■ Original article

Epileptic Disord 2022; 24 (3): 517-530

Epileptic Disorders

Somatomotor or somatosensory facial manifestations in patients with temporo-basal epilepsies

Delphine Taussig[1], Olivier David[2,3], Ana Maria Petrescu[1], Anca Nica[4], Eric Seigneuret[5], Georg Dorfmüller[6], Mohamed Choukri[7], Nozar Aghakhani[7], Viviane Bouilleret[1,8]

[1] Université Paris Saclay-APHP, Neurophysiologie et Epileptologie, Le Kremlin Bicêtre, France
[2] Univ. Grenoble Alpes, Inserm, GIN, Grenoble Institut des Neurosciences, 38000, Grenoble, France
[3] Aix Marseille Univ, Inserm, INS, Institut de Neurosciences des Systèmes, Marseille, France
[4] Service de Neurologie, CHU de Rennes, France
[5] Service de Neurochirurgie, CHU de Grenoble, France
[6] Hôpital Fondation Rothschild, Neurochirurgie pédiatrique, Paris, France
[7] Université Paris Saclay-APHP, Neurochirurgie, Le Kremlin Bicêtre, France
[8] Université Paris-Saclay, CEA, CNRS, Inserm, BioMaps, Orsay, France

Received May 14, 2021; Accepted December 18, 2021

ABSTRACT

Objective. The semiology of temporo-basal epilepsy has rarely been analysed in the literature. In this paper, we report three patients with proven basal temporal epilepsy with somatomotor or somatosensory facial ictal semiology, highly suggestive of insulo-opercular onset.

Methods. The three patients had a temporobasal lesion and their drug-resistant epilepsy was cured with resection of the lesion (follow-up duration: 7-17 years). We reviewed the medical charts, non-invasive EEG data as well as the stereoelectroencephalography (SEEG) performed in two patients. Quantitative analysis of ictal fast gamma activity was performed for one patient.

Results. Early ictal features were orofacial, either somatomotor in two patients or ipsilateral somatosensory in one. The three patients had prior sensations compatible with a temporal lobe onset. Interictal and ictal EEG pointed to the temporal lobe. The propagation of the discharge to the insula and operculum before the occurrence of facial features was seen on SEEG. Facial features occurred 7-20 seconds after electrical onset. Quantitative analysis of six seizures in one patient confirmed the visual analysis, showing statistically significant fast gamma activity originating from basal areas and then propagating to insulo-opercular regions after a few seconds.

Significance. We report three cases of lesional temporo-basal epilepsy responsible for orofacial semiology related to propagation of insulo-opercular ictal discharge. In MRI-negative patients with facial manifestations, this origin should be suspected when EEG is suggestive. These observations may contribute to our understanding of brain networks.

Key words: SEEG, localization, ictal semiology, epilepsy surgery, epileptogenicity map

• **Correspondence:**
Delphine Taussig
Université Paris Saclay-APHP, CHU Bicêtre,
Unité de neurophysiologie clinique et d'épileptologie (UNCE)
<delphine.taussig@aphp.fr>

doi:10.1684/epd.2022.1413

Elementary orofacial signs are encountered in operculo-insular epilepsy, notably in the central part of this complex [1, 2]. They are often associated with laryngeal signs. We report three patients with elementary orofacial features related to temporo-basal epilepsy. All of them had a lesion localized to the occipito-temporal gyrus. The objective was to study whether early insulo-opercular propagation could account for this semiology.

Methods

Three patients with proven basal temporal epileptogenic zones and ictal semiology, highly suggestive of operculo-insular epilepsy, were identified in three tertiary epilepsy surgery centres. All three suffered from drug-resistant epilepsy and underwent a comprehensive presurgical investigation including the collection of clinical, MRI and long-duration scalp video-electroencephalogram (EEG) data; the recording of all types of seizures being a requirement. The presurgical procedure included an ^{18}F-FDG positron emission tomography (PET) scan in one patient only. A seizure conference was held for each patient to decide on surgery or SEEG, and in those cases, an implantation plan was designed. The SEEG results were also discussed in a multidisciplinary meeting. Surgery was performed based on the decision from the seizure conference. Seizure outcome was assessed using Engel scoring [3].

We retrospectively reviewed the clinical, EEG and SEEG data and performed quantitative analyses.

Epileptogenicity mapping from SEEG recordings, sampled at 500 Hz and co-registered to the pre-operative MRI, was performed for Case 2 in whom six seizures were recorded [4]. First, ictal onset was manually positioned to the first fast discharge that could be detected. Second, visual inspection of the time-frequency power averaged over seizures showed that the earliest ictal discharges occurred in the 80-180-Hz frequency band. Third, significant power in this frequency band, in a sliding time window with 2-second width spanning the first 20 seconds of each seizure, was detected at all channels by comparing ictal power to the baseline chosen between 30 and 10 seconds before ictal onset. Finally, group statistics of ictal high gamma power were then obtained using fixed-effect group analysis based on seizures, and a propagation map, indicating the latency of occurrence of significant (p<0.001) high gamma group statistics over peri-onset time, was computed to visualise seizure propagation (see [4] for further methodological details).

