Clinical commentary

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Surgical treatment of extra-hypothalamic epilepsies presenting with gelastic seizures

Holger Joswig [1], Patricia Rejas-Pinelo [1,2], Ana Suller [1], Richard S. McLachlan [1], David A. Steven [1]

[1] Epilepsy Program, Department of Clinical Neurological Sciences, London Health Sciences Centre, University Hospital, London, ON, Canada
[2] School of Medicine, Research and Innovation Centre of the Faculty of Health Sciences, Universidad Peruana de Ciencias Aplicadas, Lima, Peru

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ABSTRACT – We provide an overview of the surgical outcome of extra-hypothalamic epilepsies with gelastic seizures based on an original case report and a summary of the literature. Twenty-two articles providing information on the outcome of resective surgery in 39 patients with extra-hypothalamic gelastic seizures from the temporal (19 patients) or frontal lobe (20 patients) were selected. We add another case of temporal lobe gelastic seizures to the literature with a video demonstrating the mirthful component of this patient's laughing seizures. Drug-refractory cases of gelastic seizures from the temporal or frontal lobes are amenable to surgical treatment following thorough investigation with imaging, as well as scalp and intracranial EEG.

Key words: epilepsy surgery, extra-hypothalamic, gelastic seizures, mirthful laughter, temporal lobe epilepsy

VIDEO ONLINE

Correspondence:
David A. Steven
London Health Sciences Centre,
University Hospital, Room A10-323,
339 Windermere Road,
London, ON, Canada
<david.steven@uwo.ca>

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Gelastic seizures (GS) are characterized by uncontrolled laughter and were first described by Trousseau in 1873. Gascon and Lombroso (1971) directly linked laughter outbursts to electroencephalographic ictal activity. Although most commonly associated with a hypothalamic origin, the seizure onset zone (SOZ) for GS is rarely in the temporal lobe or in the frontal lobe (Tassinari *et al*., 1997; Cheung *et al*., 2007). Here, we describe the presurgical and subsequent surgical management of a patient with temporal-onset mirthful laughter, followed by a literature review on extra-hypothalamic epilepsies with GS and surgical outcome using the PubMed search term "gelastic". Only English language articles were considered. Additional studies were identified by reviewing the reference lists of relevant articles from the search results.

Case study

A predominantly right-handed man first presented with possible secondary generalized tonic-clonic seizures with postictal aphasia at the age of 30. These improved promptly with antiepileptic medications (AEDs) and resolved over the next six years. However, he also developed focal non-motor seizures with or without impairment of awareness at the age of 32. The seizures were characterized by a deep sigh, nonsensical speech, and oral automatisms, occasionally preceded by mirthful laughter. These episodes lasted up to one minute and were followed by postictal confusion (*video 1*). Occasionally, he experienced an aura of a sensation at the back of his tongue or throat. These seizures tended to occur predominantly while falling asleep and upon awakening once or twice a day. Fatigue and sleep deprivation, as well as an occasional AED dose omittance, were identifiable seizure triggers. A combined antiepileptic regimen of phenytoin, topiramate, and clobazam and later lacosamide and perampanel was ineffective in terms of clear seizure control. Therefore, at the age of 41, he was admitted to our epilepsy monitoring unit (EMU) for a comprehensive investigation.

Scalp video-EEG demonstrated interictal left anterior temporal spikes and polyspikes with supra-sylvian spread, as well as rare right temporal spikes. A total of five stereotypical mirthful laughter seizures were recorded with an onset characterized by polyspikes over the left anterior temporal region (*figure 1A*). While magnetic resonance imaging (MRI) showed no structural epileptogenic lesion, and single-photon emission computed tomography (SPECT) was unremarkable, ^{18}F-fludeoxyglucose positron emission tomography (^{18}F-FDG PET) revealed distinct left temporal hypometabolism (*figure 2A*). A neuropsychological assessment suggested mild to moderate left neocortical and mesial temporal dysfunction and possibly frontal and subcortical dysfunction. To precisely delineate the SOZ, the patient underwent depth electrode implantation for stereoelectroencephalography (SEEG) according to our institutional protocol (Joswig *et al.*, 2018a, 2018b), covering the left temporal and orbitofrontal lobe, the opercular region, as well as the insula (*figure 2B, C*). Interictal activity showed widely distributed spikes in the left temporal neocortex, orbitofrontal cortex, and inferior insula. Independent spikes also occurred synchronously in the hippocampus and amygdala. The SOZ was determined to be left neocortical temporal and mesiotemporal after 23 recorded stereotypical seizures (*figure 1B*). Extraoperative electrocortical stimulation via the depth electrodes (stimulation mode: bipolar; 300-μs pulse

