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Clinical and Electrographic Features of Sunflower Syndrome

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Abstract

Background—Sunflower Syndrome describes reflex seizures – typically eyelid myoclonia with or without absence seizures – triggered when patients wave their hands in front of the sun. While valproate has been recognized as the best treatment for photosensitive epilepsy, many clinicians now initially treat with newer medications; the efficacy of these medications in Sunflower Syndrome has not been investigated. We reviewed all cases of Sunflower Syndrome seen at our institution over 15 years to describe the clinical course, electroencephalogram (EEG), and treatment response in these patients.

Methods—Search of the electronic medical record and EEG database, as well as survey of epilepsy providers at our institution, yielded 13 cases of Sunflower Syndrome between 2002–2017. We reviewed the records and EEG tracings.

Results—Patients were mostly young females, with an average age of onset of 5.5 years. Seven had intellectual, attentional or academic problems. Self-induced seizures were predominantly eyelid myoclonia +/− absences and 6 subjects also had spontaneous seizures. EEG demonstrated a normal background with 3–4Hz spike waves +/− polyspike waves as well as a photoparoxysmal response. Based on both clinical and EEG response, valproate was the most effective treatment for reducing or eliminating seizures and improving the EEG; 9 patients tried valproate and 66% had significant improvement or resolution of seizures. None of the nine patients on levetiracetam or seven patients on lamotrigine monotherapy achieved seizure control, though three patients had improvement with polypharmacy.

Conclusions—Valproate monotherapy continues to be the most effective treatment for Sunflower Syndrome and should be considered early. For patients who cannot tolerate valproate, higher doses of lamotrigine or polypharmacy should be considered. Levetiracetam monotherapy, even at high doses, is unlikely to be effective.

Keywords

Epilepsy; reflex seizures; reflex epilepsy; self-induced seizures; eyelid myoclonia; absence seizures

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1. Introduction

Of the 10% of epilepsy patients with photosensitivity, a small subset self-induce seizures by seeking out photic triggers (Koepp et al., 2016). Patients will stare at a light source and wave abducted fingers in front of their faces, rapidly blink as they slowly close their eyes, or perform other behaviors that create a similar flicker effect (Ames and Saffer, 1983). Typically, seizures include eyelid myoclonia (EM) with or without absences seizures, though some patients can go on to have generalized tonic-clonic seizures (GTCs) (Belcastro and Striano, 2014; Panayiotopoulos, 2005). This constellation of symptoms has been termed “Sunflower Syndrome” due to the sun-seeking behaviors of the patients and the characteristic way in which they bend their faces up toward the sun. It is unclear if Sunflower Syndrome is a distinct clinical entity or is related to other conditions with photosensitivity (Striano et al., 2009), particularly Jeavons Syndrome.

Valproate is widely recognized as the most effective treatment for photosensitive epilepsies (Covanis et al., 2004; Koepp et al., 2016), though ethosuximide, benzodiazepines, lamotrigine, and more recently levetiracetam have also proven effective in eliminating the photoparoxysmal response on EEG and stopping seizures (Striano et al., 2008). In contrast to other photosensitive seizures, self-induced seizures are extremely refractory to treatment (Andermann et al., 1962; Covanis et al., 2004; Hutchison et al., 1958; Ng, 2002), even when patients are compliant with medication. Covanis et al. (2004) note that while 75% of patients with Jeavons Syndrome respond to valproate monotherapy, only 40% of patients with self-induced seizures respond to this treatment. Caraballo et al. (2009) described 63 patients with EM; of the six patients in this cohort who self-induced seizures, at least four were refractory to treatment. Case reports and small series report improvement with other medications, including fenfluramine (Aicardi and Gastaut, 1985; Boel and Casaer, 1996), pimozide (Binnie, 1988), and stimulant therapy (Fernández-Mayoralas et al., 2011), as well as various forms of psychotherapy (Ng, 2002). Environmental measures – such as sun avoidance, use of sunglasses or specific blue-lens (Z1) glasses, or occlusion of one eye –also prevent seizures. The Z1 lenses, which have been evaluated predominantly in Europe, additionally eliminate the photoparoxysmal response on EEG (Belcastro and Striano, 2014; Capovilla et al., 2006). Many of the larger case series of Sunflower Syndrome were published in the 1960s–1980s, with relatively little written in the last fifteen years (Bebek et al., 2006; Belcastro and Striano, 2014; Caraballo et al., 2009; Covanis et al., 2004). In particular, there is a paucity of data on response to newer anti-seizure medications which are now frequently prescribed instead of valproate. We reviewed the clinical, EEG, imaging, and laboratory data of children seen at our center for self-induced photic seizures to describe clinical course and treatment response.

