



# Ictal and postictal semiology in patients with bilateral temporal lobe epilepsy

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## ABSTRACT

Bilateral temporal lobe epilepsy is characterized by evidence of seizure onset independently in both temporal lobes. The main aim of the present study was to determine whether patients with evidence of independent bilateral temporal lobe epilepsy (biTLE) can be identified noninvasively on the basis of seizure semiology analysis. Thirteen patients with biTLE, as defined by invasive EEG, were matched with 13 patients with unilateral temporal lobe epilepsy (uniTLE). In all 26 patients, the frequency of predefined clusters of ictal and periictal signs were evaluated: ictal motor signs (IMs), periictal motor signs (PIMs), periictal vegetative signs (PIVs), the frequency of early orolimentary automatisms (EOAs), and the duration of postictal unresponsiveness (PU). Some other noninvasive and clinical data were also evaluated. A lower frequency of IMs was noted in the group with biTLE (patients = 46.2%, seizures = 20.7%) than in the group with uniTLE (patients = 92.3%, seizures = 61.0%) ( $p = 0.030$ ;  $p < 0.001$ , respectively). The individual IMs average per seizure was significantly lower in the group with biTLE (0.14; range = 0–1.0) than in the group with uniTLE (0.80; range = 0–2.6) ( $p = 0.003$ ). Postictal unresponsiveness was longer than 5 min in more patients (75.0%) and seizures (42.9%) in the group with biTLE than in the group with uniTLE (patients = 30.8%, seizures = 18.6%) ( $p = 0.047$ ;  $p = 0.002$ ). The frequency of EOAs, PIMs, PIVs, and other clinical data did not differ significantly. There is a lower frequency of ictal motor signs and longer duration of postictal unresponsiveness in patients with biTLE.

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## 1. Introduction

Bilateral temporal lobe epilepsy or also known as bitemporal epilepsy is often vaguely characterized by the existence of independent seizure-onset zones in both temporal lobes. Bitemporal epilepsy is not clearly defined and is usually suspected when independent bilateral temporal seizures are recorded in scalp EEG. Bitemporal epilepsy has been defined by depth electrodes, as clear clinical and scalp EEG differences that could noninvasively distinguish bitemporal epilepsy from unilateral temporal lobe epilepsy have not been established [1]. Patients with bitemporal epilepsy are generally considered to be poorer surgery candidates than patients with unilateral temporal lobe epilepsy [2–5]. Seizure semiology is an important part of the presurgical assessment of epilepsy surgery candidates. To the best of our knowledge, the seizure semiology in

bitemporal epilepsy and that in unilateral temporal lobe epilepsy have not been compared in detail. The main goal of this study was to reveal potential differences between the patients with bitemporal epilepsy and those with unilateral temporal lobe epilepsy in terms of history data, semi-invasive EEG findings, and seizure semiology.

## 2. Methods

### 2.1. Group definition

We reviewed all of the patients with temporal lobe epilepsy (TLE) who underwent invasive video-EEG at one of two epilepsy centers in Czech Republic: the Brno Epilepsy Center at St. Anne's University Hospital between 1999 and 2012 and the Epilepsy Center Motol at the University Hospital Motol in Prague between 2006 and 2012. We defined the following criteria for identifying the patients in the group with bitemporal epilepsy (biTLE): independent bitemporal seizure origin, defined on the basis of invasive EEG as (1) spontaneous clinical seizures arising independently from both temporal lobes (electrographic

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seizures were not included in this analysis because their clinical significance is not yet clearly determined [6]) and/or (2) habitual complex partial seizures (CPSS) elicited by the electrical stimulation of the temporal lobe contralateral to the spontaneous seizures. For comparison purposes, we formed a control group of patients with unilateral temporal lobe epilepsy (the group with uniTLE). The subjects in this group were patients with uniTLE who were completely seizure-free for at least two years after epilepsy surgery. Patients from the group with uniTLE were matched with patients from the group with biTLE in terms of age at the onset of epilepsy, age at evaluation, duration of epilepsy, and gender. We selected a group of 26 patients who fulfilled these matching criteria (13 in the group with biTLE and 13 in the group with uniTLE). The study was approved by the Ethics Committee of St. Anne's University Hospital.

## 2.2. Presurgical evaluation

All 26 patients underwent a comprehensive presurgical evaluation, including detailed history and neurological examination, neuropsychological testing, magnetic resonance imaging (MRI), and scalp video-EEG monitoring. Bilateral carotid sodium amobarbital/methohexital testing was performed in 11 patients of the group with biTLE and 12 patients of the group with uniTLE; unilateral testing was available in one patient of the group with biTLE. Interictal and/or ictal single-photon emission computed tomography (SPECT) was performed in 10 patients of the group with biTLE and in seven patients in the group with uniTLE. Fluorodeoxyglucose positron emission tomography (FDG-PET) was performed in 12 patients from the group with biTLE and in seven patients from the group with uniTLE.