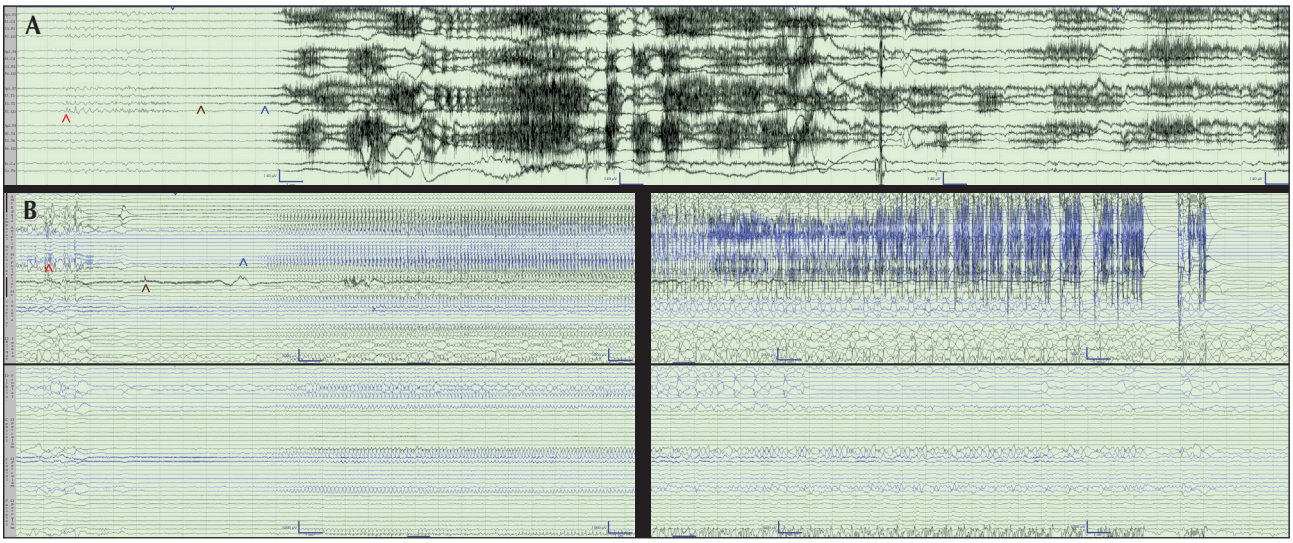

Figure 1. Scalp EEG and stereo-EEG recordings of a 41-year-old patient with mirthful laughter seizures. (A) Ictal scalp-EEG: a total of five stereotypical mirthful laughter seizures were recorded in which EEG changes preceded clinical changes. Seizures began over A1-T3-F7 with polyspikes and spread to C3-P3 over five seconds. This was followed by generalized low-voltage fast activity with maximum left temporal activity, followed by muscle artefacts when the patient started laughing. (B) Ictal stereo-EEG (bipolar montage): during two weeks of intracranial electroencephalographic monitoring, 23 seizures were recorded, all electrographically stereotyped and preceding the clinical onset by three to four seconds. Onset with 1.5 Hz and high-voltage discharges were located in the left neocortical temporal region, and -to a lesser degree- in the amygdala, insula, and orbitofrontal cortex (compare with *figure 2B*). This was followed by low-voltage fast activity in the same electrodes (maximum in the neocortical temporal regions) for two seconds. After that, there was a diffuse attenuation in all the contacts for five to six seconds. Finally, spikes progressively built up in the temporal neocortex, which then spread to the rest of the temporal region, as well as to the amygdala, insula, and orbitofrontal cortex. In order to downsize the figure, 51 seconds of the ictal stereo-EEG have been cropped. Red marker: electrographic onset; brown marker: awaking from sleep; blue arrow: clinical seizure onset (laughter); hc: hippocampus.

