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Semiology of hyperkinetic seizures of frontal *versus* temporal lobe origin

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ABSTRACT – *Aims*. Hyperkinetic seizures are usually associated with frontal lobe epilepsy. However, some patients have hyperkinetic seizures of temporal lobe origin. The semiological differences in hyperkinetic seizures between frontal and temporal lobe epilepsy have not been well studied. Here, we retrospectively assessed ictal semiology in order to distinguish between hyperkinetic seizures of frontal lobe origin and those of temporal lobe origin.

Methods. We retrospectively reviewed data on patients who had undergone surgery for hyperkinetic seizures of temporal or frontal lobe origin and achieved favourable seizure outcomes (Engel Class I) with a minimum postoperative follow-up of 24 months. We reviewed seizure histories, imaging reports, video-EEG monitoring data, operative records, and pathological findings. We analysed and compared the hyperkinetic semiology of video-recorded seizures of temporal lobe origin and those of frontal lobe origin.

Results. Forty hyperkinetic seizures in eight patients (seven adult patients and one 12-year-old patient) with temporal lobe epilepsy and 45 hyperkinetic seizures in nine patients (eight adult patients and one 16-year-old patient) with frontal lobe epilepsy were analysed. Emotional facial expressions (such as fear, laughing, or anger), bilateral forceful elbow flexion, bilateral forceful grasping, facial flushing, and bilateral facial contraction were observed significantly more frequently in seizures of frontal lobe origin. Oroalimentary automatisms, seizures during wakefulness, salivation, and bilateral drop of the corners of the mouth were observed significantly more frequently in seizures of temporal lobe origin.

Conclusions. Observation of a number of signs during hyperkinetic manifestations may help to predict whether a seizure originates from the frontal lobe or the temporal lobe.

Key words: epilepsy surgery, frontal lobe epilepsy, hyperkinetic, semiology, temporal lobe epilepsy

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Hyperkinetic seizures involve predominantly the proximal limb or axial muscles, producing irregular sequential ballistic movements, such as pedalling, pelvic thrusting, thrashing, rocking movements, and increase in the rate of ongoing movements or inappropriately rapid performance of a movement (Blume *et al*., 2001). Penfield and Erickson described a case of a violent seizure that was characterized by marked confusion, weeping, and violent struggling, followed apparently by amnesia (Penfield and Erickson, 1941). In this case, the patient had a scar, caused by a head injury in the right superior frontal and precentral gyri; stimulation at a triangular gyrus, lying anterior to the scarred area and including area 6a beta, reproduced the signs observed in the patient's habitual attacks. Such hyperkinetic seizures, which are characterized by violent movements, such as kicking of the arms and legs, rocking back and forth, struggling, and uncontrollable running, have been reported to be characteristic of frontal lobe epilepsy (Tharp, 1972; Geier *et al*., 1975; Ludwig *et al*., 1975; Williamson *et al*., 1985). However, some hyperkinetic seizures originate from the temporal lobe and other brain regions (Holthausen and Hoppe, 2000; Nobili *et al*., 2004; Ryvlin *et al*., 2006; Nishibayashi *et al*., 2009). These hyperkinetic seizures originating from brain regions other than the frontal lobe have been considered to be similar to those originating from the frontal lobe, and differences in semiology between hyperkinetic seizures of temporal lobe origin (TLHKSs) and hyperkinetic seizures of frontal lobe origin (FLHKSs) have not been extensively analysed.

Here, we show the differences in semiology between TLHKSs and FLHKSs. Our results may help in differentiating between the two.

Methods

Study design

This was a retrospective study approved by the institutional review board of the National Epilepsy Center (Shizuoka Institute of Epilepsy and Neurological Disorders). We searched the medical records at the National Epilepsy Center and analysed video recorded hyperkinetic seizures of patients with favourable postoperative seizure outcomes.

Selection criteria

Patients were included if they had the following:

– Temporal Lobe HyperKinetic Seizures (TLHKS) or Frontal Lobe HyperKinetic Seizures (FLHKS);

– long-term monitoring of video-scalp EEG recording or intracranial EEG recording, with capture of at least five analysable hyperkinetic seizures;

– postoperative Engel Class I outcome after a minimum follow-up of 24 months.

