Original article

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Ictal EEG modifications in temporal lobe epilepsy

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ABSTRACT – Temporal lobe epilepsy is the most common type of epilepsy in adults with medically intractable, localisation-related epilepsy, amenable to surgery. Together with clinical and neuroimaging data, presurgical ictal scalp-EEG findings are often sufficient to define the epileptogenic zone. It is widely believed that ictal scalp-EEG findings in temporal lobe epilepsy are represented by 5-9-Hz lateralised rhythmic theta activity or 2-5-Hz lateralised rhythmic delta activity. On the basis of experimental models and experience with intra-cerebral EEG recordings, the pattern of low-voltage fast activity is considered to be the electrophysiological hallmark of the epileptogenic zone. We reviewed the ictal scalp-EEG data relating to 111 seizures in 47 patients with temporal lobe epilepsy who underwent video-EEG recordings during presurgical work-up. We found that 35 patients (74.4%) showed flattening, low-voltage fast activity or fast activity as the initial EEG pattern. When visible, the rhythmic delta or theta activity followed the fast activity. Low-voltage fast activity, flattening or fast activity occurs in the majority of patients with temporal lobe epilepsy and represents the main ictal EEG pattern. Low-voltage fast activity (or similar) is also identifiable as the initial ictal EEG pattern in scalp-EEG recordings.

Key words: low-voltage fast activity, ictal scalp-EEG, temporal lobe epilepsy, epileptogenic zone, epilepsy surgery, video-EEG recording

Temporal lobe epilepsy is the most common type of epilepsy in adults with drug-resistant, localisation-related epilepsy, amenable to surgery. Together with clinical and neuroimaging data, the presurgical evaluation of interictal and ictal scalp-EEG findings is often fundamental for defining the epileptogenic zone (EZ). However, ictal EEG modifications are now considered to be more important than interictal EEG abnormalities in localising the EZ (Engel *et al*., 2003; Jan *et al*., 2010).

The identification of any specific activity in scalp-EEG recordings depends on the interaction of various factors, such as the electrical properties of the conductive tissues of the head, background activity, the setting in which the recording is made, and the patient's condition (Jan *et al*., 2010; Cosandier-Rimélé *et al*., 2012). However, the evaluation of EEG findings is complete and reliable only when it is strictly related to ictal onset because the secondary propagation of ictal discharges can involve different and distant brain areas.

Published studies of the subject date back some decades and describe

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only a few ictal EEG patterns. The ictal scalp-EEG findings in temporal lobe epilepsy are historically described as 5-9-Hz lateralised rhythmic theta activity or 2-5-Hz lateralised rhythmic delta activity (Blume *et al*., 1984; Risinger *et al*., 1989; Ebner and Hoppe, 1995; Ebersole and Pacia, 1996; Pacia and Ebersole, 1997; Jan *et al*., 2010). More recent studies have not dealt with the subject directly and electrophysiological findings are typically described on the basis of previous reports (Bentes *et al*., 2008; Jung *et al*., 2009; Rodin *et al*., 2009; Napolitano and Orriols, 2010; Adamolekun *et al*., 2011). Ictal scalp-EEG propagation is generally more evident and easily identifiable, but determining the ictal onset discharge is more complicated because of the presence of artefacts and low signal-to-noise ratio in scalp-EEG (Pacia and Ebersole, 1997; Rodin *et al*., 2009; Jan *et al*., 2010; Cosandier-Rimélé *et al*., 2012). Low-voltage fast activity (LVFA) is characterised by elevated frequency (20-80 Hz) and low amplitude (no more than 20 μV) and is often considered as an electrophysiological hallmark of EZ.

In fact, studies on experimental models have demonstrated that LVFA plays a role in epileptogenesis and correlates with the EZ (Wendling *et al*., 2003; Bartolomei *et al*., 2008; Jacobs *et al*., 2008; Bragin *et al*., 2010). Moreover, it has been demonstrated that LVFA can be identified in intracranial EEG recordings, thus opening up a new perspective for interpreting seizure onset. In particular, many studies have demonstrated that LVFA does not depend on the underlying pathology (Jacobs *et al*., 2008; Jacobs *et al*., 2009) and that the surgical prognosis is related to the removal of brain areas producing it (Lee *et al*., 2000; Jacobs *et al*., 2008; Wetjen *et al*., 2009; Bartolomei *et al*., 2010; Jacobs *et al*., 2010). However, to the best of our knowledge, no studies have yet investigated the presence of LVFA in scalp-EEG recordings, despite its recognised value in localising the EZ.

