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Research Article

Stereo-EEG features of temporal and frontal lobe seizures with loss of consciousness

Nuria Campora[1,2,*], Juan Pablo Princich[1,2], Alejandro Nasimbera[1,2], Santiago Cordisco[2], Manuela Villanueva[2], Silvia Oddo[1,2], Brenda Giagante[1], Silvia Kochen[1,2]

[1]Neuroscience Department, El Cruce Hospital, Florencio Varela, Argentina
[2]Studies in Neuroscience and Complex Systems (ENyS), CONICET, Florencio Varela, Buenos Aires 1888, Argentina

*Corresponding author. Av Calchaqui 5401, Florencio Varela, Buenos Aires, Argentina. E-mail: nuriacampora@yahoo.com.ar

Abstract

The loss of consciousness (LOC) during seizures is one of the most striking features that significantly impact the quality of life, even though the neuronal network involved is not fully comprehended. We analyzed the intracerebral patterns in patients with focal drug-resistant epilepsy, both with and without LOC. We assessed the localization, lateralization, stereo electroencephalography (SEEG) patterns, seizure duration, and the quantification of contacts exhibiting electrical discharge. The degree of LOC was quantified using the Consciousness Seizure Scale. Thirteen patients (40 seizures) with focal drug-resistant epilepsy underwent SEEG. In cases of temporal lobe epilepsy (TLE, 6 patients and 15 seizures), LOC occurred more frequently in seizures with mesial rather than lateral temporal lobe onset. On the other hand, in cases of frontal lobe epilepsy (7 patients; 25 seizures), LOC was associated with pre-frontal onset, a higher number of contacts with epileptic discharge compared to the onset count and longer seizure durations. Our study revealed distinct characteristics during LOC depending on the epileptogenic zone. For temporal lobe seizures, LOC was associated with mesial seizure onset, whereas in frontal lobe epilepsy, seizure with LOC has a significant increase in contact showing epileptiform discharge and a pre-frontal onset. This phenomenon may be correlated with the broad neural network required to maintain consciousness, which can be affected in different ways, resulting in LOC

Keywords: consciousness; stero-EEG; epilepsy

Introduction

The term "consciousness" has its origin in the Latin word "conscıentıa" which means "knowledge shared with others" (Fabbro et al. 2019). Definitions of consciousness vary across different academic fields, such as philosophy and psychology. In neurosciences, consciousness is generally defined as a wakeful state characterized by the ability to perceive, interact, and communicate with the environment and others in the integrated manner that wakefulness normally implies (Gloor 1986, Dennett 1995, Searle 2000, Baars 2005, Monaco et al. 2005, Ali et al. 2012, Beniczky et al. 2016). Although much has been written about consciousness, it continues to be a topic of ongoing discussion (Searle 2000, Tononi 2005, Ali et al. 2012, Campora and Kochen 2016).

Epilepsy is a prevalent disorder that affects approximately 1 in 200 people (Espinosa-Jovel et al. 2018) within the population. The loss of consciousness (LOC) represents one of the most dramatic clinical manifestations of seizures, including both generalized and focal seizures. However, the underling mechanisms behind LOC are not fully understood. Despite conceptual uncertainties, the assessment of consciousness is undeniably fundamental in epilepsy (Johanson et al. 2011, Beniczky et al. 2016). Previously, our group developed a survey which includes the evaluation of memory and subjective perceptions during seizures (Campora and Kochen 2016). The survey aims to provide an assessment of different components of consciousness. Furthermore, in previous studies, our group analyzed intracerebral signals also in both temporal and frontal epilepsy using quantified methods. We observed a dramatic drop of the phase-locking value before seizure onset (SO) within the epileptogenic zone (EZ), indicating the synchronization of intracerebral signals across different brain regions during seizures (Cámpora et al. 2019). We also characterized the time-locked index to quantify the degree of harmonicity between frequency bands during seizures (Dellavale et al. 2020). Subsequently, we studied the relationship between frequency bands during seizures and identified an increase in variance after seizure onset, with the predominance of power in the theta and alpha bands in seizures with LOC (Capitán et al. 2019, Cámpora et al. 2019, Maidana Capitán et al. 2020).

Recent studies in the field of consciousness have gathered data regarding the connection between intracerebral signals

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(Bartolomei and Naccache 2011). One prominent theory is the Global Workspace Theory, which proposes that conscious processing results from coherent neuronal activity between widely distributed brain regions, with a particular emphasis on fronto-parietal-associative cortices.