From a database of patients operated on for intractable focal epilepsy at the National Epilepsy Center between January 1983 and December 2014, we identified eight eligible patients who had suffered from TLHKSs (all seizures had been recorded during long-term intracranial EEGs). We also identified nine eligible patients who had suffered from FLHKSs from January 2001 to December 2014 (seizures in seven patients had been recorded during long-term intracranial EEG monitoring, and seizures in the remaining two had been recorded during long-term video-scalp EEG monitoring). There were six males and two females in the group with TLHKSs, and seven males and two females in the group with FLHKSs; there was no significant difference in sex between the two patient groups (*table 1*).

Three patients with TLHKSs (37.5%) had an epileptogenic zone on the left side and five patients (62.5%) on the right side, whereas five patients with FLHKSs (55.6%) had an epileptogenic zone on the left side and four patients (44.4%) on the right side; there was no significant between-group difference (p=0.64). The mean age at epilepsy onset in the temporal lobe epilepsy group was significantly older than that in the frontal lobe epilepsy group (13.3 ± 7.1 *versus* 4.1 ± 1.3 [mean ± SD]; p=0.003). Four TLHKS patients (50%) had daily seizures and four (50%) had weekly or monthly seizures, whereas six FLHKS patients (67%) had daily seizures and three (33%) had weekly or monthly seizures; there was no significant between-group difference (p=0.64). The eight patients with TLHKSs underwent anterior temporal lobectomy with mesio-temporal resection (n=4) (*figure 1A*), lesionectomy (n=2) (*figure 1B, D*), lateral temporal resection (n=1) (*figure 1C*), or total temporal lobectomy (n=1). No patients were diagnosed with mesial temporal lobe epilepsy. The nine patients with FLHKSs underwent frontal corticectomy (n=3) (*figure 1E-H, K, L*), prefrontal lobectomy (n=4) *(figure 1I, J*), or frontal lobectomy (n=2) (*figure 1M, N*) (*tables 2 and 3*). Histological examination of resected specimens of TLHKSs patients showed dysembryoplastic neuroepithelial tumour in four patients, focal cortical dysplasia in one, and non-specific lesions in three, whereas there was focal cortical dysplasia in all patients with FLHKSs (*tables 2, 3*). Findings based on patient images are also described in *tables 2*, *3*.

Seizure analysis

We reviewed the first five analysable recorded hyperkinetic seizures for each patient. Video recorded seizures were carefully reviewed in order to study the characteristics of hyperkinetic seizures and the presence or absence of accompanying signs. We evaluated

Table 1. Demographic background of patients with hyperkinetic seizures of temporal lobe or frontal lobe origin.

Demographic item	Temporal lobe seizure patients (*n* = 8)	Frontal lobe seizure patients (*n* = 9)	*p* value
Sex			
Female	2 (25.0%)	2 (22.2%)	1.00
Male	6 (75.0%)	7 (77.8%)	
Laterality of epileptogenic zone			
Left	3 (37.5%)	5 (55.6%)	0.64
Right	5 (62.5%)	4 (44.4%)	
Age at epilepsy onset, years	13 (5-27)	4 (2-6)	0.003
Age at surgery, years	28 (12-41)	27 (19-40)	0.56
Seizure frequency			
Daily	4 (50%)	6 (67%)	0.64
Weekly or monthly	4 (50%)	3 (33%)	

Categorical data are shown as numbers (percentages of samples) and continuous data are shown as mean (range).