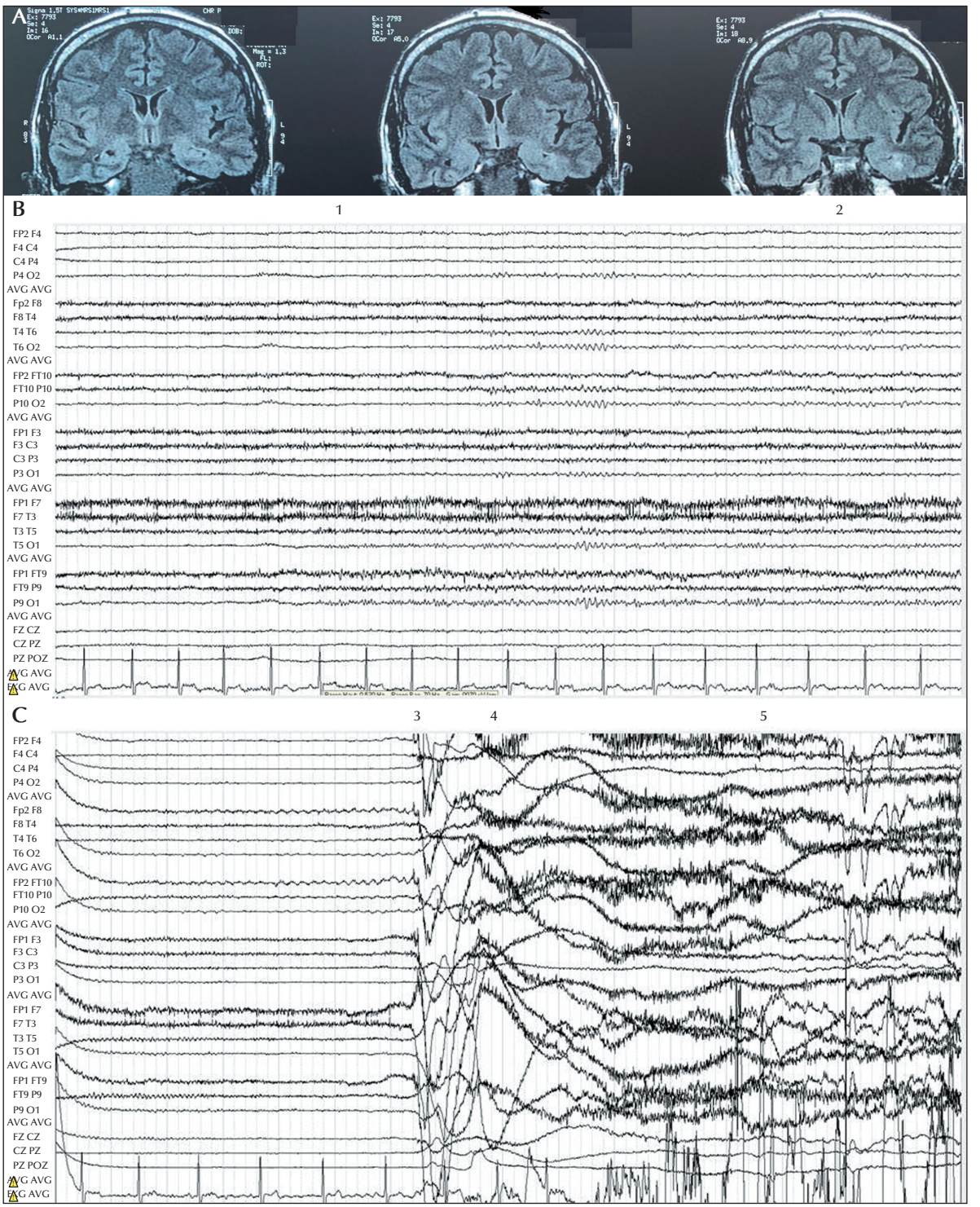
Results

The main non-invasive data are summarized in *table 1*. After non-invasive investigation, Patient 3 underwent a lesionectomy based on satisfactory electroclinical correlations while Patients 1 and 2 underwent SEEG due to unclear correlations between the electroclinical features and the lesion. In the following, we provide further details about the electroclinical data. Patient 1 felt a strange feeling described as a dream or an inability to think, lasting one to two seconds. During scalp EEG, five seizures were recorded. For type 1 (four seizures), the patient became bradycardic, had rapid eyelid movements and smiled at the end. For type 2 seizures, he had bradycardia and immediately afterwards had a contraction of the right side of his mouth, agitation with rubefaction and a smile followed by a laugh. He never had post-ictal aphasia. On EEG recorded with the 10-20 system and supplementary temporo-basal electrodes, all seizures were characterized by a left temporal fast discharge, predominant on the anterior part, followed by bilateral anterior and basal temporal theta activity which was more pronounced on the left (*figure 1B, C*). The ictal semiology suggested involvement of the anterior insula (based on the bradycardia and agitation) and the anterior cingulate (based on the smile and occasional laughter, the agitation and the rubefaction). The frontal operculum was believed to play a key role because of the early facial contraction as well and the mirth and laughter. The frontobasal region, that accounts for less specific semiology, could also be a candidate area, because of its strong connections with the previously discussed areas. However, because of the initial fast discharge, the interictal abnormalities and the MRI results, a temporal onset could not be excluded. The aim with SEEG was to explore this last hypothesis, by possibly excluding a frontal or insular epileptogenic zone. During SEEG, the electrodes were used to explore the whole temporal lobe, including the lesional and perilesional cortices, the orbital region, the middle frontal gyrus, the insula (two electrodes) and the frontal operculum. *Figure 1A* shows the lesion and *figure 1D* the electrode implantation. There were continuous slow waves and spikes from the contacts exploring the lesion, highly suggestive of a focal cortical dysplasia, and a few spikes from the other electrodes. One type 1 seizure and two type 2 seizures were recorded. In the type 1 seizure, the ictal discharge started at the contacts exploring the lesion with propagation to the temporal electrodes (*figures 1E, F*). In the type 2 seizures that lasted 35 seconds, beta activity was recorded at the onset at the contacts exploring the lesion. In the following seconds, the discharge slowed down with propagation to the amygdala and hippocampus. The facial contraction occurred less than one second after the clinical onset, immediately after a reacceleration from the contacts exploring the lesion with propagation manifesting as fast discharge on the insula and two seconds afterwards on the frontal operculum (*figures 1G, H*). Electrical stimulations were performed with the following parameters: 50 Hz frequency, impulse duration of 0.5 milliseconds and duration of 5 seconds. These elicited type 2 seizures when stimulating within the lesion (Lp6-Lp7, Lp7-Lp8 and Bp5-Bp at 6 1.6 mA)

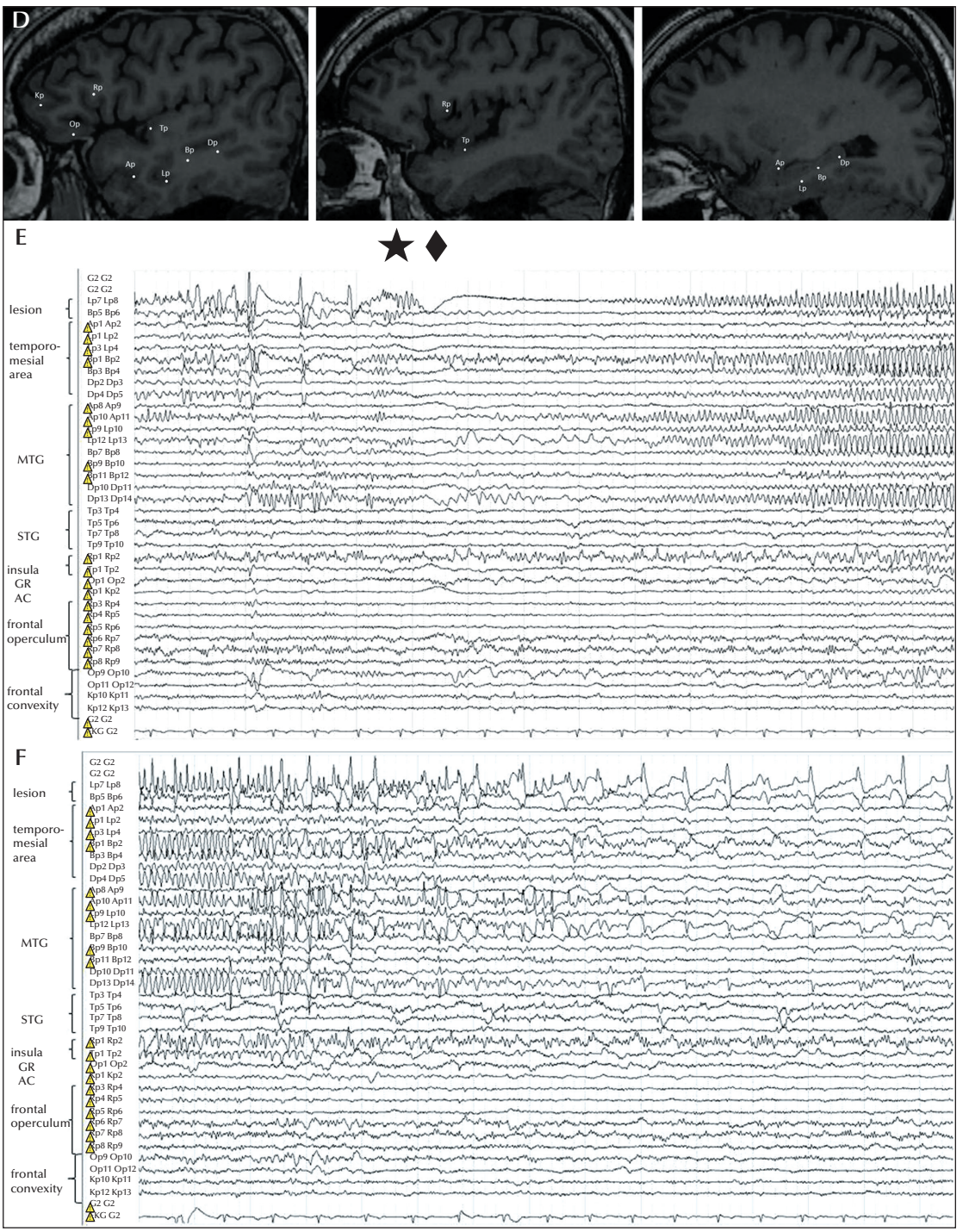
▼ **Table 1.** Clinical data.

