Surgery for Epilepsy Involving Rolandoic and Peri-Rolandic Cortex

Shize Jiang (jiangshize1990@163.com)  
Huashan Hospital Fudan University  
https://orcid.org/0000-0001-6287-7834

Liqing Lang  
Fudan University Huashan Hospital Department of Neurosurgery

Bing Sun  
Fudan University Huashan Hospital Department of Neurosurgery

Dongyan Wu  
Fudan University Huashan Hospital Department of Neurology

Rui Feng  
Fudan University Huashan Hospital Department of Neurosurgery

Juanjuan He  
Fudan University Huashan Hospital Department of Neurosurgery

Jie Hu  
Fudan University Huashan Hospital Department of Neurosurgery

Liang Chen  
Fudan University Huashan Hospital Department of Neurosurgery

Ying Mao  
Fudan University Huashan Hospital Department of Neurosurgery

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Abstract

Purpose

To evaluate the risk factors associated with motor deficit following surgeries involving rolandic & peri-rolandic cortex and to introduce our surgical experiences dealing with lesions in this region.

Methods

We retrospectively reviewed patients who experienced drug-refractory epilepsies and received surgeries in our hospital. Medical records were carefully studied, and patients with lesions located in the rolandic & peri-rolandic cortex were screened. Those with detailed follow-up information were included. Lesion locations, resected regions, and invasive exploration techniques were studied to assess their relationship with the postoperative motor deficit.

Results

A total of 41 patients with lesions located in the rolandic or peri-rolandic cortex were included in this study. Of all these patients, 23 (56.10%) patients suffered from a transient motor deficit and 2 (4.88%) with a permanent disability after surgery. All eight patients with the anterior bank of precentral sulcus resected experienced motor deficit, and six of them gradually recovered within half a year. Seven patients with the anterior half of precentral gyrus resected did not experience permanent disability. A total of 14 (34.15%) patients received invasive exploration, and one of them had a permanent disability.

Conclusions

The anterior bank of the central sulcus is indispensable for motor functions, and the destruction of this region would inevitably cause a motor deficit. The upper part of the central sulcus could also be removed without significant neurological impairment if there is an epileptogenic lesion.

Introduction

Epilepsy is one of the most common types of neurological disorder, and around 30% of them could not be controlled well despite many antiepileptic drugs (AEDs) applied [1, 2]. For patients with drug-resistant epilepsies, surgery is the most effective method. For example, anterior temporal lobe resection is one of the most common epilepsy surgeries, with the seizure freedom rate of 50–70% for mesial temporal lobe epilepsies (MTLE) [3, 4]. Other surgical techniques for epilepsy include cortical lesion resection, disconnection, and modulation surgeries [5, 6].

The goal of epilepsy surgery is to eliminate seizures or limit their severity with or without medications. To achieve this goal, a complete resection or disconnection of the epileptogenic zone is needed [7–9]. For seizures arising from the non-eloquent cortex, to achieve a better surgical control rate, the potential epileptogenic cortex's maximum resection is encouraged [10–14]. However, for seizures arising from the rolandic and peri-rolandic cortex, we face the dilemma of maximum resection and maximum brain function protection [15, 16].

Several studies concerning epilepsy surgery arise from rolandic and peri-rolandic cortex [17, 18], and most of these studies focused on the pediatric population [19–21]. However, children tend to recover better than adults for surgeries involving the rolandic cortex because of brain plasticity [22]. So experience from children could not be applied to adult populations directly. Some other studies mainly focused on the prognosis of seizures. They rarely studied factors that will help preserve motor-related functions. One case series concerning precentral gyrus resection and neurological deficits [17]. However, the benign and malignant lesions are mixed within their case series. Since seizures due to benign and malignant reasons might have different surgical strategies, they should be considered separately. Therefore, we summarized our epilepsy surgical cases involving rolandic and peri-rolandic cortex with pathological proved benign lesions. Surgical considerations, together with factors that may influence the protection of motor functions, were introduced.