2. Materials and Methods

We searched the electronic medical records and EEG database of Lucile Packard Children’s Hospital, in Palo Alto, CA from January 1, 2002–November 1, 2017 for the following keywords or phrases: Sunflower Syndrome; Jeavons Syndrome; fixation off; eyelid myoclonia/myoclonus +/− absence seizures; photic reflex epilepsy/seizures; and self-

induced seizures. In addition, our epilepsy clinicians were surveyed for cases. Patients between ages 0–18 years of age whose medical records mentioned self-induction of seizures with light were included; those with other forms of self-induction (i.e. reading epilepsy) were excluded.

Of the 105 charts reviewed, 13 children met inclusion criteria. We reviewed seizure semiology, age of onset, neurodevelopmental status, family history of neurologic disorders, treatment history, and suspected etiology (i.e. MRI, metabolic or genetic findings). We reviewed all available EEG tracings to evaluate the background, interictal activity, seizures, and response to hyperventilation and intermittent photic stimulation (IPS); we reviewed EEG reports for similar data on tracings done outside our institution.

3. Results

3.1 Clinical History

We have summarized key clinical findings of the 13 patients in Table 1. Most patients were female (77%). Age of onset varied between 2–8 years of age. Six had normal development, two had processing difficulties, four had attention deficit hyperactivity disorder (ADHD), and one had intellectual disability (ID). Four had migraines during adolescence. Two patients were initially misdiagnosed as having tics.

3.2 Family History

Six patients had no family history of epilepsy. One patient had a brother with psychogenic non-epileptic spells, one had a brother with febrile seizures, and two had aunts/uncles with a history of convulsions. One patient's mother had unusual sensations when watching video games and one patient's grandfather had a history of "flicker-induced vertigo." Additionally, a patient who had initially been misdiagnosed with tics also had a grandfather with a diagnosis of tics.

3.3 Seizure Semiology

Self-induced seizures consisted of EM without absence for five patients, EM with absence for six patients and absence alone for two patients. Self-induction techniques included staring at the sun, waving the hand in front of a light source, or rubbing the forehead. Seven patients (54%) also had spontaneous seizures.

3.4 Etiology

There was no clear etiology found for any patient, though work-up was often limited. Birth history was non-contributory in the 11 patients for which it was available. Five patients had neuroimaging, all of which was normal. Two epilepsy gene panels revealed no known pathogenic variants, including no variants in the CHD2 gene (which has been associated with self-induced photosensitive seizures) (Galizia et al., 2015).

3.5 Burden of Disease

Table 1 includes an approximate seizure frequency. All patients had seizures at least daily, and many had seizures multiple times per hour when in bright sunlight. At least 3 patients

noted that seizures significantly interfered with class and homework. Five remarked that seizures prevented normal participation in recess, gym or outdoor activities; one patient frequently triggered seizures while swimming. Three children felt embarrassed by their seizures and most commented that peers questioned them about these events.