Patient	1	2	3
Sex	M	M	M
Manual dominance	R handed	R handed	R handed
Age at investigation (years)	40	14	43
Age at onset (years)	9	7	37
Epilepsy duration (years)	31	7	6
Seizure frequency	1 seizure/night	Clusters twice a month	Daily seizures
Medical history	None	None	Crohn's disease
Seizure semiology	Brief sensation then contraction of the right side of the mouth and motor agitation. Previous fast occurring tonic-clonic generalisation.	Several initial sensations: either sees a familiar scene, feels a contraction of the jaw, or feels that his R hand no longer moves normally, followed by tachycardia, contraction and clonic movement of the mouth and limb contractions.	Fear, tachycardia and heat sensation of the face, the R side of the neck and the R upper limb, followed by inconstant speech arrest.
Non-invasive investigation			
Interictal EEG	Frequent theta and delta waves and spikes in the L temporal anterior and basal region.	Frequent theta and delta waves and spikes in the R temporal anterior and basal region.	No abnormalities
Ictal EEG	5 recorded seizures. L temporal fast discharge starting in the anterior region; during the course of the seizure, bilateral temporal anterior and basal temporal theta discharge is evident.	Recording of a cluster of 10 seizures: -8 seizures with R temporal discharge after clinical onset; -2 seizures with R temporal discharge before clinical onset.	4 recorded seizures. Attenuation of R background activity then R temporal posterior rhythmic theta activity.
MRI	Lesion in the L occipito-temporal gyrus. FCD?	Lesion in the R occipito-temporal gyrus. FCD?	Cavernoma in the posterior part of the R occipito-temporal gyrus.
PET-scan	ND	Hypometabolism within the lesion	ND
Surgery			
Type	Lesionectomy	Lesionectomy	Lesionectomy
Follow-up	17 years	10 years	7 years
Outcome	Ia	Ia	Ia
Pathology	FCD IIb	DNET	cavernoma

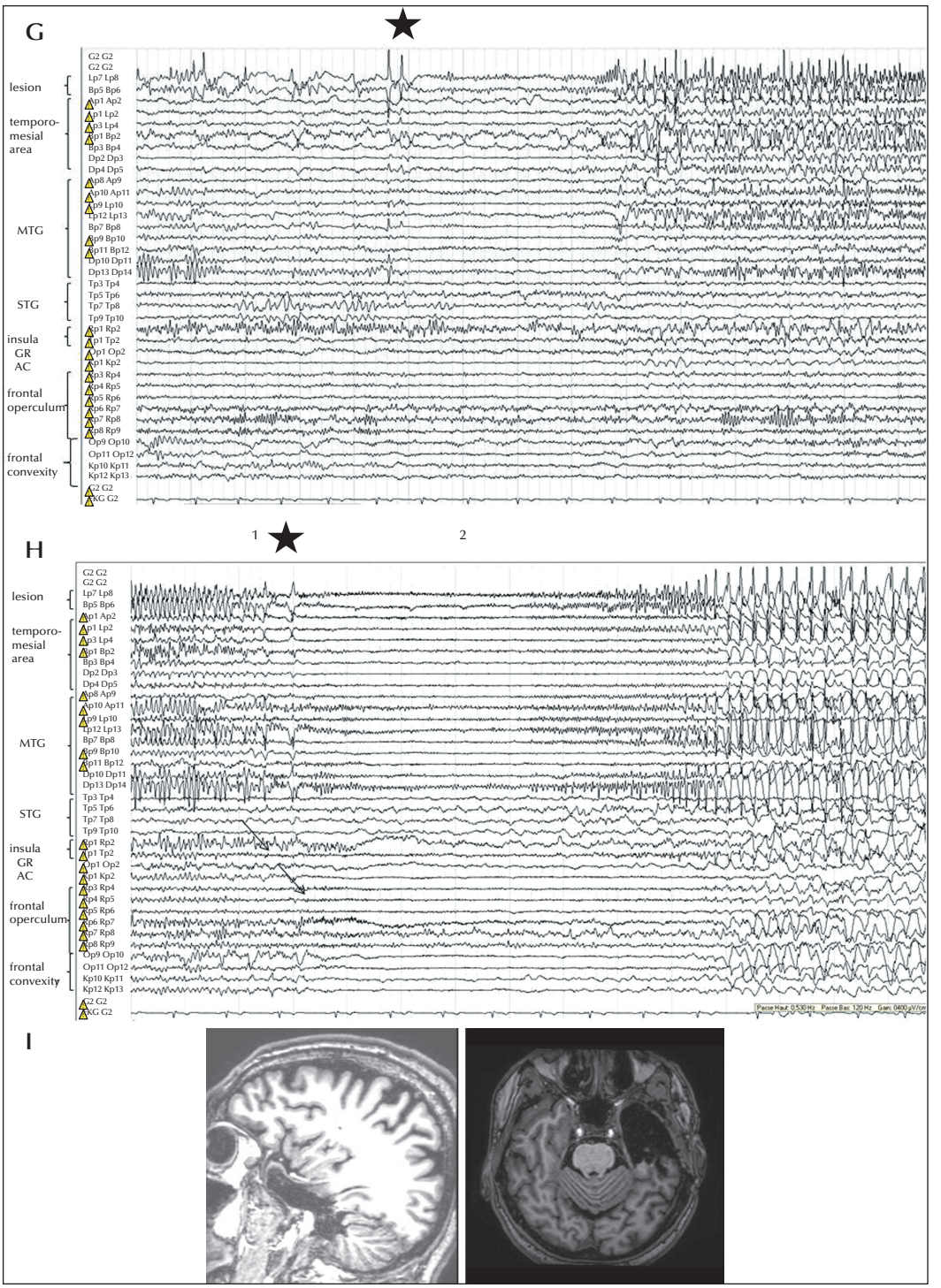
DNET: dysembryoplastic neuroepithelial tumour; FCD: focal cortical dysplasia; L: left; R: right; ND not done.



■ **Figure 1.** Patient 1. (A) Preoperative MRI (coronal view; T2 weighted sequences). (B, C) Scalp EEG (10-20 system with supplementary temporo-basal electrodes; longitudinal montage; amplitude: 150 µV/cm, low-pass filter: 70 Hz, high-pass filter: 0.530 Hz). (B) Seizure onset with electrical onset showing left temporal rhythmic activity from temporal anterior and basal electrodes (1) and decrease in heart frequency (2). (C) Seizure continuation with opening of the eyes 19 seconds after the electrical onset (3), right facial contraction (4) and left temporal delta activity (5).



■ **Figure 1.** (D) Electrode implantation (left panel: lateral view; right panel: insular view; lower panel: hippocampal view). (E-H) SEEG recording (amplitude: 400 µV/cm, low-pass filter: 120 Hz, high-pass filter: 0.530 Hz). MTG: middle temporal gyrus; STG: superior temporal gyrus; GR: gyrus rectus; AC: anterior cingulate. (E, F) Type 1 seizure. (E) Onset showing beta activity from the contacts exploring the lesion (Lp7-Lp8) (star) followed by a fast discharge at the same contacts (diamond) with propagation in the shape of a spike discharge from the temporal electrodes. The patient does not provide any warning, does not answer immediately during the discharge and describes having a brief non-specific sensation after the seizure. (F) Seizure continuation and end (*continued*).



