Epileptic Disord 2013; 15 (3): 338-41

Seizure semiology reflects spread from frontal to temporal lobe: evolution of hyperkinetic to automotor seizures as documented by invasive EEG video recordings

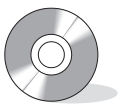
Fadime Irsel Tezer [1,2], Kadriye Agan [1,3], Ingo Borggraefe [1], Soheyl Noachtar [1]

[1] Epilepsy Center, Dept of Neurology, University of Munich, Munich, Germany
[2] Dept of Neurology, Hacettepe University, School of Medicine, Ankara
[3] Dept of Neurology, Marmara University, School of Medicine, Istanbul, Turkey

Received January 24, 2013; Accepted July 19, 2013

ABSTRACT – This patient report demonstrates the importance of seizure evolution in the localising value of seizure semiology. Spread of epileptic activity from frontal to temporal lobe, as demonstrated by invasive recordings, was reflected by change from hyperkinetic movements to arrest of activity with mild oral and manual automatisms. [*Published with video sequences*]

Key words: hyperkinetic seizure, hypermotor seizure, automotor seizure, seizure semiology, temporal lobe epilepsy, frontal lobe epilepsy



Correspondence:
Soheyl Noachtar
Epilepsy Center,
Dept of Neurology,
University of Munich,
Marchioninistr 15,
81377 Munich, Germany
<noa@med.uni-muenchen.de>

The analysis of seizure semiology is an important part of evaluation of patients considered for resective epilepsy surgery. Temporal lobe epilepsy (TLE) is typically characterised by abdominal and psychic auras, which may evolve into motionless staring and eventually manual and oral automatisms (Henkel *et al*., 2002). The seizure semiology originating from frontal lobe is more variable and includes so called "*hyperkinetic*" (Engel, 2001) or "*hypermotor*" (Lüders *et al*., 1998) seizures, which may easily be mistaken for non-epileptic pseudoseizures because of their clinical appearance (Williamson *et al*., 1985; Kanner *et al*., 1990; Manford *et al*., 1996; Lüders *et al*., 1998). Hyperkinetic seizures consist of motor automatisms which involve predominantly proximal parts of the limbs. The movements often appear violent, bizarre, and frenetic (Williamson *et al*., 1985; Manford *et al*., 1996). The supplementary sensorimotor area is thought to generate hyperkinetic seizures (Williamson *et al*., 1985; Manford *et al*., 1996; Lüders *et al*., 1998).

Although hypermotor seizures are typical of frontal lobe epilepsy (FLE),

doi:10.1684/epd.2013.0597

there are well documented exceptions to this. Depth electrode recordings in the temporal and frontal cortex demonstrate spread of seizure activity from the amygdala to the frontal lobe. The hypermotor phase of the seizure starts when the epileptic activity reaches the frontal lobe. The analysis of seizure evolution provides more localising information than the analysis of single seizure types (Henkel *et al*., 2002).

This report shows the change of seizure semiology during spread of epileptic activity from the frontal to the ipsilateral temporal lobe.

Case study

A 33-year-old female was admitted to our epilepsy monitoring unit because of intractable seizures since the age of 23 years. Typically, she had nocturnal seizures characterised by proximal trunk movements with impairment of consciousness. Sometimes the seizures evolved into generalised tonic-clonic seizures. Frequently, she had an aura consisting of an indescribable sensation which heralded the seizures before consciousness was impaired. Carbamazepine, levetiracetam, lamotrigine, and zonisamide failed to control the seizures. Her average seizure frequency was 3-4 per day. Her history was unremarkable for trauma, febrile seizures, and meningitis. No family history of epilepsy was known. Her systemic and neurological examinations were normal.

