■ Electroclinical reasoning report

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SEEG re-exploration in a patient with complex frontal epilepsy with rapid perisylvian propagation and mixed "startle - reflex" seizures

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ABSTRACT – The SEEG International Course, organised in 2017, focused on the investigation and surgery of insulo-perisylvian epilepsies. We present one representative complex case that was discussed. The patient had seizures displaying startle/reflex components. He was MRI negative, while other non-invasive investigations offered only partially concordant data. Initial SEEG exploration resulted in an incomplete definition of the epileptogenic zone. A second SEEG followed, which led to a thorough assessment of the seizure onset zone and the epileptic network, localised to the lateral inferior premotor cortex, explaining the incongruent data obtained beforehand. This was the basis of a tailored resection with a favourable outcome. The patient has been seizure-free for five years without any motor nor cognitive deficits, but with pharmacodependence to one AED. The electroclinical reasoning is presented, accompanied by relevant commentaries and recommendations from the tutors. [*Published with video sequences*].

Key words: epilepsy surgery; insular-opercular epilepsy; premotor epilepsy; startle seizures; reflex seizures; focal cortical dysplasia; SEEG

VIDEOS ONLINE

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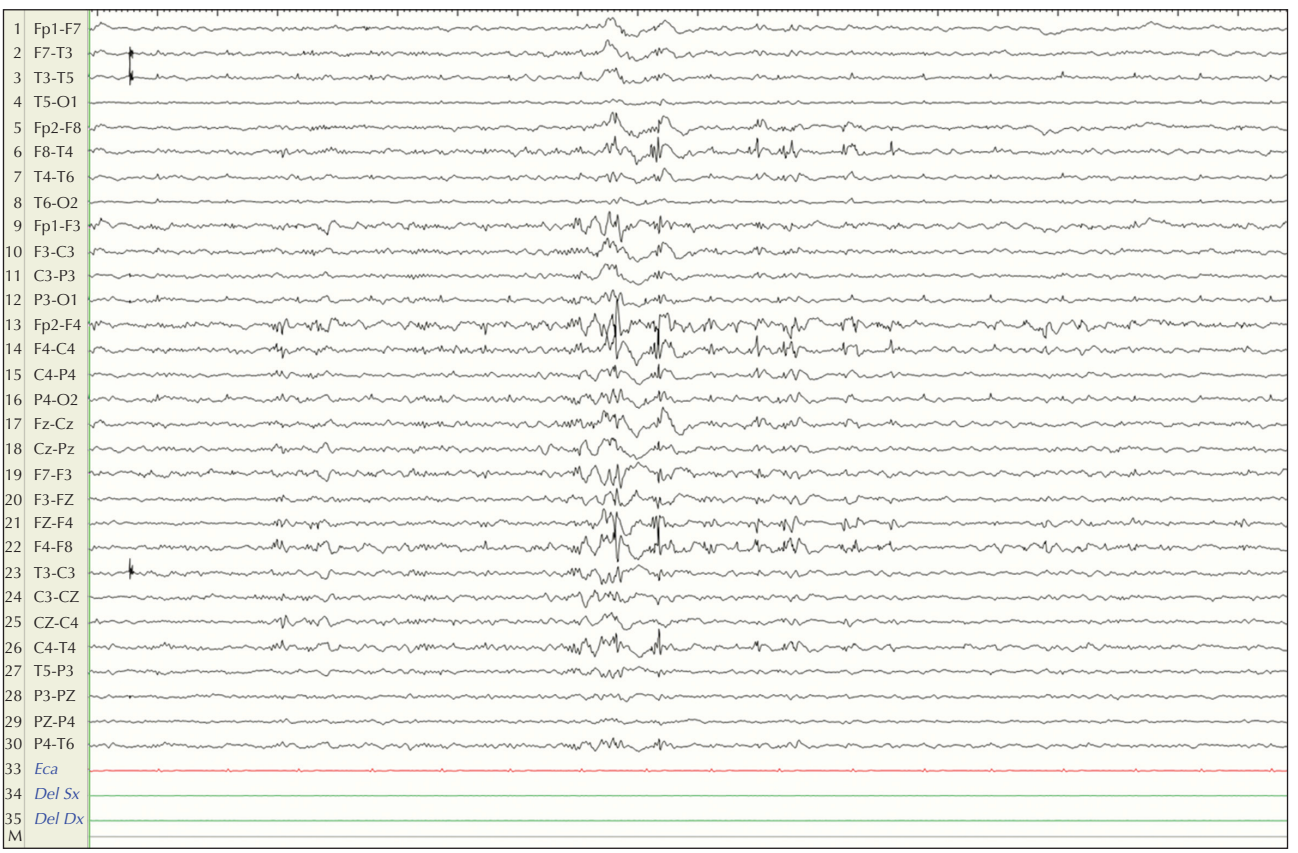
We present the case of a 23-year-old, right-handed man investigated at the GOM Niguarda Hospital, Milano, Italy for drug-resistant epilepsy. This case was part of the SEEG International Course, Venice 2017 that focused on the investigation and surgery of insulo-perisylvian epilepsies. The case is therefore accompanied by discussion around different strategies proposed by the participants as well as relevant commentaries and recommendations from the tutors. Throughout the text, the various parts of the epileptogenic network were defined according to SEEG standards [1]. The seizure onset zone (SOZ) was defined as the cortex explored by the contacts involved in seizure generation. The epileptogenic zone (EZ) was defined as containing the contacts of primary ictal organisation. The irritative zone (IZ) was the neural tissue where spikes and pathological fast rhythms were recorded in the interictal period, while the lesional zone (LZ) contained the cortex displaying pathological slow activity.