■ **Figure 1.** (G, H) Type 2 seizure. (G) Electrical onset (star) showing beta activity from the contacts exploring the lesion. In the following seconds, the discharge slows down with propagation to the amygdala and hippocampus. (H) Seizure continuation, 14 seconds after the electrical onset, corresponding to clinical onset (eyes open and slowing of heart rate) (1) and facial contraction (17 seconds after electrical onset) (2). Immediately after the clinical onset, the ictal discharge speeds up from the contacts exploring the lesion (star) with a simultaneous fast discharge on the insula and frontal operculum (arrows). (I) Post-operative MRI (*continued*).

or the parahippocampal gyrus (Lp3-Lp4 at 1.6 mA). No other stimulation evoked a seizure. Stimulation of the insula did not cause a response elicited at Tp1-2 (intensity up to 3 mA) and stimulation of Rp1-Rp2 (3 mA) evoked a feeling of heaviness in the throat associated with a local afterdischarge lastingf 6 seconds. Stimulation of Rp3-Rp8 evoked speech arrest associated with a local afterdischarge lasting 2 to 8 seconds, confirming the location of the electrode in the pars triangularis.

Patient 2 described several auras: either he saw a familiar scene, he felt that his right hand was not moving freely, or he felt a contraction of his jaw. He reported most frequently no sensation. During scalp EEG, a cluster of 10 seizures was recorded. Five seizures occurred during wakefulness and five during sleep. When the patient was awake, it seemed as though he felt something, but he only twice reported a vague sensation in his mouth. This was followed by clonic movements of the mouth that appeared to be bilateral, but were lateralized to the left on two occasions. Afterwards, hypersalivation, rapid shaking of the right hand and occasional hypertonia of the right upper limb as well as dystonia of the left hand occurred. The seizures started with rhythmic right temporal delta activity which was visible before the clinical onset in eight seizures and afterwards in two (*figures 2B, C*). In four seizures, a left temporal discharge with suprasylvian propagation and another frequency different from the right discharge occurred during the course of the seizures. The sensation of seeing a familiar scene was highly suggestive of a temporomesial seizure. Interictal and ictal EEG were compatible with this diagnosis, but the objective ictal semiology pointed to an insulo-opercular onset. SEEG was performed based on the hypothesis of seizure onset within the lesion with emphasis on temporomesial involvement, but also with the aim of excluding insulo-opercular onset. SEEG was employed to explore the lesion on the right side, the whole temporal lobe, the occipital and parietal convexities, the insula (three electrodes with insular contacts), the frontal and motor operculum and the post-central gyrus. Because of some ictal features that were difficult to lateralize and the left discharge seen in four seizures, two contralateral electrodes were implanted: one facing the lesion and one insulo-opercular (*figure 2D*).

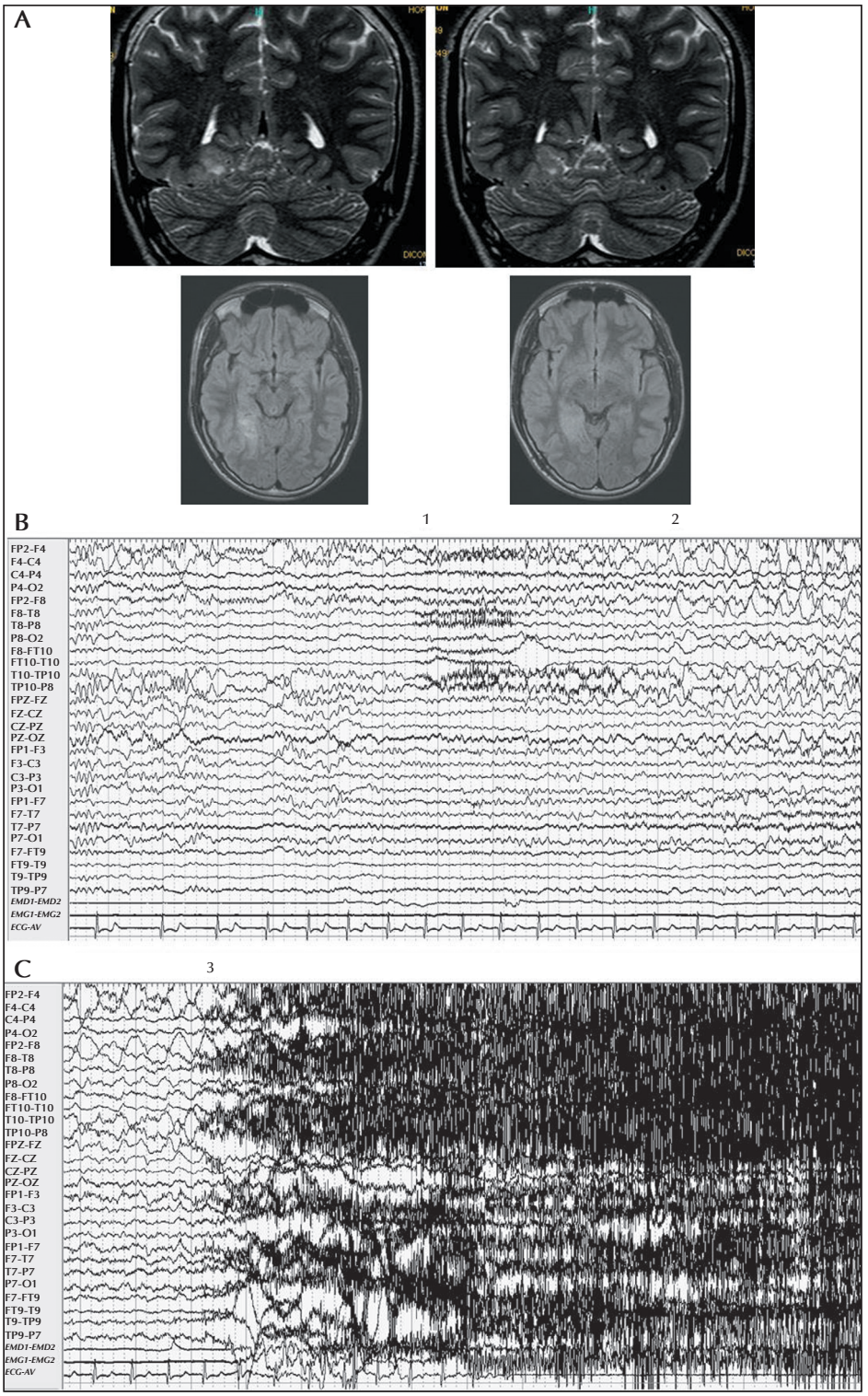
Spikes and beta bursts were detected from the contacts exploring the posterior part of the lesion (OI 1-2). There were also slow waves and spikes on the temporal pole and superior temporal sulcus. Polyspikes were detected during sleep from the contacts located in the right amygdala and hippocampus. A cluster of 13 seizures in three days was recorded. Only one occurred during wakefulness; the patient warned of the seizure but did not explain thereafter what he felt. For the 12 others out of sleep, the patient had no sensation. The seizures lasted, on average, one minute, and involved contraction of the mouth, followed by clonic movements of the face, throat-clearing sounds and dystonia of the right arm. The seizures started with a fast discharge from electrodes exploring the lesion (OI 1-2), rapidly propagating to the temporal pole (*figures 2E, F*). One second after the electrical onset, there was a fast discharge on the insula with rhythmic activity from the opercular electrodes. At the time of facial contraction, seven seconds after the electrical onset, theta activity was recorded from the contacts exploring the lesion, the amygdalo-hippocampal complex, the frontal and central operculum and the electrode facing the lesion on the left. The time-frequency analysis pointed to early insular involvement, four seconds after the electrical onset seen from the contacts exploring the lesion (*figure 3*).

Patient 3 reported a heat sensation on the face, the right side of the neck and the right upper limb. After the seizure, when asked what he felt, he described an associated fear, but never reported this during seizures. On scalp EEG, activity was modified on the right temporal region before a warning from the patient, followed by a build-up of right temporal posterior rhythmic activity. During the only seizure recorded with few muscle artefacts, suprasylvian propagation was visible (*figure 4*).

