

The Application of Video-Electroencephalographic Monitoring Combined with Multimodal Imaging in Surgical Management of Secondary Epilepsy in Children

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Research Article

Keywords: pediatric epilepsy surgery, video-electroencephalographic monitoring (VEEG), magnetic resonance imaging (MRI), positron emission tomography-computed tomography (PET-CT), electrocorticography (ECoG), stereoelectroencephalography (SEEG)

Posted Date: October 14th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-11032460/v1>

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Additional Declarations: No competing interests reported.

Abstract

Objective To explore the clinical value of video-electroencephalography (VEEG) coupled with multimodal imaging for pre-surgical epileptogenic-zone localization, surgical planning and prognostic evaluation in children with drug-resistant epilepsy.

Methods: A retrospective analysis was performed on 42 pediatric patients who underwent epilepsy surgery. Pre-operative workup included brain magnetic resonance imaging (MRI), long-term scalp VEEG, and PET-CT for selected participants. For diagnostically challenging cases, stereoelectroencephalography (SEEG) depth electrodes or electrocorticography (ECoG) cortical electrodes were implanted to refine epileptogenic-zone identification. Cortical electrical stimulation (CES) and somatosensory evoked potentials (SEP) were applied to map eloquent brain regions. Intra-operative cortical electrophysiological monitoring guided lesion resection. Individualized surgical strategies were adopted according to lesion characteristics. Post-operative follow-up spanned 1-4 years, and seizure outcomes were assessed using the modified Engel classification.

Results: Pre-operative brain MRI revealed heterogeneous epileptogenic etiologies, most frequently temporal-lobe signal abnormalities (17 cases) and focal cortical developmental anomalies (12 cases). Long-term VEEG allowed detailed recording of background electroencephalographic rhythms; sleep markedly elevated the yield of interictal epileptiform discharges. Interictal discharges were captured in 35 patients, and spontaneous clinical seizure discharges were successfully recorded in 15 patients. Among 21 patients receiving PET-CT, focal hypometabolism was observed within most lesion territories. Combined long-term VEEG with intracranial SEEG/ECoG electrodes precisely delineated discharge onset zones, while functional-mapping techniques ensured operative safety. Ten distinct individualized surgical protocols were implemented tailored to underlying pathology. Intra-operative ECoG detected sharp-wave activity in 35 patients, informing the extent of surgical resection. Integrating MRI findings with post-surgical histopathology clarified the etiological diagnosis for all patients. After 1-4 years of follow-up, 33 patients attained Engel class I outcomes, indicating satisfactory overall seizure control.

Note: Developmental anomalies encompass structural malformations and signal alterations, collectively termed cortical developmental abnormalities herein.

Conclusions: Video-electroencephalography combined with multimodal imaging including MRI and PET-CT enables reasonably accurate (though not absolute) dual anatomical-electrophysiological localization of epileptogenic zones. This multimodal framework provides robust evidence for individualized surgical planning and bears considerable clinical utility.

1.Introduction

Drug-resistant pediatric epilepsy represents a prevalent chronic neurological condition in child neurology, with secondary epilepsy constituting a major subset of refractory childhood epilepsy. Recurrent prolonged seizures inflict irreversible harm on the developing pediatric brain, manifesting as intellectual

regression, cognitive deterioration and behavioral disturbances, while severely impairing patients' daily functioning, academic performance and long-term psychosocial well-being ^[1]. For children whose seizures persist despite two or more adequate trials of antiseizure medications, surgical resection of the epileptogenic zone constitutes a key therapeutic modality to achieve long-term seizure remission or potential cure ^[2].

Successful pediatric epilepsy surgery hinges critically on precise localization of the epileptogenic zone's anatomical substrate and electrographic onset. Single-modality investigations carry notable limitations. Brain MRI vividly depicts structural cerebral lesions yet cannot confirm whether a given lesion acts as the genuine epileptogenic source. VEEG captures epileptiform electrographic events but offers no direct anatomical information. PET-CT infers epileptogenic territories via cerebral metabolic changes yet lacks sufficient localization accuracy when used in isolation. The pediatric brain undergoes ongoing maturation; eloquent cortex and epileptogenic networks are intricately intertwined, and multifocal or bilateral discharge patterns are frequently encountered. Reliance on any single imaging or electrophysiological test risks localization error, potentially yielding incomplete resection, persistent post-operative seizures, or iatrogenic injury to vital neural structures with permanent sequelae.

Current clinical guidelines endorse multimodal comprehensive evaluation integrating anatomical imaging, metabolic imaging and prolonged VEEG. Intracranial electrode implantation and electrical-stimulation functional mapping are added when indicated, enabling cross-validation among anatomical, electrographic and metabolic findings. This retrospective study enrolled 42 children with drug-resistant epilepsy treated surgically. Pre-operative brain MRI, long-term scalp VEEG and PET-CT data were synthesized; SEEG or ECoG intracranial electrodes were deployed for high-resolution localization in complex cases, and intra-operative electrophysiological monitoring guided tailored resection. This work aimed to delineate the clinical utility of VEEG combined with multimodal imaging for epileptogenic-zone localization, surgical decision-making and long-term prognostic assessment, generating evidence to optimize pre-surgical evaluation pathways for children with refractory epilepsy.

2. Materials and Methods

2.1 Pre-operative Assessment

Forty-two pediatric epilepsy patients managed in the Department of Neurosurgery, Tangdu Hospital, Air Force Medical University were retrospectively recruited. All participants received a confirmed epilepsy diagnosis based on brain MRI and prolonged scalp VEEG. Twenty-one patients underwent interictal PET-CT of the brain; 8 received long-term SEEG monitoring following depth-electrode implantation; 4 received long-term ECoG monitoring after placement of subdural cortical electrodes. This study was performed following the Declaration of Helsinki. The research protocol was approved by the Institutional Review Board of Tangdu Hospital, Air Force Medical University (approval number: K2025-123-32).

