Yawning is a common physiological event in mammals. It may increase vigilance when drowsiness occurs and is probably important for social communication. It has been reported in different pathological conditions (Daquin et al. 2001). The occurrence of yawning in relation to epileptic seizures has been rarely described (Penfield and Jasper 1954, Goldie and Green 1961, Donat and Wright 1991, Muchnik et al. 2003). We report a patient with non-dominant for speech hemisphere seizures and irresistible, repetitive and forceful post-ictal yawning.

**Case report**

A 48-year-old, left-handed woman with no neurological antecedents began having intractable epileptic attacks at age 18. She described five types of seizures:

- **Seizure type 1**, isolated myoclonic jerks of the left arm,
- **Seizure type 2**, left arm numbness,
- **Seizure type 3**, type 2 progressing to dystonic posturing of the left arm with non-forceful head deviation to the left, and brief unresponsiveness, occasionally followed by a transitory left arm weakness,
- **Seizure type 4**, sudden falls without warning,
- **Seizure type 5**, rare secondarily generalized tonic-clonic seizures (GTCS).

Surface EEG recordings showed interictal epileptic activity over the right centro-parieto-temporal regions. Her neurological examination was normal. Medical history was remarkable for asymptomatic hepatitis C. At 30, she underwent invasive EEG studies, which disclosed epileptiform abnormalities over the right parietal operculum. Brain CT and MR imaging were normal. Neuropsychological evaluation revealed right hemisphere dysfunction (sodium amobarbital test confirmed a left hemispheric dominance for speech). A right inferior parietal and posterior temporal resection did not lead to improvement. Pathological studies showed no specific
changes. At 31, she had a second resection at the temporal edge of the previous operation, again with no improvement.

In 2004, she underwent further telemetry recordings. Thirty-eight seizures were recorded: 9 were myoclonic jerks; 6, left arm sensory seizures (duration, < 10 s); 22, sensory attacks followed by left hand dystonia, head deviation and loss of awareness (mean duration, 15 s). No falls were recorded but she had one GTCS. Ictal scalp EEG showed no changes with types 1 and 2, and disclosed low voltage fast activity over the right centro-parieto-temporal areas without depression of background activity with all type 3 seizures (figure 1). Attacks occurred at random during the day when she was alert and active, and alpha activity was seen before and after each of the 37 seizures that did not generalize. Complete blood count, electrolytes, renal and thyroid function tests were normal, but hepatic enzymes were mildly elevated. Oxygen blood saturation and CO2 were not measured during yawning, but routine venous blood parameters were normal.

**Yawning.** In 19 type 3, and in two type 2 seizures, she had repetitive, irresistible and forceful yawning starting from one to 30 seconds (mean, 8.5 s) after the seizure offset. No yawning was detected with myoclonic jerks, and the single GTCS was not included in the analysis. Yawning lasted from five to 60 seconds. There was no change of cardiac rhythm. There was no obvious change in blood pressure, respiration and no skin changes occurred during these episodes. Yawning started with a deep inspiration through her wide-open mouth (figure 2). She was alert during all yawning episodes. She received oxcarbazepine 1,800 mg/d during telemetry and her valproate was gradually withdrawn. Her husband had witnessed similar parasymptomatic yawning for approximately six years occurring typically at the end or just after type 3 seizures when she received different medications singly or in combinations. Physiological yawning was observed when the patient was tired or bored, with a quality similar to her peri-ictal yawning, except for lack of clustering and irresistibility. We found no episodes of peri-ictal yawning when reviewing the earlier (1986 and 1987) prolonged surface and intracranial video-EEG studies. Her seizures remained the same over the years, including those followed by yawning except for lack of clustering and irresistibility. We received different medications singly or in combinations.

**Yawning and epilepsy.** We describe a woman with repetitive yawning as an unusual, isolated, autonomic, post-ictal phenomenon. Yawning appeared immediately or seconds after cessation of seizures, was irresistible and was observed after most (86%) of the focal sensory-motor and some (33%) sensory attacks. We cannot explain why yawning appeared late (approximately 24 years after onset of her seizures and 11 years after her second surgery) during the course of her disorder.

**Physiology of yawning.** Yawning is a complex spatio-temporal reflex, and neural structures involved are presumably located in the brainstem near respiratory and vasomotor centers, and those that control facial mimicry, mastication, throat movements and stretching. The purpose and mechanism of yawning are not well understood. Three theories have been proposed: a relation to respiration, alertness or communication (Daquin et al. 2001). The hypothalamic paraventricular nucleus contains nerve endings that belong to the incerto-hypothalamic dopaminergic system and project to oxytocinergic neurons located in this nucleus. These in turn project to the hippocampus, pons and medulla oblongata and mediate the expression of yawning. Oxytocinergic neurons can be modulated by several neurotransmitters and neuropeptides, such as dopamine, oxytocin, prolactin, excitatory amino acids, acetylcholine and nitric oxide to name the most important ones (Argiolas and Melis 1998). Oxytocin injected in the CA1 field of the hippocampus induced yawning (Melis et al. 1986). Acetylcholine and cholinomimetic drugs can induce yawning in rats and muscarinic antagonists can prevent it. Finally, prolactin facilitates and opioids inhibit yawning.