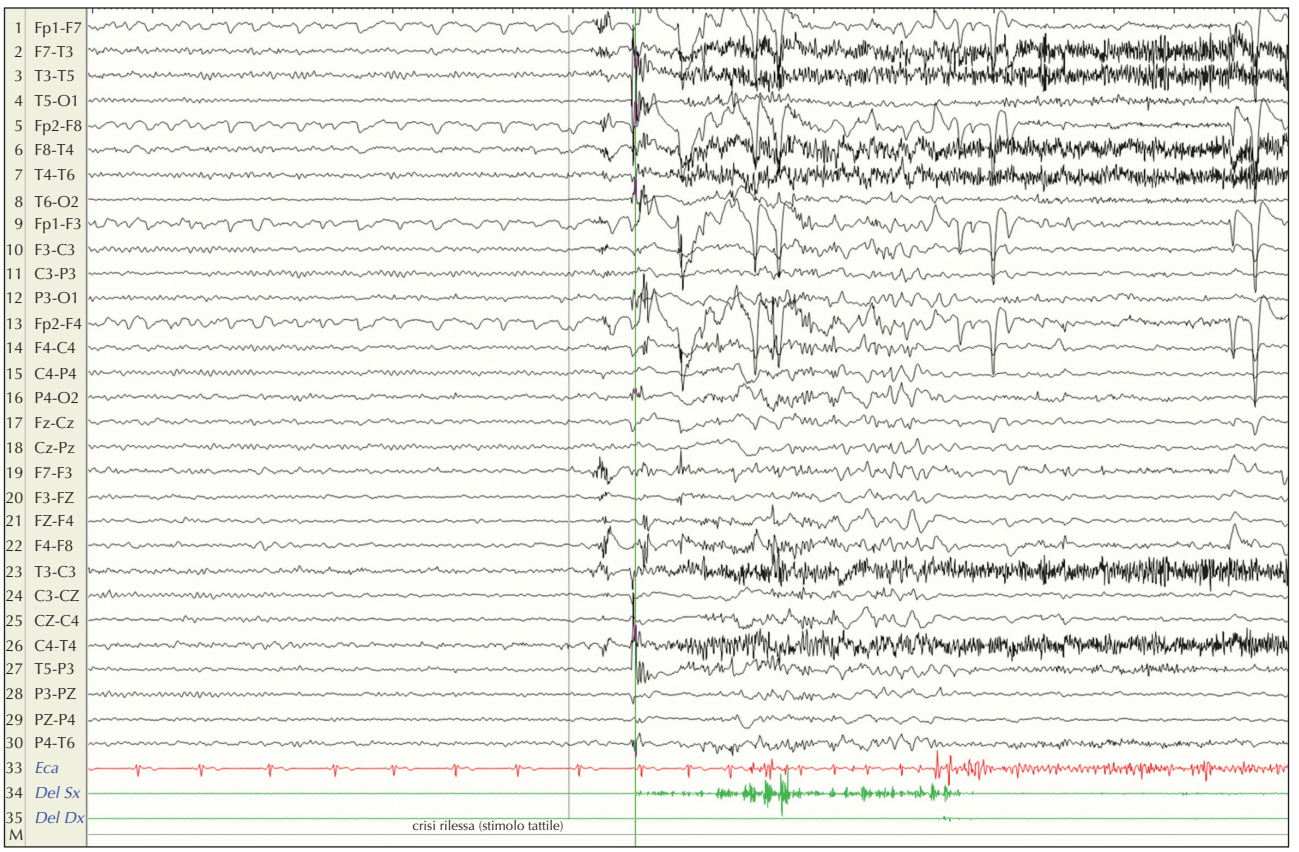
Clinical history did not reveal any personal nor family risk factors for epilepsy, and the patient did not have any comorbidities. His epilepsy started at 17 years, with two generalised tonic-clonic seizures on awakening. This type of seizure remained predominant during the first years, but became exceptional over time, being replaced by two other types of seizures: brief events characterized by hand stiffening and more prolonged events with a similar onset, followed by a contraction of the jaw, myoclonic jerks of the mouth and head deviation towards the left. Contact was preserved, and the patient reported being able to understand but not speak. The episodes lasted between 10 and 20 seconds and occurred predominantly during daytime, being usually precipitated by sudden noises or other unexpected stimuli, such as somatosensory stimulations. From a subjective perspective, the patient felt that the progression from the brief to the more prolonged seizures could be halted by the shaking of the left hand.
Several antiepileptic drugs were tried, including phenytoin, valproic acid, carbamazepine and benzodiazepines. He was taking a combination of levetiracetam and lamotrigine when he was admitted for presurgical evaluation.

Non-invasive evaluation

Interictal EEG showed a normal, symmetrical background activity. During wakefulness, he had rare right-sided theta activity on the frontal and central derivations. During sleep, bursts of fast activity, as well as sequences of spike-and-wave discharges were recorded on the same EEG channels but tended to be more extended (*figure 1*). Several spontaneous and provoked seizures (sudden sensory or auditory stimulations) were captured (*figure 2, video sequences 1-3*). Clinically, the patient reported a sensation in the left hand, accompanied by stiffening and reactive shaking of the same hand. Concerning the EEG, besides the muscular artefact, a fast discharge was identifiable on the right fronto-central channels.



■ **Figure 1.** Interictal EEG during sleep displaying right frontal polyspikes with a phase reversal at F4, also visible over the midline and homologous contralateral territories. Bipolar longitudinal followed by bipolar transversal montages, followed by EKG and EMG electrodes for the left and right deltoids. HPF: 1.6 Hz; LPF: 120 Hz; notch: 50 Hz.