In order to identify any change before the first clinical manifestation (corresponding to ictal onset), we reviewed the ictal EEGs of patients with temporal lobe epilepsy who had undergone video-EEG recordings during the presurgical work-up. The aim of this study was to determine whether LVFA can also be identified in scalp-EEG recordings and evaluate its role in defining the EZ.

Patients and methods

Patients

Between September 2005 and July 2011, 636 patients were investigated in the Laboratory of the "Claudio Munari" Epilepsy Surgery Centre as part of their presurgical work-up or in order to determine correct diagnosis. The digitalised EEG data were available for review (Nihon Kohden System). For each patient, one or more seizures were recorded and all were followed for at least one year after surgery.

For the purposes of this study, we selected 47 patients with medically intractable temporal lobe epilepsy and reviewed their clinical, anatomical, and neurophysiological data in detail (*i.e*. their clinical history, neurological examination results, seizure semiology, brain magnetic resonance imaging (MRI) findings, neuropsychological assessment results, and neuropathological information). Twenty-four patients (51.1%) did not have any significant risk factors for seizures (*i.e*. a family history of epilepsy, pre- or perinatal brain damage, febrile seizures, or head trauma), and 9 (19.1%) had a history of febrile seizures. Twelve patients (25.5%) reported a seizure-free period of at least one year during the disease.

Forty-three patients (91.5%) had positive brain MRI findings. Twenty-five patients (53.2%) underwent left and 22 (46.8%) right temporal lobe surgery. The main clinical characteristics, neuropathological data, and outcomes are shown in *table 1*.

Methods

Video-EEG monitoring and EEG analysis

All of the patients underwent video-EEG monitoring at the Claudio Munari Epilepsy Surgery Centre, with electrodes positioned according to the 10-20 international system (Fpz as reference). No sphenoidal electrodes were used. During long-term monitoring, impedance and the possible presence of artefacts were constantly checked. The neurophysiological findings were retrospectively reviewed by two competent independent investigators.

Video-EEG monitoring was performed by means of a referential montage, thus allowing the recordings to be reviewed in numerous bipolar and referential montages. During the review, digital filtering and gain adjustment were used to optimise the EEG display. The sampling rate was 500 Hz. The low filter was always 1.6 Hz, and depending on the technical "aspect" of the EEG, the range of the high filter was 30-120 Hz. Every patient underwent baseline EEG with stimulation tests (hyperventilation, intermittent photic stimulation, and auditory stimulation). If necessary, sleep deprivation and/or antiepileptic drug withdrawal was used to increase the probability of a seizure.

We analysed interictal abnormalities during wakefulness and sleep but, above all, we evaluated the ictal scalp-EEG findings in order to identify any change before the first clinical (subjective or objective) manifestation (ictal onset) and subsequent ictal EEG propagation.

Table 1. Main clinical characteristics of patients.

Males/Females (%)	32/15 (68/32)
Age at epilepsy onset	10.26±8.53 (0-37)
Duration of epilepsy	21.42±12.43 (1-47)
Age at surgery	31.72±15.01 (2-57)
Number of seizures/month	15.28±17.06 (2-60)
Number of AEDs	2.15±0.72 (1-4)
NEUROPATHOLOGY	14 isolated HS (29.8%) 7 FCD IIIa (14.9%) 5 MCD (10.7%) 4 low-grade brain tumour (8.5%) 4 FCD I (8.5%) 3 FCD IIIb (6.4%) 2 double pathology (4.2%) 8 negative (17%)
OUTCOME (months) **follow-up 40.2±20.6 (12-86)**	35 Ia+Ic (74.6%) 2 Ib (4.2%) 1 Id (2.1%) 2 II (4.2%) 7 III (14.9%)

HS: hippocampal sclerosis; FCD: focal cortical dysplasia; MCD: malformation of cortical development.
Neuropathological data are reported according to Blümcke classification (Blümcke *et al.*, 2011). Outcome is reported according to Engel classification (Engel *et al.*, 1993).

Ictal EEG classification

We retrospectively reviewed a total of 2,884 hours of EEG recordings and 111 seizures (2.3 seizures/patient). We analysed all the seizures of the patients, but due to certain circumstances (position or activities of the patient, technical and muscular artefacts), the EEG of ictal onset was not clear in 42 seizures (37.8% of the total number) and consequently not included in our analysis. Nevertheless, only in 4 patients (8.5%) was the ictal onset (considered as "no visible pattern") not identified. For the majority of the patients (26 patients; 55.3%), we recorded at least two seizures and the other patients (17 patients; 36.2%) each had one seizure which was suitable for analysis. The total number of analysed seizures was 69.