During non-invasive video-EEG monitoring, 12 hyperkinetic seizures were recorded, which showed non-lateralised (*n*=2), left temporal (*n*=7), left frontotemporal (*n*=2), and right frontotemporal (*n*=1) EEG seizure onset. Three of these seizures were preceded by her habitual auras. One seizure evolved into a generalised tonic-clonic seizure. Left head and eye version with a "sign of four" manifested before evolution into secondary generalised tonic-clonic seizures, pointing to right hemisphere seizure onset. Interictal epileptiform discharges were recorded in the right (68%) and left (32%) anterior temporal regions during non-invasive video-EEG monitoring. Her cranial magnetic resonance imaging (MRI) revealed a lesion in the right frontal lobe suggestive of focal cortical dysplasia and subtle right mesial temporal sclerosis without atrophy. Her ictal SPECT showed hyperperfusion in the right frontobasal area and PET revealed hypometabolism in the same region. Invasive EEG evaluation with subdural electrodes covering the right orbito-frontal and lateral frontal and right mesial and lateral temporal regions was performed in order to further localise the seizure onset (*figure 1*). In addition, sphenoidal electrodes and scalp electrodes covered the left temporal and frontal areas. Interictally, most of the invasive interictal epileptiform discharges were recorded in the right mesiotemporal and basal temporal areas (78%). During invasive monitoring, she had 23 hypermotor seizures during eight days. All except three of these seizures occurred during sleep. The seizures mostly started with fearful facial expression, followed by hyperkinetic movements predominantly involving the proximal legs. In the later phase of the seizures, hyperkinetic automatisms subsided and she had an arrest of her activity with mild oral or manual automatisms for 20 seconds (*see video sequence*). All invasively recorded seizures showed EEG seizure onset in the right orbitofrontal region (*figure 2A*) which preceded the clinical onset by 1-26 seconds. The EEG seizure pattern migrated to the right mesiotemporal region 48-56 seconds later (*figure 2B*). At that time, the orbitofrontal EEG seizure ceased. This migration was consistently associated with a change of seizure semiology; the hyperkinetic automatisms ceased and arrest of activity with mild oral and manual automatisms prevailed.

The seizure pattern propagated to the contralateral temporal lobe *via* involvement of the right temporal region, in two of her 23 seizures.

The patient underwent a right frontal resection including the orbitofrontal and frontopolar cortex based on the results of invasive EEG recordings and electrical stimulation of the cortex, sparing somatosensory cortex. Histopathology of the resected specimen revealed focal cortical dysplasia type IIb (Palmini *et al*., 2004). She has been seizure-free without medication for five years during the follow-up period.

Discussion

This report shows that the spread of epileptic activity from frontal to temporal lobe is reflected by the change of seizure semiology. Determining the exact localisation of the epileptogenic zone between temporal and frontal lobes is a common issue in the evaluation of patients considered for epilepsy surgery since temporal and frontal lobe epilepsy are the most common epilepsy syndromes amenable to resective epilepsy surgery (Noachtar and Borggraefe, 2009). Seizure semiology provides valuable information about the localisation and lateralisation of the epileptogenic zone. However, spread of epileptic activity may blur the picture and lead to controversy regarding seizure onset based on seizure semiology (Blume *et al*., 2001). This case demonstrates the importance of seizure evolution in the evaluation of patients for epilepsy surgery. This is particularly important since the patient's MRI revealed mild ipsilateral mesial temporal sclerosis in addition to right frontal focal cortical dysplasia and non-invasive interictal EEG revealed exclusively bilateral temporal interictal epileptiform discharges which