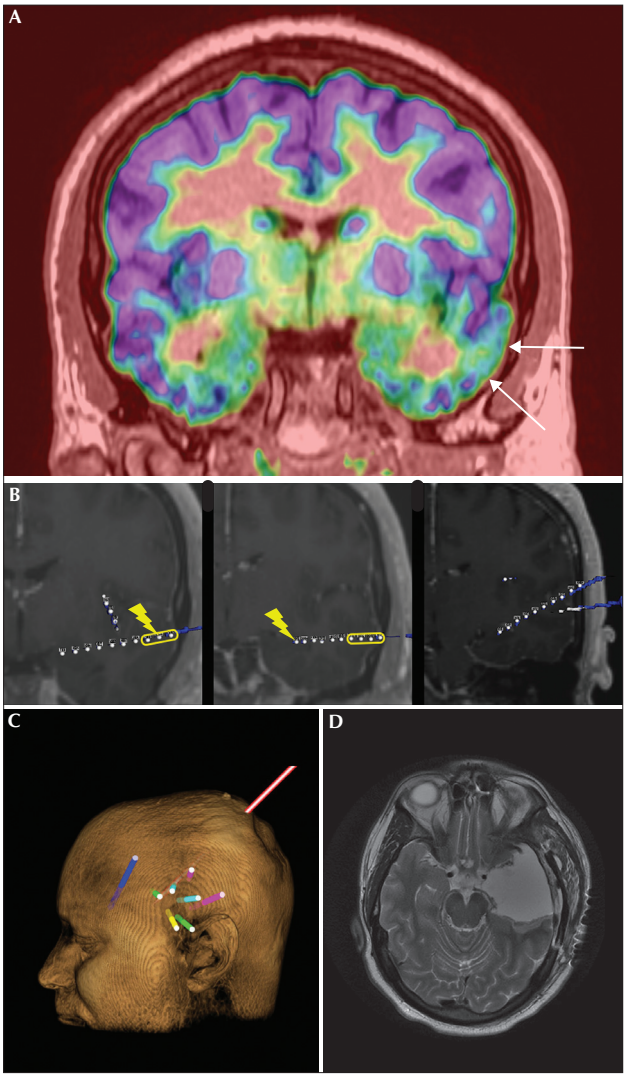

Figure 2. (A) Left temporal hypometabolism (arrows) in a patient with mirthful laughter seizures demonstrated by ^{18}F-fludeoxyglucose positron emission tomography. (B) The left, middle, and right panel show the depth electrode contacts at the level of the amygdala, as well as the anterior and posterior hippocampus, respectively. The neocortical seizure onset zones are highlighted with yellow boxes. Stimulation of one contact in the anterior superior temporal gyrus neocortex electrode (left panel; lightening symbol) triggered a typical laughing seizure at 2 mA. Stimulation of the anterior hippocampus electrode contact (middle panel; lightning symbol) with 2 mA triggered epigastric rising, nausea, and induced speech arrest, and made the patient feel as if he was going to have a laughter seizure. (C) Stereoelectroencephalography implantation scheme covering the left temporal (lower row: yellow/green/pink electrodes) and orbitofrontal lobe (dark blue electrode), the opercular region (upper row: light green/light blue/pink electrodes), as well as the insula (light blue and oblique red electrode). (D) T2-weighted magnetic resonance imaging after left anterior temporal lobectomy.

width; 50-Hz frequency; 2-mA current) determined left hemispheric language dominance. Stimulation of the anterior superior temporal gyrus neocortex (*figure 2B*) triggered a typical laughing seizure at 2 mA. Stimulation of the anterior hippocampus with 2 mA triggered an epigastric rising sensation with nausea and speech arrest and made the patient feel as if he was going to have a laughing seizure. In view of the temporal SOZ, an awake left anterior temporal lobectomy (ATL) with electrocortical stimulation for language mapping was performed (*figure 2D*).

Pathology revealed Chaslin's gliosis and focal cortical dysplasia type IIa in the subpial neocortical regions. The postsurgical course was unexpectedly complicated by a transient but severe delirium which fully resolved over two weeks. Minor expected postoperative short-term memory problems improved readily as did some transient word-finding difficulties. He has remained seizure-free at the last follow-up visit at one year (Engel Class IA) on lacosamide and perampanel.

Discussion

Mirthful laughter of temporal onset with subsequent ATL resulting in seizure freedom, as in this case, is a rarely reported scenario. The body of literature on surgical cases of extra-hypothalamic mirthful laughter seizures is summarised in *table 1* and in the following discussion.