We observed the following ictal EEG patterns:

- no visible pattern;
- background attenuation (disappearance of background activity mainly localised in posterior regions);
- reduction/disappearance of interictal EEG abnormalities;
- rhythmic spikes;
- rhythmic slow activity (theta or delta);
- flattening (clear reduction of focal amplitude without any distinct fast activity, with different localisation);
- fast activity (generally beta activity);
- low-voltage fast activity (fast activity associated with a clear reduction in amplitude; no more than 20 μv).

Statistical analysis

The associations between ictal EEG patterns and postsurgical outcomes or neuropathological findings were evaluated using the Pearson's χ^2 test with Yates' correction. One-way analysis of variance (ANOVA) was used to evaluate any differences in age at epilepsy onset, illness duration, seizure rate or seizure freedom, in relation to postsurgical outcome.

Results

Ictal EEG

Analysis of the ictal EEGs revealed two distinct phases: i) ictal onset; and ii) subsequent discharge development and possible contralateral propagation.

The following EEG patterns were observed in relation to ictal onset (*figures 1-5*):

- no visible pattern in 4 patients (8.5%);
- background attenuation in 4 patients (8.5%);
- reduction in interictal EEG abnormalities in 3 patients (6.3%);
- fast activity in 2 patients (4.2%) (*figure 4*);
- rhythmic spikes in 1 patient (2.1%);
- flattening in 23 patients (48.9%) (*figures 1 and 5*);
- LVFA in 10 patients (21.5%) (*figures 2 and 3*).

EEG flattening, LVFA, and fast activity were observed in a total of 35 patients (74.4%), of whom 27 (77.1%) had favourable postsurgical outcome (Engel class I). None of the patients showed rhythmic slow activity (theta or delta) at seizure onset. All of the patterns were clearly localised on the EEG unless otherwise specified, and all the discharges were clearly confined to the temporal lobe (F8-T4, T4-T6 and F7-T3, T3-T5).

A favourable outcome was observed in 18/23 patients with EEG flattening (78.3%), 8/10 with LVFA (80%), and 1/2 with fast activity (50%); *i.e.* 71.1% of all patients with favourable outcome. The other favourable outcomes were observed in the 4 patients with background attenuation, the 3 patients with reduced interictal abnormalities, and the patient with rhythmic spikes.

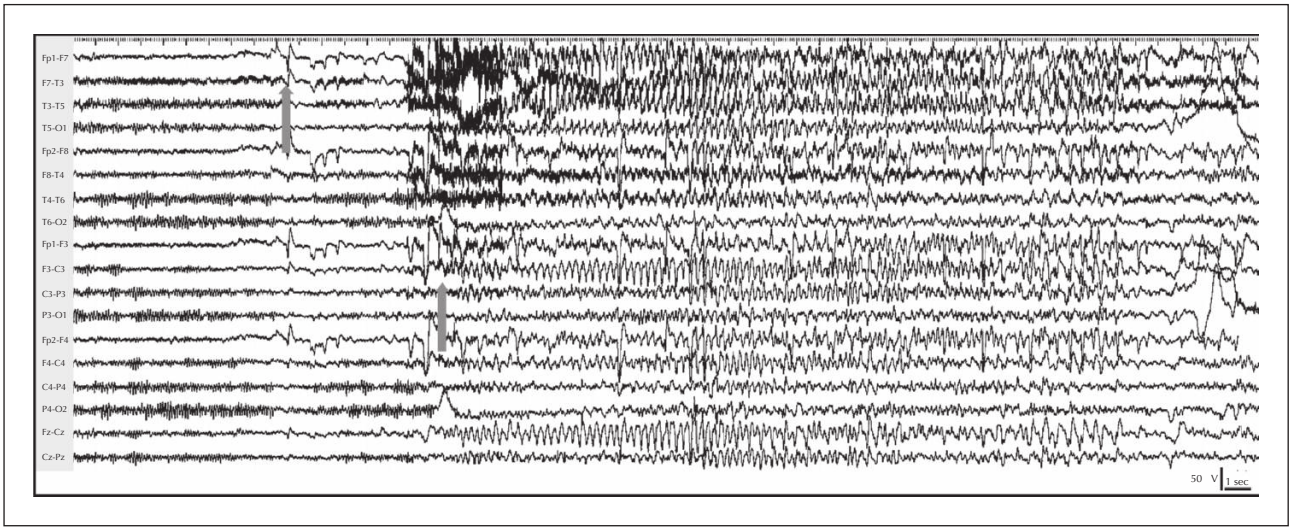

Figure 1. Left temporal EEG flattening. We observed rhythmic delta activity ten seconds later.