3.6 EEG Data without Medications

Ten patients had a total of eleven EEGs done without medication (summarized in Table 2). Nine studies were done to diagnose the disorder and two were done to follow-up after medications had been stopped. The background was normal for age in all studies except for intermittent right posterior focal slowing in one patient. All ten patients had generalized 3–4Hz spike wave discharges occurring in brief runs, usually with a bifrontal predominance; nine of the ten had multiple runs lasting longer than 3 seconds. Some patients had longer studies (i.e. ambulatory EEGs), and these patients clearly had more frequent and longer runs of spikes when outside. Eight patients had polyspikes or frontal fast activity. Hyperventilation in three of four studies elicited generalized spike or polyspike wave discharges. IPS led to a photoparoxysmal response in six of seven patients. Seven EEGs in six patients captured seizures with EM associated with an increase in frontal spike and polyspike wave activity lasting up to 8 seconds, with one patient having concomitant posterior polyspikes. For three of the four patients without tracings or full reports (not included in Table 2), the chart describes the unmedicated EEG as “abnormal” or abnormal due to “spike and wave complexes” or “photoparoxysmal response.”

3.7 Treatment Response

The clinical response is discussed in Table 1. In addition, we reviewed nineteen EEGs from eleven patients receiving antiepileptic therapy with details presented in Table 3.

3.7.1 Levetiracetam—Levetiracetam was the first medication tried in nine patients and added to topiramate in a tenth. No patients achieved seizure freedom on levetiracetam monotherapy. Six patients reported no change in seizure frequency. One reported a decrease in seizures from hourly to 10 times per day; ethosuximide was then added. Another reported subjective improvement in seizures frequency, but the EEG was unchanged and she switched to the more effective valproate. A final patient noticed “significant” improvement, but levetiracetam was stopped due to suicidal ideation. Four children on levetiracetam monotherapy (30–83mg/kg/day) had abnormal EEGs with persistence of the photoparoxysmal response. Three of the four children were brought outside during their EEG and all self-induced seizures in sunlight.

Patients on polytherapy including levetiracetam had a better clinical response than those on levetiracetam monotherapy. One patient had hundreds of seizures per day on topiramate (5mg/kg/day) but achieved seizure freedom and EEG normalization with the addition of levetiracetam (25mg/kg/day). As above, a second patient failed high dose levetiracetam (80mg/kg/d) monotherapy, but improved on levetiracetam (53mg/kg/d) combined with ethosuximide (40mg/kg/d) with resolution of seizures, a normal EEG during wakefulness, but persistent photoparoxysmal response; this child had recurrence of spikes waves on a follow-up EEG.

3.7.2 Lamotrigine—Lamotrigine was trialed in seven patients and we reviewed the EEG of three. Lamotrigine was discontinued in two due to rash; in one, it was too early to determine its effect on seizures. Three patients noticed no difference in seizure frequency and two felt their seizures worsened. One child felt it significantly improved seizure frequency, but she had resolution of seizures only when valproate was added. We reviewed four EEGs from three children on lamotrigine. Two children on lamotrigine monotherapy (~3mg/kg/day) did not have improvement in their EEGs, but one had normalization of the EEG after valproate was added. A third patient on a higher lamotrigine dose (5mg/kg/day) had fewer discharges compared to the diagnostic EEG. Two children could still self-induce seizures in sunlight; the third was not tested in sunlight but had persistent photoparoxysmal response.

3.7.3 Valproate—Nine patients tried valproate and eight ultimately continued this treatment, though it was the initial therapy for only one child. Therapeutic doses ranged from 12.5–43mg/kg/day with levels of 83–130μg/mL. Five patients achieved seizure freedom, though two continued to have hand-waving without EEG correlate. One patient had elimination of self-induced seizures, but persistent spontaneous EM in the car. Two patients had trouble tolerating valproate due to stomach discomfort; in one, a lower dose was tried with less clinical efficacy and in the second, valproate was discontinued. The child with ID had improvement in GTCs but minimal improvement in self-induced seizures with valproate.