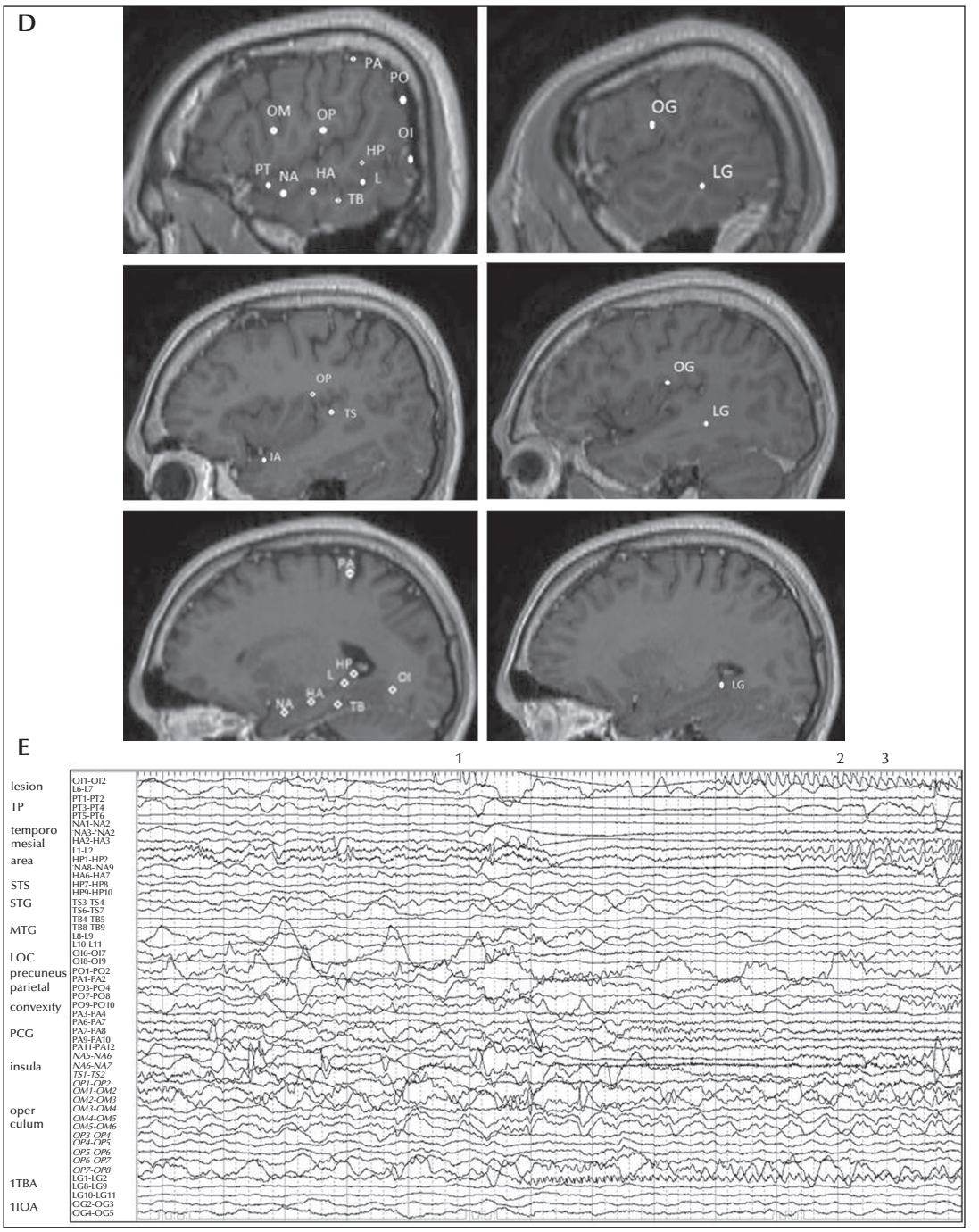
A lesionectomy was performed on all three patients. The pathology showed FCD type IIb in Patient 1, a dysembryoplastic neuroepithelial tumour in Patient 2 and a cavernoma in Patient 3. All of them have been seizure-free with 7 to 17 years of follow-up (Engel Class 1). Postoperative MRI is presented in *figures 1I, 2G and 4D*.

Discussion

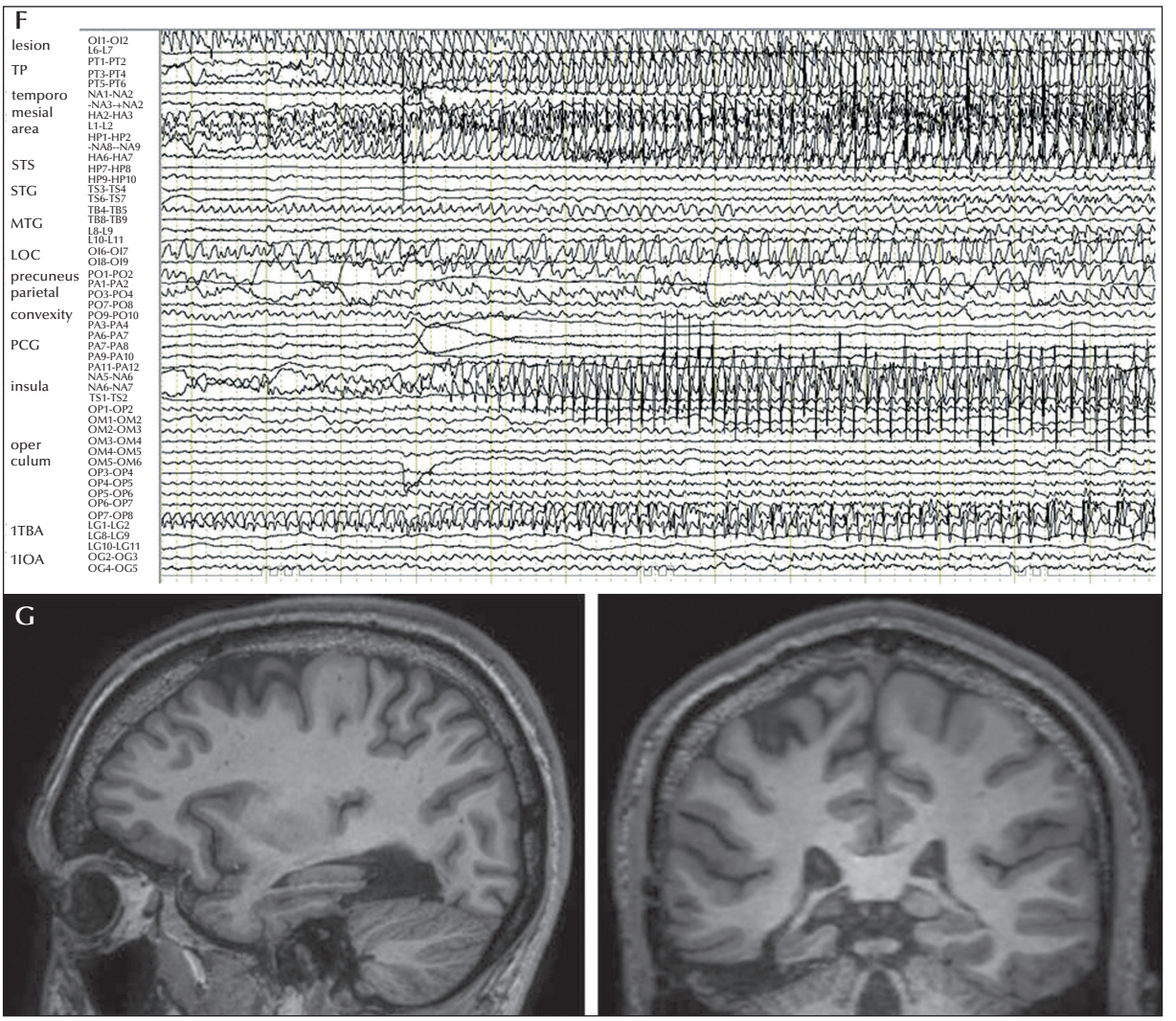
We report early facial somatosensory or somatomotor features in three patients suffering from temporal epilepsy related to a lesion located on the occipito-temporal gyrus. The semiology of temporobasal seizures is poorly described in the literature. They are indeed usually included in the group of neocortical epilepsy types [5, 6]. They have also been mixed with epilepsy types originating in the parahippocampal gyrus [7]. Patients similar to ours, with epilepsy related to lesions lateral to the collateral sulcus and at least 3 cm posterior to the temporal pole, have only been previously reported by Usui *et al*. [8]. Among them, eight patients had basal epilepsy as opposed to seven patients labelled as lateral in whom lesions were located in the middle temporal gyrus and superior temporal gyrus. These eight patients had no homogeneous clinical features. None had any facial symptomatology except for two