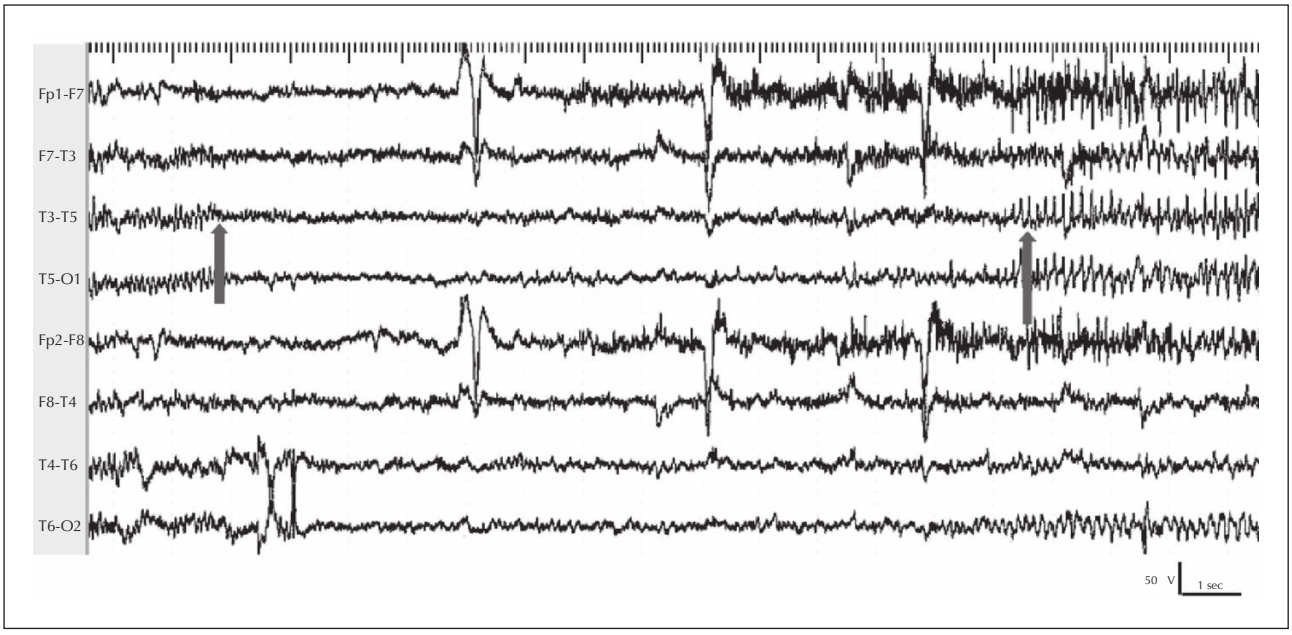

Figure 2. Low-voltage fast activity in left temporal regions. Rhythmic theta activity becomes visible 13 seconds later.

The following patterns were observed in relation to subsequent discharge development or propagation:
- rhythmic slow activity (theta) in 30 patients (63.8%) (*figures 2* and *5*);
- rhythmic slow activity (delta) in 10 patients (21.3%) (*figures 3 and 4*);
- rhythmic spikes in 6 patients (12.8%);
- fast activity in 1 patient (2.1%).

In 9 patients (19.1%), 4 of whom had unfavourable surgical outcome, the propagation of discharge involved contralateral regions or was diffuse.

Twenty-one of the 35 patients with EEG flattening, LVFA or fast activity showed rhythmic theta activity, of whom 17 had favourable outcome (81%).

Considering the total number of seizures, we observed the following at ictal onset:
- no visible pattern in 42 seizures (37.8%);
- background attenuation in 4 seizures (3.6%);
- reduction in interictal EEG abnormalities in 5 seizures (4.5%);
- fast activity in 2 seizures (1.8%);