2.2 Patient Demographics

The cohort comprised 27 female and 15 male subjects, aged 8 months-18 years (mean 10.5 years). Disease duration ranged from 3 months to 12 years. All patients received 1-5 antiseizure agents pre-operatively.

2.3 Clinical History

Twenty-eight patients exhibited no additional epilepsy-related historical features beyond lesions identified on neuroimaging. Fourteen patients presented with relevant antecedents: 8 had a history of febrile convulsions (one concurrent with prior encephalitis and a positive family history of epilepsy; one with febrile convulsions plus familial epilepsy); 3 had prior traumatic brain injury (overlapping with one febrile-convulsion case and one case with a family history of epilepsy); 4 reported a positive family history of epilepsy (including one patient with traumatic brain injury and one with prior encephalitis and febrile convulsions); one patient had early-childhood intracranial hemorrhage; one had perinatal hypoxia.

2.4 Seizure Semiology

(1) Seizure-occurrence state: 18 patients experienced seizures in both wakefulness and sleep; 17 had seizures exclusively during wakefulness; 7 seized only during sleep.

(2) Seizure frequency: 8 patients had daily seizures; 28 had weekly-to-monthly episodes; 3 had annual seizures; 3 presented with isolated single seizures.

(3) Aura: 17 patients reported no aura; 5 younger children could not describe subjective pre-ictal phenomena; 20 experienced auras including unilateral limb numbness or weakness, focal limb jerking, facial twitching, auditory or visual hallucinations, dizziness, blurred vision, fear, nausea, and déjà-vu or non-specific discomfort.

(4) Seizure types: 4 patients presented with focal-onset seizures; 7 evolved to generalized tonic-clonic seizures (GTC) following aura; 16 developed GTC without preceding aura; 5 had GTC episodes either with or without aura; 10 manifested staring accompanied by automatisms (laughing, vocalization, wandering, blank staring), occasionally progressing to secondary GTC (6 without aura, 4 with aura).

2.5 Pre-operative MRI

All 42 patients underwent epilepsy-protocol brain MRI on a 3.0-T GE scanner, acquiring axial, coronal and sagittal T1-weighted, T2-weighted and FLAIR sequences. Post-operatively, all patients received follow-up CT or MRI.

2.6 Long-Term VEEG

Pre-operative prolonged scalp VEEG was performed for every patient using an Optoelectronics 9200 video-EEG system. Thirty-six patients were monitored with the international 10-20 electrode system supplemented by bilateral sphenoidal electrodes. Six children younger than 5 years omitted sphenoidal electrodes and were monitored solely with the 10-20 montage. Recording duration was no less than 24 hours, adjusted according to individual seizure frequency.

2.7 PET-CT

Twenty-one of the 42 patients underwent interictal PET-CT scanning. The remaining 21 patients skipped PET-CT because neuroimaging had already demonstrated well-defined epileptogenic lesions.

2.8 Surgical Procedures

Eight patients underwent SEEG recording after depth-electrode implantation; among them, 7 subsequently received craniotomy for epileptogenic-zone resection, and one underwent SEEG-guided radiofrequency thermocoagulation. Four patients had subdural electrodes placed for ECoG monitoring. Six children under age 5 did not receive intracranial electrodes and were assessed via scalp 10-20 VEEG. Intracranial recording lasted ≥ 24 hours, adapted to seizure frequency.

2.9 Histopathological Evaluation

Resected epileptogenic-zone tissue specimens from 27 patients were submitted for neuropathological examination.

2.10 Follow-up

Post-operative follow-up covering 1-4 years was accomplished via outpatient clinic visits and telephone interviews.

3.Results

3.1 Pre-operative Imaging: MRI Findings

Pre-operative brain MRI demonstrated the following lesion distribution across the 42 patients: 17 temporal-lobe mass-like or structural abnormalities; 9 frontal- or occipital-lobe space-occupying lesions; 12 focal cortical dysplasias; 2 gray-matter heterotopias; one Sturge-Weber syndrome; one hypothalamic hamartoma. Correlating intra-operative observations and histopathology, among the 17 temporal-lobe signal-abnormality cases, 10 harbored hippocampal sclerosis with gliosis, 4 exhibited tumor-like temporal-lobe lesions, and 3 had temporal-lobe arachnoid cysts. Imaging-based anatomical localization and pathological diagnoses were concordant for the remaining cases.

3.2 Pre-operative Electrophysiology: Scalp VEEG Findings

All patients completed pre-operative long-term scalp VEEG. Analyses encompassed background rhythms, interictal discharges and ictal discharges.

3.2.1 Background Rhythms

During quiet wakefulness, background rhythms matched age-appropriate norms in 37 patients; 5 showed mildly slowed background activity.

3.2.2 Interictal Discharges

Twenty-two patients displayed no epileptiform discharges while awake; epileptiform discharges were detected in 20 awake patients. Seven patients were free of epileptiform activity in both wakefulness and sleep; 35 patients demonstrated interictal discharges during sleep. Of these 35 patients, 26 had discharges confined to the lesional lobe or ipsilateral hemisphere; 3 exhibited bilateral-region discharges; 6 manifested multifocal or widespread epileptiform activity; 15 had discharges captured by sphenoidal electrodes.

3.2.3 Ictal Discharges

Clinical seizures were successfully recorded in 15 patients. For 11 of these 15, the dominant ictal discharge region or hemisphere agreed with interictal findings and with the MRI-defined lesion site. Four recordings could not lateralize or localize seizure onset.