Methods

Data recruitment and ethics

This study was approved by the institutional review board of Huashan Hospital. We retrospectively reviewed surgical cases with suspected epileptogenic zone located in the rolandic cortex and peri-rolandic cortex at Huashan Hospital from Dec. 2012 till Dec. 2019. To be included in this study, all patients need to be: 1) with suspected epileptogenic zone located in or immediately adjacent to precentral and/or postcentral gyrus; 2) all received resective surgery; 3) with detailed presurgical and postsurgical imaging information (MRI, CT or intraoperative images) to confirm seizure focus and the location of resected areas; 4) have detailed follow-up information. Patients with gliomas above grade II were excluded from this study due to surgical considerations.

Presurgical evaluation procedure

A comprehensive presurgical evaluation was included in all patients, including 1) detailed seizure semiology analysis, 2) high-resolution MRI (including T1, T2, and Flair), 3) video-EEG analysis, 4) neuropsychological evaluation, and 5) FDG PET if possible. The operation strategies were made based on the conclusion reached in the preoperative multidisciplinary conference. Patients were referred to invasive evaluation if the available evidence is discordant.

Surgical strategies

All patients underwent epilepsy surgery by a single surgeon who specialized in functional neurosurgery. General anesthesia or awake anesthesia was applied. Direct brain stimulation (DCS) was performed for all awake surgical cases with an Ojemann stimulator. The stimulation parameters were as follows: biphasic square wave pulses (pulse width, 1ms) with the constant current were applied. Cortical mapping starts from 1mA with a step of 1 mA and up to a maximum of
15 mA. Somatosensory evoked potential (SSEP) was applied for all cases to identify the central sulcus. Motor evoked potential (MEP) was applied for all patients with lesions located in the motor-related region (primary motor & premotor).

**Follow-up**

Postoperative follow-up is one of the essential parts to assess the outcome of epilepsy surgeries. All patients were instructed to follow-up in the outpatient clinic at three months, six months, one year, and two years respectively, after the operation. Seizure outcome was assessed according to Engel's classification. Postoperative MR images were usually acquired three months after surgery. AEDs were gradually tapered off if no seizure occurred for at least two years after surgery with a neurologist's instruction.

**Image acquisition and processing procedures**

High-resolution MRIs were acquired with a 3T MRI device. Freesurfer (https://surfer.nmr.mgh.harvard.edu/) was applied to create a surface model of the brain. 3D slicer was applied to show three-dimensional reconstructed models of the brain and related models. Resected areas were manually delineated with MRICron (https://www.nitrc.org/projects/mricron) by two experienced neurosurgeons. The volume of interest (VOI) was then normalized to MNI space with SPM12 (http://www.fil.ion.ucl.ac.uk/spm/). Each VOI was then binarized to create the resection mask and resection masks located within the right hemisphere were flipped to the left side. After that, all VOIs were added up to create the distribution map of all resected areas.

**Results**

**Demographics data**

Forty-seven patients with drug-refractory epilepsies who received surgery in our center were included. However, 6 of them were excluded because detailed follow-up information or medical images were not available. Finally, a total of 41 cases were studied in our case series.

There are 28 (68.29%) male and 13 (31.71%) female within our case series, with a mean age of 23.88 ± 9.43 years. The mean duration of epilepsy was 12.96 ± 8.72 years. Of all these patients, 15 (36.59%) with lesions located in the left hemisphere, and 26 (63.41%) were located in the right hemisphere. Thirteen (31.71%) received awake surgery, while 28 (68.29%) with surgery under general anesthesia. Twenty-seven (65.85%) patients received non-invasive presurgical evaluation and 14 (34.15%) patients needed further invasive exploration, with 8 (19.51%) stereoelectroencephalography (SEEG) / 6 (14.63%) subdural electrodes, before surgical removal of the suspected lesion. Clinical characteristics of all patients were summarized in table 1.

**Topography of resections**

Of all 41 patients included, those resected cortexes were mainly located around the rolandic cortex or adjacent to the rolandic cortex, slightly extended to the posterior part of the frontal cortex and the anterior part of the parietal cortex (Fig. 1).

**Surgical interventions and motor functions**

Of all these 41 patients, 25 (60.98%) suffered from a motor deficit after surgery, among which 23 with transient motor impairment and 2 with a permanent disability. As for those 23 patients with transient motor deficit after surgery, 6 (26.09%) removed lesions located in the anterior half of the precentral gyrus, 3 (13.04%) with lesions located in the central sulcus, 3 (13.04%) with lesions in the premotor cortex and 3 (13.04%) with lesions involved both precentral and postcentral gyrus, 4 (17.39%) with lesions in the parietal lobe, 4 (17.39%) with lesions in the premotor cortex and 3 (13.04%) with lesions in the paracentral lobule or supplementary motor area (SMA). Transient motor deficit range from one week to 6 months (Table 2). The precentral gyrus was destructed from the anterior bank to the posterior end around the hand knob region in both patients with a permanent motor deficit.