Temporal lobe-onset GS

Oehl *et al*. (2009) decribed a 42-year-old male with GS characterized by a sense of mirth during progression of electric discharges to the temporobasal regions, whereas motor components of laughter occurred later. The patient underwent resection taylored to the inferior temporal gyrus and mesial structures, which resulted in seizure freedom. The temporobasal regions were previously shown to produce symptomatic laughter upon subdural recording of seizure activity in an ictally unaware patient (Umeoka *et al*., 2008) or upon stimulation with a mirthful component in two cases (Arroyo *et al*., 1993). An early case of a 33-year-old soldier (Sethi and Rao, 1976) with a tumour in the left middle and inferior temporal gyrus highlighted the importance of the temporal lobe in processing emotions. This patient presented with ictal laughter, crying or a combination of both, as well as running (cursive epilepsy). Lesionectomy, confirming a Grade I astrocytoma, resulted in seizure freedom. Loiseau *et al*. (1971) reported an extra-axial cause (meningioma) of temporal-onset GS. Another case of GS related to the temporal lobe reported by Dericioglu *et al*. (2005) concerned a young man with GS without a sense of mirth, characterized by short laughter attacks with loss of consciousness and postictal amnesia. MRI revealed

Table 1. Overview of surgical cases of extra-hypothalamic epilepsy with gelastic seizures.

Author & year	Patient	Mirth	Seizure onset zone	Surgery & pathology	Seizure outcome & follow-up
Oehl *et al.*, 2009	RHD M ageSx: 42 ageSz: 16	+	R lateral temporal	Resection of the R temporomedial and inferior gyrus FCD type IIA	**Engel 1A** up to 12 months follow-up with lamotrigine
Sethi and Rao, 1976	M ageSx: 33 ageSz: 31	-	L middle temporal gyrus/inferior temporal gyrus + L fusiform/ parahippocampus	Lesionectomy Astrocytoma Grade I	**ILAE 1A** up to six months without AEDs
Loiseau *et al.*, 1971	F ageSx: 25 AgeSz: 25	?	L temporal lobe	Lesionectomy Meningioma	**?**
	F ageSx: 6 ageSz: 2.5	?	Anterior cingulate gyrus	Lesionectomy Astrocytic cyst	**?**
Dericioglu *et al.*, 2005	RHD M ageSx: 22 ageSz: 14	-	R inferior temporal gyrus	R ATL Cortical dysplasia	**Engel 1A**
Kovac *et al.*, 2015	M ageSx: 44 ageSz: 12	?	L medial temporal	L ATL Hippocampal sclerosis	**ILAE 1**
	M ageSx: 36 ageSz: -	?	L medial temporal	L ATL Hippocampal sclerosis	**ILAE 1**

Table 1. Overview of surgical cases of extra-hypothalamic epilepsy with gelastic seizures (*Continued*).

Gutierrez *et al.*, 2016	M ageSx: 60 ageSz: -	?	R anterior temporal	R ATL Temporal lobe: reactive changes Hippocampus: reactive gliosis	**Engel 1**
	F ageSx: 45 ageSz: -	?	R anterior temporal	R ATL Temporal lobe: reactive changes Hippocampus: insufficient tissue	**Engel 1**
	F ageSx: 36 ageSz: -	?	L anterior temporal	L ATL Temporal lobe: reactive changes Hippocampus: insufficient tissue	**Engel 1**
	M ageSx: 30 ageSz: -	?	R anterior temporal	R ATL Temporal lobe: reactive changes Hippocampus: insufficient tissue	**Engel 1**
	M ageSx: 39 ageSz: -	?	L anterior temporal	L ATL Temporal lobe: reactive changes Hippocampus: insufficient tissue	**Engel 1**
	M ageSx: 31 ageSz: -	?	R anterior temporal	R ATL Temporal lobe: reactive changes Hippocampus: extensive neuronal loss in CA1 and CA3 with moderate neuronal loss in CA2 and CA4	**Engel 1**
	M ageSx: 15 ageSz: -	?	R anterior temporal	R ATL Temporal lobe: reactive changes Hippocampus: insufficient tissue	**Engel 2**
	F ageSx: 45 ageSz: -	?	L lateral temporal	L ATL Pathology not available	**Engel 3**
	F ageSx: 33 ageSz: -	?	R anterior temporal	R ATL Temporal lobe: reactive changes Hippocampus: insufficient tissue	**Engel 4**

Table 1. Overview of surgical cases of extra-hypothalamic epilepsy with gelastic seizures (*Continued*).