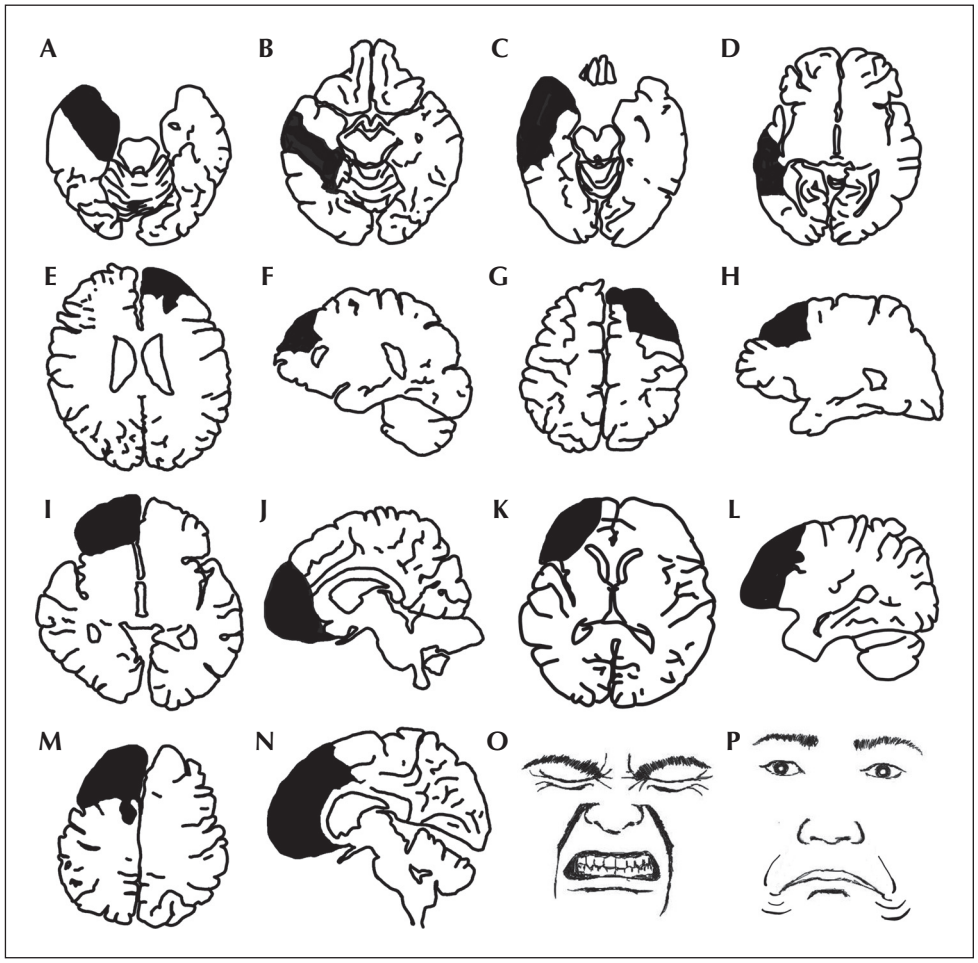

Figure 1. Resection areas in patients with hyperkinetic seizures of temporal lobe origin (A-D) or frontal lobe origin (E-N). Bilateral facial contraction (O) and bilateral drop of the corners of the mouth (P). Note that bilateral facial contraction is often accompanied by emotional expressions and forceful eye closure. Refer to the text and the tables for further details. These facial expressions were adopted and modified from drawings by Faigin (1992).

Table 2. Characteristics of patients with hyperkinetic seizures of temporal lobe origin.

Localization of epileptogenic zone	Age at epilepsy onset (years)	Age at surgery (years)	Preoperative imaging findings	Neurosurgical procedure	Histopathological results	Engel class
R temporal	16	38	CT: calcification in R uncus. MRI: high-intensity lesion in R temporal tip with R hippocampal atrophy	R ATL + mesio-temporal resection (*figure 1A*)	DNT	Ia
R temporal	11	16	MRI: high-intensity lesion in R basal and lateral temporal regions on T2WI	R ATL + mesio-temporal resection	DNT	Ia
L temporal	27	41	No lesion	L ATL + mesio-temporal resection	Non-specific	Ic
R temporal	5	12	CT: high-density lesion in R medial temporal lobe. MRI: high-intensity lesion in R mesio-temporal lobe with R hippocampal atrophy on T2WI	R Mesio-temporal lesionectomy (*figure 1B*)	DNT	Ia
L temporal	15	30	MRI: high-intensity cystic lesion in L middle temporal gyrus on T2WI	L total temporal lobectomy	Non-specific	Ia
R temporal	16	34	No lesion	R lateral temporal resection (*figure 1C*)	Non-specific	Ia
R temporal	11	29	MRI: high-intensity lesion in R middle temporal gyrus on FLAIR	R posterolateral temporal lesionectomy (*figure 1D*)	DNT	Ia
L temporal	5	27	No lesion	L ATL + mesio-temporal resection	FCD IIb	Ia

ATL: anterior temporal lobectomy; CT: computed tomography; DNT: dysembryoplastic neuroepithelial tumour; FCD: focal cortical dysplasia; FLAIR: fluid attenuated inversion recovery; L: left; MRI: magnetic resonance imaging; R: right; T2WI: T2 weighted image.

Table 3. Characteristics of patients with hyperkinetic seizures of frontal lobe origin.