Awake craniotomy was performed in 12 of these patients. However, 10 of them suffered from transient motor impairment, none with a permanent disability with long-term follow-up. Fourteen patients were treated with invasive evaluation (subdural electrodes/SEEG) before resectioning the suspected epileptogenic zone, and the surgical decision was made according to intracranial EEG results. However, one of them (patient 24) suffered from monoplegia after surgery (Table 2).

**Subjects with persistent disability**

Two patients (patients 24 & 28) suffered from persistent motor disability in our case series. For patient 24 (Fig. 3), preoperative MRI indicated a lesion located in the left superior frontal gyrus and invaded the precentral gyrus's anterior bank. This patient received subdural electrodes exploration, and EEG results indicated the lesion to be the epilepsy responsible region. The surgical plan was made based on The posterior part of the superior frontal gyrus and the anterior part of the precentral gyrus (with the anterior bank of the central sulcus destructed in the hand knob region) were resected in order to prevent seizures. This patient suffered from monoplegia of the left arm immediately after surgery. Although he received active rehabilitation treatment after surgery, his left hand suffered from a permanent deficit (muscle strength grade II).

A total of 6 patients (patients 8, 13, 17, 21, 33, 36) with lesions mainly located within the central sulcus or paracentral lobule (Fig. 4). All patients received awake craniotomy. Navigation and intraoperative electrophysiological monitoring were applied during surgery. Transient monoplegia immediately after surgery was found in all of them, and five patients gradually recovered within three months except one (patient 33) recovered in 6 months.

**Etiology and seizure outcome**

Surgical findings of all these 41 patients includes 23 (56.10%) FCD, 10 (24.39%) gliosis, 2 (4.88%) ganglioglioma, 2 (4.88%) vascular malformation and 5 (12.20%) other types of pathological change. Thirty-one patients (75.61%) achieved Engel class I outcome and eight patients (19.51%) with the Engel class II
outcome at the last follow-up. The pathological findings and the surgical results were summarized in Table 1.

General Surgical Complications

One patient in our case series suffered from intracranial hemorrhage needs hematoma evacuation. Two patients suffered from intracranial infection and need antibiotics. No death occurred in this series.

Case illustration

This is a 47-year-old right-handed male patient (patient 33) who suffered from intractable seizures for four years. Most seizures consisted of left hand somatosensory symptoms, which he described as ‘tightening’ or ‘shaking’. Seizure mainly involved the left hand and even the left limb. Sometimes may evolve into generalized tonic/clonic, especially during the night. Although multiple AEDs, such as valproate, carbamazepine, and clonazepam, were applied, the seizure is poorly controlled. He still experienced a daily seizure before admission.

A detailed preoperative evaluation was taken on admission. Structure MRI indicates lesion located within left central sulcus, slightly high signal in T2 Flair image (Fig. 5A, B & C). Surgery was performed under awake anesthetics. During the operation, direct stimulation of the brain was performed and electric stimulation of the lesion with muscle response of left hand. Detailed functional mapping was not available during the surgery because intraoperative seizures were induced by electric stimulation. The continuous motor evoked potential was applied to monitor motor functions. Surgical resection of the lesion was taken under great care. The patient experienced paralysis of the left upper limb after the operation. He could move his arm a little bit one week after surgery and gradually raise his left arm one month later. Three months after surgery, the muscle strength of his left hand reached grade V.

Discussion

Surgical removal of lesions located around the rolandic cortex remains a challenge for neurosurgeons because of the high risk of neurological deficit [18]. Techniques, such as awake anesthetics, could help preserve the brain's functions during surgery but not enough. In our case series, 41 patients received surgical removal of the epileptogenic cortex involving the rolandic and peri-rolandic cortex and 39 patients without a persistent motor deficit in long-term follow-up. This result is achieved based on a good understanding of the cortex's cytoarchitectonic basis and detailed peri-surgical evaluation techniques. We introduced our experiences dealing with cases around the rolandic cortex and hope our experience could help motor function protection in clinical practice.