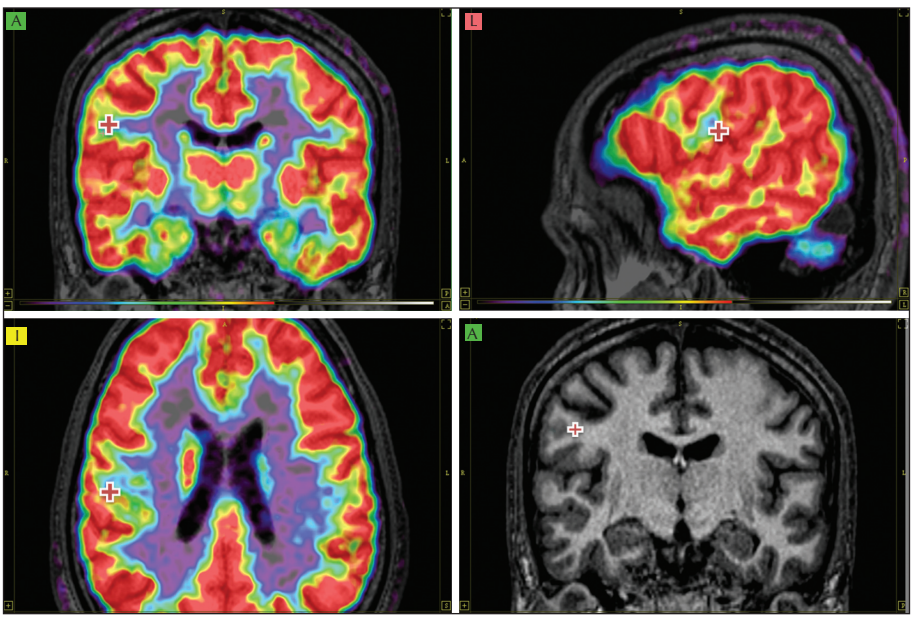
■ **Figure 2.** Ictal EEG during wakefulness displaying a startle-reflex seizure elicited by a sensory stimulus (sudden touch of the left leg: black vertical bar). Note the right ictal activity starting with a fast-rhythmic discharge, most prominent in the fronto-central channels, followed by a polymorphic diffuse slowing. The green vertical bar marks the clinical onset. Note the associated myogenic potential in the contralateral deltoid. Bipolar longitudinal, followed by bipolar transversal montages, followed by EKG and EMG electrodes for the left and right deltoids. HPF: 1.6 Hz; LPF: 15 Hz; notch: 50 Hz.

Brain 1.5 T MRI, performed according to our epilepsy protocol [2], did not reveal any structural abnormalities. 18 FDG-PET, however, demonstrated right frontal ventral premotor hypometabolism (*figure 3*).

The neuropsychological examination showed a right-handedness (100%, Edinburgh scale). Long-term memory and attention were normal, but the patient exhibited a slight deficit in verbal working-memory and a more pronounced deficit in his verbal phonetic fluency. This deficit was not considered to be related to the epilepsy's localisation as there was no reason to suspect non-classic language lateralisation in this patient. Clinical arguments were complete hand dominance for the right hand, as well as intact receptive ictal and postictal language functions, and also the immediate restoration of expressive language functions in the postictal period. Finally, there was no family history of left-handedness. Taking all these arguments into consideration, his "inability to speak" was considered a sign of ictal anarthria. That is also the reason why a non-invasive language lateralisation investigation, such as Wada-testing or language fMRI, was not proposed. However, the further proposed invasive exploration with intracranial electrodes allowed for the testing of this presumption.

Hypothesis

The main hypothesis for the epileptic focus for all of the participants was the suprasylvian operculum [3, 4] as well as the primary motor and sensory face and hand area. These were suspected based on the clinical semiology and the PET interictal hypometabolism. However, there was a discrepancy between these data and interictal EEG findings that showed an epileptiform activity centred over the F4 electrode. An alternative hypothesis could point to more



■ **Figure 3.** MRI co-registered with 18 FDG-PET, demonstrating right frontal premotor ventral hypometabolism (cross sign). The corresponding T1-weighted MRI image does not show any obvious abnormality.

anterior frontal structures, such as the lateral premotor and the dorsolateral prefrontal cortex. Moreover, although the seizure semiology was more indicative of lateral frontal involvement, medial frontal structures such as the dorsomedial prefrontal cortex, the pre-supplementary sensory-motor area (pre-SMA) and the supplementary sensory-motor area (SMA), and the cingulate cortex should be taken into account as well [5]. Arguments for these localizations were the startle component of the seizures and the very fast bilateral involvement. SMA should also be considered because of the mixed sensory-motor hand semiology. The unilateral and brachio-facial tonic contraction were arguments more in favour of a perisylvian and ventral lateral premotor origin than a dorsal lateral or medial premotor one [6]. However, the fast propagation between these two systems makes this differentiation artificial [7].

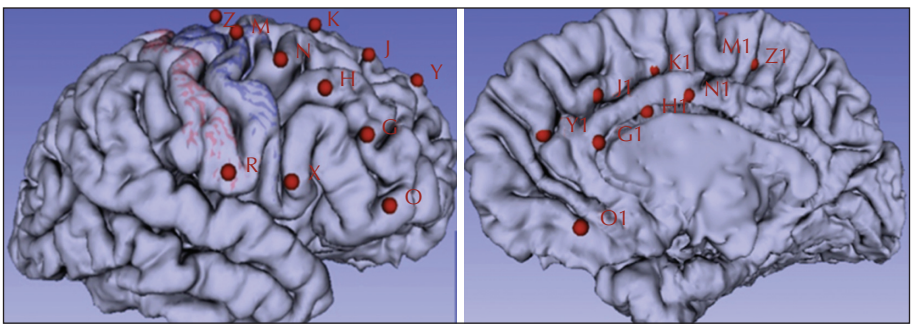
Based on these hypotheses, and taking into account that the non-invasive data remained insufficient, all the participants agreed to go further with an invasive evaluation. All the teams considered a strictly unilateral implantation. This was appropriate due to the high lateralizing value of the ictal semiology - left hand sensation and contraction- as well as electrophysiological and metabolic data. The participants proposed a unilateral right-sided SEEG evaluation as the optimal way to map both medial and lateral structures of the frontal and parietal lobes, including the activity from the depth of the sulci of these regions.