The objectives of this study was to analyse and describe the intracerebral electrophysiological activity during focal seizures associated to LOC. This research aims to contribute in a better understanding of the neural network dynamics at play during loss of consciousness.

Methodology

This study received approval from the Ethics Review Board of El Cruce Hospital, in accordance with the principles outlined in the Declaration of Helsinki. Prior to participation, all patients provided written informed consent that had been approved by the ethics board, following standard procedures.

We selected a total of 13 adult patients diagnosed with drug-resistant focal epilepsy who required SEEG as part of their clinical evaluation. All patients underwent a detailed medical record and neurological examination, neuropsychological testing, routine magnetic resonance image (MRI), scalp EEG, and SEEG.

Assessment of clinical LOC

To evaluate the level of consciousness, we utilized the Consciousness Seizure Scale (CSS) (Arthuis et al. 2009, Bartolomei and Naccache 2011). This scale assesses consciousness through eight items, which were rated independently by two epileptologists (N.C. and S.K.). Items 1–7 were scored 0 or 1, while the eighth item was scored from 0 to 2. This scoring system results in a possible total score ranging from 0 to 9. Based on the total score, we categorized patients into three groups: those without LOC (≤1), those with an intermediate LOC (2–5), and those with LOC (≥6).

Evaluation of epileptogenic zone

In our study, we adopted the theoretical concept (Kahane et al. 2006) that defines the epileptogenic zone (EZ) as the region from which the epileptogenic discharge originates and immediately spreads. The concept is based on various factors including the localization of the onset of the ictal discharge, the immediate propagation pattern, ictal semiology, interictal discharge, and brain MRI findings.

Electrode implantation

We utilized depth electrodes (Ad Tech) which came in two configurations: (i) 8 or 10 platinum contacts with inter-contact center to center distance of 5 or 10 mm, contact length of 2.4 mm, and a diameter of 1.1 mm, or (ii) 9 platinum contacts with 3 mm distance between the first and the second contact and 6 mm inter-electrode distance from the second to the last and the electrode diameter of 1.28 mm. Each electrode was identified by a letter of the alphabet, and individual contacts within an electrode were typically labeled with numbers starting from the deepest to the base.

As an indicator of spatial extent of ictal recruitment, we considered the number of intracerebral contacts involved in the epileptogenic discharge. While this measure has its limitations, it provides valuable insight into the approximate cerebral tissue involved in the neuronal network.

Anatomic localization of electrode positions

The placement of electrodes during surgery was determined by pre-established hypotheses regarding the likely site of SO and propagation. These hypotheses were based on a combination of electroencephalography, clinical, and MRI data. Therefore, the positions of the electrodes were not standardized. In Fig. 1, we have depicted all the electrodes that were analyzed, along with their respective localization.

Intracranial electroencephalography recordings

We recorded SEEG signals using Cervello 1.04.200 software at a sampling rate of 2000 Hz. Identification of the SO was performed independently by two epileptologists (N.C. and S.K.).

The SO was recognized as the initial SEEG changes characterized by sustained rhythmic discharges or repetitive spike-wave discharges that could not be attributed to changes in the patient's

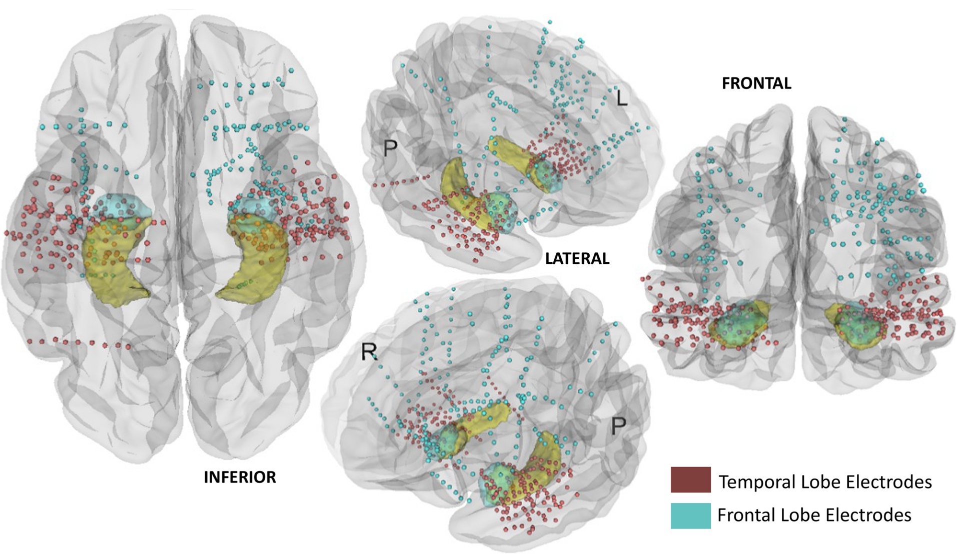

Figure 1. Inferior, lateral, and frontal view of all electrodes from 13 patients normalized and overlaid on a template 3D brain model. Contacts were labeled using iElectrodes open-Source Toolbox (https://www.nitrc.org/projects/ielectrodes/), extracted from post-implant CT scans. Hippocampus and amygdala are also depicted in the model for anatomical reference