The precentral gyrus as a whole is not all indispensable for motor function. Traditionally, the precentral gyrus was thought to be responsible for movement, and the postcentral gyrus was responsible for sensory information processing [23–25]. However, this is not always the case in the real setting. According to the cytoarchitectonic organization of the brain, Brodmann divided the brain into different sub-areas. Based on Brodmann’s observation, BA4, characterized by giant pyramid neurons, is responsible for voluntary movement. BA6, situated anterior to the primary motor cortex, is responsible for planning complex movements [26]. Penfield performed direct cortical stimulation to confirm functional representation regions with awake craniotomy and found motor response areas mainly situated around the central sulcus, but not purely in the precentral gyrus [23], which is similar to Brodmann’s findings. However, the boundary between BA4 and BA6 is not always consistent with anatomical landmarks. Zilles further studied the morphology and cytoarchitecture of the central sulcus with 32 human brains and found BA4 mainly located in the anterior bank of central sulcus [27]. That is, the anterior bank of the central sulcus might be the indispensable cortex for motor function. So, surgical removal of the precentral gyrus’s anterior part might not lead to a persistent motor deficit.

The lower part of the precentral gyrus is also safe to remove, even located in the central sulcus’s anterior bank. According to Penfield’s early observations, stimulation in the face representation area of the precentral gyrus could induce a bilateral facial motor response, but no ipsilateral motor response was observed with stimulation in the representation area of limb [23, 28]. So, motor responses are not always controlled by the unilateral hemisphere. This could explain why surgical removal of the lower part of the precentral gyrus will not cause permanent disability. In addition to identifying the motor representation cortex with DCS, it is also crucial to bear in mind that we need to find the boundary between the hand representation area and face representation area because the motor cortex below this boundary is relatively safe to remove.

An epileptogenic lesion which is located within the central sulcus is not always a contra indicator for surgery. The epileptic lesion might destroy the brain's function locally or at a considerable distance away [7]. This is most probably because of the interictal discharges and seizure spreading effects [29, 30]. So the epileptogenic lesion, especially those benign lesions that occurred early during the lifespan, may not harbor any function, even if they are located in the center of the eloquent cortex. Much attention should be paid to the perioperative evaluation stage to delineate the boundary of the lesion and the eloquent cortex to achieve a better prognosis, which is the case with our case series.

Whether invasive exploration, such as subdural electrodes or SEEG, helps protect the brain’s function for epilepsy surgeries involving the rolandic cortex is doubtful. In our early cases, subdural electrodes were applied to identify the potential epileptogenic zone and help us identify the eloquent cortex to avoid motor deficits. Surgical removal of the posterior part of the superior and middle frontal gyrus, together with the upper part of the precentral gyrus (the anterior bank of central sulcus was involved), was performed (supplemental figure). Successful control of epilepsy was achieved after surgery. However, two patients (patient 6 & patient 24) experienced a persistent left hand movement deficit. This might be because the commercially available subdural strips/grid is not precise enough for functional mapping in the eloquent cortex. Currently, each contact's diameter is 2.5mm, and the distance between two adjacent subdural strips/grid contacts is 10mm, which is inexactitude for delineating the boundary of the eloquent cortex. We need a more precise method to delineate the epileptogenic zone boundary and eloquent zone, which is the core problem in epilepsy surgeries. High-density ECoG grid/strip might help this scenario, but lots more work needs to be done.

Intraoperative neuro-electrophysiological monitoring is crucial in identifying the boundaries of the eloquent cortex and preserving the intact functions of the brain [31]. From our perspective, SSEP and MEP monitoring is a must, while awake surgery with DCS is recommended but not essential for motor function.
protection. For seizure arising from the rolandic cortex, it is good to identify the motor representation cortex as described before. However, it is quite common to induce a seizure for DCS, especially in the motor cortex[32, 33]. We need to be quite careful with DCS in the motor cortex. SSEP helps identify the central sulcus location, and continuous MEP monitoring is essential in helping protect motor functions. In our case series, all of them have SSEP and MEP monitoring. Twelve surgeries were performed under awake anesthesia, and none of them experienced long-term motor deficits.