Localization of epileptogenic zone	Age at epilepsy onset (years)	Age at surgery (years)	Preoperative imaging findings	Neurosurgical procedure	Histopathological results	Engel class
L frontal	3	40	MRI: high-intensity area around L superior frontal sulcus on FLAIR	L frontal corticectomy (*figure 1E, F*)	FCD type IIb	Ia
L frontal	5	29	MRI: high-intensity area around L superior frontal sulcus and middle frontal gyrus on FLAIR	L frontal corticectomy (*figure 1G, H*)	FCD type IIb	Ia
R frontal	4	25	MRI: high-intensity area in R orbitofrontal region on FLAIR	R prefrontal lobectomy (*figure 1I, J*)	FCD type IIb	Ia
L frontal	4	35	MRI: high-intensity lesion in L cingulate gyrus and L frontal operculum on T2WI and FLAIR	L frontal lobectomy	FCD type IIb	Ia
R frontal	5	28	MRI: high-intensity lesion in R prefrontal region on T2WI	R prefrontal lobectomy	FCD type IIb	Ia
L frontal	3	19	MRI: abnormal sulcus with thickening of cortex in L superior frontal gyrus	L prefrontal lobectomy	FCD type IIa	Ia
L frontal	5	25	No lesion	L prefrontal lobectomy	FCD type IIa	Ia
R frontal	2	20	MRI: gyral malformation with thickening of grey matter in R inferior frontal gyrus and operculum	R frontal corticectomy (*figure 1K, L*)	FCD type IIb	Ia
R frontal	6	23	MRI: high-intensity area, mainly in R anterior cingulate gyrus on FLAIR	R frontal lobectomy (*figure 1M, N*)	FCD type IIb	Ia

FCD: focal cortical dysplasia; FLAIR: fluid attenuated inversion recovery; L: left; MRI: magnetic resonance imaging; R: right; T2WI: T2 weighted image.

the clinical signs in 40 TLHKSs and 45 FLHKSs (all clinical signs and results are given in *Appendix tables 1-3*). Ictal emotional facial expressions were analysed by visual inspection. Auras were investigated by reviewing the medical records of each patient.

Statistical analysis

Statistical analyses were performed to determine which clinical signs had frequencies that differed significantly between TLHKSs and FLHKSs. Fisher's exact test was used to test differences between categorical variables, and the Mann-Whitney U test was used to compare groups with regards to ordinal or continuous variables, with a level of significance at $p<0.05$.
Calculations were performed using JMP 13 (SAS Institute Inc., Cary, NC, USA).

Results

A total of 40 TLHKSs were analysed in eight patients and 45 FLHKSs were analysed in nine patients. Because the ictal semiology varied slightly across hyperkinetic seizures in each patient, we analysed the clinical signs not per patient, but per seizure. The results are presented in *table 4* and *Appendix tables 1-3*.
Based on the analysis, emotional facial expression (fear, laughing, or anger) (21/38 *vs* 39/44; $p=0.001$), bilateral forceful elbow flexion (6/39 *vs* 23/44; $p<0.001$), bilateral forceful grasping (0/38 *vs* 9/41; $p=0.003$), flushing (7/26 *vs* 31/43; $p<0.001$), and bilateral facial contraction (11/40 *vs* 41/45; $p<0.001$) (*figure 1O*) were observed significantly more frequently in FLHKSs than in TLHKSs, whereas oroalimentary automatism (8/39 *vs* 1/42; $p=0.01$), salivation (5/28 *vs* 0/43; $p=0.008$), and bilateral drop of the corners of the mouth (12/40 *vs* 4/45; $p=0.02$) (*figure 1P*) were observed significantly more frequently in TLHKSs than in FLHKSs, respectively (*table 4*). Because truncal movements consisted of rocking back and forth, rotating, and pelvic thrusting -in a random fashion- in many seizures, we were unable to analyse these features statistically. Before seizure onset, for more than half of the TLHKSs, the patient had been awake, whereas only eight of the 45 FLHKSs occurred during wakefulness (22/40 *vs* 8/45, respectively; $p<0.001$) (*table 4*).
The latency from the onset of hyperkinetic seizures to the beginning of vocalization, such as screaming, groaning, speaking, and laughing, was significantly shorter for TLHKSs ($n=36$) than FLHKSs ($n=36$) (2.6±3.8 *vs* 4.8±4.3 [mean±SD], respectively; $p=0.01$) (*table 4*). Although the latency from the beginning of initial manifestation to the onset of hyperkinetic manifestations tended to be longer in TLHKSs ($n=40$) than FLHKSs ($n=45$), the difference was not statistically significant (11.6±20.2 *vs* 5.3±7.0 [mean±SD], respectively; $p=0.55$) (*Appendix table 3*). The latency from the onset to the end of the hyperkinetic seizure did not differ between the two groups of TLHKSs ($n=39$) and FLHKSs ($n=45$) (32.7±16.6 *vs* 28.4±19.5 [mean ± SD], respectively; $p=0.06$) (*Appendix table 3*). The results for the latency from the end of the hyperkinetic seizures to the beginning of communication are shown only as