3.3 Pre-operative Metabolic Imaging: PET-CT Findings

Twenty-one patients received interictal PET-CT (7 of whom later underwent intracranial-electrode implantation). Twenty-one lesion sites demonstrated focal hypometabolism; 14 showed concomitant ipsilateral hippocampal hypometabolism. One patient exhibited lesional hypometabolism plus additional contralateral-hemisphere focal hypometabolism. These metabolic observations informed estimation of epileptogenic-zone extent.

3.4 Refined Localization with Intracranial VEEG and Functional Mapping

For diagnostically complex patients, 8 underwent SEEG and 4 underwent ECoG. Intracranial recordings precisely mapped interictal discharge fields and the earliest ictal-onset zones. Cortical electrical stimulation delineated eloquent cortex. In 3 ECoG-monitored patients, somatosensory evoked potentials localized the central sulcus, lowering the risk of peri-operative neurological deficit.

3.5 Individualized Surgical Strategies

Ten customized surgical approaches were formulated based on lesion location, pathology and epileptogenic-network features:

Unilateral anterior temporal lobectomy combined with amygdaloseptotomy (10 cases)

Temporal-lobe lesionectomy plus ipsilateral anterior temporal lobectomy and amygdaloseptotomy (4 cases)

Temporal-lobe lesionectomy, anterior temporal lobectomy and amygdalectomy (hippocampus preserved) (1 case)

Temporal-lobe lesionectomy and anterior temporal lobectomy (hippocampus and amygdala preserved) (1 case)

Isolated temporal-lobe lesionectomy (1 case)

Resection of frontal-occipital space-occupying lesions with adjacent epileptogenic-zone clearance (9 cases)

Resection of cortical-dysplasia lesions plus resection of surrounding epileptogenic tissue (12 cases)

Resection of gray-matter-heterotopia foci (2 cases)

Epileptogenic-zone resection for Sturge-Weber syndrome (1 case)

SEEG-guided radiofrequency thermocoagulation of hypothalamic epileptogenic tissue (1 case)

3.6 Intra-operative Electrophysiological Guidance for Resection

Intra-operative ECoG monitoring was performed for 35 patients, revealing sharp-wave epileptiform activity. Surgeons adjusted resection margins dynamically according to sharp-wave distributions to optimize epileptogenic-zone removal.

3.7 Long-Term Post-operative Outcomes (1-4-Year Follow-up)

Seizure prognosis was graded via the modified Engel scale. Thirty-three patients achieved class I (seizure-free or solely isolated auras); 4 attained class II (infrequent non-disabling post-operative seizures); 3 reached class III (markedly reduced seizure burden); 2 remained class IV (no meaningful seizure improvement). Overall surgical efficacy was robust.

4. Discussion

Etiologies underlying pediatric secondary epilepsy are heterogeneous. Epileptogenic zones frequently combine structural anatomical abnormalities and pathological electrographic activity. Isolated imaging or scalp-EEG data are insufficient for precise localization and surgical margin definition. This study implemented a multimodal pre-surgical evaluation workflow integrating VEEG, anatomical MRI and metabolic PET-CT. SEEG/ECoG intracranial electrodes added high-fidelity tracing for difficult cases, complemented by real-time intra-operative electrophysiology. Cross-validation among anatomical, electrographic and metabolic data supported individualized surgical planning. Extended post-operative follow-up demonstrated seizure-free outcomes for most patients, confirming the merits of this multimodal paradigm for pre-surgical pediatric epilepsy assessment and supporting its broader clinical adoption.

For children with suspected epileptic seizures, we routinely perform 3.0-T epilepsy-protocol brain MRI (axial, coronal, sagittal T1-, T2- and FLAIR sequences). This protocol delivers superior structural detail compared with conventional MRI and facilitates identification of seizure-associated lesions. Next, prolonged VEEG is acquired using the 10-20 system; sphenoidal electrodes are added for most patients, with recording duration ≥ 24 hours. In this cohort, interictal epileptiform-discharge yield was 47.6% (20/42) during wakefulness and 83.3% (35/42) during sleep. Twenty patients exhibited epileptiform discharges in both wakefulness and sleep; 15 manifested discharges exclusively during sleep. Compared with routine short-duration EEG, prolonged VEEG captures continuous wake-sleep electrographic data and increases the likelihood of recording clinical seizures (15 events captured here). Concurrent video further improves the reliability of epileptogenic-zone inference. Sphenoidal electrodes enhance detection of temporal-lobe discharges, as illustrated by 16 patients in this study whose temporal-lobe epileptiform activity was visible only on sphenoidal channels. Across interictal and ictal epochs, 34 patients (81%) showed dominant epileptiform discharges confined to the lesional lobe or ipsilateral hemisphere, exhibiting high concordance with MRI localization. This lateralizing-localizing signal constitutes the predominant electrophysiological pattern.

Temporal-lobe epilepsy represented a large subgroup in this series (17/42, 40.5%). Selective temporal-lobe resection targeting temporal cortex together with the hippocampus-amygdala complex constitutes the most established surgical procedure for this entity, associated with low surgical-complication risk and favorable therapeutic response^[3]. For pediatric temporal-lobe epilepsy, our combined-modality workup suggests the following interpretive framework(As in Fig. 1 and Fig. 2, Case 1 and Case 2). When MRI shows marked unilateral hippocampal or amygdala signal-volume alterations, VEEG documents prominent ipsilateral-sphenoidal epileptiform discharges, and PET-CT demonstrates hypometabolism within the lesion plus ipsilateral mesial-temporal hypometabolism, the ipsilateral hippocampus-amygdala complex should be included within the resection volume. By contrast, if sphenoidal-electrode discharges are minimal or absent, MRI shows preserved hippocampal-amygdala volume-signal characteristics, and PET-CT shows only lesional hypometabolism without mesial-temporal hypometabolism, the hippocampus-amygdala may be preserved. Multimodal synthesis across scalp VEEG, MRI, PET-CT and clinical semiology guides the final decision. Prominent sphenoidal-electrode epileptiform activity serves as an important indicator for mesial-temporal resection yet cannot be interpreted in isolation.