Neuro-navigation is also recommended in epilepsy surgery involving rolandic cortex surgery but not enough. Brain shift due to loss of CSF is a big problem for neuro-navigations[34]. We can not rely on neuro-navigations to identify surgical boundaries if we could not correct brain shift related problems. 'Surface navigation' based on relative anatomical structures, such as vessels, bones, and gyrus, could supplement the current navigation system[35].

En bloc resection of the lesion is recommended. This is because the seizure is believed orientated from grey matter and corticectomy is enough for seizure control. Destructions of extra white matter in the rolandic region may lead to a persistent motor deficit. We need to find the boundary between the grey matter and the white matter to remove the epileptic grey matter purely. Thus, we could start from the relatively safe areas where the interface between grey matter and white matter is straightforward. The resected grey matter thickness could serve as the reference for the remaining cortex to be removed. We could also follow the boundary from relative safe areas to highly eloquent areas.

Limitations

In this study, we only included epilepsy surgeries with benign lesions. Seizures due to malignant lesions, such as GBM, were not included. This is because, for benign lesions, we could only remove regions that are responsible for seizure generation. Thus, functional preservation is of vital importance with minimum resection. However, for malignant lesions, such as GBM, we need to balance between tumor recurrent and function preservation. Sometimes aggressive resection is needed, and we have to sacrifice some functions of the brain. So, our result might not be suitable for malignant tumor-induced epilepsies in the rolandic area. Secondly, a relatively short follow-up time is also a disadvantage of our study. We will still work on this topic and provide more cases with a longer follow-up time later.

**Abbreviations**

AEDs, antileptic drugs

MTLE, mesial temporal lobe epilepsies

DCS, Direct brain stimulation

VOI, volume of interest

SEEG, stereoelectroencephalography

M.S., muscle strength

AHGP, anterior half of precentral gyrus

SFG, superior frontal gyrus

MFG, middle frontal gyrus

IFG, inferior frontal gyrus

SSEP, Somatosensory evoked potential

SMA, supplementary motor area

**Conclusion**

The central sulcus's anterior bank but not the precentral gyrus is indispensable for motor functions from the cortex's cytoarchitectonic basis. Besides, the facial representation area is bilaterally controlled. Thus, protection of the upper part of the central sulcus's anterior bank is vital for motor protection. However, Epileptic lesions might alter the function of the brain through interictal spikes and seizure spreading mechanisms. So if there is a lesion located in the upper part of the central sulcus, it could also be removed without significant neurological impairment with the assistant of intraoperative neurophysiological techniques.

**Declarations**

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Code availability: NA
Author contributions:

This study was completed under the supervision of Ying Mao. Jie Hu, Liang Chen, and Shize Jiang contributed to the study conception and design. Material preparation, data collection and analysis were performed by Shize Jiang, Liqin Lang, Bing Sun, Dongyan Wu, Rui Feng and Juanjuan He. The first draft of the manuscript was written by Shize Jiang and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Ethics approval: This study was approved by the institutional review board of Huashan Hospital.

Consent to participate: All participants were inform consented.

Consent for publication: All authors reviewed the paper and approved for publication

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Tables

Table 1 Demographic and clinical characteristics of all patients
|                          |       |
|--------------------------|-------|
| **Age**                  | 23.88±9.43 |
| **Sex**                  |       |
| Male                     | 28 (68.29%) |
| Female                   | 13 (31.71%) |
| **Seizure duration**     | 12.98±8.72 |
| **Side of surgery**      |       |
| Left                     | 15 (36.59%) |
| Right                    | 26 (63.41%) |
| **Anesthesia**           |       |
| Awake surgery            | 13 (31.71%) |
| General anesthesia       | 28 (68.29%) |
| **Invasive exploration** |       |
| SEEG                     | 8 (19.51%) |
| Subdural electrodes      | 6 (14.63%) |
| No                       | 27 (65.85%) |
| **Motor deficit**        |       |
| Transient motor deficit  | 23 (56.10%) |
| Permanent motor deficit  | 2 (4.88%) |
| **Pathology**            |       |
| FCD                      | 23 (56.10%) |
| Gliosis                  | 10 (24.39%) |
| Ganglioglioma            | 2 (4.88%) |
| Vascular malformation    | 2 (4.88%) |
| Others                   | 5 (12.20%) |
| **Seizure outcome**      |       |
| Engel I                  | 31 (75.61%) |
| Engel II                 | 8 (19.51%) |
| Engel III                | 1 (2.44%) |
| Engel IV                 | 1 (2.44%) |