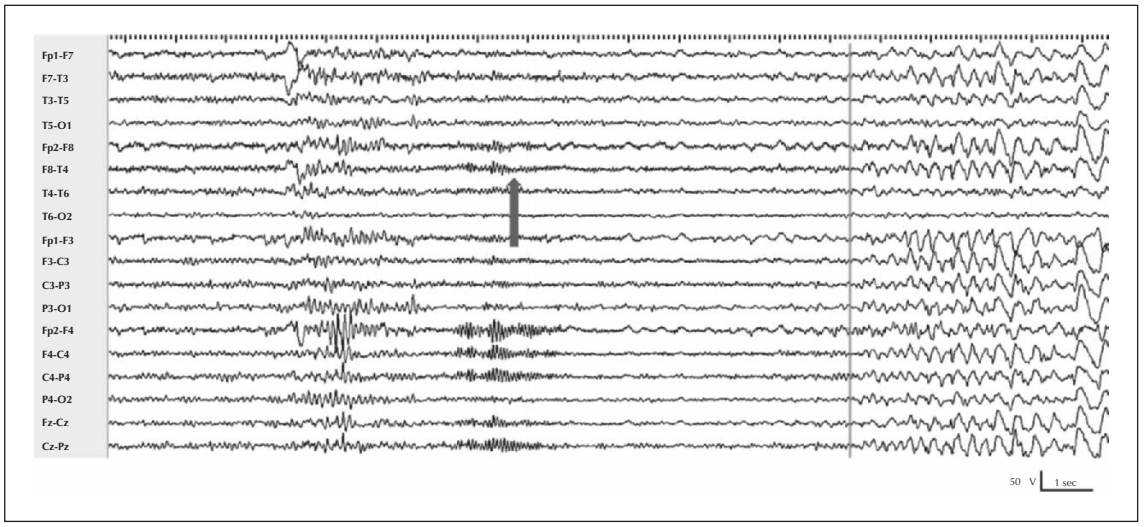

Figure 3. Low-voltage fast activity in right temporal regions. Rhythmic delta activity appears seven seconds later.

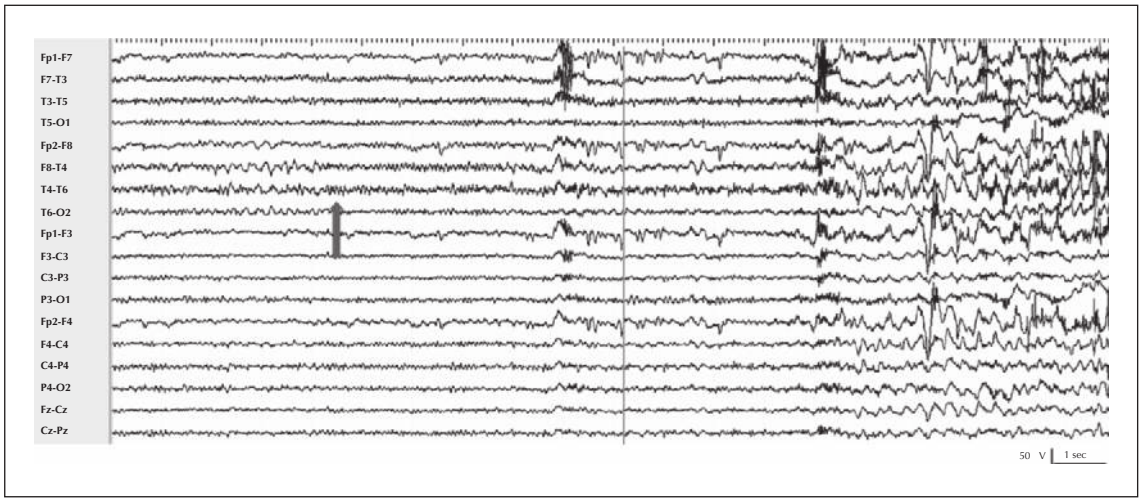

Figure 4. Fast activity in right temporal regions.
The subsequent development of the ictal discharge (ten seconds later) is characterised by rhythmic delta activity.

- rhythmic spikes in 1 seizures (0.9%);
- flattening in 35 seizures (31.6%);
- LVFA in 22 seizures (19.8%);
- in 59/111 seizures (53.2%), we observed EEG flattening, LVFA or fast activity.

Moreover, we analysed the extent of localisation of the initial ictal onset relative to the subsequent EEG patterns (65 seizures; EEGs in which there was "no visible pattern" or no localised background attenuation were excluded). In 59 seizures (90.8%), localisation of the initial ictal onset was better defined, with respect to the delimitation of the subsequent pattern. In 6 seizures (9.2%), localisation of the initial ictal onset and subsequent pattern was identical. Localisation of the subsequent pattern was consistently less defined than that of the initial ictal onset.

Finally, we evaluated the duration of the ictal EEG pattern. The median duration of initial ictal EEG pattern observed was 13±8 seconds (mean±standard deviation; range: 2-50 seconds).