■ **Figure 2.** Patient 2. (A) Preoperative MRI (upper panel: coronal view, T2-weighted sequences; lower panel: axial view, FLAIR sequences). (B, C) Scalp EEG (10-20 system with supplementary temporo-basal electrodes; longitudinal montage; amplitude: 100 µV/cm, low-pass filter: 70 Hz, high-pass filter: 0.530 Hz). (B) Seizure onset during sleep corresponding to awakening (clinical onset) (1) and right fronto-temporal delta activity (electrical onset), starting five seconds after the clinical onset (2). (C) Seizure continuation with facial contraction occurring 11 seconds after clinical onset (3).



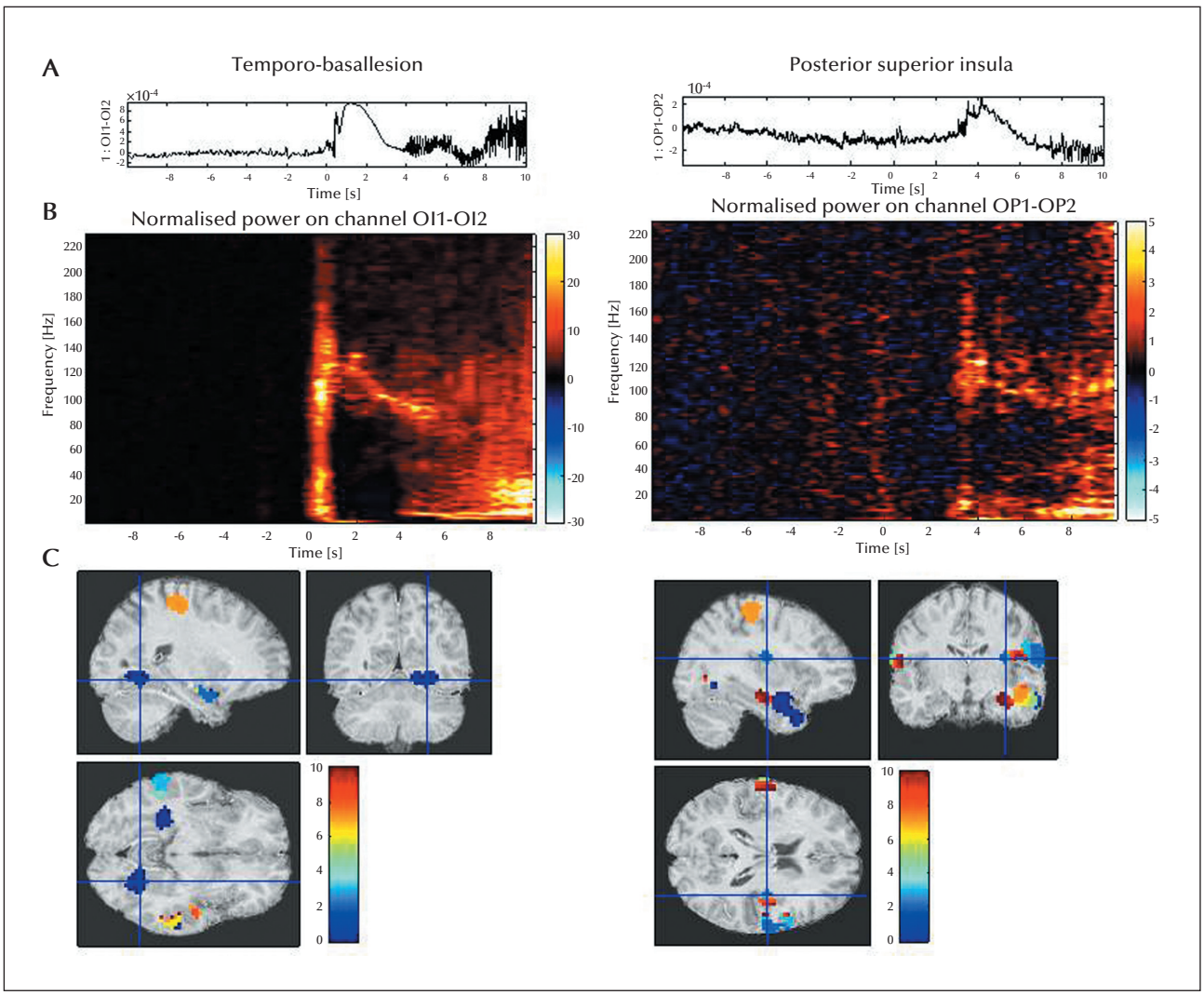
■ **Figure 2.** (D) Electrode implantation. Left panels: implantation in the right hemisphere showing lateral view (upper), insular view (middle) and hippocampal view (lower). Right panels: implantation in the left hemisphere showing lateral view (upper), insular view (middle) and hippocampal view (lower). (E, F) SEEG recording (amplitude: 750 µV/cm, low-pass filter: 120 Hz, high-pass filter: 0.530 Hz). TP: temporal pole; STS: superior temporal sulcus; STG: superior temporal gyrus; MTG: middle temporal gyrus; LOC: lateral occipital cortex; PCG: post-central gyrus; tab: left temporo-basal area; lIOA: left insulo-opercular area. (E) Seizure onset showing electrical onset with a fast discharge from the electrodes exploring the lesion (OI 1-2) (1) with fast propagation on the temporal pole, fast discharge on the insula (arrow) at clinical onset (6 seconds after the electrical onset) (2), with movement, tachycardia and facial contraction (7 seconds after the electrical onset) (3). Note the rhythmic activity from the opercular electrodes and the theta activity from OG1 facing the lesion on the left (*continued*).



■ **Figure 2.** (F) Seizure continuation and end. The patient shows head and trunk deviation to the left and hypertonia of the right side of the body. The discharge has spread out and predominates from the electrodes exploring not only the lesion, the temporal pole and the amygdalo-hippocampal complex, but also the insula and the central operculum explored by OP. (G) Postoperative MRI (T1-weighted sequences). Left panel: sagittal view; right panel: coronal view (*continued*).

patients with oroalimentary automatisms. Furthermore, they were investigated with subdural electrodes, and no insulo-opercular exploration was performed.