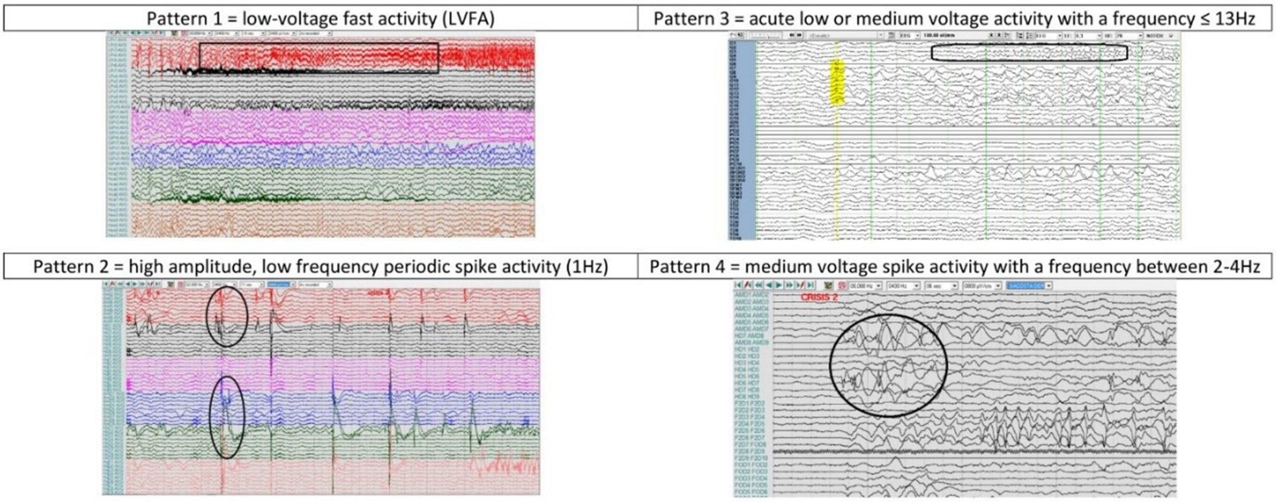

Figure 2. Examples of stereoelectroencefalography patterns

state. These changes resulted in habitual seizure symptoms consistent with those reported in previous studies. The SO zone was defined based on the contacts where these early ictal SEEG changes were observed.

Periods of interest

We analyzed the seizure onset defined as the first 5 s following the initial change observed in SEEG and maximal propagation, which refers to the maximum number of electrode contacts involved during a seizure.

The determination of the onset of LOC was rigorously established by two independent evaluators (N.C. and S.K.). This determination was made when the patient was interacting with a family member or technician but ceased to respond appropriately to the technician, who initiated the application of the scales previously described. Additionally, we presented the seizure video to the patient, and they confirmed the moment when LOC occurred. In cases where there was uncertainty in determining this moment, those cases were excluded from the analysis for this paper (N = 8).

SEEG signals

We categorized SEEG patterns into four types based on previous reports (Perucca et al. 2014) (Fig. 2):

Pattern 1: Low-voltage fast activity (LVFA)

Pattern 2: High-amplitude, low-frequency periodic spike activity (1 Hz)

Pattern 3: Acute low or medium voltage activity with a frequency ≤ 13 Hz

Pattern 4: Medium voltage spike activity with a frequency between 2 and 4 Hz

We excluded seizures with secondary generalization or those induced by electrical stimulation. Electrodes with significant artifacts were disregarded. Seizures with poor quality videos, evaluations conducted too late, or cases where more than 2 CSS items were not scored were also excluded. The SEEG data were independently analyzed by two reviewers (N.C. and S.K.), who were blinded to clinical information. Consensus was reached through discussion.