Postsurgical outcome

Postsurgical outcome was favourable in 38 patients (80.9% in Engel class I). All of the 14 patients with hippocampal sclerosis had a favourable outcome and the presence of hippocampal sclerosis was statistically significant (p=0.0087). Four patients with a negative histological examination had an unfavourable outcome,

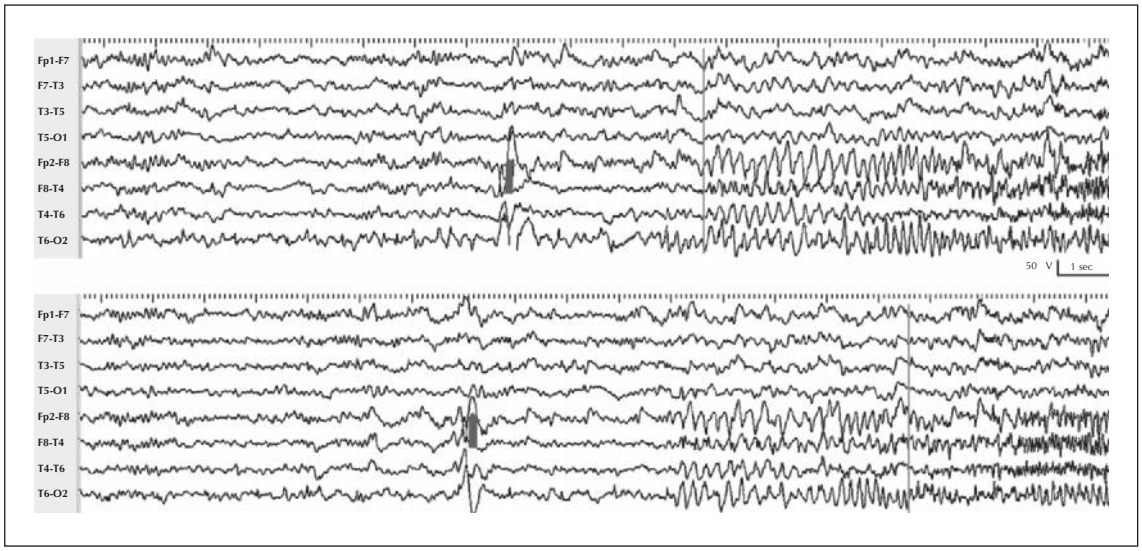

Figure 5. EEG of two different seizures of the same patient. The ictal EEG pattern is identical and starts with a slow wave of elevated amplitude followed by EEG flattening with right temporal localisation. Also, in these seizures, the subsequent development of the ictal discharge appears four seconds later and is characterised by rhythmic theta activity.

but this association was not statistically significant (p=0.052). We did not find any significant correlations between neuropathology and the ictal patterns.

The associations between outcome and age at surgery, illness duration or seizure frequency were not statistically significant (p=0.65, p=0.68, and p=0.10, respectively), nor were the associations between outcome and interictal spikes during wakefulness or sleep (p=0.73 and p=0.70, respectively).

Discussion

We reviewed the ictal EEGs of 47 patients with drug-resistant temporal lobe epilepsy who, on the basis of clinical, anatomical, and video-EEG findings, underwent temporal cortectomy. Our aim was to analyse and describe early ictal EEG patterns, determine whether LVFA is also identifiable on scalp-EEG, and assess its value in defining the EZ.

The analysis of scalp-EEG recordings of 111 seizures allowed us to identify ictal onset (*i.e.* EEG changes occurring before or at the same time as the first clinical signs) in all but four patients (8.5%).

LVFA is considered the electrophysiological signature of the EZ (Fisher *et al.*, 1992; Wendling *et al.*, 2003; Bartolomei *et al.*, 2008) and does not depend on the underlying pathology (Bragin *et al.*, 2004; Jacobs *et al.*, 2008; De Curtis and Gnatkovsky, 2009; Jacobs *et al.*, 2009; Cosandier-Rimélé *et al.*, 2012). Moreover, it has been shown that surgical prognosis is related to the removal of brain areas generating LVFA (Lee *et al.*, 2000; Wetjen *et al.*, 2009; Bartolomei *et al.*, 2010).

The frequency of scalp-EEG LVFA is clearly different from that observed in intra-cranial EEG recordings, but the concept and significance remain the same. The differences are mainly due to the presence of artefacts and the electrical properties of the conductive media in the head. It was commonly held that LVFA cannot be seen on scalp-EEGs because of artefacts and the low signal-to-noise ratio (Cosandier-Rimélé *et al.*, 2012). However, we found that the main early ictal EEG pattern was LVFA, flattening or fast activity (variants of the principal pattern), which we distinguished as different expressions of the same EZ activity that depended on the patients' condition (position of the patient, *i.e.* standing, sitting, lying; what the patient was doing, *i.e.* eating, speaking, sleeping; where the recordings were performed; and the presence of the technical and muscular artefacts). LVFA was observed in 21.5% of patients, and flattening, LVFA or fast activity in 35 patients (74.4%), of whom 27 (77.1%) had favourable surgical outcome. None of the patients showed rhythmic theta/delta activity at the time of ictal onset.