We reviewed seven EEG tracings from five children on valproate. Two children had normalization of the EEG and two others showed marked improvement. One child's EEG improved on high but not low dose valproate. The fifth child, with ID, stood apart with a quite abnormal EEG on two occasions. Three of five children had resolution of the photoparoxysmal response; two children were recorded outside and neither could self-induce seizures with hand-waving. As above, a sixth child had normalization of the EEG with lamotrigine plus valproate.

3.7.4 Ethosuximide—Five patients tried ethosuximide. Four discontinued for inefficacy or non-compliance. One had improvement with ethosuximide plus levetiracetam therapy (described above).

3.7.5 Other agents—Four patients trialed benzodiazepines (clonazepam or clobazam) and only one patient continued clobazam for control of his GTCs. Two patients stopped zonisamide monotherapy therapy due to persistent seizures; an EEG in one showed improvement in interictal discharges from baseline and resolution of the photoparoxysmal response. One patient tried felbamate, which was ineffective.

3.7.6 Non-pharmacologic Therapies—Three developmentally-normal patients significantly benefited from counseling on trigger avoidance (i.e. wearing sunglasses and hats). A fourth patient had marginal improvement, but even with polarized sunglasses, still had seizures in the car; the patient had been unable to obtain the Z1 lenses. A fifth patient did not benefit from sunglasses. Four patients were offered Comprehensive Behavioral

Intervention for Tics (CBIT); two declined and the other two did not complete therapy due to logistical issues.

4. Discussion

Sunflower Syndrome, or self-induced photosensitive epilepsy, is a rare condition that is difficult to treat. Larger case series are now 30–50 years old and there have been various new anti-seizure medications introduced in the interim. We present the clinical and electrographic data of thirteen children seen at our institution over the last 15 years with particular attention to response to anti-seizure medications.

4.1 Clinical Features

Our population shared many clinical similarities with prior case series. The group was predominantly (77%) female, consistent with some (Andermann et al., 1962) but not all (Darby et al., 1980; Green, 1966) reports. Age of onset was also similar, ranging from 2–8 years of age. There was a clear family history of seizures in about one quarter of our patients and a family history of photosensitivity without clearly documented seizures in an additional 15%. Our group had better cognitive abilities than previously reported. Prior literature has suggested a high rate of ID, particularly in patients with early seizure onset. Andermann et al. (1962) noted that 50% of 21 patients had ID. More recently, Caraballo et al. (2009) described that four of six patients with self-induced seizures had ID and onset of epilepsy in the first 3 years. In contrast, only one patient in our group had ID, though six other children had academic difficulties or ADHD. The patient with ID had the earliest age of seizures onset (2.5 years) but there were other children with early onset with normal development or ADHD. Several children with delayed diagnosis had normal cognition.

4.2 Diagnosis of Seizures

EM, absence seizures or a combination of the two were induced by various maneuvers in natural light, similar to previous reports (Kent et al., 1998). Four patients were accomplished ballerinas and yet were not bothered by bright stage lighting, highlighting the stimulating effects of natural light in this group. Given this, our neurophysiology laboratory has made it routine practice to obtain an ambulatory recording or bring children into the sunlight during their video EEG. This practice has been helpful in both establishing a diagnosis and determining treatment response. Two patients continued to have hand-waving during everyday activities, even though this movement no longer activated the EEG, and therefore behavioral therapies were recommended instead of medication escalation. The fact that hand-waving may continue even after it fails to induce seizures highlights that EEG should be used in conjunction with clinical reports in assessing therapy.

4.3 Treatment

Similar to other photosensitive epilepsies (Covanis et al., 2004), children with Sunflower Syndrome have the best clinical response to valproate. All nine children who tried valproate had clinical benefit, and only one weaned off due to stomach upset. Valproate was typically effective at relatively low doses (13–25mg/kg/day), though a higher dose was required for one patient. Valproate may not be as effective in the subset of patients with self-induced

seizures and ID. This work in conjunction with prior case series suggest that valproate should be the first line agent for children presenting with self-induced seizures.