Statistical analysis

Qualitative variables were analyzed using Chi test or Fisher test as appropriate. Quantitative variables were analyzed using *t*-test and ANOVA. Differences in the number of electrode contacts with epileptiform discharge at the ictal onset and at the beginning of LOC were analyzed using paired samples *t*-test. Statistical significance levels were set at $P \leq 0.05$. The SPSS 20.0 for Windows was used for all the statistical analyses.

RESULTS

Demographic and clinical data

We included a total of 13 patients (56 seizures) who were considered as surgical candidates at the Hospital El Cruce, between 2012 and 2017.

The average age of the patient was 28.25 ± 6.86 years-old, the average age at first seizure was 12.42 ± 6.5 years-old and the average time of epilepsy was 16.67 ± 9.23 years. Of the included patients, 41.7% were female.

MRI findings indicated that 41.7% of the patients had normal MRI results, 25% (three) exhibited focal cortical dysplasia, 16.7%

Table 1. Demographic characteristics

	Temporal lobe epilepsy (6 patients, 15 seizures)	Frontal epilepsy (7 patients, 25 seizures)	P
Seizures with LOC (N)	8	19	
Seizures without LOC (N)	7	6	
Aged (average ± SD, years)	28 ± 7.53	29.71 ± 7.52	0.679
Sex female/male	4/2	2/5	0.22
	MRI		
Normal (5/13)	3	2	
Cortical retraction (2/13)	1	1	
Low-grade tumor (1/13)	1	0	
Hippocampal alteration (1/13)	1	0	
Focal cortical dysplasia (4/13)	0	4	

SD = standart desviation, P = patients, sz = seizures.

(two patients) displayed cortical retraction and 8.3% (one patient) had a low-grade tumor. On average, the electrodes implanted were 5.67 ± 2.23 and the contacts implanted were 43.42 ± 8.05 (Tables 1 and 2).

Out of the 40 seizures analyzed, 13 seizures were without LOC and 27 with LOC.

Based on the EZ localization, we divided patients in two groups: temporal lobe epilepsy (TLE, 6 patients) and frontal epilepsy (FLE, 7 patients). There were no significant demographic differences between these two groups (Table 1).

Temporal epilepsy

Seven seizures without alteration of consciousness (AOC), 4 profound AOC and 3 with moderate AOC (Fig. 1).

Duration of seizure: Although seizures with profound AOC were longer 4638 ± 1125 s versus 2986 ± 9.37 s without AOC), there was no significant difference ($P = 0.692$).

Topography: At ictal onset, hippocampal involvement was significantly greater in seizures with profound AOC (87.5%), compared to seizures without AOC (12.5%) ($P = 0.035$). This is the same as the time of propagation of activity when AOC (profound AOC 100% versus without AOC 28.6%, $P = 0.007$) (Fig. 1).

Cortical size: The median of contact at the beginning of the seizure was 6.38 ± 3.57, and at the moment of AOC was 8.67 ± 3.58. We did not find relationship between number of contact and the grade of LOC (P=0.12).

Lateralized: Seizure with *profound AOC* began 25% at the left hemisphere and 75% at the right hemisphere. At the time of AOC, 75% had left involvement (50% bilateral).

The seizure *without LOC* began at right hemisphere 71.4% and left hemisphere 28.6%, without contralateral propagation. iEEG patterns: At seizure-onset, the most frequent pattern was 4 both in profound AOC (62.5%) and without AOC (57.1%).

At the time of AOC, the most frequent pattern was Pattern 3 (55.6%), both in without AOC (57.1%) and profound AOC (50%).

Temporal lobe epilepsy

($N = 6$, seizures = 15): Within this subgroup, eight seizures were with LOC and seven seizures were without LOC.

Localization of epileptogenic discharge

In terms of localization, a substantial majority (87.5%, 7/8) of seizures with LOC originated within the hippocampus, with only one case originating in the lateral temporal cortex. Importantly, across all instances, there was consistent engagement of the hippocampus in the propagation of ictal activity. Among seizures without LOC, 71.4% (5/7) initiated in the lateral temporal cortex, while 28.6% (2/7) began in the hippocampus and remained in the same region throughout all seizures.

So, seizures with LOC were significantly more likely to involve the hippocampus at any point during the seizure (8/8 seizures) compared to those without LOC (2/7 seizures, Fisher test $P = 0.007$).

Number of contacts with epileptic discharge

Seizures with LOC showed no significant change between electrical onset (8.38 ± 4.03) and maximal propagation (9.38 ± 3.46; *t*-paired test $P = 0{,}227$).