Tran *et al.*, 2014	RHD M ageSx: 40 ageSz: 1.4	?	L medial temporal	L amygdala resection Normal pathology	**ILAE 1** up to two years follow-up without AEDs
	RHD M ageSx: 25 ageSz: 6	?	R inferior frontal gyrus + superior anterior insula	1st operation: R frontal polar cortectomy 2nd operation: R inferior frontal gyrus + superior anterior insula 1st: FCD type II 2nd: normal pathology	1st operation: **ILAE 4** 2nd operation: **ILAE 1** up to 19 months follow-up without AEDs
	RHD M ageSx: 26 ageSz: 10	?	R lateral temporal	R superior and medial temporal gyrus inferior frontal gyrus resection FCD	**ILAE 1** up to four years follow-up without AEDs
	RHD M ageSx: 38 ageSz: 26	?	R inferior frontal gyrus + anterior insula	R inferior frontal gyrus + superior anterior insula resection Pathology not available	**ILAE 1** up to 18 months follow-up with AED
	RHD F ageSx: 20 ageSz: 10	?	R medial temporal	R sAHE Hippocampal sclerosis	**ILAE 1** up to two years follow-up with AED
	RHD F ageSx: 28 ageSz: 9	?	R anterior insula + posterior orbitofrontal	R anterior insula + posterior orbitofrontal resection Pathology not available	**ILAE 1** up to 17 months follow-up without AEDs
	RHD F ageSx: 39 ageSz: 5	?	L inferior frontal gyrus + anterior insula	L frontal operculum-anterior insula resection FCD	**ILAE 1** up to 12 months follow-up with AED
Uribe-San-Martin *et al.*, 2015	M ageSx: 45 ageSz: 14	+	L temporal	L sAHE Hippocampal sclerosis associated with cortical dysplasia	**ILAE 1** for one year, then relapse with **ILAE 4**
	M ageSx: 35 ageSz: 29	-	L mesial frontal lobe	Resection of L frontal pole Neuroysticercosis	**ILAE 1** up to five years follow-up and three years without AEDs

Table 1. Overview of surgical cases of extra-hypothalamic epilepsy with gelastic seizures (*Continued*).

Cheung *et al.*, 2007	F ageSx: 29 ageSz: 5	-	R frontal	Resection of R superior frontal cortical dysplasia FCD with subcortical ballooned astrocytes	**Engel 1** up to 15 months follow-up with AED
Hu *et al.*, 2011	RHD M ageSx: 15 ageSz: 5	+	R frontal	Resection of the anterior part of the R inferior frontal gyrus FCD type II	**Engel 1A** up to three months follow-up under continued AED (oxcarbazepine 1200 mg per day)
Marashly *et al.*, 2017	RHD F ageSx: 3 ageSz: 3	-	R frontal	R frontal lobectomy sparing the motor regions FCD type IIA	**Engel 1A** up to 15 months follow-up without AEDs (first six months with phenobarbital).
Dubey *et al.*, 2015	RHD M ageSx: 6 ageSz: 6	?	L frontal	Resection of L frontal epileptogenic focus FCD type II	**Engel 1A**
Zhou *et al.*, 2017	M ageSx: 3 ageSz: <1	?	L frontal	L partial frontal lobectomy FCD type IIB	**Engel 1A** up to 14 months follow-up under continued AED (topiramate)
Unnwongse *et al.*, 2010	RHD M ageSx: 66 ageSz: Childhood	?	R orbitofrontal	R superior and middle frontal gyrus as well as cingulate and orbitofrontal gyrus resection FCD type IIB	**Engel 1A** up to 12 months follow-up without AEDs
Umeoka *et al.*, 2008	F ageSx: 49 ageSz: 43	?	R frontal (posterior orbitofrontal)	Partial R prefrontal lobectomy (orbitofrontal + anterior perforating substance) Mixed oligoastrocytoma	**ILAE 1A** up to 33 months follow-up without AEDs
Kurle and Sheth, 2000	RHD F ageSx: 6 ageSz: -	-	R superior frontal gyrus - medial frontal gyrus (anterior to paracentral) and anterior cingulate gyrus	Lesionectomy Dysembryoplastic neuroepithelial tumour	**ILAE 1A** up to 18 months without AEDs

Table 1. Overview of surgical cases of extra-hypothalamic epilepsy with gelastic seizures (*Continued*).