First SEEG study

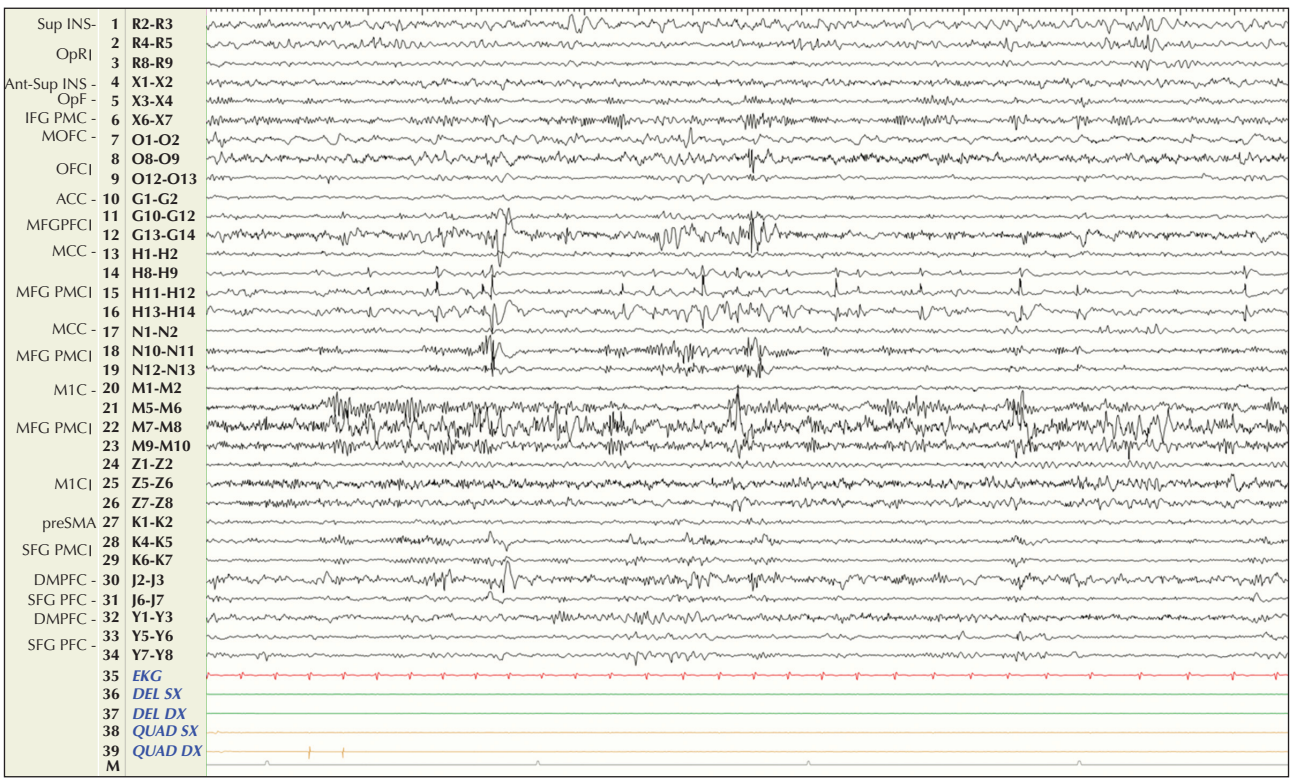
Unilateral SEEG implantation was performed with 11 intracerebral electrodes, of which two targeted the perisylvian structures including the right frontal and Rolandic operculum and the adjacent insular cortex. The remaining nine electrodes aimed at evaluating laterally the premotor, prefrontal and orbitofrontal cortices, and medially the rectus gyrus, anterior and mid-cingulate cortex, paracentral lobule, SMA, pre-SMA and dorsomedial prefrontal cortex (*figure 4*). Note that the functional area where the electrodes were located was decided based on the results of the functional mapping with direct electrical stimulations (for example, differentiating between primary motor cortex and premotor cortex; premotor cortex and prefrontal cortex).

SEEG recordings showed subcontinuous interictal spiking activity, interspaced by voltage suppression over the lateral premotor cortex at the level of the posterior middle frontal gyrus (MFG) and inferior frontal sulcus (IFS) (*figure 5*). The primary motor cortex showed mild irritative abnormalities such as small spikes, fast rhythms and some sharp waves, rarely linked to the premotor anomalies. Note that fast rhythms and sharp graphoelements are often found in intracranial physiological electrical activity of the primary motor cortex.