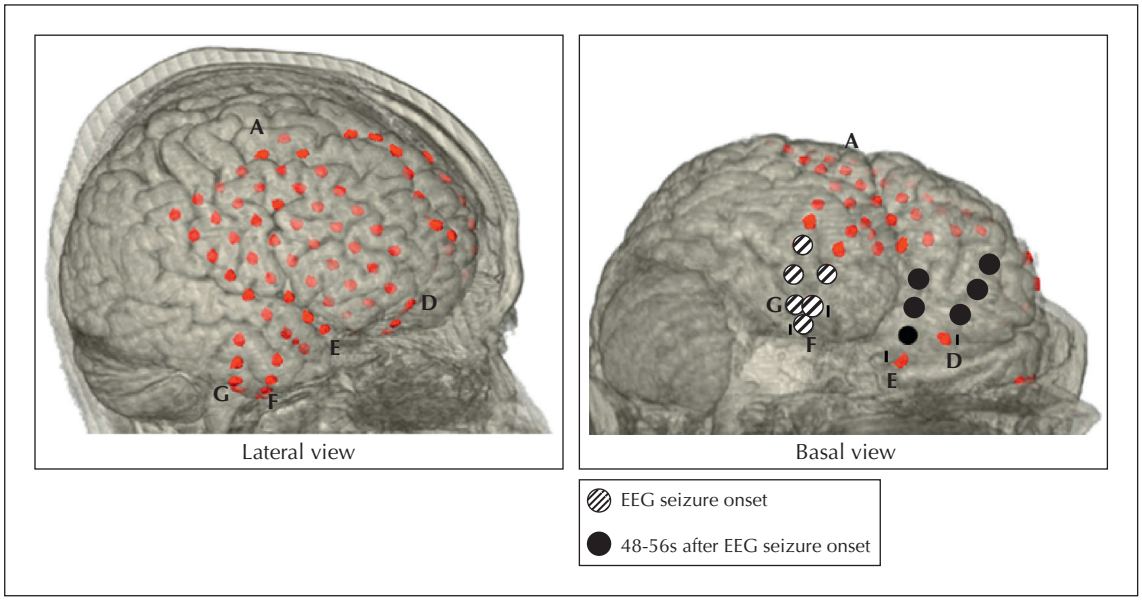


Figure 1. Lateral and basal view of the subdural electrodes (red) covering the right orbitofrontal (E, D), lateral frontal (A), and right mesial and lateral temporal (F, G) regions.
The electrodes showing the orbitofrontal EEG seizure onset during hyperkinetic seizure are marked in black (E, D). The electrodes (G, F) showing EEG seizure activity in the temporal lobe, 48 to 56 seconds later during oral and manual automatisms, are shaded.

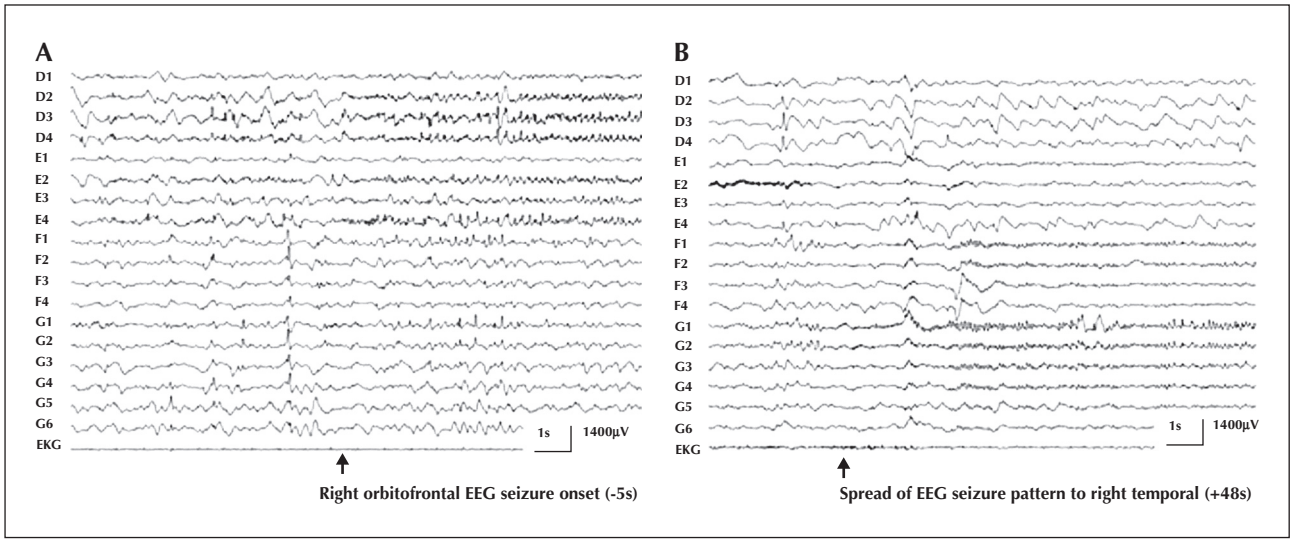


Figure 2. Selected channels of an 87-channel subdural recording, referenced to electrode CPZ. (A) The EEG seizure onset pattern in the right orbitofrontal region (electrodes E2-4 and D2-4) preceded the clinical onset by five seconds. (B) The EEG seizure pattern spread to the right mesiotemporal region 48 seconds later. The ictal EEG activity in the frontal region ceased at that time. The migration of ictal activity to temporal lobe was associated with mild oral and manual automatisms. For localisation of the electrodes, see *figure 1*.

were observed in only 16% of patients with frontal lesions (Remi *et al*., 2011).