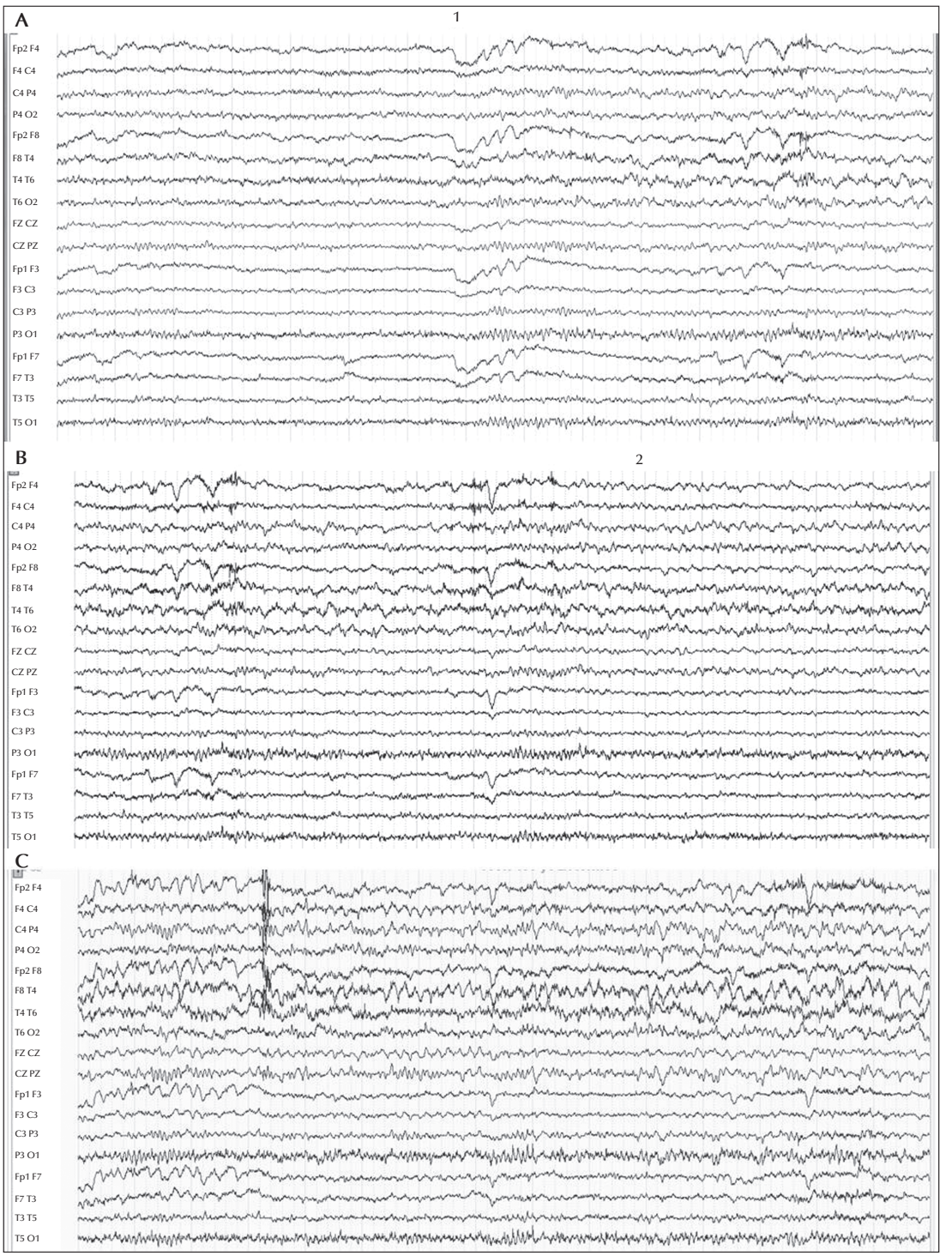
Facial semiology has been studied in mesial temporal epilepsy, and somatomotor manifestations have been reported in seven patients with temporomesial epilepsy [9]. However, the electrode implantation at the time did not encompass the insulo-opercular region. More recently, ictal facial contraction has been reported in a patient investigated with subdural electrodes [10]. The patient experienced right-sided tonic facial movements and ictal crying. Seizures were confirmed to originate from the left amygdala hippocampal region and to propagate to the left cingulate region. The facial contraction was considered to be related to this cingulate propagation. However, this patient is not really comparable to ours, as his epilepsy was mesial and not basal. Moreover, the insula was not explored because of the implantation method. Even so, as the propagation from the insula to the cingulate is rapid, insulo-opercular commitment is highly probable. Similarly, Perven *et al.* reported that 8% of patients with temporomesial epilepsy have somatosensory auras, of which some are not cured with surgery. They suggested an insular onset, however, this was not confirmed because of the absence of invasive exploration and the lack of insular electrodes during invasive exploration [11]. Another



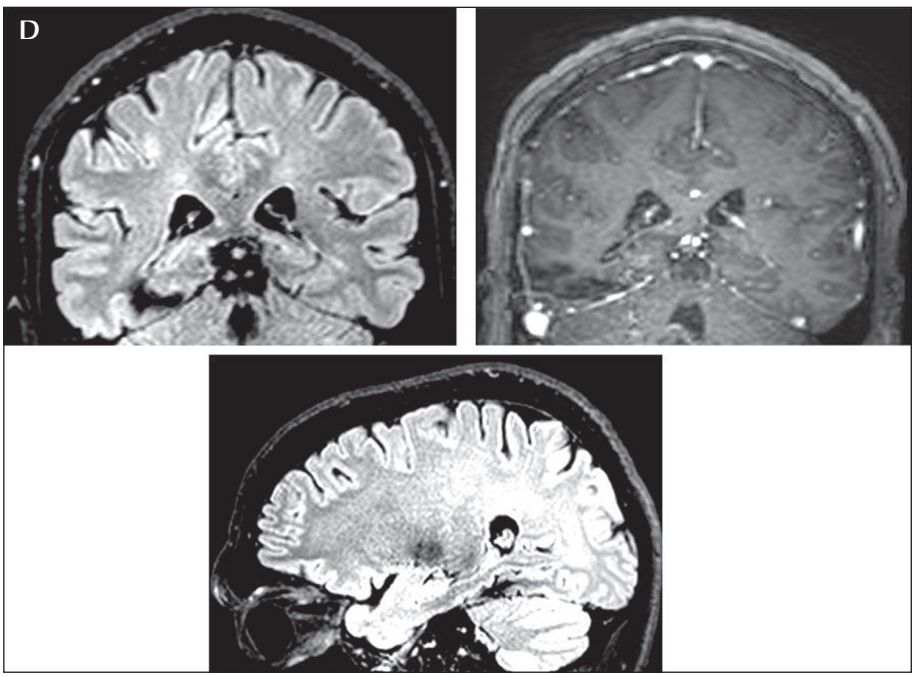
■ **Figure 3.** Comparison of seizure dynamics between the lesion (channel OI1-OI2) and posterior superior insula (channel OP1-OP2) in Patient 2. (A) Time series of a seizure onset showing the first 10 seconds. (B) SEEG power in time-frequency domain, z-scored over the baseline and averaged across the seizures. Ictal changes are stronger in the lesion and propagate to the insula after around three seconds. (C) Group propagation map of high-gamma (80-180 Hz) power indicating the onset, in seconds, of significant ictal changes of power. The pattern of propagation is relatively complex.

article reported a patient suffering from left temporomesial epilepsy with a right somatosensory aura [12]. The somatosensory aura was associated with propagation of the discharge to the somatosensory and posterior cingulate cortices. Lastly, Aupy *et al*. showed that oroalimentary automatisms in temporomesial seizures are related to insulo-opercular propagation of the discharge [13].