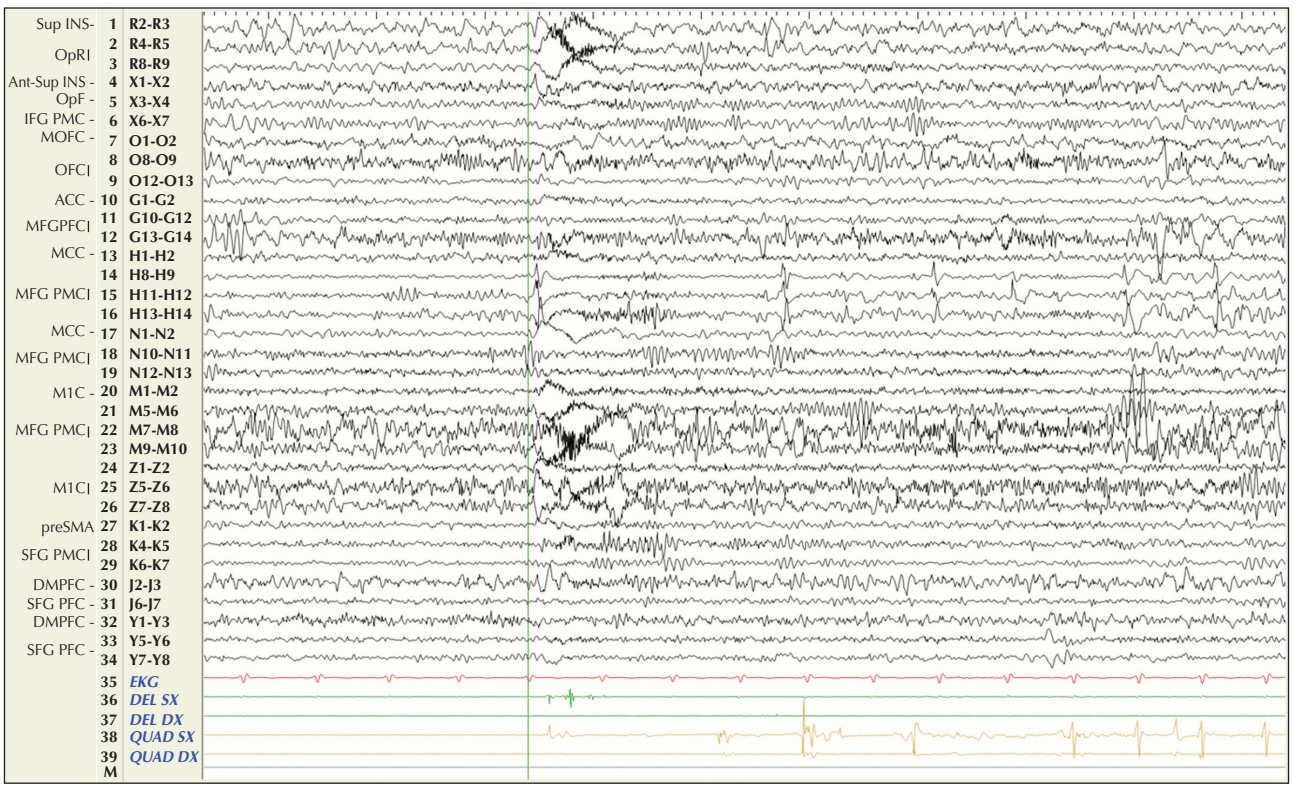
Several seizures were recorded, showing low-voltage fast activity (LVFA) superimposed on a slow wave in the Rolandic operculum and motor hand area, with



■ **Figure 4.** Personalized volumetric reconstruction of the patient's right hemisphere displaying the stereotactic localization of the 11 intracerebral electrodes (first SEEG study). The reconstruction was performed with FreeSurfer software and the colours mark the precentral gyrus (blue) and the postcentral gyrus (red).



■ **Figure 5.** Interictal SEEG activity showing continuous spiking over the lateral contacts of electrodes H (premotor accessory inferior frontal gyrus [IFG]) and N (premotor MFG gyrus). Intracranial bipolar channels between adjunct electrodes exploring key cortical structures followed by EKG and EMG channels for left and right deltoid and quadriceps. HPF: 0.001 Hz; LPF: 300 Hz; notch: 50 Hz. ACC: anterior cingulate cortex; Ant-Sup INS: anterior-superior insular cortex; DMPFC: dorsomedial prefrontal cortex; IFG PFC: lateral prefrontal cortex, frontal inferior gyrus; IFG PMC: lateral premotor cortex, frontal inferior gyrus; M1C: primary motor cortex (precentral gyrus); MCC: middle cingulate cortex; MFG PFC: lateral prefrontal cortex, frontal middle gyrus; MFG PMC: lateral premotor cortex, frontal middle gyrus; MOFC: medial orbitofrontal cortex; OFC: orbitofrontal cortex; OpF: frontal operculum; OpR: Rolandic operculum; SFG: superior frontal gyrus; SFG PFC: lateral prefrontal cortex, frontal superior gyrus; SFG PMC: lateral premotor cortex, frontal superior gyrus Sup INS: superior insular cortex.



■ **Figure 6.** Ictal SEEG activity showing LVFA (green vertical line marks the debut), superimposed on a slow wave at the middle and lateral contacts of electrodes M (primary motor hand area) and internal and middle contacts of the electrode R (insula and Rolandic operculum). The pathological interictal H and N electrodes display a propagation pattern composed of small polyspikes that precede the LVFA on M and R, followed by a large spike and a flattening. Montage and filters as for *figure 5*.

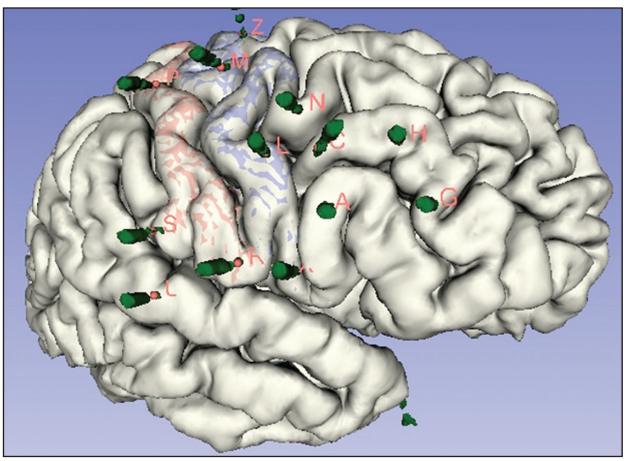
an activation of electrode N01-02 (the mid-cingulate cortex) (*figure 6*). The electrodes displaying interictal pathological activity were not predominantly involved, exhibiting only an electrodecrement followed by interictal-like spikes.

Conclusion of the first SEEG study

SEEG findings showed conflicting results. Interictal abnormalities were localized more anteriorly and superiorly in the premotor area (in accordance with the scalp EEG spikes that showed a phase reversal at the electrode F4). By comparison, the ictal activities were localized more posteriorly and inferiorly in the precentral gyrus and Rolandic operculum (in accordance with the PET hypometabolism). The normal background SEEG activity of the primary motor cortex, however, raised concerns about localizing the SOZ at this level. The main conclusion was that the SOZ could be located within an area not investigated by intracerebral electrodes, possibly between the explored premotor and motor cortices. As suggested by most of the participants, a second SEEG study was proposed, aiming at better covering the inferior premotor and ventrolateral prefrontal cortex, as well as the suprasylvian motor and sensory face areas. Some of the groups, however, proposed to proceed directly to surgery, or chose to add intracerebral electrodes during the current investigation. These options are presented in the supplementary material.