Seizures arising from the frontal lobes are typically brief, lasting for under one minute with little or no postictal confusion. They often have a nocturnal preponderance. Warnings are frequent but mostly non-specific. Complex motor automatisms, including waving, rotating, stepping, cycling and rocking movements, are prominent and begin abruptly with a frenetic or agitated, but stereotyped, appearance (Williamson *et al*., 1985). Other motor seizures such as tonic, clonic and versive seizures also occur in FLE.

Seizures arising from the temporal lobes often begin with abdominal or psychic auras followed by arrest of activity, impairment of consciousness, staring, and oral and manual automatisms (Henkel *et al*., 2002).

However, there is some considerable overlap of seizure semiology between FLE and TLE (Manford *et al*., 1996). Furthermore, as a result of seizure activity spread from the frontal to temporal region, the temporal automatisms can be masked by the more obvious hyperkinetic semiology of frontal origin. In our patient, the ictal hyperkinetic activity associated with frontal lobe epileptic activity ceased before propagation of epileptic activity to the temporal lobe, which then gave rise to the manual and oral automatisms characteristic of seizures of temporal lobe origin.

In conclusion, meticulous analysis of seizure semiology with a focus on seizure evolution provides valuable localising information which is important for the evaluation of patients considered for resective epilepsy surgery. □

Disclosures.
The authors report no conflict of interests.

Legends for video sequences

The seizure started with fearful facial expression, followed by hyperkinetic movements predominantly involving the trunk and proximal legs. Later, the hyperkinetic movements subsided and she had an arrest of activity with mild oral and manual automatisms.

Key words for video research on www.epilepticdisorders.com

Syndrome: focal non-idiopathic frontal (FLE)
Etiology: dysplasia (cytoarchitectural)
Phenomenology: hypermotor seizure; automotor (distal, mouth or tongue) seizure
Localization: frontal lobe (right)

References

Blume WT, Ociepa D, Kander V. Frontal lobe seizure propagation: scalp and subdural EEG studies. *Epilepsia* 2001; 42: 491-503.

Engel J Jr. A proposed diagnostic scheme for people with epileptic seizures and with epilepsy: report of the ILAE Task Force on Classification and Terminology. *Epilepsia* 2001; 42: 796-803.

Henkel A, Noachtar S, Pfander M, Lüders HO. The localizing value of the abdominal aura and its evolution: a study in focal epilepsies. *Neurology* 2002; 58: 271-6.

Kanner AM, Morris HH, Lüders H, *et al*. Supplementary motor seizures mimicking pseudoseizures: some clinical differences. *Neurology* 1990; 40: 1404-7.

Lüders H, Acharya J, Baumgartner C, *et al*. Semiological seizure classification. *Epilepsia* 1998; 39: 1006-13.

Manford M, Fish DR, Shorvon SD. An analysis of clinical seizure patterns and their localizing value in frontal and temporal lobe epilepsies. *Brain* 1996; 119: 17-40.

Noachtar S, Borggraefe I. Epilepsy surgery: a critical review. *Epilepsy Behav* 2009; 15: 66-72.

Palmini A, Najm I, Avanzini G, *et al*. Terminology and classification of the cortical dysplasias. *Neurology* 2004; 62: S2-8.

Remi J, Vollmar C, de Marinis A, Heinlin J, Peraud A, Noachtar S. Congruence and discrepancy of interictal and ictal EEG with MRI lesions in focal epilepsies. *Neurology* 2011; 77: 1383-90.

Williamson PD, Spencer DD, Spencer SS, Novelly RA, Mattson RH. Complex partial seizures of frontal lobe origin. *Ann Neurol* 1985; 18: 497-504.