In *seizures without LOC*, the number of contacts with epileptogenic activity remained stable during all the seizures (seizure onset with 6.43 ± 2.82 and maximal propagation with 6.71 ± 3.4; *t*-paired test, $P = 0.35$).

We did not find any relation between the amount of contacts and LOC.

Seizure duration

Seizures *without LOC* had an average duration of 2986 ± 9.37 s and those *with LOC* lasted for 4638 ± 1125 s. Although seizures with LOC were longer, there was no significant difference (t-test, $P = 0.692$).

LOC typically began within the first 10 s from seizure onset, with an average onset time of 7.38 ± 4.56 s.

Hemisphere compromised

In terms of laterality, six (75%) of the seizures with LOC originated in the right temporal lobe and only one remained confined in this hemisphere, while 5/6 propagated to the left hemisphere. Two seizures (25%) originated in the left temporal lobe and stayed within the same hemisphere during propagation. Thus, regarding discharge spread, 87.5% (7/8) of seizures involved the left temporal lobe, with only one seizure remaining confined to the right hemisphere. Seizures that began in the left temporal lobe stayed in the same hemisphere throughout the seizure. In seizures without LOC, 71.5% (5/7) began in the right temporal lobe, and 28.5% (2/7) in the left temporal lobe, with the activity staying within the same lobe during all seizures.

Table 2. Data of the patients

Patient	Gender	EZ	Side	Electrodes (amount)	Contacts (amount)	Number of analyzed seizures	Engel	Time from surgery (years)
Patient 1	M	FL	L	1	44	4	I	6
Patient 2	M	FL	L	5	38	6	III	4
Patient 3	M	FL	L	5	35	8	Surgery pending	N/A
Patient 4	M	FL	L	6	38	3	III	2
Patient 5	F	FL	R	5	44	8	II	6
Patient 6	M	FL	L	7	43	8	I	4
Patient 7	F	FL	R	5	28	1	I	1
Patient 8	M	TL	L	6	43	3	II	4
Patient 9	M	TL	L	6	45	4	I	3
Patient10	F	TL	R	11	67	4	II	3
Patient 11	F	TL	R	5	40	1	II	6
Patient 12	F	TL	R	5	40	5	II	2
Patient 13	F	TL	R	6	44	2	I	3

M: male, F: female, FL: frontal lobe, TL temporal lobe, L: left, R: right, PET: positron emission tomography, N/A: not applicable.

SEEG patterns

At seizure onset, those with LOC had pattern 4 in 5/8, pattern 3 in 2/8, and pattern 2 in 1/8. During propagation, the patterns were pattern 3 in 4/8, pattern 1 in 3/8, and pattern 4 in 1/8. We did not find statistical significance (pattern onset 3 and 4, Fisher $P = 0.121$).

In seizures without LOC, the onset pattern was pattern 3 in 4/7, and patterns 1, 2, and 4 had 1/7 each of them. The same patterns remained consistent through the seizure.

Frontal epilepsy

($N = 7$, seizures = 25): Within this subgroup, there were six seizures without LOC and 19 with LOC.

Localization of epileptogenic discharge

In seizures with LOC, the SO was prefrontal in 78.9% (15/19) of cases, while it was the motor/premotor/insular cortex in 21.1% (4/19) of cases. Regarding the propagation of activity in the group with LOC, the prefrontal cortex was engaged in 94.7% (18/19) of seizures, with only one instance retaining activity within the motor/premotor/insular cortex. In the group without LOC, 83.3% (5/6) had the onset in the motor/premotor/insular cortex, and one seizure began in the prefrontal cortex (Fisher, $P = 0.001$). Activity remained in the same cortex throughout all seizures. All patients had unilateral homolateral hippocampal electrode, and we did not identify any epileptiform activity in them.

Number of contacts with epileptic discharge

In seizures with LOC, the amount of electrodes with epileptic discharge at seizure onset was 3.62 ± 1.6 and at maximal propagation, it was 7.38 ± 3.66 (*t*-paired test, $P = 0.007$).

The amount of contacts with epileptiform activity remained similar throughout *seizures without LOC* (seizure onset 5 ± 1.89 versus maximal propagation 5.67 ± 1.94, $P = 0.178$).

The amount of contacts with epileptiform discharge increased in seizure with LOC compared to the seizure onset (*t*-paired test, $P = 0.007$).

Seizure duration

The average duration of *seizures without LOC* was 2657 ± 7.8 s, while those *with LOC* had an average duration of 94.15 ± 12.11 s. Seizures with LOC were significantly longer than those without LOC (*t*-test, $P = 0.002$).

