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Review

# Ictal vomiting as a sign of temporal lobe epilepsy confirmed by stereo-EEG and surgical outcome☆

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ABSTRACT

Vomiting is uncommon in patients with epilepsy and has been reported in both idiopathic and symptomatic epilepsies. It is presumed to originate in the anterior part of the temporal lobe or insula. To date, 44 cases of nonidiopathic focal epilepsy and seizures associated with ictal vomiting have been reported. Of the 44 cases, eight were studied using invasive exploration (3 stereo-EEG/5 subdural grids).

Here, we report a 4-year-and-7-month-old patient with a history of febrile convulsion in the second year of life and who developed episodes of vomiting and complex partial seizures at 3 years of age. Scalp EEG showed no electrical modification during vomiting while the complex partial seizure displayed a clear right temporal origin. Brain MR showed hippocampal volume reduction with mild diffuse blurring of the temporal lobe. Stereoelectroencephalography study confirmed the mesiotemporal origin of the seizures and showed that the episodes of vomiting were strictly related to an ictal discharge originating in the mesial temporal structures without insular diffusion. The patient is now seizure-free (18 months) after removal of the right anterior and mesial temporal structures.

In all the reported patients, seizures seemed to start in mesial temporal structures. The grid subgroup is more homogeneous, and the most prominent characteristic (4/5) is the involvement of both mesial and lateral temporal structures at the time of vomiting. In the S-EEG group, there is evidence of involvement of either the anterior temporal structures alone (2/3) or both insular cortices (1/3).

Our case confirms that vomiting could occur when the ictal discharge is limited to the anterior temporal structure without insular involvement. Regarding the pathophysiology of vomiting, the role of subcortical structures such as the dorsal vagal complex and the central pattern generators (CPG) located in the reticular area is well established. Vomiting as an epileptic phenomenon seems to be related to the involvement of temporal structures, mainly mesial structures (amygdala) and with an uncertain role of the insula. An intriguing hypothesis is that the ictal discharge in mesial structures determines seizure manifestation that could be explained not only by tonic activation of the cortex, but also by 'release' (reduction of inhibition?) of the CPG responsible for involuntary motor behaviors.

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## 1. Introduction

Nausea is the unpleasant sensation of having the urge to vomit, and vomiting is the forceful expulsion of gastric contents mediated by integrated autonomic and motor function mechanisms [1,2]. While nausea is relatively frequent as a symptom of seizure, vomiting is uncommon, in both adults and children with epilepsy.

Since the first description by Panayiotopoulos et al. [3], it was clear that vomiting is a key feature of benign childhood epilepsy. Photosensitive occipital lobe epilepsy is another, less frequent, condition in which ictal vomiting may occur [4].

Ictal vomiting has also been observed in both adults and children with temporal lobe epilepsy (TLE) of presumed symptomatic origin (mainly hippocampal sclerosis) [5,6]. Frequency of ictal vomiting was approximately 2% in temporal lobe epilepsies [5].

Lateralizing value of ictal vomiting was reported in TLE without consistent results. Ictal vomiting was reported from involvement of both the nondominant [5,7–12] and dominant temporal lobes [13–16].

☆ The work has not previously been presented at any meeting.

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It was also recorded as a symptom of insular epilepsy as it was correlated with a discharge in both anterior insular cortexes during a stereoelectroencephalography (S-EEG) procedure [17].

To date, three cases of ictal vomiting investigated with intracerebral electrodes have been reported [10,14,17], as well as five cases investigated with subdural grids [5,10].

We present a patient who had right TLE with ictal vomiting documented during S-EEG study: the epileptic discharge was confined to the anterior hippocampal region and amygdala. Moreover, we critically reviewed the previously reported cases in order to better define the electroclinical and anatomical correlation of ictal vomiting. To date, 44 cases with nonidiopathic focal epilepsy and seizures associated with ictal vomiting have been reported [5,8–19]. Out of the 44 cases, eight were studied with invasive exploration (S-EEG/subdural grids) and considered in the discussion.

## 2. Case report

This is a 4-year-and-7-month-old right-handed female patient with a normal birth and psychomotor development. Neurologic examination was normal. At the age of 18 months, she had one episode of febrile status epilepticus. At the age of 3 years, she started to present brief focal seizures with staring and automatism. Seizures were resistant to carbamazepine, valproate, and clobazam, and were characterized by staring, loss of consciousness, fearful facial expression, flushing, and oral automatisms, followed by right head orientation and retching. Ictal EEG showed a rhythmic fast discharge in the right temporal lobe spreading into the suprasylvian EEG traces. A rhythmic slow activity appeared in the second half of the seizure, involving both hemispheres and terminating later on the left side (Fig. 1A). Interictal EEG showed sharp waves and spikes over the right temporal lobe with anterior suprasylvian diffusion. Several episodes of vomiting without impairment of consciousness and no EEG modifications were recorded. These episodes were described in the patient's clinical history; gastrointestinal evaluation performed at the time showed no pathologic results. Brain MR (3-T magnet) revealed a slight decrease in the size of the right hippocampus, with a more widespread posterior blurring. Griffiths Mental Development Scales (GMDS) revealed a general quotient of 102.