Table 4. Video-recorded semiological features of hyperkinetic seizures of temporal lobe and frontal lobe origin.

Video-recorded semiological feature	Temporal lobe seizures (*n*)	Frontal lobe seizures (*n*)	*p* value
Emotional facial expression	21/38	39/44	0.001
Oroalimentary automatism	8/39	1/42	0.01
Bilateral forceful elbow flexion	6/39	23/44	<0.001
Bilateral forceful grasping	0/38	9/41	0.003
Seizure when awake	22/40	8/45	<0.001
Facial flushing	7/26	31/43	<0.001
Salivation	5/28	0/43	0.008
Bilateral facial contraction	11/40	41/45	<0.001
Bilateral drop of corners of the mouth	12/40	4/45	0.02
Latency from onset of HKS to vocalization (secs)	2.6 ± 3.8 (36)	4.8 ± 4.3 (36)	0.01

Categorical data are shown as number/number of samples that were evaluable on video. Data for latency are shown as mean ± SD (number of samples that were evaluable on video).

Table 5. Auras associated with hyperkinetic seizures of temporal and frontal lobe origin.

Aura	Temporal lobe origin (*n*=8)	Frontal lobe origin (*n*=9)	*p* value
Visual	1	0	0.47
Auditory	1	0	0.47
Visceral	1	0	0.47
Emotional	0	3	0.21
Unclassifiable	3	3	1.00
None	2	3	1.00

a reference, because in many seizures there was no intervention by a physician, nurse, or family member (*Appendix table 3*). As shown in *Appendix tables 1-3*, the incidences of many other signs did not differ significantly between TLHKSs and FLHKSs.

We also investigated auras on a per-patient basis (*table 5*). Visual auras (blurred vision) (1/8 *vs* 0/9; p=0.47), auditory aura (auditory hallucination) (1/8 *vs* 0/9; p=0.47), and visceral sensation (choking sensation or a sensation of squeezing in the abdomen) (1/8 *vs* 0/9; p=0.47) were observed in patients with TLHKSs but not in patients with FLHKSs, whereas emotional auras (fear in two patients, and a startled feeling in one patient) were observed in patients with FLHKSs but not in patients with TLHKSs (0/8 *vs* 3/9, respectively; p=0.21). The two patients with fear auras exhibited frightened facial expressions during their seizures, whereas the patient with a startled aura had a fierce look or laughed during seizures.

Discussion

Although the incidence of many clinical signs during hyperkinetic seizures did not differ significantly between temporal lobe epilepsy and frontal lobe epilepsy, we found that four signs were observed more frequently in TLHKSs and five others were observed more frequently in FLHKSs (*table 4, Appendix tables 1-3*).

Nevertheless, the lack of significant difference in the rates of occurrence of many signs between TLHKSs and FLHKSs (*Appendix tables 1-3*) suggests that the two have the same hyperkinetic symptomatogenic zones (Bartolomeil *et al*., 2002). Previous studies have reported that hyperkinetic behaviour appears when ictal discharge in the temporal lobe spreads to extratemporal structures, such as the cingulate gyrus, supplementary motor area, and orbitofrontal area (Nobili *et al*., 2004; Ryvlin *et al*., 2006).

