

Surgical Strategies in Patients with the Supplementary Sensorimotor Area Seizure

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Objective : This study was designed to analyze surgical strategies for patients with intractable supplementary sensorimotor area(SSMA) seizures.

Methods : Seventeen patients who had surgical treatment were reviewed retrospectively. Preoperatively, phase I (non-invasive) and phase II (invasive) evaluation methods for epilepsy surgery were done. Seizure outcome was assessed with Engel's classification. The mean follow-up period was 27.2 months (from 12 months to 54 months).

Results : An MRI identified structural abnormality in eight patients and 3D-surface rendering revealed abnormal gyration in three. PET, SPECT, and surface EEG could not delineate the epileptogenic zone. Video-EEG monitoring with a subdural grid or depth electrodes verified the epileptogenic zone in all patients. Surgical procedures consisted of a resection of the SSMA and simultaneous callosotomy in two patients, a resection of the SSMA extending to the adjacent area in seven, a resection of a different area without a SSMA resection in seven, and a callosotomy in one. Seizure outcomes were class I in 11 (65%), class II in five (29%), class III in one (6%).

Conclusion : In patients with intractable SSMA seizure, surgery was an excellent treatment modality. Precise delineation of the epileptogenic zone based on multimodal diagnostic methods can provide good surgical outcomes without neurological complications.

KEY WORDS : Supplementary sensorimotor area · Seizure · Outcome.

Introduction

Patients with seizures originating in the supplementary sensorimotor area(SSMA) and adjacent areas frequently display medical intractability and a high seizure frequency. Ictal semiology suggestive of seizures from the SSMA is characterized by the sudden and brief tonic posturing of abduction and external rotation of the shoulder with flexion at the elbow, with or without head turning. These seizures are usually abrupt and brief, attacks are frequent, and awareness is preserved in most cases, though they commonly occur during sleep. These clinical manifestations are clues and of value in predicting seizures from SSMA¹⁶⁾. Electrophysiologic definition of the epileptogenic zone of the SSMA, however, is complex due to the geographic location of the SSMA in the depths of the interhemispheric fissure and the rapid propagation of ictal discharges from the SSMA to adjacent areas and the contralateral hemisphere, or

from adjacent areas and the contralateral hemisphere to the SSMA. Surface EEG recording is almost impossible to differentiate whether the seizures arise from the SSMA or spread to the SSMA from adjacent or remote areas¹⁸⁾. Therefore, the precise localization of the epileptogenic zone is essential for surgery. The ictal onset from the SSMA and the propagation of the ictal discharges from adjacent areas to the SSMA could develop the seizures just as the presentation from the SSMA. Intracranial EEG recording with subdural grid electrodes rather than depth electrodes are useful to delineate the ictal onset zone and the location of the eloquent area near the SSMA¹⁵⁾.

Surgical resection must include the SSMA or adjacent area producing the SSMA seizures for seizure control. During surgery, electrocorticography(ECoG) and functional mapping of the eloquent area is mandatory to avoid new development of neurologic deficit^{1,4)}. Authors report surgical outcomes in patients with SSMA seizure developed from the SSMA and/or

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adjacent areas, based on the results from multimodal diagnostic modalities.

Materials and Methods

Patients

From January 2000 to June 2005, a total of seventeen patients received a final diagnosis of SSMA seizure based on the characteristic ictal semiology and underwent surgical treatment. We included the patients that showed semiology characterized by sudden and brief tonic posturing of either unilateral or bil-

ateral extremities. Face and body trunk were occasionally involved, and awareness was preserved^{3,4,16}. The term SSMA seizure is usually used to identify seizures that present symptomatology suggesting that they are the result of the epileptic activation of the SSMA. An SSMA seizure is not the result of epileptiform discharges arising from the SSMA. When the epileptogenic zone is in the SSMA, it is called SSMA epilepsy¹³.

Preoperative evaluation methods

Preoperative evaluation methods included both Phase I and Phase II. Phase I evaluations were composed of noninvasive

Table 1. Patients profile, preoperative evaluation, extent of resection and surgical outcome