Published literature notes that multifocal or bilateral lesions do not universally preclude epilepsy surgery^[4]. Resection remains feasible when a single primary epileptogenic zone can be reliably identified. Nevertheless, patients with multifocal disease present substantial localization challenges. For instance, many children with tuberous sclerosis bear multiple potentially epileptogenic tubers; distinguishing the seizure-generating tuber may be impossible, rendering them ineligible for resective surgery. Multimodal integration of VEEG, imaging and clinical semiology can sometimes pinpoint the dominant ictal-onset tuber. One patient in our cohort with tuberous sclerosis harbored MRI-visible tubers in the right frontal, right temporal and left parietal lobes(As in Fig. 3, Case 3). Scalp VEEG demonstrated abundant

right-frontal sharp-and-spike-wave interictal activity; seizures were aura-free generalized tonic-clonic events with early right-frontal ictal predominance. The patient was right-handed with intact language function, consistent with a non-dominant right hemisphere. Since PET-CT would likely show hypometabolism across all three tubers without additional localizing value, PET-CT was omitted. The child underwent craniotomy for right-frontal tuber resection guided by intra-operative ECoG targeting areas of frequent epileptiform discharges, and remained seizure-free for more than three-year follow-up.

When epileptogenic zones abut or partially overlap eloquent cortex, scalp EEG only provides coarse regional localization, complicating surgical planning. In these circumstances intracranial-electrode implantation is indicated (As in Fig. 4 and Fig. 5, Case 4 and Case 5). Subdural cortical grids can be placed over the lesional cortex; alternatively SEEG depth electrodes are stereotactically inserted into and around the lesion including functionally-eloquent territories [5]. Prolonged intracranial VEEG delineates interictal discharge distributions and ictal-propagation patterns. Intra-monitoring electrical stimulation maps cortical function, because post-craniotomy visual inspection of gyral anatomy (atrophy, discoloration, malformation) is often misleading. For sensorimotor cortex, somatosensory evoked potentials via subdural electrodes further identify the central sulcus. Collectively these techniques facilitate maximal safe resection while preserving neurological function.

In this cohort, 79% (33/42) achieved post-operative seizure freedom; another 17% (7/42) attained partial seizure reduction, yielding high caregiver satisfaction. Post-operative surveillance included annual VEEG to track resolution of epileptiform discharges alongside antiseizure-medication stewardship. Among patients seizure-free for ≥ 2 years, 36% (15/42) demonstrated substantially improved or normalized EEG findings, supporting individualized medication-tapering and discontinuation protocols.

In summary, surgical intervention benefits carefully selected children with secondary epilepsy and improves quality of life. Earlier surgery capitalizes on greater developmental brain plasticity and fosters optimal neurodevelopment [6–9]. Video-electroencephalography combined with multimodal imaging (brain MRI, PET-CT) enables robust anatomical-functional epileptogenic-zone localization in pediatric secondary epilepsy. When complemented by intracranial-electrode recording and intra-operative ECoG-guided tailored resection, this diagnostic-surgical pathway yields excellent long-term seizure outcomes. It represents a preferred pre-surgical assessment algorithm for children with secondary epilepsy and merits widespread clinical implementation [10–11].

Abbreviations

VEEG

video-electroencephalographic monitoring

MRI

magnetic resonance imaging

PET-CT

positron emission tomography-computed tomography

ECoG
electrocorticography
SEEG
stereoelectroencephalography

Declarations

6. Conflict of Interest

The authors declare no conflict of interest.

7. Author Contributions

Chao Wang and Jia-hua Zhou¹ led study design and prepared the manuscript; Xiao-rong Jing carried out the experiments and performed statistical analysis; Chen Yao performed data analysis and interpretation; Wei Zhang provided data collection. All authors read and approved the final manuscript.

8. Availability of data and materials

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

9. Acknowledgments

We acknowledge the assistance from the Tangdu Hospital, Air Force Medical University.

10. Ethics approval and consent to participate

The research protocol was approved by the Institutional Review Board of Tangdu Hospital, Air Force Medical University (approval number: K2025-123-32).

All patients provided written informed consent prior to the examinations.

All participants gave informed consent. The article does not involve personal information such as the names of the participants.

11. Funding

This study was not supported by any funds.

12. Copyright Transfer Statement

All authors have read and approved the final version of this manuscript, and agree to submit it to BMC Medical Imaging for publication.