Emotional facial expression was observed more frequently in FLHKSs than in TLHKSs. Furthermore, emotional auras, such as fear and startled sensation, were observed in patients with FLHKSs but not in those with TLHKSs (*table 5*). Bancaud and Talairach commented that anterior cingulate gyrus seizures are accompanied by intense fright, with a facial expression of fear (Bancaud and Talairach, 1992). Two of our three FLHKS patients with emotional auras had lesions in the cingulate gyrus on MRI. Bartolomei *et al*. reported that intense emotional alterations, such as intense agitation, screaming, and facial expressions of rage, fear, or anger, were associated with a loss of synchrony between the orbito-frontal cortex and the amygdala (Bartolomei *et al*., 2005). Our results suggest that such disruption of functional connections occurs more frequently in FLHKSs than in TLHKSs. Our patients with TLHKSs did not have auras of fear. Gil-Nagel and Risinger reported auras of fear in hippocampal temporal lobe seizures (HTSs), but not in extrahippocampal temporal lobe seizures (ETSs) (Gil-Nagel and Risinger, 1997). The fact that all of our patients with TLHKSs had ETSs may therefore explain why they had no auras of fear.

Oroalimentary automatisms are commonly observed in patients with temporal lobe epilepsy and are considered indicative of mesial temporal lobe involvement, and especially involvement of the amygdala (Wieser, 1983; Engel *et al*., 2008). A previous study found that hyperkinetic behaviour is followed by typical oral automatisms in six of 12 patients with TLHKSs (Carreño *et al*., 2005). Epileptic discharges in TLHKSs may more easily propagate to the mesial temporal lobe and cause oral automatisms than those in FLHKSs. Another explanation is that a transient dysfunction of higher cortical centres by epileptic seizures can release the brainstem masticatory central pattern generators (CPG) responsible for oroalimentary automatisms that are normally inhibited by the cortical centres (Tassinari *et al*., 2009). Epileptic discharges in TLHKSs may more easily inhibit the mature neopallium that controls the brainstem masticatory CPG than those in FLHKSs.

Gardella *et al*. examined the prevalence of ictal grasping in FLHKSs, frontal lobe seizures other than FLHKSs, temporal lobe seizures, and extrafrontal/extratemporal seizures (Gardella *et al*., 2006). They reported that the prevalence of ictal grasping was significantly higher in FLHKSs than in other seizure groups. Irrepressible exploratory reaching/grasping movements can be elicited by direct electrical stimulation in the vicinity of the contralateral cingulate sulcus, from the anterior cingulate motor areas or pre-supplementary motor area (Chassagnon *et al*., 2008). As for bilateral forceful elbow flexion, electrical stimulation of the ventral banks of the cingulate cortex produces tonic flexion

of the contralateral wrist and elbow (Basha *et al*., 2013). We speculate that the epileptic discharges in FLHKSs can more easily affect the cingulate motor areas bilaterally via the corpus callosum than those in TLHKSs; there is thus more frequent bilateral grasping and forceful flexion of the elbows in FLHKSs than in TLHKSs. Since protective reflexes such as grasping are proposed to be carried out by subcortical CPG (Tassinari *et al*., 2009), ictal grasping may be a release phenomenon caused by loss of control of neocortical structures on the CPG.

TLHKSs have been reported to be associated with sleep (Nobili *et al*., 2004; Mai *et al*., 2005). Mai *et al*. reported that TLHKSs frequently occurred during sleep (Mai *et al*., 2005). However, 55% of TLHKSs occurred during wakefulness in our study; this was significantly more frequent than in FLHKSs (17.8%). TLHKSs are thus not always nocturnal.

As a paediatric autonomic sign in temporal lobe epilepsy and extratemporal lobe epilepsy, ictal flushing has been reported to be of no value for either lateralizion or localizion of seizure origins (Fogarasi *et al*., 2006). However, our results suggest that ictal flushing might be a useful localizing clue when differentiating between FLHKSs (in which it was more common) and TLHKSs.

Hypersalivation has been observed in patients with focal impaired awareness seizures of temporal lobe origin, as well in seizures of insular-opercular, orbitofrontal, or dorsolateral frontal origin (Delgado-Escueta *et al*., 1991; Satow *et al*., 2004; Shah *et al*., 2006; Proserpio *et al*., 2011). We observed hypersalivation only in TLHKSs, suggesting that it may be a clue for localization when comparing FLHKSs and TLHKSs.