In comparison to valproate, patients had a less robust response to lamotrigine therapy. Low doses (<4mg/kg/day) had no effect on seizure frequency or the EEG, though higher doses (≥5mg/kg/day) or polytherapy did have better effects. Levetiracetam monotherapy, even at doses of 80mg/kg/day, failed to control self-induced seizures or alter the EEG. In contrast, moderate doses of levetiracetam combined with other agents led to significant clinical and EEG improvement. In our cohort, zonisamide, ethosuximide, and benzodiazepines were rarely used, but did not lead to good seizure control.

4.4 Limitations

The conclusions in this study are limited by the retrospective nature of the data collection. We used broad search criteria targeted at identifying patients who had photosensitivity by either clinical or EEG criteria and then reviewed charts in detail for a description of a self-induction technique. As we do not routinely ask every patient about self-induction, our search method only identifies patients in which the behavior was described by themselves or their parents. It is possible that we missed patients who surreptitiously induced seizures or whose self-induced seizures were not noticed by family; such patients may have milder courses and respond to different medications. Even though we selected for more severe cases, the data we present are still relevant for practitioners as it is these children whose lives are most impacted by self-induced seizures and who present to epilepsy clinic for treatment.

5. Conclusion

Sunflower Syndrome is a challenge to treat. Valproate has historically proven to be the most effective medication for photo-sensitive epilepsies, but among our patient population, newer seizures medications are consistently prescribed first. Regardless of initial agent choice, most patients ultimately switched to valproate, which offered the best chance of seizure freedom and EEG improvement. If a patient cannot use valproate, higher doses of lamotrigine or possibly polypharmacy should be considered. Levetiracetam monotherapy is unlikely to control seizures. Finally, at a minimum, neurologists should counsel patients about trigger avoidance, and other non-pharmacologic therapies can be explored.

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Highlights

- Self-induced seizures in photosensitive patients are difficult to treat.
- Typical patients are school-aged and often have cognitive/attention difficulties.
- Seizures consist of eyelid myoclonia +/− absence; 50% have spontaneous seizures.
- Consider valproate or polypharmacy early; avoid levetiracetam monotherapy.
- Counsel on trigger avoidance. Measure treatment response with EEGs done outside.

TABLE 1

Clinical Characteristics

This table describes the clinical characteristics and treatment trials of every patient.

ID#	Sex	Age at Onset (yr)	Seizure Semiology	Trigger	Baseline Frequency	Spontaneous Seizures	Developmental Problems	Final Medication Regimen (mg/kg/d)	Seizure Control	Failed Medications (mg/kg/d)	Non-Drug Therapies
1	M	5	EM	Stares at sun	>Hourly		Slow processing	VPA 500mg BID (32)	Sz Free	LEV 1000mg BID (58)	MD counseling
2	F	8	EMA	Waves hand	>Daily		No	VPA 375mg BID (25)	Sz Free; still waves hand	None	None
3	M	8	EM	Stares at sun	>Daily		Slow Processing	VPA 375mg BID (25)	No change	LEV 700mg BID (56) LTG 50mg BID (4)	CBIT
4	F	5.5	EMA	Waves hand	>Hourly		No	LTG 50mg BID (3.3)	No change	LEV 500mg BID (40)	MD counseling
5	F	5	EMA & UE My	Waves hand	Hourly		Severe ADHD	VPA 500mg BID (43)	Sz reduced	LEV 250mg BID (20); ETX 250mg BID (20); FEL 400mg BID (40); ZNS 75mg BID (6.5); CLB	CBIT
6	F	4	EM	Stares at sun	>Hourly	GTC, MY	No	LEV 1500mg BID (25); TPM 150mg BID (5)	Sz Free	None	MD counseling
7	F	<10	EMA	Waves hand	>Daily		ADHD	VPA 1000mg BID (26)	Sz Free	LEV	None
8	M	2.5	EMA & UE MY	Stares at sun	>Hourly	GTC	ID	VPA 500mg BID (13); CLB (0.5)	No change	LEV, ETX, LTG, CLZ	None
9	F	3	EMA	Stares at sun	>Hourly	GTC	ADHD	VPA 350mg BID (25) LTG 50mg BID (3.5)	Sz Free	LEV, ETX, ZNS	None
10	F	5	EM	Rub forehead	>Daily	ABS, GTC	No	VPA 750mg QD (13)	Sz reduced	LTG; LEV 500mg BID (27); ETX	None
11	F	5	EM	Waves Hand	>Daily	GTC	No	CLZ 0.5mg TID (0.01)	Sz reduced	VPA 250mg BID (11); LTG 25mg BID (<1m/k/d)	None
12	F	6	ABS	Rub forehead	Hourly		No	LTG 150mg BID (7)	Sz Free; still waves hand	CLZ	None
13	F	7	ABS	Waves hand	>Daily	GTC	ADHD	LEV 1000mg BID (47) ETX 750mg BID (37)	Sz reduced	None	MD counseling