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Figures

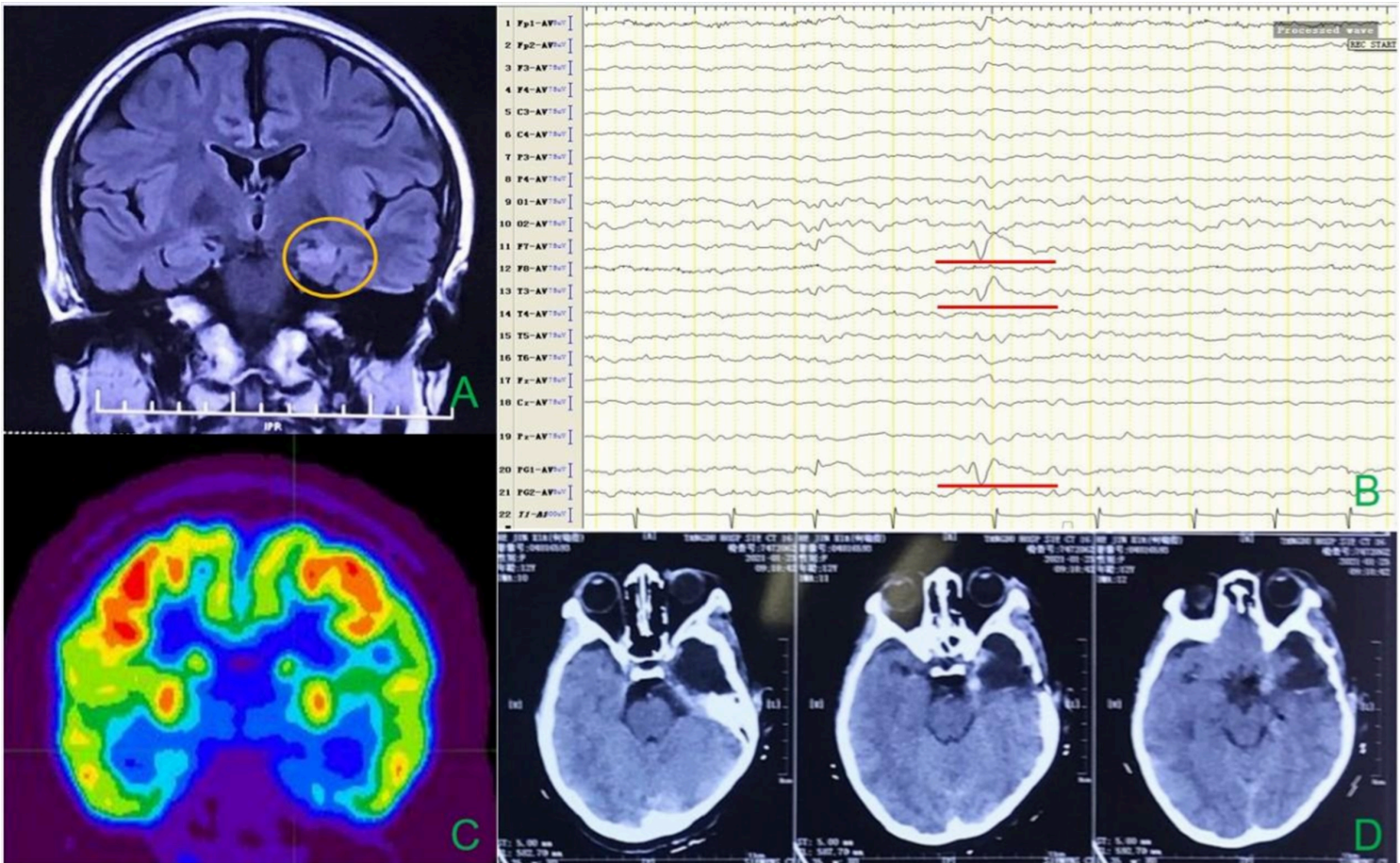


Figure 1

(Case 1: Left hippocampal sclerosis) A. Preoperative head Flair sequence MRI shows a high signal in the left hippocampus (marked by the yellow circle); B. Preoperative scalp EEG shows a large number of spike waves in the left sphenoid bone electrode (PG1), left anterior temporal (F7), and left middle temporal (T3) during interictal periods; C. Preoperative PET-CT shows significant hypometabolism in the medial left temporal lobe; D. Postoperative CT shows that the left anterior temporal hippocampal amygdala has been resected.

(Pathology: Local neurons are arranged abnormally along with glial cell proliferation, and the left hippocampal cortex shows developmental malformation.)