## 2.3. Scalp EEG, semi-invasive EEG, and invasive EEG procedures

In all 13 patients in the group with biTLE, invasive video-EEG monitoring was a part of the presurgical evaluation. In the patients from the group with uniTLE, depth EEG was performed in four patients because their noninvasive data were insufficient to proceed directly to surgery. Evidence indicates that all 26 patients had mesial temporal lobe epilepsy: in all of the patients in the group with biTLE and in four of the patients from the group with uniTLE who underwent invasive EEG (patients 14, 15, 18, and 23), the seizure-onset zone (SOZ) was found within the hippocampus, amygdala, or temporal pole (i.e., antero-mesio-temporal onset) (see Table 1); in the remaining nine patients from the group with uniTLE (patients 16, 17, 19, 20, 21, 22, 24, 25, and 26) the lesion localization, long-term video-EEG monitoring with scalp/sphenoidal electrodes, and FDG-PET findings led us to consider them to be patients with mesial temporal lobe epilepsy.

Scalp/sphenoidal EEG was performed using the international 10–20 electrode placement system. Multicontact depth electrodes inserted orthogonally or diagonally into both amygdalohippocampal complexes were used in all patients of the group with biTLE. A combination of two stereotactically implanted depth electrodes and subdural strip electrodes was used in two patients (patients 12 and 13).

Only preoperative EEG data were used for further analysis in all of the patients.

## 2.4. Surgery and outcome measure

Eight patients from the group with biTLE and all of the patients from the group with uniTLE underwent resective surgery. We recorded the

**Table 1**

Demographic and clinical characteristics, histopathology, and outcome in the group with biTLE (patients: 1–13) and in the group with uniTLE (patients: 14–26). M – male; F – female; TBI – traumatic brain injury; PI – perinatal insult; FSs – febrile seizures; M/E – meningitis/encephalitis; LD – language dominance according to the Wada test; L – left; R – right; \* – only unilateral testing performed; SOZ – localization of seizure-onset zone proven by invasive EEG (if performed); AHC – amygdalohippocampal complex; Tpol – temporal pole; HS – hippocampal sclerosis; DNET – dysembryoplastic neuroepithelial tumor; TL – temporal lobe; MCD – malformation of cortical development; HA – hippocampal atrophy; MAng – meningioangiomatosis; HIMAL – hippocampal malrotation; FCD – focal cortical dysplasia; VNS – vagus nerve stimulation; AMTR – anteromedial temporal lobe resection; LE – lesionectomy.

Patient	Sex/age at evaluation (years)	Insult	LD	Side-SOZ identified by invasive EEG	MRI finding	Side/surgery	Histopathology	Follow-up surgery (years)	Follow-up VNS (years)	Outcome (Engel)	Outcome (Mc Hugh)
<i>Bitemporal group</i>											
1	M/33	TBI	L	L-AHC; R-AHC	Normal	–	–	–	14	–	I
2	M/41	PI, FSs	L	L-AHC; R-AHC	HS	L/AMTR	HS grade IV	3	2	III A	V
3	F/29	–	L	R-AHC; L-AHC	Tumor	R/AMTR	DNET	3	–	I B	–
4	F/25	–	L	R-AHC; L-AHC	AHC hyperintensity	R/AMTR	Gliosis	10	–	I A	–
5	M/23	–	L	L-Tpol, AHC; R-AHC	TL hypotrophy	L/AMTR	FCD IA	2	1	IV	V
6	F/19	–	L	L-Tpol, AHC; R-AHC	Normal	L/temporal pole resection	Normal	2	1	IV	III
7	F/41	M/E	L	R-AHC; L-AHC	HS	R/AMTR	HS grade III	2	–	II A	–
8	F/33	–	L*	R-AHC; L-AHC	MCD	–	–	–	2	–	V
9	M/47	PI	–	L-AHC; R-AHC	HA	–	–	–	2	–	II
10	F/51	–	L	R-AHC; L-AHC	TL lesion	R/AMTR	MAng	1	–	III A	–
11	F/29	PI	L	R-AHC; L-AHC	HIMAL	–	–	–	4	–	V
12	M/41	–	L	L-Tpol, AHC; R-AHC	Suspected FCD	L/AMTR	FCD IA	5	–	IV	–
13	M/43	PI, M/E	L	L-Tpol, AHC; R-AHC	HS	–	–	–	–	–	–
<i>Unitemporal group</i>											
14	M/33	–	L	R-AHC	HA	R/AMTR	Normal	9	–	I A	–
15	M/29	PI, FSs	–	–	HS	R/AMTR	HS grade uncertain	10	–	I A	–
16	M/41	M/E, FSs	L	L-AHC	HS	L/AMTR	HS grade uncertain	9	–	I A	–
17	M/21	PI	L	–	HA	L/AMTR	Normal	3	–	I A	–
18	F/41	–	L	L-AHC	Normal	L/AMTR	FCD IA	3	–	I A	–
19	M/40	–	L	–	Cavernoma	R/extended LE	Cavernoma	9	–	I A	–
20	F/36	PI, FSs	L	–	HS	R/AMTR	HS grade uncertain	8	–	I A	–
21	M/31	–	L	–	HS	L/AMTR	HS grade III	7	–	I A	–
22	F/24	–	L	–	Tumor	R/extended LE	DNET	9	–	I A	–
23	F/47	–	L	L-AHC	Normal	L/AMTR	Normal	2	–	I A	–
24	M/33	–	L	–	Normal	R/AMTR	Normal	5	–	I A	–
25	M/43	–	L	–	Normal	R/AMTR	FCD IA	3	–	I A	–
26	M/36	M/E	L	–	HA	L/AMTR	Normal	4	–	I A	–

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