Considering the total number of seizures (111), we observed flattening, LVFA or fast activity in 59 seizures (53.2%). Moreover, we observed that the initial ictal pattern was more localised compared to the subsequent EEG pattern in 59/65 seizures (90.8%).

Unlike previously published findings, our data suggest that scalp-EEG patterns can also play an important role in localising the EZ. It is important to underline the significance of greater accuracy in localising LVFA (or its variants) which should be considered in order to carefully investigate these ictal EEG patterns.

Moreover, despite one of the limitations of our study which was certainly the small number of patients, we suggest that scalp-EEG LVFA is probably related to favourable outcome, as demonstrated by intracranial EEG recordings.

The first ictal discharge in patients with temporal lobe epilepsy is usually described as rhythmic theta/delta activity (Risinger *et al*., 1989; Walczak *et al*., 1992; Ebner and Hoppe, 1995; Pacia and Ebersole, 1997; Vossler *et al*., 1998; Tatum *et al*., 2008; Jan *et al*., 2010), however, although we observed rhythmic theta/delta activity in 85.1% of the patients, this appeared on average 13 seconds after the LVFA (or similar activity). We therefore consider this to be a subsequent EEG pattern that corresponds to ictal discharge development (or propagation, if contralateral).

This is of importance with regards to localising the "true" onset of ictal discharge since, clinical signs may otherwise be overlooked and the EZ may be incorrectly localised. Considering the long time (mean: 13 seconds) between the initial and subsequent ictal EEG discharges, there is a high risk of overlooking some important clinical and neurophysiological characteristics, not only because the development of an ictal discharge appears later, but also because it can involve different, and in some cases even contralateral, regions. Nine of our patients (19.1%) showed discharge propagation involving the contralateral regions or diffuse involvement, four of whom had unfavourable surgical outcome. This underlines the need to search for the "true" onset of discharge which may be found once the first clinical sign has been identified.

Identifying the ictal onset discharge is complicated because of the presence of artefacts and the low signal-to-noise ratio of scalp EEG, however, this is not a major limitation as we failed to identify the ictal onset in only 8.5% of our patients. Probably, this limitation can be further reduced by increasing the number of recorded seizures of each patient (generally at least two seizures).

It therefore appears that, in clinical practice, the ictal onset should always be carefully investigated by analysing any subtle EEG modifications before the first clinical sign. □

Disclosures.

None of the authors have any conflict of interest to disclose.

References

Adamolekun B, Afra P, Boop FA. False lateralization of seizure onset by scalp EEG in neocortical temporal lobe epilepsy. *Seizure* 2011; 20: 494-9.

Bartolomei F, Chauvel P, Wendling F. Epileptogenicity of brain structures in human temporal lobe epilepsy: a quantified study from intracerebral EEG. *Brain* 2008; 131: 1818-30.

Bartolomei F, Cosandier-Rimélé D, McGonigal A, *et al*. From mesial temporal lobe to temporoperisylvian seizures: a quantified study of temporal lobe seizure network. *Epilepsia* 2010; 51: 2147-58.

Bentes C, Pimentel J, Costa J, Santos R, Rolo V. Ictal singing: case report and reappraisal of the literature. *Epileptic Disord* 2008; 10: 113-8. doi: 10.1684/epd.2008.0185.

Blümcke I, Thom M, Aronica E, *et al*. The clinicopathologic spectrum of focal cortical dysplasias: a consensus classification proposed by an ad hoc Task Force of the ILAE Diagnostic Methods Commission. *Epilepsia* 2011; 52: 158-74.

Blume WT, Young GB, Lemieux JF. EEG morphology of partial epileptic seizures. *Electroencephalogr Clin Neurophysiol* 1984; 57: 295-302.

Bragin A, Wilson CL, Almajano J, Mody I, Engel Jr. J. High-frequency oscillations after status epilepticus: epileptogenesis and seizure genesis. *Epilepsia* 2004; 45: 1017-23.

Bragin A, Engel J, Staba RJ. High-frequency oscillations in epileptic brain. *Curr Opin Neurol* 2010; 23: 151-6.

Cosandier-Rimélé D, Bartolomei F, Merlet I, Chauvel P, Wendling F. Recording of fast activity at the onset of partial seizures: depth EEG *vs* scalp EEG. *Neuroimage* 2012; 59: 3474-87.