Bilateral facial contraction was observed more frequently in FLHKSs than in TLHKSs. Souirti *et al*. investigated (mainly by using stereoencephalography) the anatomical neural network underlying ictal pouting, with the mouth turned downwards as a "*chapeau de gendarme*," in frontal lobe epilepsy (Souirti *et al*., 2014). Hypermotor seizures occurred in seven of their nine patients with ictal pouting, and vegetative signs, including rubefaction, were also seen. The authors concluded that ictal pouting is sustained by reciprocal mesial and lateral frontal interactions involved in emotional and cognitive processes, in which the anterior cingulate cortex plays a pivotal role. Bilateral drop of the corners of the mouth was observed more frequently in TLHKSs than in FLHKSs, although we could not find any description about this symptom in the literature. During bilateral drop of the corners of the mouth, patients closed their mouth and opened their eyes with no apparent emotional expression, whereas during bilateral facial contraction, the facial muscles were strongly contracted with deep nasolabial folds, sometimes with firmly closed eyes as part of the strong facial contraction. It seems that there is emotional involvement in most of the seizures with bilateral facial contraction during HKS.

Ictal vocalization occurred in not only FLHKSs, but also TLHKSs (*Appendix table 1*). The latency from the onset of hyperkinetic seizures to the beginning of vocalization (such as screaming, groaning, speaking, or laughing) was significantly shorter in TLHKSs than in FLHKSs (*table 4)*. In some cases, vocalization began earlier than hyperkinetic manifestations in TLHKSs. The significance of the earlier onset of vocalization in TLHKSs is unclear, and further studies are needed to clarify this.

Although the sample sizes were small, visual and auditory auras and visceral sensations were observed in patients with TLHKSs but not in those with FLHKSs (*table 5*). Auditory and visual hallucinations are more common in ETSs than in HTSs, whereas epigastric symptoms are more common in HTSs (O'Brien *et al*., 1996; Gil-Nagel and Risinger, 1997). Because all of our patients with TLHKSs had ETSs, it is not surprising that auditory or visual hallucinations were observed in our patients with TLHKSs. Although auditory and visual hallucinations have been reported in patients with frontal lobe epilepsy, they are not common in this condition, and our patients with FLHKSs did not experience them (La Vega-Talbot *et al*., 2006; Ferri *et al*., 2014). One patient with TLHKSs experienced visceral sensations as auras, suggesting that the seizure spreads to the mesial temporal region or the insular cortex -where viscerosensitive responses have been reported to be evoked by stimulation- at an early stage (Ostrowsky *et al*., 2000).

Gibbs *et al*. reported that the mean duration of clinically observable ictal manifestations and the clinically observable delay between the first video-detectable movement and onset of hypermotor manifestations were significantly shorter in frontal sleep-related hypermotor epilepsy (SHE) than in extrafrontal SHE (Gibbs *et al*., 2018). Although there were no statistically significant differences, the duration of HKS and the latency from initial manifestation to HKS tended to be shorter in FLHKSs than in TLHKSs in our study.

To our knowledge, this is the first study to have evaluated and compared the semiology of TLHKSs and FLHKSs. TLHKSs are rare; they were observed in one or two of 23 patients with hyperkinetic seizures (Holthausen *et al*., 2000; Tao *et al*., 2010). Conversely, in patients with temporal lobe epilepsy, hyperkinetic seizures were observed in 12 of 502 patients (Carreño *et al*., 2005). This low prevalence of TLHKSs probably makes it difficult to evaluate the semiology of hyperkinetic seizures for the purpose of distinguishing between TLHKSs and FLHKSs. Here, we found

only eight patients with TLHKSs who had postoperative Engel Class I outcome. We intend to confirm our results in a larger study.

In summary, we identified several differences in semiology between TLHKSs and FLHKSs. Emotional facial expression, bilateral facial contraction, bilateral forceful elbow flexion, bilateral forceful grasping, and facial flushing were more commonly associated with FLHKSs, whereas bilateral drop of the corners of the mouth, oroalimentary automatism, salivation, and diurnal seizures were more commonly associated with TLHKSs. These findings, which remain to be confirmed in a larger study, might help to infer the epileptogenic zone in hyperkinetic seizures, in particular, in non-lesional cases. □

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

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TEST YOURSELF

(1) Does the semiology of hyperkinetic seizures of temporal lobe origin differ from that of frontal lobe origin?

(2) What signs are more frequent in seizures of frontal lobe origin?

(3) What signs are more frequent in seizures of temporal lobe origin?

Note: Reading the manuscript provides an answer to all questions. Correct answers may be accessed on the website, www.epilepticdisorders.com, under the section "The EpiCentre".