ABS=absence; ADHD=Attention Deficit Hyperactivity Disorder; CBIT=Comprehensive Behavioral Intervention for Tics; CLB=clobazam; CLZ=clonazepam; ETX=ethosuximide; GTC=generalized tonic clonic seizure; EM=eyelid myoclonia; EMA=eyelid myoclonia with absence; ID=intellectual disability; LTG=lamotrigine; LEV=levetiracetam; mg/kg/d=milligram/kilogram/day; My=myoclonus; Sz=seizure; TPM=topiramate; VPA=valproate; ZNS=zonisamide

TABLE 2

EEG Characteristics while not taking Seizure Medications

This table shows characteristics of EEGs performed without seizure medication. Most of these EEGs were diagnostic EEGs while two were done after a medication wean. The EEG# (column 1) indicates the order of the EEGs, also accounting for EEGs described in Table 3. When reporting about seizures, +SI indicates that the patient was still able to elicit seizures in the sun while "." indicates that the patient was not brought into sunlight for the EEG. Fragments are unilateral spikes/polyspikes that were felt to be unilateral manifestations of generalized discharges rather than true focal discharges. Patient 12 had two unmedicated EEGs (one at diagnosis and a second after weaning medications).

ID#/EEG#	Age (yr)	Interictal 3–4 Hz Spikes	Interictal Polyspikes/Frontal Fast	Maximum Spike Wave Duration (sec)	Runs >3 Sec (#/hour)	Focal Findings	Seizures Induced in Sun	HV	PPR
2/1	8	+	−	6	~6	−	.	.	+
3/1	9	+	+ Cz	5	43	Fragments	+ SI	.	.
4/1	7.5	+ BF/BP	+ BF	16	10	−	+ SI	.	.
5/1	7.5	+ BF/BP	+ BF	7	13	−	+ SI	.	+
7/3	16	+ BF	+ BF	8	30	−	+ SI	.	.
9/2	5.4	+	−	2	0	−	.	−	−
10/1	12.1	+ BF	+ BF/BP	10	9	Fragments	.	SW	+
11/1	7.4	+	+	5	4	Focal slowing	.	PSW	+
12/1	8.6	+ BF	+ BF/BP	10	15	Fragments	+ SI	.	+
12/3	10.7	−	+ BF/BP	6	7	Fragments	+ SI	.	.
13/1	11.3	+	+	5	Multiple	−	+ SI	S/PSW	+

BF=bifrontal; BP= biposterior; Cz=central; HV=hyperventilation; PPR=photoparoxysmal response; PSW=polyspike waves; Sec = second; SI=self-induced; S/PSW=spike and polyspike waves; SW=spike waves;.=not tested