In frontal seizures, LOC typically began after 10 seconds from frontal seizure onset, with an average of 37.29 ± 25.21 s. When compared to temporal seizures, where LOC occurs in 7.38 ± 4.56 s in temporal seizures, we can determine that LOC begins earlier in temporal seizures than in frontal seizures (assessed using an Independent samples ttest, with the test Levene yielding a value of 0.010 and a *P*-value of 0.04).

Hemisphere compromised

All patients had only unilateral electrodes, which limited the analysis of laterality in LOC. Then we describe seizures from each hemisphere separately.

Seizures originating from right hemisphere showed that 7/9 were associated with LOC, while 2/9 occurred without LOC.

Most seizures originating from the left hemisphere have LOC (12/16), while 4/16 occurring without LOC.

Although we could not analyze the relationship between LOC and laterality, it is notable that most seizures originating from the left hemisphere were associated with LOC.

SEEG patterns

At seizure onset, those with LOC had pattern 3 in 10/25 seizures, pattern 1 in 8/25, and pattern 2 in 1/25. During propagation, in seizures with LOC, the patterns were pattern 1 in 7/25, pattern 3 in 6/25, and pattern 2 and 4 in 3/25 each.

In frontal seizures without LOC, the onset pattern was pattern 3 in 5/6 and pattern 2 in 1/6, and these patterns remained consistent throughout the seizure.

We did not find relation between SEEG patterns and LOC.

Discussion

The international classification of epilepsy identified LOC as a cornerstone for distinguishing the main categories of focal seizures (Angeles 1981, Berg et al. 2010, Beniczky et al. 2016, Fisher et al. 2017). It emphasizes an essential characteristic that significantly impacts the quality of life of patients. Seizures characterized by LOC provide a unique model for studying reversible changes of conscious in vivo, particularly in the context of intracerebral EEG data. In clinical practice, consciousness is generally defined as a state of wakefulness with the ability to perceive, interact, and communicate with the environment and others in the integrated manner that wakefulness normally implies (Kahane et al. 2006).

In this study, we included patients with temporal and frontal epilepsies who were candidate for surgery. We analyzed LOC and the characteristics of intracerebral electroencephalographic signals. Various theories were proposed regarding LOC (Yamauchi 1998, Tononi 2008, Yu and Blumenfeld 2009, Bartolomei and Naccache 2011, Danielson et al. 2011). One theory previously applied to epilepsy is the Global Workspace (GW) theory (Bartolomei and Naccache 2011). GW explains LOC as oversyncronization between cortical areas and deep structure. This theory was previously applied both to temporal and frontal epilepsies, where it identified the significant roles of the hippocampus, prefrontal and parietal cortical areas, and the thalamus (Arthuis et al. 2009), (Bonini et al. 2016).

In TLE, LOC was more common when the hippocampus was involved at any time during the seizure, consistent with previous reports (Lux et al. 2002, Englot et al. 2010). There is consensus of the important role of the hippocampus in consciousness. In this work, we were unable to establish the involvement of other extratemporal structures, such as parietal cortex and thalamus, which were previously identified to be affected during LOC in temporal epilepsy (Arthuis et al. 2009, Lambert et al. 2012).

We did not find a relation between laterality and LOC. The relationship between laterality and consciousness remains a subject of debate (Lux et al. 2002, Bauerschmidt et al. 2013). Some reports described a greater occurrence LOC in left-sided seizures (Gazzaniga 1988), while others identified bilateral temporal involvement (Gloor 1980, Munari 1980, Bancaud and Talairach 1993). Some studies did not find any clear relationship (Lux et al. 2002). Additionally, it is important to consider that language integrity is often necessary for evaluation of consciousness, which can give the left hemisphere more prominence (Blumenfeld 2012).

In the frontal epilepsy group, we observed the involvement of the prefrontal area at seizure onset or during propagation in seizures with LOC. This finding aligns with the established role of the prefrontal cortex in the processing of consciousness. Several studies identified prefrontal as well as parietal areas as main cortical regions involved in consciousness (Laureys et al. 1999, Di Perri et al. 2014, Bonini et al. 2016). None of the studied frontal seizures involved the hippocampus. On the other hand, frontal seizures with spared consciousness had onset mainly in pre-motor/motor (non-prefrontal) areas. This is consistent with previous reports that have suggested that the motor cortex is not functionally involved in LOC (Blumenfeld et al. 2004a, 2004b, Bonini et al. 2014).