Neilson *et al.*, 2014	RHD M ageSx: 10 ageSz: 10	+	R frontal	Lesionectomy Dysembryoplastic neuroepithelial tumour mixed with cortical dysplasia	**Engel 1A** up to 18 months follow-up
Mohamed *et al.*, 2007	M ageSx: 17 ageSz: 10	-	L anterior cingulate gyrus	Tumour resection Pleomorphic xanthoastrocytoma	**ILAE 1A** up to 12 months follow-up without AEDs
Nicolae *et al.*, 2010	RHD M ageSx: 27 ageSz: 25	-	R middle cingulate gyrus	Lesionectomy Grade II astrocytoma	**ILAE 1A** up to three months follow-up without AEDs
Arroyo *et al.*, 1993	F ageSx: 35 ageSz: 15	-	L superior and medial frontal gyrus + dorsal anterior cingulate gyrus	Lesionectomy plus adjacent anterior cingulate gyrus Cavernoma+gliosis	**ILAE 1A** up to 16 months without AEDs; one relapse during weaning off AED
Jayalakshmi *et al.*, 2018	M ageSx: 43 ageSz: 41	-	L inferior frontal gyrus	Partial lesionectomy Cavernoma	**Engel 1A** up to 18 months follow-up; relapse during weaning off AED **Engel 1A** up to 10 years follow-up with one AED
Chassagnon *et al.*, 2003	RHD M ageSx: 24 ageSz: 4	?	L supplementary motor area and cingulate motor area	Stereotactic monopolar electric radiofrequency lesions No pathology	**Engel 1A** up to 27 months follow-up under continued AED (topiramate)

AED: antiepileptic drug; ageSx: age at surgery; ageSz: age at seizure onset; F: female; FCD: focal cortical dysplasia; L: left; M: male; RHD: right-hand dominant.

right inferior temporal cortical dysplasia. Following an intracarotid amobarbital test, a successful right ATL was performed. In the largest case series of GS (Kovac *et al.*, 2015), two out of 19 patients underwent ATL for a temporal epileptogenic zone resulting in seizure freedom. Gutierrez *et al*. (2016) reviewed 16 cases of GS, of which nine underwent ATL. Outcome was Engel Class I in six, Engel Class II in one, Engel Class III in one, and Engel Class IV in one. Similarly, in the case series of the Centre Hospitalier Université de Montréal (Tran *et al.*, 2014), three patients with temporal GS achieved seizure freedom. The favourable outcomes in the majority of these patients support the role of ATL in the treatment of GS with extra-hypothalamic temporal SOZ. Whether selective amygdalhippocampectomy (sAHE) may work just as well remains unanswered at this point. While the only patient from the Gutierrez case series who underwent sAHE remained seizure-free, Uribe-San-Martin *et al*. (2015) reported a less favourable outcome. Their patient with hippocampal sclerosis undergoing sAHE had a relapse one year after initial seizure freedom and ultimately achieved only a 50% reduction compared to his presurgical average.

In summary, 19 patients with extra-hypothalamic temporal-onset GS had a male:female ratio of 2.2, a mean age at seizure onset of 14.8 ± 8.7 years (range: 1.4 to 31 years), and mean age at surgery of 35.1 ± 10.9 years (range: 15 to 60 years). Laterality was equal: 10 (52.6%) right-sided and nine (47.4%) left-sided. Follow-up time was available for six patients: 1.8 ± 1.3 years (range: 0.5 to 4 years). Seizure outcome was determined in all patients as follows: Engel Class 1 in 14 (77.8%); Engel Class II in one (5.6%); Engel Class III in one (5.6%); and Engel Class IV in two (11.1%).