TABLE 3

EEG Characteristics while taking Seizure Medications

This table reviews EEG features seen on different medication regimens. The first column provides the medication type while the second column provides the dose. The column on the far right describes the change in EEG compared to an EEG in the same patient without medication. Please note that several patients have multiple EEGs, either on different medications or different doses of the same medication; the EEG# (third column) indicates the order of the EEGs, also accounting for any EEGs described in Table 2. When reporting about seizures, +SI indicates that the patient was still able to elicit seizures in the sun while −SI indicates that the patient tried but was unable to elicit seizures. Fragments are unilateral spikes/polyspikes that were felt to be unilateral manifestations of generalized discharges rather than true focal discharges.

Med	Dose (mg/kg/d)	ID#/EEG#	Age (yr)	Interictal 3–4Hz Spikes	Interictal Polyspikes/Frontal Fast	Maximum Spike Wave Duration (sec)	Runs >3 Sec (#/ hour)	Focal Findings	Seizures Induced in Sun	HV	PPR	Change from EEG off Med
ZNS	6.5	5/2	7.6	+ BF/BP	−	2	0	−	.	Gen SW	−	Imp
TPM&LEV	T: 1.5 L: 42	6/1	15.0	−	−	0	0	−	.	−	−	NL
LEV	30	1/1	7.3	+ BF	+ BF	15	36	R SW	.	R Slow	+	.
	40	4/2	7.9	+ BF/BP	+ BF	15	26	−	+ SI	Gen SW	+	NC
	57	7/1	11.3	+ BF	+ BF	5	<1	−	+ SI	.	.	NC
	83	13/2	11.6	+	+	10	1	−	+ SI	−	+	NC
ETX & LEV	E: 40 L: 43	13/3	13.0	−	−	3	3	−	.	+ S/PSW	+	IMP
	E: 27 L: 37	13/4	15.5	+	+	2	0	−	+ SI	−	+	NC
LTG	3.3	4/3	8.5	+ BF/BP	+ BF/BP	20	30	−	+ SI	+ ABS	−	NC
	3.5	9/2	6.3	+ BF/BP	−	9	4	−	.	−	−	NC
	5	12/2	9.4	−	−	<1	0	Fragments	− SI	.	.	Imp
LTG & VPA	L: 3.5 V: 25	9/3	6.7	−	−	0	0	−	.	−	−	NL
VPA	25	2/2	9.2	−	−	0	0	−	.	−	−	NL
	22	5/3	7.8	+BF	+ BF	6	10	−	.	−	−	NC
	44	5/4	8.3	+ BP	+ BP	<0.5	0	−	− SI	−	−	Imp
	26	7/2	15.5	−	−	0	0	−	.	−	−	NL
	25	8/1	6.1	+	−	.	.	C3 spikes	.	.	+	.

Med	Dose (mg/kg/d)	ID#/EEG#	Age (yr)	Interictal 3–4Hz Spikes	Interictal Polyspikes/Frontal Fast	Maximum Spike Wave Duration (sec)	Runs >3 Sec (#/ hour)	Focal Findings	Seizures Induced in Sun	HV	PPR	Change from EEG off Med
	20	8/2	8.2	+ L>R	+ L>R	.	.	C3 spikes	.	.	+	.
	13	10/2	16.1	+ BP	+	6	<1	Fragments	−SI	−	+	Imp

BF=bifrontal; BP= biposterior; Cz=central; C3=left central; ETX=ethosuximide; HV=hyperventilation; Imp=improved; LTG=lamotrigine; LEV=levetiracetam; mg/kg/d=milligram/kilogram/day; Med=antiseizure medication(s); NC=no change; NL=normalized; PPR=photoparoxysmal response; PSW=polyspike waves; Sec = second; S=spike; SI=self-induced; SW=spike waves; TPM=topiramate; VPA=valproate; ZNS=zonisamide; + = present; − = absent;. = not tested