Although the number of contacts with epileptic activity has a lot of limitations, we found a significant increase in their firing during epileptic propagation in frontal seizures with LOC. We could hypothesize that LOC will occur during a seizure when a critical volume of cortical tissue within essential areas is involved by a hypersynchronized discharge, disrupting normal local functioning and/or deactivating distant structures necessary for the regulation of cognitive processes and maintaining vigilance. This is aligns with the GW theory (Bartolomei et al. 2014, Bonini et al. 2016).

Frontal seizures with LOC were observed to be longer than those without LOC, a difference not seen in temporal epilepsy. Furthermore, the onset of LOC in frontal lobe epilepsy occurred later in seizure compared to TLE.

A limitation of this study is the lack of evaluation of non-epileptogenic electrical changes, such as slow waves. Previous studies have related a slowdown in activity in bifrontal and ipsilateral parietal areas in TLE (Blumenfeld et al. 2004b) with LOC. Additionally, the sample sizes were relatively small, and these results should be further verified and investigated in larger sample sizes in future studies.

In conclusion, temporal seizures with LOC are characterized by hippocampal involvement at any time of the seizure. In frontal epilepsy, seizures with LOC are characterized by the involvement of prefrontal areas, an increase in contacts with epileptogenic discharge, longer duration, and no hippocampal compromise.

Future studies applying other methodologies, both analogical and quantitative, for signal analysis are necessary to characterize functional connectivity between spatially distributed regions and to explore the pathophysiological mechanisms during LOC.

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Conflict of interest

We declare we have no competing interests.

References

Ali F, Rickards H, Cavanna AEJE, Behavior. The assessment of consciousness during partial seizures. *Epilepsy Behav* 2012;**23**:98–102.

Angeles DJE. Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia* 1981;**22**:489–501.

Arthuis M, Valton L, Regis J *et al.* Impaired consciousness during temporal lobe seizures is related to increased long-distance cortical–subcortical synchronization. *Brain* 2009;**132**:2091–101.

Baars BJ. Global workspace theory of consciousness: toward a cognitive neuroscience of human experience. *Prog Brain Res* 2005;**150**:45–53.

Bancaud J, Talairach J, Paris. Crises du lobe temporal. 1993.

Bartolomei F, McGonigal A, Naccache LJE, Behavior. Alteration of consciousness in focal epilepsy: the global workspace alteration theory. *Epilepsy Behav* 2014;**30**:17–23.

Bartolomei F, Naccache L. The global workspace (GW) theory of consciousness and epilepsy. *Behav Neurol* 2011;**24**:67–74.

Bauerschmidt A, Koshkelashvili N, Ezeani CC *et al.*, Behavior. Prospective assessment of ictal behavior using the revised Responsiveness in Epilepsy Scale (RES-II). *Epilepsy Behav* 2013;**26**: 25–8.

Beniczky S, Neufeld M, Diehl B *et al.* Testing patients during seizures: a European consensus procedure developed by a joint taskforce of the ILAE–Commission on European Affairs and the European Epilepsy Monitoring Unit Association. *Epilepsia* 2016;**57**: 1363–8.

Berg AT, Berkovic SF, Brodie MJ *et al.* Revised terminology and concepts for organization of seizures and epilepsies: report of the ILAE Commission on Classification and Terminology, 2005–2009. Wiley Online Library *Epilepsia* 2010;**51**:676–85.

Blumenfeld H. Impaired consciousness in epilepsy. *Lancet Neurol* 2012;**11**:814–26.

Blumenfeld H, McNally KA, Vanderhill SD *et al.* Positive and negative network correlations in temporal lobe epilepsy. *Cereb Cortex* 2004a;**14**:892–902.

Blumenfeld H, Rivera M, McNally K *et al.* Ictal neocortical slowing in temporal lobe epilepsy. *Neurology* 2004b;**63**:1015–21.

Bonini F, Lambert I, Wendling F *et al.* Altered synchrony and loss of consciousness during frontal lobe seizures. *Clin Neurophysiol* 2016;**127**:1170–5.

Bonini F, McGonigal A, Trébuchon A *et al.* Frontal lobe seizures: from clinical semiology to localization. *Epilepsia* 2014;**55**:264–77.

Campora N, Kochen S, Behavior. Subjective and objective characteristics of altered consciousness during epileptic seizures. *Epilepsy Behav* 2016;**55**:128–32.

Cámpora NE, Mininni CJ, Kochen S *et al.* Seizure localization using pre ictal phase-amplitude coupling in intracranial electroencephalography. *Sci Rep* 2019;**9**:1–8.

