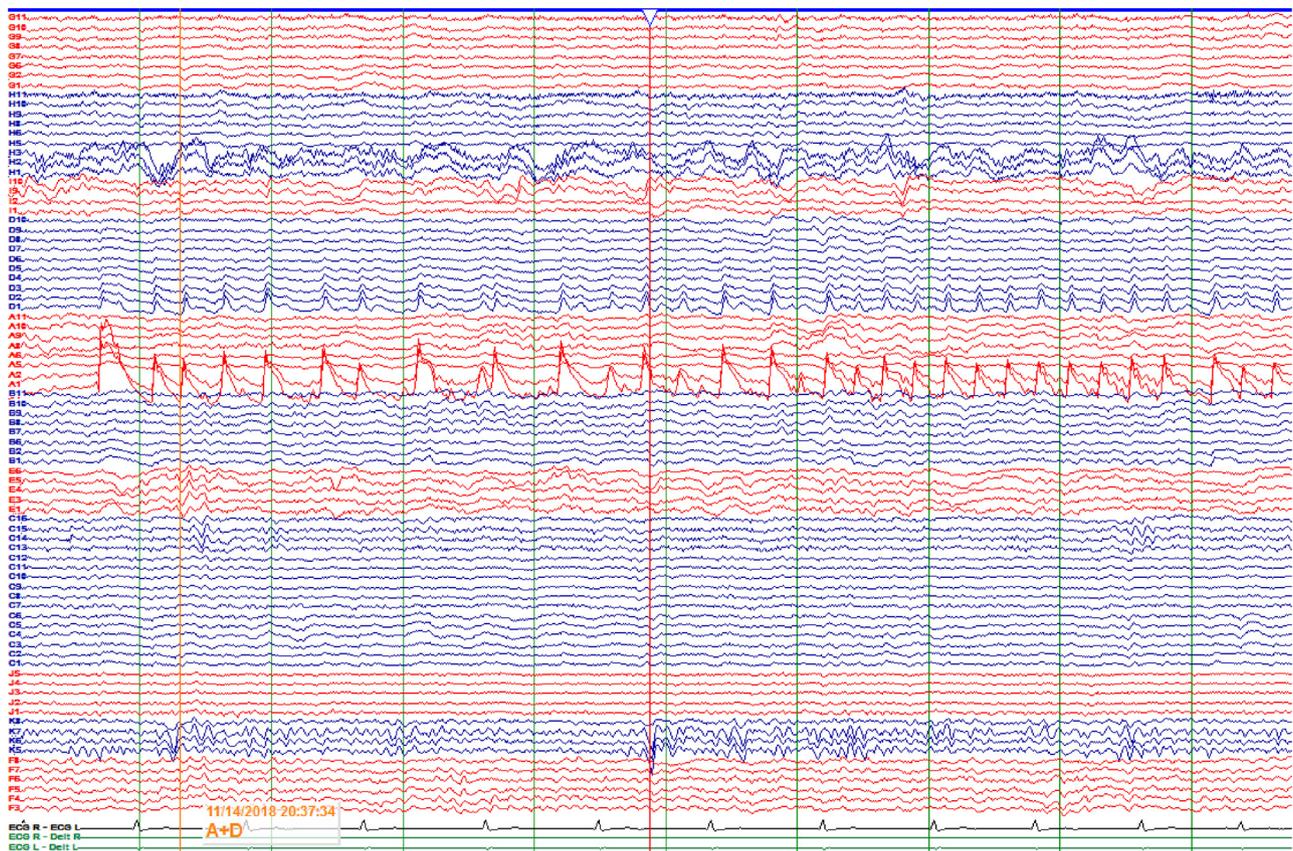


Fig. 2. Evolving rhythmic activity seen at electrode A (deepest contacts A1/2) left anterior hippocampus, with spread seen to involve electrode D (contacts D1/2/3) left amygdala.

hemispheres. Implantation of 10 Dixi® depth electrodes was undertaken with the aim of a ‘temporal plus’ implantation covering the left amygdala and hippocampus, lateral temporal lobe, insula, face motor area and right amygdala and hippocampus.

The inter-ictal EEG demonstrated continuous slow wave activity (2 Hz) with superimposed fast rhythms seen at electrode H1,2 & 3 (right anterior hippocampus). High voltage spike and sharp waves plus slow waves are seen at the same 3 contacts. These high amplitude slow waves can occasionally spread to involve electrode G (right amygdala).

Almost continuous discharges are seen at A1 and A2 (left anterior hippocampus) (Fig. 1).

Multiple subclinical seizures were captured arising from A1 and A2. In the two clinical seizures the first ictal EEG changes were seen at A1 and A2 with early spread to the B1 (left posterior hippocampus). It was upon involvement of the left insula (electrode C) that the clinical semiology (retching) presented. The seizures originated in the left hippocampus (A1/A2). Fig. 2. A limitation is that the right insula was not implanted due to increased vascular risk with bilateral insula

implantation. The results demonstrate spread to the right hemisphere ten seconds after left hemispheric involvement and semiological onset, thus the likelihood of right insula early involvement is low. This patient went on to have a left temporal lobectomy. Six months post-operatively he has not had any day-time seizures only a few night-time seizures without vomiting.

## 2. Conclusion

This study has eloquently demonstrated that the seizure semiology of retching was a dominant hemisphere phenomenon and arose upon spread to the dominant insula. Ictal vomiting is typically considered a localizing sign with non-dominant lateralization in patients with partial seizures of temporal lobe origin. Two cases using intracranial electrode mapping indicated that ictal vomiting can occur as a manifestation of left temporal onset seizures in left hemisphere-dominant patients and one case demonstrated ictal vomiting upon activation of the dominant hemisphere anterior hippocampus [3,4].

Scalp EEG alone cannot accurately lateralize seizure onset in cases

where ictal vomiting is the first semiological sign. Intracranial depth electrode studies are beginning to illustrate that in some cases, ictal vomiting may be a dominant hemisphere phenomenon.

## Declaration of Competing Interest

None.

## References

- [1] Devinsky O, Frasca J, Pacia SV, Luciano DJ, Paraiso J, Doyle W. Ictus emeticus: further evidence of nondominant temporal involvement. *Neurology* 1995;45:1158–60.
- [2] Schindler K, Wieser HG. Ictal vomiting in a left hemisphere language-dominant patient with left-sided temporal lobe epilepsy. *Epilepsy Behav* 2006;8(February (1)):323–7.
- [3] Schauble B, et al. Ictal vomiting in association with left temporal lobe seizures in a left hemisphere language-dominant patient. *Epilepsia* 2002;43(November (11)):1432–5.
- [4] Pietrafusa, et al. Ictal vomiting as a sign of temporal lobe epilepsy confirmed by stereo-EEG and surgical outcome. *Epilepsy Behav* 2015;53(December):112–6.