Patient No. and age/sex	Seizure			Preoperative evaluation					Surgery	Postoperative neurologic complication	Postoperative outcome (Engel's classification)
	Age of onset (yrs)	Duration (yrs)	Frequency	FDG-PET	SPECT	EEG with depth electrodes and SDG	Amytal test	Magnetic resonance image			
1(38/f)	3	35	1~4/day	—	—	Lt. SSMA	—	Lt. frontal	Lt. SSMA lesionectomy & CC	Rt. hemiparesis	I
2(28/M)	15	13	0.5/month	—	Lt. T	Rt. SSMA & Rt. MFG	L : Lt. m : Rt.	—	Rt. SSMA & MFG lesionectomy	Rt. hemiparesis	I
3(24/f)	12	12	1/month	Rt. F-P	—	Lt. SSMA & Lt. orbitofrontal	L : Rt. m : Rt.	Lt. frontal	Lt. SSMA & frontal lesionectomy	—	I
4(5/f)	1	4	4/month	—	Rt. F	Rt. SSMA & SFG & MFG/	—	—	Rt. SSMA & MFG & SFG lesionectomy	Lt. arm weakness	I
5(40/M)	16	24	5/month	Lt. T Bilateral P	Rt. F	Lt. SFG	L : Lt. m : Rt.	—	Lt. SSMA & SFG lesionectomy	—	III
6(28/f)	14	14	2/day	Lt. anterior F	—	Lt. frontal premotor area	—	Lt. frontal	Lt. SSMA & frontal lesionectomy	—	II
7(34/f)	7	27	5~6/day	—	Rt. T	Rt. SSMA	L : Lt. m : Rt.	—	Rt. SSMA & MFG & SFG lesionectomy	—	I
8(31/M)	12	19	1/day	—	Rt. F	Rt. frontal	L : Lt. m : Lt.	Rt. frontal	CC	—	I
9(19/M)	18	1	1~4/day	Lt. anterior F-P	—	Lt. frontal	L : Rt. m : Rt.	Lt. F-P	Lt. MFG & IFG lesionectomy	—	II
10(33/f)	6	27	1/day	Lt. P Bilateral T	—	Lt. frontal	L : Lt. m : Lt.	—	Lt. SSMA & MFG & SFG lesionectomy	Rt. hemiparesis	I
11(20/M)	5	15	5/month	Lt. F-T-P	—	Lt. central & precentral	L : Rt. m : Rt.	Lt. F-P	Lt. Central & precentral lesionectomy	—	II
12(58/M)	3	55	1/month	Rt. T	—	Lt. prefrontal	L : Rt. m : Rt.	Lt. frontal	Lt. orbitofrontal lesionectomy	—	I
13(21/f)	17	4	1/day	Bilateral T	—	Rt. orbitofrontal	L : Lt. m : Lt.	—	Rt. orbitofrontal lesionectomy	—	I
14(17/M)	3	14	5/day	Rt. T	—	Rt. orbitofrontal	L : Lt. m : Lt.	—	Rt. orbitofrontal lesionectomy	—	II
15(38/M)	13	25	3/day	Lt. T	Lt. F-T	Lt. MFG	L : Lt. m : Lt.	Lt. frontal	Lt. MFG lesionectomy/	—	II
16(23/f)	4	19	1/day	Bilateral P	Lt. T	Lt. parietal	L : Lt. m : Rt.	—	Lt. parietal lesionectomy	Transient apraxia	I
17(33/M)	8	25	3/day	—	—	Rt. SSMA	—	—	Rt. SSMA lesionectomy & CC	—	I

No : number, M : male, f : female, Lt. : left, Rt. : right, f : frontal, T : temporal, P : parietal, yrs : years, FDG-PET : fluorodeoxyglucose-positron emission tomography, SPECT : single photon emission computerized tomography, SDG : subdural grid, EEG : electro-encephalography, L : language, m : memory, SSMA : supplementary sensorimotor area, MFG : middle frontal gyrus, SFG : superior frontal gyrus, CC : corpus callosotomy, — : preoperative evaluation was not done

methods such as history taking, neurologic examinations, brain MRI, 3D-surface rendering with MRI, fluorodeoxyglucose-positron emission tomography (FDG-PET) or single photon emission computerized tomography (SPECT), chronic video-EEG monitoring with surface electrodes, neuropsychological testing, and an amygdala test. And Phase II evaluations were composed of invasive methods like chronic video-EEG monitoring with subdural grids and depth electrodes. During surgery, ECoG and functional mapping were done to decide the extent of the resection margin in correlation with the intracranial EEG data and to identify the eloquent area.

Operation

A resection of the SSMA and simultaneous callosotomy were done in two patients (11.7%). A resection of the SSMA extending to an adjacent area, such as the middle frontal gyrus, orbitofrontal gyrus, superior frontal gyrus, prefrontal cortex and middle & superior frontal gyrus, were performed in seven (41.1%). A resection of a different area without the SSMA resection was done in seven patients (41.1%). A callosotomy was done in one (5.8%) who had previous surgery in the premotor cortex.