Second SEEG study

The second SEEG implantation was similar to the previous one, with supplementary electrodes covering the temporal and posterior Rolandic operculum, the posterior IFG and the precentral and postcentral gyrus at the supposed location of the primary face and hand areas (*figure 7*). This was chosen for a more precise mapping of the supposed SOZ and exploration of the sensory-motor and perisylvian networks. Some of



■ **Figure 7.** Personalized volumetric reconstruction of the patient's right hemisphere, displaying the intracerebral stereotactic location of the second SEEG implantation. Note the new electrodes in comparison to Figure 3. A: posterior IFG; C: posterior IFS; L: motor face area; P: sensory hand area; S: posterior Rolandic operculum; L: temporal operculum and the disappearance of electrodes K, J, Y in the prefrontal cortex and O in the orbitofrontal cortex. Reconstruction and colours are the same as for *figure 3*.

the areas that did not show pathological activity, such as the prefrontal and orbitofrontal cortices, were not re-investigated.

SEEG recordings showed interictal repetitive spiking activity with superimposed fast rhythms and isolated fast activity in the lateral premotor area (MFG, posterior IFG and IFS). Ictal findings were concordant with the irritative zone, with a rapid propagation to the lateral motor cortex and the Rolandic operculum (*figure 8*). These areas of propagation were the apparent areas of ictal onset in the prior implantation.

Conclusion of the second SEEG study

The second intracranial exploration enabled us to draw a much more coherent hypothesis for the epileptic network, explaining the divergent findings of the first exploration. The IZ was centred in the premotor cortex but included connected zones where interictal spikes propagated, such as the internal contacts of R and X (anterior insula) and the internal contacts of N (middle cingulate). Supplementary contacts in the electrode U (temporal operculum) and in electrodes G and H (prefrontal cortex) were considered to display pathological slow activity - LZ. The SOZ appeared to be located in the premotor MFG and IFG gyrus and in the precentral sulcus, explored by the electrodes C, A, and L. Despite very consistent ictal activation at electrodes M and R (primary motor cortex), there were several reasons for sparing this region and thus avoiding a permanent motor deficit. The first was the normal interictal background activity recorded over these electrodes, suggesting preserved tissue architecture. The second was the sustained LVFA observed over the electrodes L, A and C, as compared with the electrodes M and R. The third was the significant time delay between the involvement of the electrodes L, A and C and the involvement of the electrodes M and R, demonstrating a premotor to motor propagation.

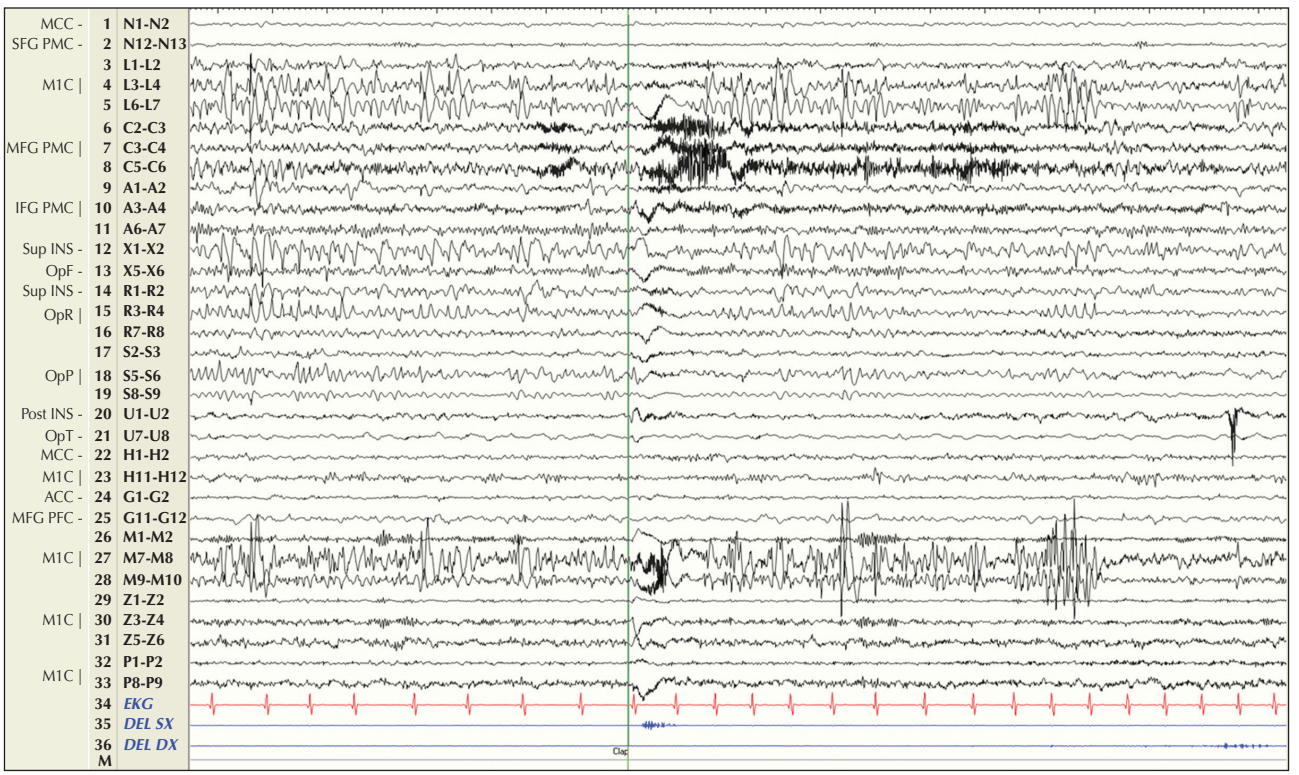
Surgical strategy

The therapeutic approach started first with a radio-frequency thermo-coagulation (RFTC) session performed over the contacts of electrodes L, A and C. The patient experienced a four-week period of seizure freedom after which the seizures relapsed with the same frequency and semiology. Although transient, this initially good result of RFTC indirectly confirmed the pivotal role of the cortex explored by these electrodes. Resective surgery was performed in a second step accordingly, including the middle and inferior premotor cortex, the frontal operculum and the precentral sulcus (*figure 9*). The superior limit was chosen in the sulcus above the electrode N, as the SEEG activity recorded here had a normal background with minimal involvement at seizure onset. The inferior limit extended to the sylvian fissure, due to the ictal involvement of the electrode X and the low risk to produce any neurological deficit. If the resection had been performed in the dominant hemisphere for language, however, the pars triangularis should have been carefully mapped before extending the resection at that level.