Capitán M, Cámpora N, Kochen S *et al.* Principal component analysis for seizure characterization in EEG signals. *Biological cibernetics* 2019;**144**:461–71.

Danielson NB, Guo JN, Blumenfeld H. The default mode network and altered consciousness in epilepsy. *Behav Neurol* 2011;**24**:55–65.

Dellavale D, Urdapilleta E, Cámpora N *et al.* Two types of ictal phase-amplitude couplings in epilepsy patients revealed by spectral harmonicity of intracerebral EEG recordings. *Clin Neurophysiol* 2020;**131**:1866–85.

Dennett D. La consciencia explicada. *Paidós Madrid*. 1995.

Di Perri C, Stender J, Laureys S *et al.*, Behavior. Functional neuroanatomy of disorders of consciousness. *Epilepsy Behav* 2014;**30**:28–32.

Englot DJ, Yang L, Hamid H *et al.* Impaired consciousness in temporal lobe seizures: role of cortical slow activity. *Brain* 2010;**133**:3764–77.

Espinosa-Jovel C, Toledano R, Aledo-Serrano Á *et al.* Epidemiological profile of epilepsy in low income populations. *Seizure* 2018;**56**:67–72.

Fabbro F, Cantone D, Feruglio S *et al.* Origin and evolution of human consciousness. *Prog Brain Res* 2019;**250**:317–43.

Fisher RS, Cross JH, D'souza C *et al.* Instruction manual for the ILAE 2017 operational classification of seizure types. *Epilepsia* 2017;**58**:531–42.

Gazzaniga MS. Brain modularity: towards a philosophy of conscious experience. *Consciousness of Contemporary Science*. Oxford University Press, 1992.

Gloor P. Loss of consciousness in temporal lobe seizures: observations obtained with stereotaxic depth electrode recordings and stimulations. In: Canger R, Penry JK and Angeleri F (eds.), *Advances in Epileptology: XI Epilepsy International Symposium*, Raven Press, 1980, 349–53.

Gloor PJE. Consciousness as a neurological concept in epileptology: a critical review. *Epilepsia* 1986;**27**:S14–S26.

Johanson M, Valli K, Revonsuo A. How to assess ictal consciousness? *Behav Neurol* 2011;**24**:11–20.

Kahane P, Landré E, Minotti L *et al*. The Bancaud and Talairach view on the epileptogenic zone: a working hypothesis. *Epileptic Disord* 2006;**8**:16–26.

Lambert I, Arthuis M, McGonigal A *et al*. Alteration of global workspace during loss of consciousness: a study of parietal seizures. *Epilepsia* 2012;**53**:2104–10.

Laureys S, Goldman S, Phillips C *et al*. Impaired effective cortical connectivity in vegetative state: preliminary investigation using PET. *Neuroimage* 1999;**9**:377–82.

Lux S, Kurthen M, Helmstaedter C *et al*. The localizing value of ictal consciousness and its constituent functions: a video-EEG study in patients with focal epilepsy. *Brain* 2002;**125**:2691–8.

Maidana Capitán M, Cámpora N, Sigvard CS *et al*. Time-and frequency-resolved covariance analysis for detection and characterization of seizures from intracraneal EEG recordings. *Biol Cybern* 2020;**114**:461–71.

Monaco F, Mula M, Cavanna AEJE, Behavior. Consciousness, epilepsy, and emotional qualia. *Epilepsy Behav* 2005;**7**:150–60.

Munari C. Impairment of consciousness in temporal lobe seizures: a stereoelectroencephalographic study. In: Naquet R, Masland RL, Porter RJ, Mattson RH, Booker H, Rafal RD (eds.), *Advances in Epileptology: XIth Epilepsy International Symposium*. United State: Raven Press, 1980, 111–4.

Perucca P, Dubeau F, Gotman JJB. Intracranial electroencephalographic seizure-onset patterns: effect of underlying pathology. *Brain* 2014;**137**:183–96.

Searle JRJA. Consciousness. *Annu Rev Neurosci* 2000;**23**:557–78.

Tononi GJ. Consciousness, information integration, and the brain. *Prog Brain Res* 2005;**150**:109–26.

Tononi G. Consciousness as integrated information: a provisional manifesto. *Biol Bull* 2008;**215**:216–42.

Yamauchi T. Impairment of consciousness during epileptic seizures with special reference to neuronal mechanisms. *Epilepsia* 1998;**39**:16–20.

Yu L, Blumenfeld H. Theories of impaired consciousness in epilepsy. *Ann N Y Acad Sci* 2009;**1157**:48–60.

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