Annexe Table 1. Video-recorded semiological complex behaviour during hyperkinetic seizures of temporal lobe and frontal lobe origin.

VIDEO-RECORDED SEMIOLOGICAL FEATURE	Temporal lobe seizures (*n*)	Frontal lobe seizures (*n*)	*p* value
Nonversive head turning			
Contralateral to epileptogenic zone	1/40	1/45	1.00
Ipsilateral to epileptogenic zone	8/40	5/45	0.37
Forceful eye closure	13/40	18/44	0.50
Vocalization	36/40	36/45	0.24
Screaming or groaning	29/40	34/45	0.24
Speaking	5/40	1/45	0.10
Laughing	2/40	1/45	0.60
Emotional facial expression			
Fearful facial expression	21/38	39/44	0.001
Mouth opening and closing	10/38	22/44	0.04
Mainly opening	13/39	24/45	0.08
Mainly closing	13/39	7/45	0.07
Oroalimentary automatism	8/39	1/42	0.01

Categorical data are shown as number/number of samples that were evaluable on video.

Annexe Table 2. Video-recorded semiological complex behaviour during hyperkinetic seizures of temporal lobe and frontal lobe origin.

VIDEO-RECORDED SEMIOLOGICAL FEATURE	Temporal lobe seizures (*n*)	Frontal lobe seizures (*n*)	*p* value
Laterality of movements of the upper extremities			
Contralateral to epileptogenic zone	2/40	8/45	0.09
Ipsilateral to epileptogenic zone	12/40	5/45	0.06
Laterality of movements of the lower extremities			
Contralateral to epileptogenic zone	1/40	6/45	0.11
Ipsilateral to epileptogenic zone	5/40	7/45	0.76
Forceful flexion of the elbow			
Bilateral	6/39	23/44	<0.001
Unilateral			
Contralateral to epileptogenic zone	6/39	1/44	0.048
Ipsilateral to epileptogenic zone	2/39	2/44	1.00
Forceful grasping			
Bilateral	0/38	9/41	0.003
Unilateral			
Contralateral to epileptogenic zone	2/38	4/41	0.68
Ipsilateral to epileptogenic zone	4/38	2/41	0.68
Pedalling	2/40	0/45	0.22
Kicking	8/40	10/45	1.00
Rhythmic repetitive movements of trunk and limbs	13/40	13/45	0.81

Categorical data are shown as number/number of samples that were evaluable on video.

Annexe Table 3. Video-recorded semiological features during hyperkinetic seizures of temporal lobe and frontal lobe origin.

VIDEO-RECORDED SEMIOLOGICAL FEATURE	Temporal lobe seizures (*n*)	Frontal lobe seizures (*n*)	*p* value
Seizure when awake	22/40	8/45	<0.001
Initial manifestation (+) before HKSs	29/40	32/45	1.00
Facial flushing	7/26	31/43	<0.001
Salivation	5/28	0/43	0.008
Head version	0/40	1/45	1.00
Bilateral facial contraction	11/40	41/45	<0.001
Bilateral drop of corners of the mouth	12/40	4/45	0.02
Facial clonic movement			
Contralateral to epileptogenic zone	0/40	0/45	No result
Ipsilateral to epileptogenic zone	2/40	8/45	0.09
Dystonic posturing			
Bilateral	0/40	5/44	0.06
Unilateral			
Contralateral to epileptogenic zone	8/40	3/44	0.11
Ipsilateral to epileptogenic zone	2/40	0/44	0.22
Tonic movement of extremities	1/40	0/45	0.47
Clonic movement of extremities	1/40	0/45	0.47
Latency from initial manifestation to HKS (secs)	11.6 ± 20.2 (40)	5.3 ± 7.0 (45)	0.55
Latency from onset of HKS to vocalization (secs)	2.6 ± 3.8 (36)	4.8 ± 4.3 (36)	0.009
Latency from onset of HKS to end of seizure (secs)	32.7 ± 16.6 (39)	28.4 ± 19.5 (45)	0.06
Latency from end of HKS to communication (secs)	26.2 ± 21.0 (19)	17.8 ± 30.8 (32)	0.01

Categorical data are shown as number/number of samples that were evaluable on video. Data for latency are shown as mean ± SD (number of samples that were evaluable on video). HKS: hyperkinetic seizure.