Table 2 Individual patient information, including patient demographic, lesion locations, surgical techniques, resected areas, and seizure outcomes
| NO. | age/sex | Lesion location | seizure duration (years) | Presurgical MS | Subdural electrodes/SEEG | Awake Surgery | resected region | MS after surgery | MS recovery to normal | follow up (months) | current muscle strengt |
|-----|---------|-----------------|--------------------------|----------------|--------------------------|--------------|-----------------|-----------------|----------------------|-------------------|-------------------------|
| 1   | 41/F    | R frontal (Precentral gyrus) | 20 | V | Subdural electrodes | No | R AHPG | left leg MS II | 1 month | 78 | V |
| 2   | 29/M    | L frontal | 10 | V | NA | Yes | posterior part of L MFG | V | NA | 20 | V |
| 3   | 17/F    | L frontal & parietal | 5 | V | NA | Yes | L premotor cortex & postcentral gyrus | 0 | 6 months | 32 | V |
| 4   | 28/M    | R parietal | 10 | V | NA | No | R posterior parietal cortex | V | NA | 20 | V |
| 5   | 23/M    | R frontal | 15 | V | SEEG | No | posterior part of R MFG & IFG + AHPG | V | NA | 47 | V |
| 6   | 24/M    | L frontal & parietal | 20 | III | Subdural electrodes | No | L precentral & post central gyrus | right arm MS 0 | 1 month | 86 | III |
| 7   | 23/M    | R parietal | 20 | V | NA | No | R precuneus | V | NA | 65 | V |
| 8   | 7/M     | R frontal & parietal | 5 | V | Subdural electrodes | No | R precentral gyrus & post central gyrus | left arm MS 0 | 6 months | 63 | V |
| 9   | 10/F    | R Frontal | 7 | V | Subdural electrodes | No | R premotor | V | NA | 83 | V |
| 10  | 26/F    | L frontal | 6 | V | NA | No | L premotor cortex | V | NA | 46 | V |
| 11  | 19/M    | R frontal | 16 | V | NA | No | R MFG & AHPG | left arm MS 0 | 3 months | 22 | V |
| 12  | 18/M    | L frontal | 14 | V | SEEG | Yes | posterior part of L IFG | V | NA | 56 | V |
| 13  | 24/M    | L frontal & parietal | 20 | V | NA | Yes | L central sulcus | right arm MS 0 | 3 months | 37 | V |
| 14  | 40/F    | R frontal | 25 | V | NA | No | posterior part of R SFG | V | NA | 15 | V |
| 15  | 25/M    | R frontal | 20 | V | NA | No | posterior part of R MFG | V | NA | 31 | V |
| 16  | 25/M    | L frontal | 15 | V | Subdural electrodes | Yes | posterior part of R SFG & AHPG | right leg MS III | 1 week | 77 | V |
| 17  | 14/M    | R parietal | 4 | V | SEEG | Yes | R anterior parietal cortex | left hemisphere MS III | 1 month | 21 | V |
| 18  | 24/M    | R frontal | 20 | V | NA | No | R premotor cortex | V | NA | 30 | V |
| 19  | 26/F    | R perietal | 13 | V | NA | No | R supramarginal gyrus & posterior part of postcentral gyrus | V | NA | 15 | V |
| 20  | 29/M    | R preial | 23 | V | SEEG | No | R precuneus | V | NA | 42 | V |
| 21  | 21/F    | L frontal | 9 | V | SEEG | Yes | L anterior part of paracentral lobule & posterior part of SFG | left foot MS 0 | 6 months | 41 | V |
| 22  | 22/M    | R frontal | 14 | V | NA | No | posterior part of R SFG & SMA | left arm MS III | 1 week | 21 | V |
| 23  | 14/F    | L frontal (intersection between L | 8 | V | NA | No | posterior part of L MFG & IFG | V | NA | 31 | V |
| Case | Age/Sex | Location | Electrode Count | Type | Stimulation | Area | Duration | Procedure | Lesion | Side | Procedure |
|------|---------|----------|-----------------|------|-------------|------|----------|-----------|--------|-------|-----------|
| 24   | 15/M    | L frontal | 5 V Subdural electrodes | No | posterior part of L SFG & precentral gyrus (hand knob) | right arm | NA | SEEG | 73 | right hand MS II |
| 25   | 14/M    | R frontal | 10 V SEEG | Yes | posterior part of R SFG & AHPG | Left arm | 3 months | 24 | V |
| 26   | 30/M    | R frontal | 15 V NA | No | R premotor cortex | left arm | 1 week | 17 | V |
| 27   | 15/M    | L parietal | 10 V NA | No | L parietal disconnected | right arm | 1 month | 16 | V |
| 28   | 17/F    | R frontal | 5 V NA | No | R premotor + primary motor | left arm | NA | 83 | left hand MS IV |
| 29   | 24/M    | L frontal | 2 V NA | No | L posterior part of SFG | V | NA | 31 | V |
| 30   | 45/F    | R frontal | 27 V NA | No | R premotor | left arm | 2 weeks | 14 | V |
| 31   | 27/F    | R frontal | 23 V NA | Yes | R posterior part of SFG & MFG + AHPG | left arm | 1 month | 54 | V |
| 32   | 17/F    | L precuneus | 3 V NA | Yes | mesial parietal +paracentral lobule (posterior part) | right foot | 1 month | 13 | V |
| 33   | 47/M    | R central sulcus | 4 V NA | Yes | R central sulcus | left arm | 3 months | 17 | V |
| 34   | 45/M    | R parietal | 40 V NA | No | R parietal cortex | left arm | 6 months | 48 | V |
| 35   | 19/F    | L frontal & parietal | 8 IV SEEG | Yes | L frontal & parietal (superior part of precentral gyrus preserved) | right arm | 1 month | 57 | IV |
| 36   | 25/M    | R central sulcus | 1 V NA | No | R central sulcus | left arm | 3 months | 14 | V |
| 37   | 17/M    | L parietal | 9 V NA | No | L superior parietal cortex | right arm | 1 week | 19 | V |
| 38   | 16/M    | R frontal | 3 V NA | No | R premotor cortex | left arm | 1 week | 57 | V |
| 39   | 16/M    | R frontal | 4 V NA | No | R premotor cortex | V | NA | 50 | V |
| 40   | 34/M    | R frontal | 30 V NA | No | R premotor cortex | left arm | 1 week | 47 | V |
| 41   | 27/F    | R frontal | 14 V SEEG | No | R premotor cortex | V | NA | 46 | V |