Stereoelectroencephalography was performed with seven intracerebral electrodes implanted in the right temporal and insulo-opercular cortices (Fig. 1B–C). Repetitive interictal spikes were recorded over the contacts between the head of the hippocampus and the amygdala, and less frequently over the contacts close to the body of the hippocampus.

We recorded episodes of vomiting without impairment of consciousness with an EEG counterpart characterized by a focal discharge beginning in the amygdala/head of the hippocampus region (electrode HA, first row Fig. 1D) and subsequently involving the body of the hippocampus but sparing the insula and the neocortex. Stimulation (50 Hz, 1 mA) of the mesial contacts of electrode HA (between the head of the hippocampus and the amygdala, Fig. 1B) elicited vomiting (Fig. 1D). The previously reported complex partial seizure showed a focal discharge beginning in the same contacts and propagating secondarily to the right temporal neocortex (Fig. 1E).

The patient underwent a right temporal lobectomy with anterior hippocampectomy. She has been seizure-free for 18 months. Histopathological examination revealed a hippocampal sclerosis associated with Type I cortical dysplasia (FCD IIIa). Her GMDS was stable.

## 3. Discussion

In order to better understand the pathophysiologic mechanisms and localization of ictal vomiting, we reviewed all articles in which ictal vomiting was documented and selected those with invasive recording. We excluded all cases in which ictal vomiting was associated with idiopathic focal epilepsies because the genesis is probably supported by a different, likely more widespread, epileptogenic zone [20].

To date, 44 cases of nonidiopathic focal epilepsy and seizures associated with ictal vomiting have been reported [5,8–19]. Out of the 44 cases, eight were studied with invasive exploration and were considered for the review (S-EEG/subdural grids) (Table 1).

In all, eight cases have been reported (for references, see Table 1). All the patients presented with TLE; five had right TLE, and three had left TLE.

Six cases were affected by symptomatic epilepsy: in four of them, epilepsy was related to gliosis and neuronal heterotopia, in one to focal cortical dysplasia, in another to tumor. In 2 cases, etiology was not reported. All patients but one had brain MRI.

Seven cases had interictal temporal abnormalities, and seven had seizures recorded during a scalp video-EEG with a clear temporal onset.

Five patients underwent presurgical evaluation with subdural grid; in four of them, ictal vomiting was seen during seizures with mesial temporal onset followed by involvement of lateral temporal neocortex [5]; in one patient, EEG changes were initially seen over the mesial contacts of the left anterior temporal strip [10].

Three patients were evaluated with S-EEG. The first two had psychomotor seizures in which vomiting occurred as soon as the epileptiform discharge involved the anterior mesial temporal structures, both bilaterally [10] and unilaterally [14]. In these two cases, the insula was not explored. Therefore, following publication of the paper by Catenoix et al. in 2008 [17] reporting a case of ictal vomiting associated with a bilateral insular discharge, the main working hypothesis was that vomiting, as an ictal phenomenon, depends mainly on the involvement of the insular region and not only the anterior temporal ones.

All patients, including the present case, underwent temporal lobectomy including the mesial structures with an excellent outcome reported in 7 (Engel Ia) of them; only one patient continued to have seizures occasionally accompanied by vomiting [5]. No lateralizing value could be extrapolated from all the reported cases.

In our case, both the mesiotemporal structures and the insular cortex were explored. Seizures presented vomiting and intense nausea as a unique feature, while the discharge was strictly limited to the anterior mesiotemporal region (amygdala/head of the hippocampus). The contacts exploring the insula showed no abnormalities.

As regards the pathophysiology of vomiting, the role of subcortical structures, such as the dorsal vagal complex and the central pattern generators (CPG) located in the reticular area, is well established [21].

In the literature, the precise cortical area responsible for vomiting is uncertain. Gastrointestinal tract motility has its cortical representation in the insula and limbic circuits, and this is also the cortical locus of visceral function [5].

In addition to temporal and insular structures, the mesial frontal region and parts of Papez's limbic circuit have been associated with the generation of nausea and vomiting [7,22].

Intracranial electrical stimulation of the insular cortex in patients with epilepsy with S-EEG never elicited vomiting [23–25].

Recently, it was proposed that seizure manifestation could be explained not only by tonic activation of the cortex, but also by 'releasing' (reduction of inhibition?) the CPG responsible for involuntary motor behaviors [26].

In conclusion, vomiting in focal nonidiopathic epilepsies seems to be related to the involvement of mainly mesial temporal structures, with an uncertain role of the insula. In our case, it was difficult to assess whether the hippocampus or the amygdala led the emesis. There have been no recent reports of emesis during intracranial stimulation, indicating that a more complex mechanism could be

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