Extra-hypothalamic: frontal-onset GS

The Montreal case series (Tran *et al.*, 2014) also comprised four surgically treated frontal GS cases. In all of them, the insula was partially involved and resected, and seizure freedom was achieved. A previous case report from our centre concerned a 29-year-old female with a long-standing history of generalized complex and partial seizures since early childhood (Cheung *et al.*, 2007). These were controlled with AEDs during pregnancy and resolved later in life. However, she then developed partial events consisting of brief periods of laughing as well as staring spells. Video-scalp and intracranial EEG, MRI, and SPECT delineated the SOZ to a resectable right frontal cortical dysplasia. Seizure freedom was documented until the last known follow-up visit, eight years after resection. Similarly, Hu *et al*. (2011) described a teenager with drug-refractory GS that resolved completely following resection of a focal cortical dysplasia. Marashly *et al*. (2017) reported an interesting case of a three-year-old female who presented with GS in the setting of new-onset refractory status epilepticus. PET and SEEG identified the SOZ in the right superior frontal gyrus. A right frontal lobectomy sparing the motor regions was performed that revealed focal cortical dysplasia and resulted in seizure freedom. Two more pediatric cases (Dubey *et al.*, 2015; Zhou *et al.*, 2017) and one adult case (Unnwongse *et al.*, 2010) of GS secondary to frontal focal cortical dysplasia, that were operated on sucessfully, were reported in the literature. In terms of oncological aetiology, Umeoka *et al*. (2008) described a patient with a mixed oligoastrocytoma in the right frontal lobe, precisely in the orbitofrontal cortex, who after a partial prefrontal lobectomy became seizure-free. In the same vein, others (Loiseau *et al.*, 1971; Kurle and Sheth, 2000; Neilson *et al.*, 2014) reported good outcomes following resection of orbitofrontal and prefrontal tumours. Two cingulate tumour surgical cases were operated on via an interhemispheric approach by Mohamed *et al*. (2007) and Nicolae *et al*. (2010) and resulted in seizure freedom. A cingulate cavernoma causing epileptic laughter and crying was reported by Arroyo *et al*. (1993). The authors elaborated on the observation of a lack of mirth with SOZ in the motor-predominant cingulate region vs. mirthful laughter with involvement of the temporabasal regions. Thus, on the grounds of a semiological accompanied sense of mirth, one may be able to deduct which area of the brain is involved. Jayalakshmi *et al*. (2018) obtained seizure freedom after partial resection of a cavernoma situated in proximity to Broca's area, as determined by intraoperative electrocortical stimulation. A less common infectious aetiology for GS, neurocysticercosis, was reported from Chile. Uribe-San-Martin *et al*. (2015) described a young man with known neurocysticercosis since his teenage years that was first controlled with antiparasitic drugs. At the age of 29, he developed sudden unmotivated laughter attacks with loss of awareness, with poor response to AEDs. Invasive monitoring with depth electrodes delineated the SOZ to the left mesiofrontal region. Following resection of the left frontal pole that included a calcified neurocysticercosis cyst, the patient became seizure-free. Chassagnon *et al*. (2003) successfully performed radiofrequency ablation in the left supplementary motor area and cingulate motor area for GS.

In summary, 20 patients with extra-hypothalamic frontal-onset GS had a male:female ratio of 1.2, a mean age at seizure onset of 13.6 ± 13.3 years (range: 0.8 to 43 years), and mean age at surgery of 25.2 ± 17.2 years (range: 3 to 66 years). Laterality was equal: 11 (55%) right-sided and eight (40%) left-sided, and one midline (5%). Follow-up time was available for 18 patients: 2.2 ± 2.6 years (range: 0.25 to 10 years). Seizure outcome in

19 patients was 100% favourable (Engel Class 1 or ILAE Class 1A).

Generally, the above-summarized outcomes of surgical cases of extra-hypothalamic GS were mostly favourable. Notably, no adverse events were reported. One should be aware of possible publication bias. Furthermore, extrapolation of the results should be undertaken with caution since dominant-hemisphere lateral fronto-temporal as well as insulo-perisylvian cortex resections may represent surgical challenges for some centres.

Conclusion

In light of the current case report and available literature, drug-refractory cases of temporal and frontal GS are amenable to surgical treatment following a thorough investigation with imaging, as well as scalp and intracranial EEG. Further surgical case publications are needed to better understand the role of epilepsy surgery and to establish realistic outcome expectations in the setting of extra-hypothalamic GS. □

Supplementary data.
Summary didactic slides are available on the www.epilepticdisorders.com website.

Disclosures.
Dr. Holger Joswig received speaker honoraria from UCB Canada and travel grants from Medtronic. The remaining authors have no conflicts of interest to declare.

Legend for video sequence

A typical mirthful laughter seizure from sleep of a 41-year-old patient in the epilepsy monitoring unit.

Key words for video research on www.epilepticdisorders.com

Phenomenology: gelastic seizure
Localisation: temporal lobe (left)
Syndrome: focal non-idiopathic temporal (TLE)
Aetiology: focal cortical dysplasia (Type II)

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TEST YOURSELF

(1) Where can the onset of gelastic seizures occur?

(2) A mirthful component is more commonly seen with gelastic seizures with onset in which region?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".