The facial manifestations were at the forefront in our three patients, however, they were not all comparable. In Patients 1 and 2, there were somatomotor features, whereas in Patient 3, an ipsilateral somatosensitive manifestation was evident. The mesial structures were not strongly involved in any of the patients. In Patients 1 and 2, the facial manifestations could be related to insular and later opercular propagation. In Patient 3, ipsilateral perception of heat was highly suggestive of insular involvement, as pointed out in the literature. Indeed, during electrical stimulations, somatosensory evoked responses can be ipsilateral when stimulating secondary somatosensory cortex or insula [14, 15]. In pure insular epilepsy, confirmed by SEEG, one patient



■ **Figure 4.** Patient 3. (A, B) Scalp EEG of a seizure occurring during hyperventilation (10-20 system; longitudinal montage; amplitude: 70 µV/cm, low-pass filter: 70 Hz, high-pass filter: 0.530 Hz). The patient was asked to refrain from warning and remain with eyes closed which he was able to do, except for occasional eyelid movements, and he described a heat sensation on the right part of the face and right upper limb as well as fear after the end of the discharge. (A) Electrical onset showing theta activity on the temporal anterior region (1). (B) Seizure continuation showing theta activity from suprasylvian electrodes (C4 P4), 10 seconds after the electrical onset (2). (C) Seizure continuation showing fronto-centro-parietal propagation (2).



■ **Figure 4.** (D) Preoperative MRI (FLAIR sequences) (upper left panel: coronal view; lower panel: sagittal view) and postoperative MRI (T1-weighted sequence) (right panel: coronal view) (*continued*).

had ipsilateral heat sensation of the face [16]. In our patient, this insular propagation could nevertheless not be proven.

The first reported subjective feature is consistent with temporal onset in each of the patients, as all of them showed variation in heart rate at the onset of seizures. During the course of the seizure, none had any features that were highly suggestive of TLE, but interictal and ictal EEG were compatible with this origin.

Our study has many limitations, the most significant being that it is limited to three patients. Additionally, one of them did not undergo an invasive investigation. In the other two, studied more than 10 years ago, the operculo-insular investigation was of poor quality and does not adhere to our present standards. We are notably not able to determine the precise localization of the involved area within the insula because of sampling error. Moreover, electrical stimulations were not performed in Patient 2, and1-Hz stimulations were not performed in any of the patients and so could not be used as a tool for tractography [17]. Lastly, quantitative analyses could not be conducted in Patient 1 due to the age of the data.

Nevertheless, our study demonstrates rapid connections between the occipital-temporal gyrus and regions which could contribute to the understanding of complex networks in epileptogenic patients. Furthermore, these observations should lead to exploring temporo-basal regions in cryptogenic epilepsy with early facial manifestations if other elements point to this localization. ■

Key points

- Temporo-basal epilepsies are rarely described in the literature.
- Patients with seizures originating from the temporo-basal area can have early elementary orofacial ictal manifestations.
- Somatomotor or somatosensory facial features in patients with temporo-basal epilepsies appear to be related to insulo-opercular fast propagation.

Supplementary material.
Summary slides accompanying the manuscript are available at www.epilepticdisorders.com.

Acknowledgements and disclosures.
We thank the physicians who referred the patients to epilepsy surgery. We thank Mrs. Aisha Yu, a native English speaker, for careful revision of the language.
None of the authors have any conflicts of interest to disclose. O David has received funding from the European Research Council under the European Union's Seventh Framework Programme (FP/2007-2013)/ERC Grant Agreement no. 616268 F-TRACT and the

European Union's Horizon 2020 Framework Programme for Research and Innovation under Specific Grant Agreement No. 785907 and No. 945539 (Human Brain Project SGA2 & SGA3).

References

1. Penfield W, Faulk M. The insula. *Brain* 1955; 78: 445-70.

2. Wang H, McGonigal A, Zhang K, Guo Q, Zhang B, Wang X, *et al*. Semiologic subgroups of insulo-opercular seizures based on connectional architecture atlas. *Epilepsia* 2020; 61: 984-94.

3. Engel JJ, Van Ness P, Rasmussen T, Ojemann L. Outcome with respect to epileptic seizures. In : Engel JJ, ed. *Surgical treatment of the epilepsies*. New York: Raven Press. 609-21.

4. David O, Blauwblomme T, Job A-S, Chabardès S, Hoffmann D, Minotti L, *et al*. Imaging the seizure onset zone with stereoelectroencephalography. *Brain* 2011; 134: 2898-911.

5. Foldvary N, Lee N, Thwaites G, Mascha E, Hammel J, Kim H, *et al*. Clinical and electrographic manifestations of lesional neocortical temporal lobe epilepsy. *Neurology* 1997; 49: 757-63.

6. Maizuliana H, Usui N, Terada K, Kondo A, Inoue Y. Clinical, semiological, electroencephalographic, and neuropsychological features of "pure" neocortical temporal lobe epilepsy. *Epileptic Disord* 2020; 22: 55-65.

7. Mirandola L, Badawy R, Saunders A, McIntosh A, Berkovic S, Jackson G. Clinical features of seizures associated with parahippocampal/inferior temporal lesions compared to those with hippocampal sclerosis. *Epilepsia* 2010; 51: 1906-19.

8. Usui N, Mihara T, Baba K, Matsuda K, Tottori T, Umeoka S, *et al*. Intracranial EEG findings in patients with lesional lateral temporal lobe epilepsy. *Epilepsy Res* 2008; 78: 82-91.

9. Bossi L, Munari C, Stoeffels C, Bonis A, Bacia T, Talairach J, *et al*. Somatomotor manifestations in temporal lobe seizures. *Epilepsia* 1984; 25: 70-6.

10. Hogan R, Rao V. Hemifacial motor and crying seizures of temporal lobe onset: case report and review of electroclinical localisation. *J Neurol Neurosurg Psychiatry* 2005; 77: 107-10.

11. Perven G, Yardi R, Bulacio J, Najm I, Bingaman W, Gonzalez-Martinez J, *et al*. The relevance of somatosensory auras in refractory temporal lobe epilepsies. *Epilepsia* 2015; 56: e143-8.

12. Merinder T, Rásonyi G, Tsiropoulos I, Jespersen B, Ryvlin P, Fabricius M, *et al*. Somatosensory phenomena elicited by electrical stimulation of hippocampus: Insight into the ictal network. *Epilepsy Behav Rep* 2020; 14: 100387.

13. Aupy J, Noviawaty I, Krishnan B, Suwankpakdee P, Bulacio J, Gonzalez-Martinez J, *et al*. Insulo-opercular cortex generates oroalimentary automatisms in temporal seizures. *Epilepsia* 2018; 59: 583-94.

14. Mazzola L, Isnard J, Mauguière F. Somatosensory and pain responses to stimulation of the second somatosensory area (SII) in humans. A comparison with SI and insular responses. *Cereb Cortex* 2006; 16: 960-8.

15. Pugnaghi M, Meletti S, Castana L, Francione S, Nobili L, Mai R, *et al*. Features of somatosensory manifestations induced by intracranial electrical stimulations of the human insula. *Clin Neurophysiol* 2011; 122: 2049-588.

16. Peltola M, Trébuchon A, Lagarde S, Scavarda D, Carron R, Metsähonkala L, *et al*. Anatomoelectroclinical features of SEEG-confirmed pure insular-onset epilepsy. *Epilepsy Behav* 2020; 105: 106964.

17. Trebaul L, Deman P, Tuyisenge V, Jedynak M, Hugues E, Rudrauf D, *et al*. Probabilistic functional tractography of the human cortex revisited. *NeuroImage* 2018; 181: 414-29.

TEST YOURSELF

(1) Early ipsilateral ictal facial somatosensory sensations:

A. are exclusively encountered in contralateral opercular epilepsies
B. occur in ipsilateral and contralateral insular and opercular epilepsies
C. are encountered in epilepsies of other origin
D. are likely to be related to insulo-opercular involvement

(2) Temporo-basal seizures:

A. have been rarely described in the literature
B. harbour a specific semiology
C. can be characterized by a progressive semiology
D. have a semiology similar to that of temporo-mesial seizures

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