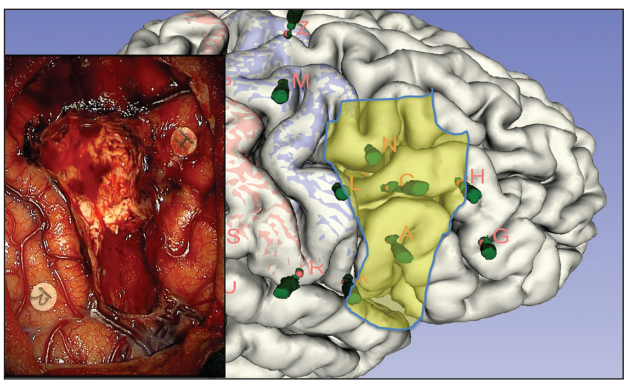
Follow-up

The patient did not experience any post-surgical deficit. The histopathological examination revealed focal cortical dysplasia (FCD) type IA, located in the anterior margin of the precentral sulcus and the precentral Rolandic opercular region with surrounding gliosis.

After three years of postoperative follow-up, the patient has remained seizure-free. Levetiracetam was tapered, but the patient still takes lamotrigine 200 mg/day. When lamotrigine was tapered to 150 mg/day, the patient presented with a seizure with complex motor semiology. This is probable due to the limits of resection imposed by functional boundaries.



■ **Figure 8.** Ictal SEEG activity showing a short startle-reflex seizure elicited by a sudden noise (hand clap: green vertical line). LVFA superimposed on a slow wave on the middle and lateral contacts of electrode C (IFS in the premotor cortex). A phase reversal was observed between C3-C4 and C5-C6. The discharge propagated over the M electrode (primary hand motor area), L electrode (primary face motor area) and A (IFG gyrus in the premotor cortex). Note the associated short myogenic potential in the contralateral deltoid. Montage and filters as for *figure 5*. ACC: anterior cingulate cortex; IFG PFC: lateral prefrontal cortex, frontal inferior gyrus; IFG PMC: lateral premotor cortex, frontal inferior gyrus; M1C: primary motor cortex (precentral gyrus); MCC: middle cingulate cortex; MFG PFC: lateral prefrontal cortex, frontal middle gyrus; MFG PMC: lateral premotor cortex, frontal middle gyrus; OpF: frontal operculum; OpR: Rolandic operculum; OpP: parietal operculum; OpT: temporal operculum; Post INS: posterior insular cortex; S1C: primary sensory cortex (postcentral gyrus); SFG PFC: lateral prefrontal cortex, frontal superior gyrus; SFG PMC: lateral premotor cortex, frontal superior gyrus; Sup INS: superior insular cortex.



■ **Figure 9.** Right: preoperative drawing of the resective surgery. Left: postoperative surgical cavity, with the position of the electrodes R and H marked with paper labels.

The return to the initial dose of lamotrigine offered seizure freedom for the last two years (Engel Class IC). The neuropsychological examination performed at 12 months post-surgery did not detect any significant deterioration in cognitive abilities.

Discussion

In this report, we describe a complex, MRI-negative case, displaying both the characteristics of startle and reflex epilepsies. Startle epilepsies are characterized by seizures produced by unexpected, sudden, multimodal stimulations (sound, sensory, visual, *etc.*). It is indeed the novelty and intensity, not the nature of the sensory stimulus, that produces this response.

The characteristic "startle response or StartReact" is clinically characterized by blinking with a bilateral short-lasting tonic arm flexion that has a characteristic "diamond shaped"- myographic presentation. Although this is considered predominantly an involuntary response, mainly integrated at the subcortical, reticulo-spinal level, it is now proven that the cortical structures participate in the voluntary response preparation and in the voluntary slow initiation of the response [8]. A few intracranial studies have showed that startle seizures arise from the medial premotor-prefrontal structures such as the SMA, pre-SMA and anterior cingulate cortex [5, 9]. Reflex epilepsies, in contrast, are provoked by stimuli that belong to one or several categories (*e.g.,* somatosensory, auditory) [10] which lead, when involving the peri Rolandic cortex, to more focal and long-lasting motor phenomena (*e.g.,* hand contraction). According to the intracranial literature, reflex seizures may arise from the primary sensory areas, the insulo-opercular cortex or the posterior cortex [11-15]. Our case exhibited clinical characteristics resembling both startle (unexpected stimuli of multiple sensory modalities) and reflex (focal prolonged motor response) seizures, and the localization of the epileptogenic zone in the lateral premotor cortex might explain this mixed and complex phenotype. This brain area, indeed, is known to have strong functional connections, not only with the primary sensorimotor cortex and insulo-opercular cortex, but also with medial premotor structures that are part of the "startle circuit" [7, 16].