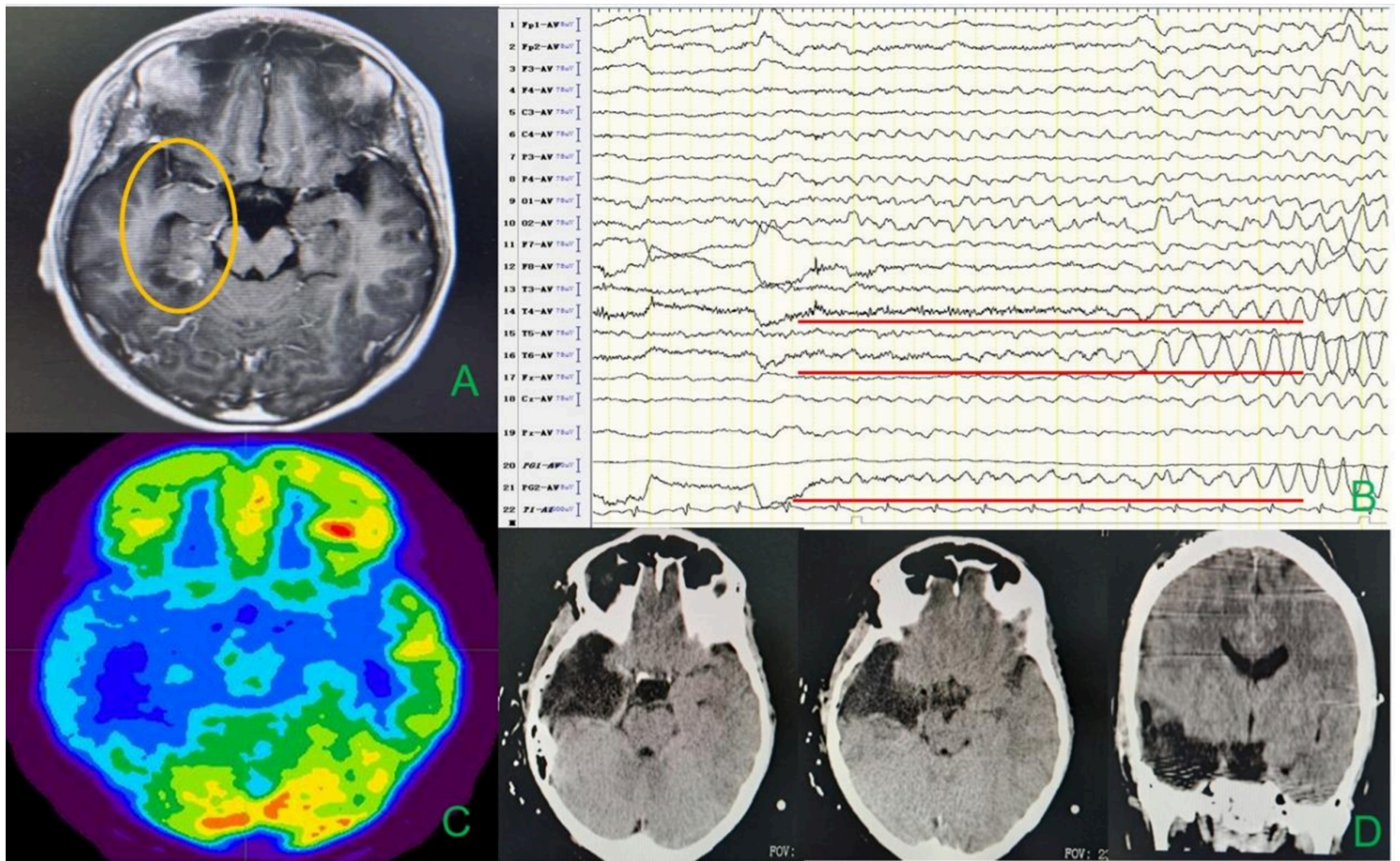


Figure 2

(Case 2: Medial temporal lobe lesion on the right side.) A. Preoperative head T1 sequence MRI shows lesion in the medial temporal lobe (hippocampal region) (marked by the yellow circle); B. Preoperative scalp EEG shows abnormal discharge at the onset of the seizure in the right sphenoid bone electrode (PG2), right middle temporal (T4), and right posterior temporal (T6) theta rhythms; C. Preoperative PET-CT shows significant hypometabolism in the medial and lateral temporal lobes of the right side; D. Postoperative CT shows the removal of the space-occupying lesion in the medial temporal lobe of the right side and the hippocampal amygdala of the anterior temporal lobe.

(Pathology: Local cortical neurons show hyperplasia and disordered arrangement, accompanied by gliosis, and there is a tendency towards cortical dysplasia.)