Surgical outcome

The surgical outcome was evaluated by using neurological examinations and Engel's seizure classification. The mean follow-up period was 27.2 months (from 12 months to 54 months).

Results

Patient population (Table 1)

The mean age of the patients was 28.8 years old (from 5 years old to 58 years old). There were nine males and eight females. The age of the seizure onset was 3 to 18 years old (mean : 9.9 years old).

Semiology

Twelve patients (70%) showed sudden onset of bilateral tonic posture evolving to generalized tonic clonic seizures. Seven patients (41%) showed hypermotor activities like thrashing which progressed to generalized tonic clonic seizures, and seven patients (41%) started with automatism evolving to SSMA seizures. A couple of patients presented different types of seizures during chronic video-EEG monitoring.

Radiologic findings (Table 1)

Brain MRIs revealed structural abnormality in eight patients (47.0%). Out of these, abnormalities were identified in the frontal lobe in six patients and fronto-parietal lobe in two patients. Nine did not show any abnormality in an MRI. Ab-

normal gyration was noticed in the 3D-surface rendering in two of nine patients without any abnormalities in the MRIs. Abnormal gyration was noticed in the frontal lobe in one and parietal lobe in one. PET and SPECT were done in 11 and 7 patients respectively. Three patients were given both a PET and SPECT and of them, only one patient showed the same abnormality in the PET and SPECT. Five patients revealed regional hypometabolism corresponding to lesions in the MRI and electrophysiological data.

EEG recordings (Table 1)

For precise localization of the epileptogenic zone prior to surgery, chronic video-EEG recordings with surface electrodes and intracranial electrodes (depth electrodes and subdural grids electrodes) were mandatory in all patients. EEGs with surface electrodes recorded ictal onset in the unilateral hemisphere in 10 patients (59%), which were not of value in localization of the epileptogenic zone. By means of invasive studies, localization or lateralization of the epileptogenic zone was possible in all patients. EEGs with subdural grid electrodes recorded focal ictal onset in six patients, regional onset in eight patients, and diffuse onset in three patients. Ictal EEG onsets were recorded from the SSMA in three patients, from the frontal lobe and the SSMA in three patients, from the frontal lobe in 10 patients, and from the parietal lobe in one patient.

Histopathologic findings

Histopathologic findings consisted of cortical dysplasia in eight patients, reactive gliosis in seven, and microdysgenesis in one.

Surgical outcome

At the average follow up period of 27.2 months (from 12 months to 54 months), overall seizure outcomes were class I in 11 (65%), class II in five (29%), class III in one (6%).

In review of the surgical procedures, all patients (2 patients) that underwent a resection of the SSMA and simultaneous ca-

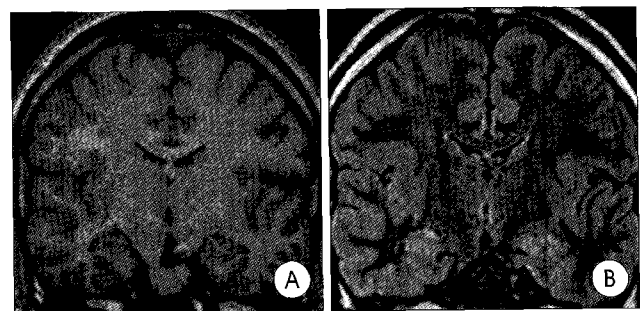


Fig. 1. T1-weighted coronal brain magnetic resonance image (A) demonstrates mild hippocampal atrophy in the right and fluid attenuated inversion recovery image (B) shows slight increase of signal intensity in the right hippocampus.