There are very limited data in the literature regarding histopathological findings in patients operated for startle or reflex epilepsy with limited cortectomies or lesionectomies, and not widespread hemispherectomies, disconnections or palliative surgeries. These are usually case reports or small case series, making generalisations very difficult. Regarding the startle cases, Job *et al*. [5] reported either normal cortex or non-specific gliosis, while Ciurea *et al*. [9] reported type IB FCD and Tian *et al*. [17] a tissue presenting degeneration of neurons and proliferation of glial cells, not further specified. Similar to our case, Sun *et al*. [18] reported a single case with type IA FCD. The histopathological findings in reflex epilepsies, benefiting from limited surgical resections, seem to be more varied. Biraben *et al*. [12] reported a vascular malformation, while Palmini *et al*. [19], focusing on only reflex seizures caused by malformations of cortical development (MCD), reported a case with subependymal heterotopia, another with double cortex, and another with perisylvian polymicrogyria. Two cases with the most limited epileptogenic zones both displayed type II FCD. Due to the low incidence of reflex seizures in their large series of MCD, the authors inferred that these lesions might be protective for these types of seizures, due to a loss of functional neurons, especially so in FCDs which are the most epileptogenic. Analysis of this small series appears to reveal an association between histopathological lesions and limited startle/reflex epilepsies. However, from our point of view, the location of the lesion and maturation of the neuronal networks are more reliable indicators of the final clinical phenotype.

This case highlights some very important red flags that should be considered when invasively exploring the motor systems in the frontal lobe epilepsies that are resumed in *table 1*.

The case was considered especially challenging as both the preinvasive and invasive investigations revealed conflicting data, making the epileptogenic zone definition difficult. As such, a second SEEG study was judged necessary, a finding which accounts for 4% of the cases in a large series of patients submitted for SEEG investigation [20]. Depending on the epilepsy surgery centre's experience, two strategies can be followed when facing an uninformative or partially informative first SEEG. The more traditional approach is to explant the patient and to redesign a second implantation at a later time. The second, available in some centres and depending on the neurosurgical risk assessment, implies the introduction of supplementary electrodes to the first SEEG, after several days of initial intracranial recording. The limited experienced

▼ **Table 1.** Semiology characteristics of motor and sensory phenomena that should be taken into consideration for central implantations.

"Red flags" when implanting primary vs. secondary motor areas
1. Although we may obtain tonic semiology upon direct electrical stimulation of the primary motor areas (M1), this rarely arises with initial ictal activation in these areas, where one more frequently sees clonic phenomena with a Jacksonian-like march. An ictal tonic motor phenomenon suggests a more global involvement of the sensorimotor circuitry.
2. A distal (vs. proximal) motor phenomenon can also appear in not-primary motor areas and should not be a strict decision criterion for excluding the premotor.
3. Frontal primary and secondary motor areas contain a large number of sensory neurons and can produce a mixed sensorimotor semiology without necessarily involving sensory areas.

accumulated by the centres where this approach is used demonstrates that it has a good safety profile. However, the consensus of the experts present at the European SEEG course, including the current authors, was that additional electrodes should be considered only if a small number is necessary. They are used to better define a partially supported hypothesis for the SOZ. For an exploration of large areas, with a high number of additional electrodes or for an ill-defined alternative hypothesis, a new SEEG exploration should be proposed. Our case had six supplementary electrodes which were considered necessary by the epilepsy team. As these were used to explore areas with a risk of permanent neurological deficit, such as in motor and sensory primary cortices, proposition of a second limited implantation, after careful analysis of the primary data, was decided as a safer strategy for the patient. However, because implantation would be close to the existing electrodes, an alternative strategy was also taken into consideration.

The benefits of supplementing with a small number of electrodes include: (1) the diagnostic benefit added to an already partially unravelled epileptogenic zone; and (2) the reduced overall medical costs, compared to two independent SEEG explorations. A second exploration offers the time for a well thought-out analysis of the primary data set and the opportunity to establish a completely new primary hypothesis (*e.g.*, in the contralateral hemisphere for a difficult-to-lateralize epilepsy), and also minimizes the risk of postoperative intracranial hypertension.

From a practical point of view, retrospectively, we can now understand that the first SEEG implantation had a bias towards the dorsolateral and medial frontal lobe as a result of interpretation of the clinical and EEG data. For this reason, we did not completely take into account the FDG-PET findings, despite the known high sensitivity of this investigation for MRI-negative FCDs located in the frontal lobe [21].

This study has several limitations. Firstly, the patient was examined by 1.5 T MRI, which was the most available MRI technology at the time when our patient was undergoing presurgical investigation. Secondly, we did not perform postprocessing analysis on the non-invasive data, such as electrical source imaging on EEG spikes, or MRI-based morphometry. Lastly, the reappearance of seizures having a modified semiology after significant drug tapering are testament to possible incomplete surgical resection, making the histopathological analysis subtotal.

Conclusion

The presented case demonstrates how cortical networks associated with MRI-negative premotor FCD can produce an electroclinical pattern mimicking a perirolandic or perisylvian epilepsy. Although rare, the startle-reflex seizures originating in this area should be invasively explored as they can be surgically curable. The study also shows that in cases of insufficient information following a first SEEG recording, a second SEEG procedure can be considered. ■

Supplementary data.
Summary didactic slides are available on the www.epilepticdisorders.com website.

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

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Legend for video sequences

Video sequence 1

Short left hand myoclonia elicited by an unexpected noise. The patient speaks immediately afterwards.

Video sequence 2

The video displays a more prolonged nocturnal spontaneous seizure. The patient has a tonic contraction of the left hand that he tries to stop by shaking and holding the fist with the right hand.

Video sequence 3

Short left hand myoclonia followed by a short tonic contraction elicited by a unexpected stimulation of his right leg. The patient speaks immediately afterwards.

Key words for video research on www.epilepticdisorders.com

Phenomenology: reflex seizures (videos 1 and 3), motor seizure (simple) (video 2)
Localization: frontal premotor lateral
Syndrome: focal non-idiopathic frontal (fle)
Aetiology: focal cortical dysplasia (type i)

TEST YOURSELF

(1) What are the ictal generators of startle and reflex seizures?

(2) What were the "red flags" for an insufficient SEEG definition of the seizure onset zone in this case?

(3) What are the strategies available when facing insufficient information based on SEEG implantation?

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".