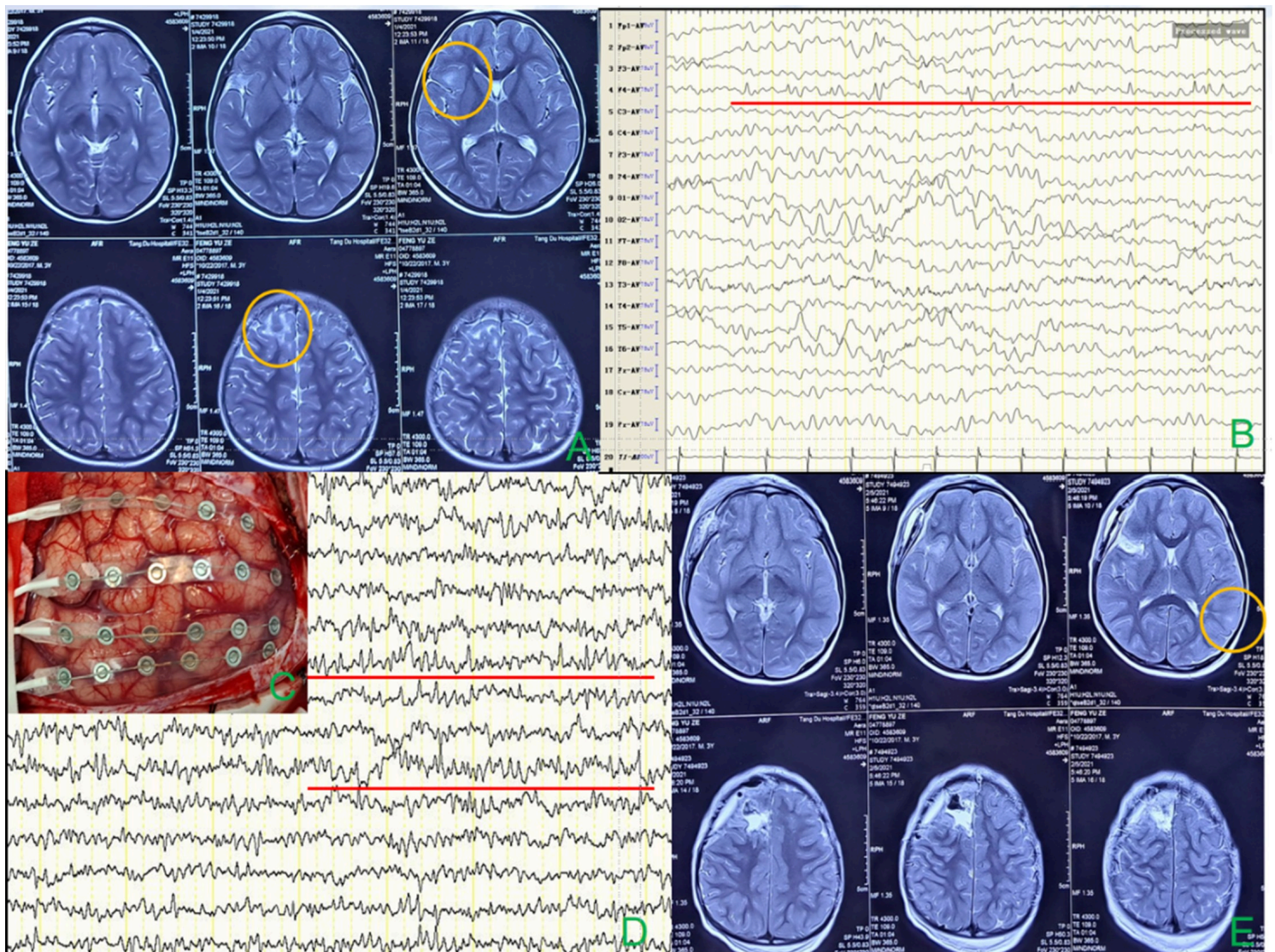


Figure 3

(Case 3: Multiple nodular sclerosis.) A. Preoperative head T2 sequence MRI shows multiple structural abnormalities in the right frontal lobe, right temporal lobe, left parietal lobe (middle posterior temporal? etc.) (as indicated by the yellow circles in Figures A and E); B. Preoperative scalp EEG shows a large number of sharp and spike waves in the right frontal and right frontal pole (the area marked by the red line); C. During the operation, cortical electrodes were placed on the cortical area of the right frontal lesion for electroencephalogram (ECoG) recording; D. ECoG recorded a large number of sharp waves in multiple leads (as indicated by the red line area); E. Postoperative MRI results after resection of the right frontal lesion and surrounding epileptic lesions.

(Pathology: 1. Right frontal lobe ganglioglioma, 2. Right frontal lobe cortical dysplasia, FCD type IIb .)

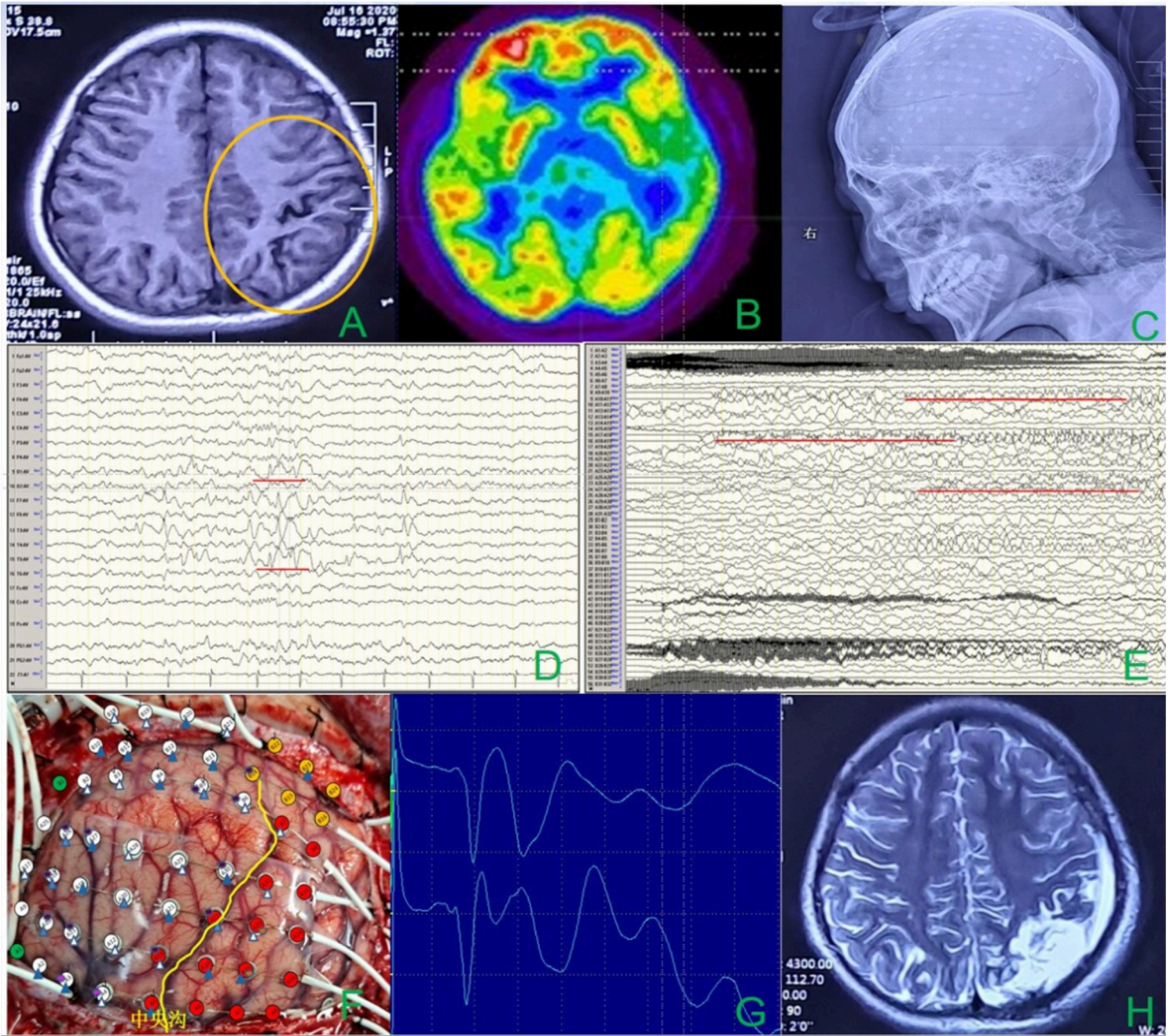


Figure 4

(Case 4: Left Temporo-Parieto-Occipital Lobe Dysplasia.) A. Preoperative head T1 sequence MRI shows abnormal structures in the left temporal-parieto-occipital lobe (marked by the yellow circle); B. Preoperative PET-CT shows significant low metabolism in the left temporal-parieto-occipital region; C. X-ray results after the first craniotomy, after implanting cortical electrodes in the region of the left temporal-parieto-occipital dysplasia; D. Preoperative scalp EEG shows a large number of sharp-slow waves in the posterior left head region (particularly at posterior temporal T5 and occipital O1); E. Cortical electrode IEEG shows that the onset of the seizure is preferentially abnormal discharges in the left posterior region after electrical stimulation; F. Schematic diagram of cortical electrical stimulation results and evoked potential SEP localization to the central sulcus after implanting cortical electrodes and locating the central sulcus; G. Representative waveform of evoked potential (SEP) located to the

central sulcus (while the waveforms recorded in the anterior and posterior central lobes also show clear phase inversion); H. MRI results after the second craniotomy to remove the epileptic lesion in the left temporal-parieto-occipital region.