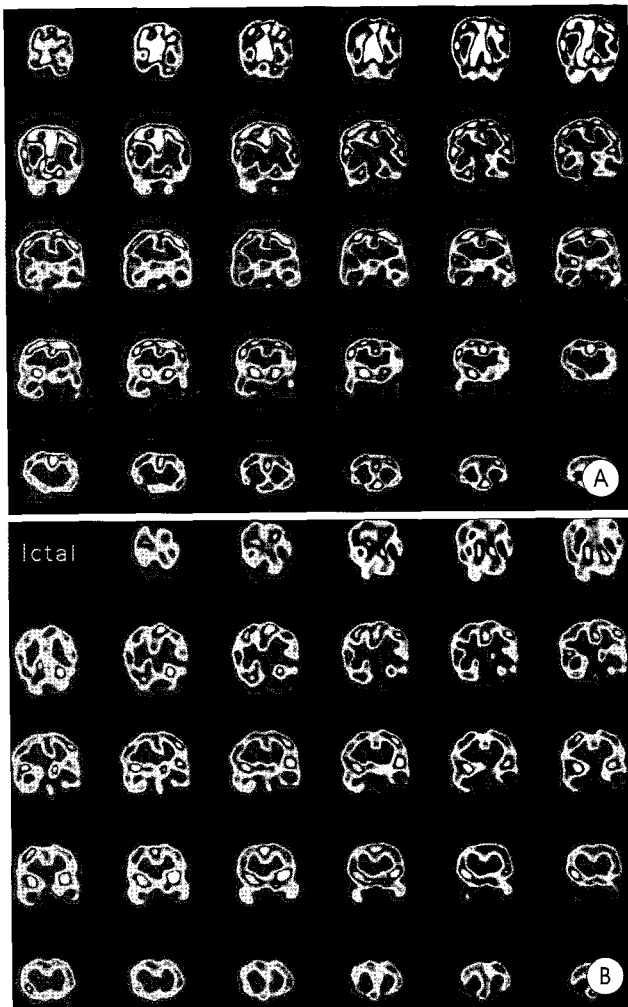


Fig. 2. Interictal single photon emission computerized tomography(SPECT) (A) shows hypoperfusion in left temporal lobe and ictal SPECT (B) does not show perfusion change.

llosotomy showed class I. Out of seven patients who underwent a resection of the SSMA extending to the adjacent area, five patients showed class I, one patient showed class II, and one patient showed class III. Out of seven patients who underwent a resection of a different area without a SSMA resection, three patients showed class I and four patients showed class II. The patient who underwent a callosotomy with previous surgery in another hospital showed class I.

In terms of the presence of abnormalities in the MRIs, out of 10 patients with structural abnormalities in the MRIs or 3D-surface rendering, five patients showed class I and another five patients showed class II. Out of seven patients without abnormal lesions in the MRIs or 3D-surface rendering, six patients showed class I and one patient showed class III. Visualization or nonvisualization of the abnormal lesions in the MRIs or 3D-surface rendering might not influence the surgical outcome in this study. In respect of the distribution of ictal EEG onset, out of 6 patients with focal EEG onset, three pa-

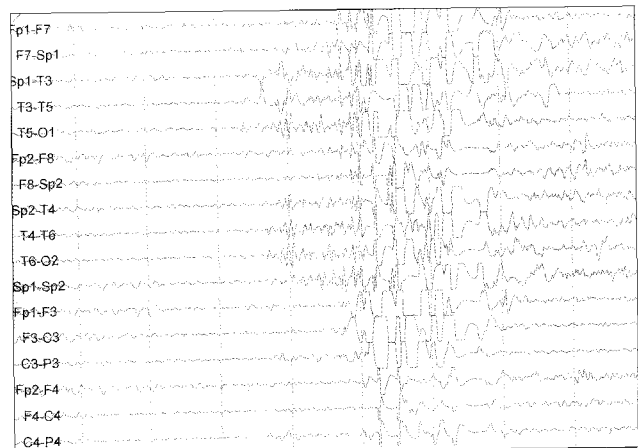


Fig. 3. Surface electro-encephalography recorded fast rhythmic activities in the bilateral hemisphere.

tients showed class I, two patients showed class II, and one patient showed class III. Out of eight patients with regional EEG onset, six patients showed class I and two patients showed class II. Out of three patients with diffuse EEG onset, two patients showed class I and one patient showed class II.

Complications

Mild hemiparesis developed in four patients and apraxia was noticed in one patient. However, these postoperative complications were improved and normalized within a few weeks (from 3 days to 3 weeks).

Illustrative case

A 28-year-old male patient was admitted with a chief complaint of complex partial seizures for 13 years. His typical seizures were a sudden onset of a versive seizure to the left side with rapid progression to the bilateral asymmetric tonic posture. Brain MR images showed mild hippocampal atrophy on the T1WI and increased signal intensity on FLAIR images in the right (Fig. 1). Interictal and ictal SPECT did not notice significant perfusion abnormality (Fig. 2). Chronic video-EEG monitoring with surface electrodes could not delineate an exact epileptogenic zone (Fig. 3). According to the clinical manifestation, subdural grid electrodes were implanted on the right frontal convexity and bilateral SSMA (Fig. 4A). An ictal EEG recorded seizure onset from the electrodes on the right middle frontal gyrus propagating to the right inferior frontal gyrus and right SSMA (Fig. 4B).