**Discussion**

We describe a woman with repetitive yawning as an unusual, isolated, autonomic, post-ictal phenomenon. Yawning appeared immediately or seconds after cessation of seizures, was irresistible and was observed after most (86%) of the focal sensory-motor and some (33%) sensory attacks. We cannot explain why yawning appeared late (approximately 24 years after onset of her seizures and 11 years after her second surgery) during the course of her disorder.
Figure 1. A) Ictal and post-ictal EEG. In this seizure (type 3), the patient demonstrated yawning 6 sec after seizure-offset (first arrow). Ictal discharge started approximately 8 to 10 sec before seizure offset, and was seen in the right centro-parieto-temporal region. There was no post-ictal depression of EEG activity. B) Post-ictal yawning.
Three mechanisms might explain peri-ictal yawning. First, activation of the brainstem structures related to a change in the level of alertness. In our patient, yawning was never caused by a change in her level of alertness. This mechanism however, could be a factor in the two patients of Muchnik and colleagues (2003). Second, a direct activation of brainstem structures by the epileptic discharge. The fact that yawning appeared immediately or just a few seconds after a right centro-parieto-temporal epileptic discharge and seizure offset, suggests a fast operating process that activated the brainstem structures. We cannot explain why yawning did not occur during the attack. Third, a seizure-mediated secretion of endogenous neurohumoral substances such as prolactin or oxytocin may cause yawning. The latter is less likely since a prolactin surge should only occur after a few minutes (Pritchard et al. 1983), and activation of oxytocin in the hypothalamic paraventricular nucleus was found to occur only 1.5 hours after kainic-induced seizures in rats (Sun et al. 1996). We did not measure plasma concentration of prolactin or oxytocin in our patient. There is no evidence that her medications were responsible for yawning since the patient’s husband and she herself did not notice any change in the yawning pattern over the previous six years when taking various drugs.

In keeping with previous reports implying preferential non-dominant hemisphere for the induction of autonomic peri-ictal symptoms such as spitting, water drinking, vomiting, urinary urge or coughing (Baumgartner et al. 2001), yawning in our patient occurred after seizures arising in her non-dominant hemisphere. However, such non-dominant lateralization may not be absolute, since a patient with yawning and seizures related to a left temporal tumor was reported (Penfield and Jasper 1954). It is remarkable that forceful yawning has been so rarely described in association with seizures.

### Legends for video sequences

#### Video 1
Type 3 seizure followed by a single brief episode of yawning (EEG, figure 1A). Before seizure onset patient is alert and watches TV. EEG onset is at 21:17:34 and offset at 21:17:43. Yawning starts 6 sec after the end of the attack, and patient remains alert throughout the event.

#### Video 2
Type 3 seizure followed by repetitive yawns and a transitory left arm weakness. EEG seizure starts at 12:22:54 and ends at 12:23:05. Ictal discharge is followed by a continuous polymorphic slow wave activity seen in the right centro-parieto-temporal area for approximately 60 sec. Forceful, repetitive yawns (and few coughs) start at 12:23:22 and persist until 12:24:30 when patient declares that she just had a seizure.

### Table 1. Patients with peri-ictal yawning.

<table>
<thead>
<tr>
<th>Ref./Sex/Age at sz onset</th>
<th>Age at yawning onset (y)</th>
<th>Yawning duration</th>
<th>Relation to seizure</th>
<th>Associated clinical manifestations</th>
<th>EEG Risk factors, imaging or pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td>2/F/23</td>
<td>23</td>
<td>Continuous (during ictal phase, time not specified)</td>
<td>Ictal</td>
<td>Headache, hiccups, urge to void, numbness L arm and mouth</td>
<td>L T astrocytoma (pathology)</td>
</tr>
<tr>
<td>2/M/?</td>
<td>-</td>
<td>3 min to 3h</td>
<td>Ictal and post-ictal</td>
<td>Sweating hands and scalp, paresthesias scalp and back, palpitations</td>
<td>Head trauma</td>
</tr>
<tr>
<td>3/F/7</td>
<td>7</td>
<td>Seconds</td>
<td>“Interictal”</td>
<td>None</td>
<td>Preceding or during bursts of 3Hz SW</td>
</tr>
<tr>
<td>4/?/infancy</td>
<td>Infancy</td>
<td>Seconds</td>
<td>Ictal</td>
<td>Infantile spasms variant (with yawning), typical spasms (without yawning)</td>
<td>During decremental activity</td>
</tr>
<tr>
<td>5/M/95</td>
<td>95</td>
<td>30 s</td>
<td>Pre-ictal</td>
<td>During drowsiness, before CPS</td>
<td>Bil T</td>
</tr>
<tr>
<td>5/F/15</td>
<td>15</td>
<td>-</td>
<td>Post-ictal</td>
<td>After CPS and generalization</td>
<td>Bil T</td>
</tr>
<tr>
<td>Our patient/F/18</td>
<td>42</td>
<td>5-60s</td>
<td>Post-ictal</td>
<td>Focal sensory-motor attacks (types 2 and 3)</td>
<td>R CTP</td>
</tr>
</tbody>
</table>

y = years, sz = seizure, min = minutes, L = left, R = right, h = hours, s = seconds, SW = spike and wave, bil = bilateral, C = central, T = temporal, P = parietal, CPS = complex partial seizure.
References