(Pathology: Due to the patient's family's financial situation, no pathological examination was conducted.)

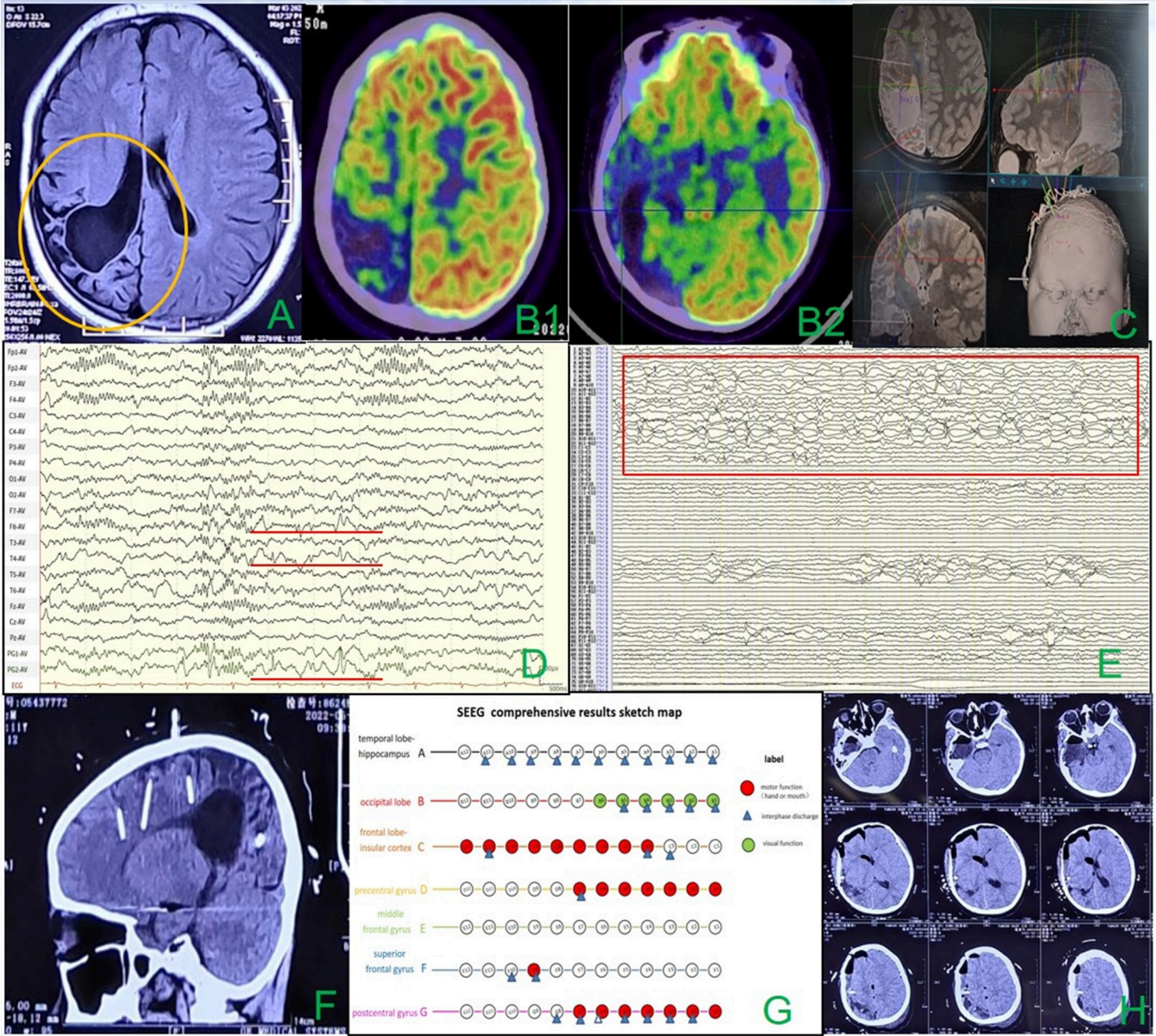


Figure 5

(Case 5: Right Temporo-parieto-occipital Lissencephaly.) A. Preoperative cranial MRI T2 sequence shows abnormal structures in the right temporal-parieto-occipital region (marked by the yellow circle); B1 & B2. Preoperative PET-CT shows significant hypometabolism in the right temporal-parieto-occipital area and the right anterior temporal lobe, hippocampus; C. An anatomical path map of the 7 brain deep electrodes

designed by the robot; D. Preoperative scalp EEG shows a large number of sharp-slow waves at the right sphenoid bone electrodes, right anterior temporal lobe, right middle temporal lobe; E. Postoperative intracranial electroencephalogram shows a large number of sharp waves in the right temporal lobe, hippocampus, occipital-parietal junction during the interictal period; F. CT shows the situation after the 7 brain deep electrode implantation surgery as designed in Figure C; G. Schematic diagram of the combined results of SEE-G and electrical stimulation; H. Postoperative CT shows the situation after the right anterior temporal lobe, hippocampus and amygdala resection + right temporal-parieto-occipital lobe disconnection surgery as implemented according to Figure G.

(Pathology: A major amputation was performed, so no pathological examination was conducted.)