AHPG, anterior half of precentral gyrus. SFG, superior frontal gyrus. MFG, middle frontal gyrus. IFG, inferior frontal gyrus. SMA, supplementary motor area

**Figures**
Figure 1
distribution probability map of resected regions of the 41 patients. The red color indicates higher density, while blue indicates a lower density. (A) lateral view, (B) medial view, (C) top view.

Figure 2
case illustration with persistent disability. One of the patients (patient 24) with persistent motor disability in our case series. The MRI before surgery show lesion is located mostly in the premotor area and partially invaded into the precentral gyrus (A & B). No significant enhancement was found after contrast (C & D). Postoperative MRIs (E, F & G) depict the resected area, with partial disruptions of the anterior bank of the central fissure. The three-dimensional map of the resected area was shown in red (H).
Figure 3
Distribution probability map of lesions within central sulcus or paracentral lobule. Of all cases included, the lesion could course along the central sulcus from the top (paracentral lobule) to the end (sylvan fissure). (A) lateral view, (B) medial view, (C) top view.

Figure 4
Case illustration of one patient with a lesion located in the central sulcus (patient 33). Preoperative sagittal (A), coronal (B), and axial (C) MRI indicate lesion located in central sulcus, a slightly high signal in T2 Flair images. Three-dimensional reconstruction of the lesion and the cerebrum indicate the lesion (in red)
located deeply in the central sulcus (D, E & F). Surface registration of intraoperative image with three-dimensional brain model (G).

Figure 5

Illustration of Brodmann's somatomotor and somatosensory representation areas. Brodmann areas were automatically parcellated with FreeSurfer. Different areas were marked with different colors, with purple for BA1, pink for BA2, blue for BA3, green for BA4, and azure for BA6. Two-dimensional images (A, axial view. D, sagittal view, and E, coronal view) together with three-dimensional images (B, pial reconstruction. C, inflated image, and F, white matter surface) were shown. G, the enlarged section across the precentral gyrus with BA1 is shown in a 3D image.