Video case report

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Post-ictal forceful yawning in a patient with nondominant hemisphere epilepsy

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ABSTRACT – Yawning has been rarely described in association with seizures and not previously documented by video-EEG. We present a 48-year-old woman with a long history of non-dominant for speech hemisphere seizures and post-ictal yawning. Yawning was irresistible, forceful and often repetitive. We reviewed the few similar epileptic cases described in the literature and discussed the possible mechanisms.

[Published with video sequences]

Key words: yawning, epilepsy, autonomic seizures, post-ictal phenomenon

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 improve-
ment. 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 de-
viation 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, electro-
ytes, 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 gradu-
ally withdrawn. Her husband had witnessed similar par-
oxysmal 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 intrac-
nuclear video-EEG studies. Her seizures remained the same
over the years, including those followed by yawning.
Repeated high quality MR failed to show a structural
lesion, except for postsurgical changes.

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.

Physiology of yawning. Yawning is a complex spatio-
temporal reflex, and neural structures involved are pre-
sumably located in the brainstem near respiratory and
vasomotor centers, and those that control facial mimicry,
mastication, throat movements and stretching. The pur-
pose and mechanism of yawning are not well understood.
Three theories have been proposed: a relation to respira-
tion, alertness or communication (Daquin et al. 2001).

The hypothalamic paraventricular nucleus contains nerve
endings that belong to the incerto-hypothalamic dopam-
inergetic system and project to oxytocinergic neurons lo-
cated in this nucleus. These in turn project to the hippec-
ampus, pons and medulla oblongata and mediate the
expression of yawning. Oxytocinergic neurons can be
modulated by several neurotransmitters and neuropep-
tides, 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 cholin-
mimetic drugs can induce yawning in rats and muscarinic
antagonists can prevent it. Finally, prolactin facilitates and
opioids inhibit yawning.

Yawning and epilepsy. We found only six, well docu-
mented cases in the literature where yawning was re-
ported in association with epilepsy (table 1). Penfield and
Jasper (1954) described two adult patients with autonomic
seizures and yawning. The first had a tumor infiltrating the
left temporal lobe and ipsilateral basal ganglia. She had
seizures beginning with a headache associated with
yawning, hiccup, urge to void and focal sensory phenom-
ena. In the second patient, attacks were ushered in by
sweating of her hands and scalp and a prickling sensation
in the scalp and back, and with repetitive yawning, palpi-
tations, visual distortions and weakness. No EEG was
reported. Golgie and Green (1961) described an adoles-
cent with “petit mal” and found an association between
yawning and 3 per second spike and wave discharges.

Donat and Wright (1991) reviewed unusual variants of
infantile spasms in 11 children (13% of their patients with
infantile spasms), and described one, with an unspecified
brain malformation, who had a variant consisting of yawn-
ing with generalized decremental activity on EEG. This
yawning variant and the child’s more typical infantile
spasms responded to ACTH. Finally, Muchnik and col-
leagues (2003) described two patients who had temporal
lobe epilepsy and yawning. In the first, yawning occurred
during drowsiness and preceded focal seizures. The pa-
tient had independent, bilateral, temporal interictal epi-
leptiform discharges, but imaging was not reported. The
other, a 17-year-old woman with an insulin-dependent
diabetes, had a normal MRI, complex partial seizures and
secondary generalization with yawning during the post-
ictal period. EEG also showed bilateral temporal spikes.
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 can not 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 however, 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.

<table>
<thead>
<tr>
<th>Ref./Sex/Age at sz onset (y)</th>
<th>Age at yawning onset (y)</th>
<th>Yawning duration</th>
<th>Relation to seizure</th>
<th>Associated clinical manifestations</th>
<th>EEG</th>
<th>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, hicups, urge to void, numbness L arm and mouth</td>
<td>-</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>-</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>
<td>None</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>
<td>“Brain malformation”</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>
<td>Diffuse brain atrophy (CT)</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>
<td>Normal (MR)</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>
<td>No lesion except surgical (MR)</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.

### 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 seizure 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.
References