De Curtis M, Gnatkovsky V. Reevaluating the mechanisms of focal ictogenesis. The role of low-voltage fast activity. *Epilepsia* 2009; 50: 2514-25.

Ebersole JS, Pacia SV. Localization of temporal lobe foci by ictal EEG patterns. *Epilepsia* 1996; 37: 386-99.

Ebner A, Hoppe M. Noninvasive electroencephalography and mesial temporal sclerosis. *J Clin Neurophysiol* 1995; 12: 23-31.

Engel J Jr, Van Ness PC, Rasmussen TB, Ojemann LM. Outcome with respect to epileptic seizures. In: Engel J Jr. *Surgical treatment of the epilepsies*. New York: Raven Press, 1993: 609-21.

Engel J, Wiebe S, French J, *et al*. Quality Standards Subcommittee of the American Academy of Neurology; American Epilepsy Society; American Association of Neurological Surgeons. Practice parameter: temporal lobe and localized neocortical resections for epilepsy: report of the Quality Standards Subcommittee of the American Academy of Neurology, in association with the American Epilepsy Society and the American Association of Neurological Surgeons. *Neurology* 2003; 60: 538-47. Erratum: 1396.

Fisher RS, Webber WR, Lesser RP, Arroyo S, Uematsu S. High-frequency EEG activity at the start of seizures. *J Clin Neurophysiol* 1992; 9: 441-8.

Jacobs J, LeVan P, Chander R, Hall J, Dubeau F, Gotman J. Interictal high-frequency oscillations (80-500 Hz) are an indicator of seizure onset areas independent of spikes in the human epileptic brain. *Epilepsia* 2008; 49: 1893-907.

Jacobs J, Levan P, Châtillon CE, Olivier A, Dubeau F, Gotman J. High frequency oscillations in intracranial EEGs mark epileptogenicity rather than lesion type. *Brain* 2009; 132: 1022-37.

Jacobs J, Zijlmans M, Zelmann R, *et al*. High-frequency electroencephalographic oscillations correlate with outcome of epilepsy surgery. *Ann Neurol* 2010; 67: 209-20.

Jan MM, Sadler M, Rahey SR. Electroencephalographic features of temporal lobe epilepsy. *Can J Neurol Sci* 2010; 37: 439-48.

Jung K, Kang J, Kim JH, Im C, Kim KH, Jung H. Spatiotemporospectral characteristics of scalp ictal EEG in mesial temporal lobe epilepsy with hippocampal sclerosis. *Brain Res* 2009; 1287: 206-19.

Lee SA, Spencer DD, Spencer SS. Intracranial EEG seizure-onset patterns in neocortical epilepsy. *Epilepsia* 2000; 41: 297-307.

Napolitano CE, Orriols MA. Graduated and sequential propagation in mesial temporal lobe epilepsy: analysis with scalp ictal EEG. *J Clin Neurophysiol* 2010; 27: 285-91.

Pacia SV, Ebersole JS. Intracranial EEG substrates of scalp ictal patterns from temporal lobe foci. *Epilepsia* 1997; 38: 642-54.

Risinger MW, Engel J. Van Ness PC, Henry TR. *Crandall PH. Ictal localization of temporal lobe seizures with scalp/sphenoidal recordings. Neurology* 1989; 39: 1288-93.

Rodin E, Constantino T, Rampp S, Modur P. Seizure onset determination. *J Clin Neurophysiol* 2009; 26: 1-12.

Tatum WO, Benbadis SR, Hussain A, *et al*. Ictal EEG remains the prominent predictor of seizure-free outcome after temporal lobectomy in epileptic patients with normal brain MRI. *Seizure* 2008; 17: 631-6.

Vossler DG, Abson Kraemer DL, Knowlton RC. Temporal ictal electroencephalographic frequency correlates with hippocampal atrophy and sclerosis. *Ann Neurol* 1998; 43: 756-62.

Walczak T, Radtke R, Lewis D. Accuracy and interobserver reliability of scalp ictal EEG. *Neurology* 1992; 42: 2279-85.

Wendling F, Bartolomei F, Bellanger JJ, Bourien J, Chauvel P. Epileptic fast intracerebral EEG activity: evidence for spatial decorrelation at seizure onset. *Brain* 2003; 126: 1449-59.

Wetjen NM, Marsh WR, Meyer FB, *et al*. Intracranial electroencephalography seizure onset patterns and surgical outcomes in nonlesional extratemporal epilepsy. *J Neurosurg* 2009; 110: 1147-52.