A resection was performed in the right SSMA and right middle frontal gyrus with the guidance of ECoG (Fig. 5). The histopathologic finding was neuronal migration disorder, Grade II (Fig. 6). Postoperatively, mild hemiparesis (Grade IV) and aphasia developed, which were relieved in 5 days. Seizures were controlled (class I) at the follow up period of 26 months.

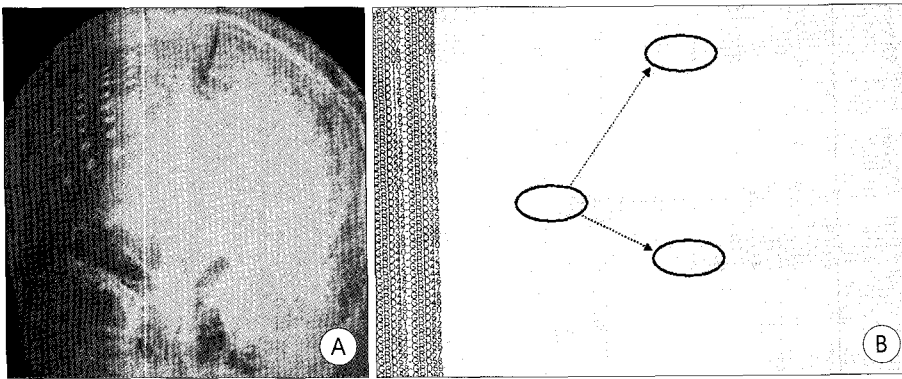


Fig. 4. Plain skull radiography after subdural grid implantation (A) shows subdural strip on right cerebral convexity and bilateral supplementary sensorimotor area (SSMA). Electroencephalography (EEG) with subdural grid electrodes (B) recorded ictal EEG onset from the right middle frontal gyrus, propagating to the right inferior frontal gyrus and right SSMA (Black circle).

Discussion

The supplementary sensorimotor area (SSMA) is a region of the cerebral cortex occupying the mesial portion of the superior frontal gyrus anterior to the primary motor cortex for the foot. It extends onto the dorsal surface of the superior frontal gyrus, and the caudal border extends inferior and posterior to the paracentral lobule¹². The SSMA has a role in planning, preparing and coordinating for the primary motor area. First described by Penfield and Welch¹⁷, SSMA seizures represent a unique constellation of very complex and stereotypic features. Classic signs of SSMA seizures include sudden abduction of the contralateral arm with flexion at the elbow and forced contralateral deviation of the head followed by forced vocalization, shrill, or speech arrest^{3,17}. Clonic activity in the contralateral face, arm, or secondary generalizations occur frequently. Versive head deviation is strongly associated with frontal seizure onset only when it occurs early in the seizure¹⁴. Speech arrest occurs only when the seizure focus involves the superior frontal gyrus on the dominant side. In spite of localizations of the epileptogenic zone distant to the SSMA, clinical manifestations are similar to each other due to rapid propagation of the ictal EEG to the SSMA. Several studies also showed that the overwhelming majority of patients with SSMA seizure have epileptogenic zones in the proximity of the

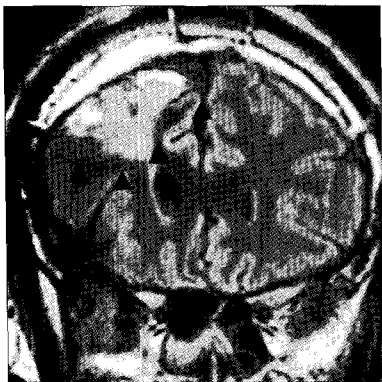


Fig. 5. Postoperative T2-weighted coronal image depicts resection area in the middle, superior frontal gyri, and right supplementary sensorimotor area (Black arrow head).

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SSMA (the mesial frontal region, the basal frontal region, the mesial parietal region, and, less frequently, the dorsal lateral convexity of the frontal lobe)^{3,7}. In our study, all patients showed ictal symptoms of sudden onset and the rapid progression of bilateral tonic posturing. Nine patients (53%) showed automatism. Versive seizures were observed in nine patients (53%). Five patients developed hypermotor seizures like thrashing. These motor symptoms seen in the present 17 patients were in good agreement with previous reports^{1,16}.

Duchowny et al.⁹ showed that in children undergoing epilepsy surgery, complete resection was the only significant prognostic factor in seizure control. Optimum results were achieved only if the lesion (if present) and the electrographically defined epileptogenic zone were excised. However, the completeness of

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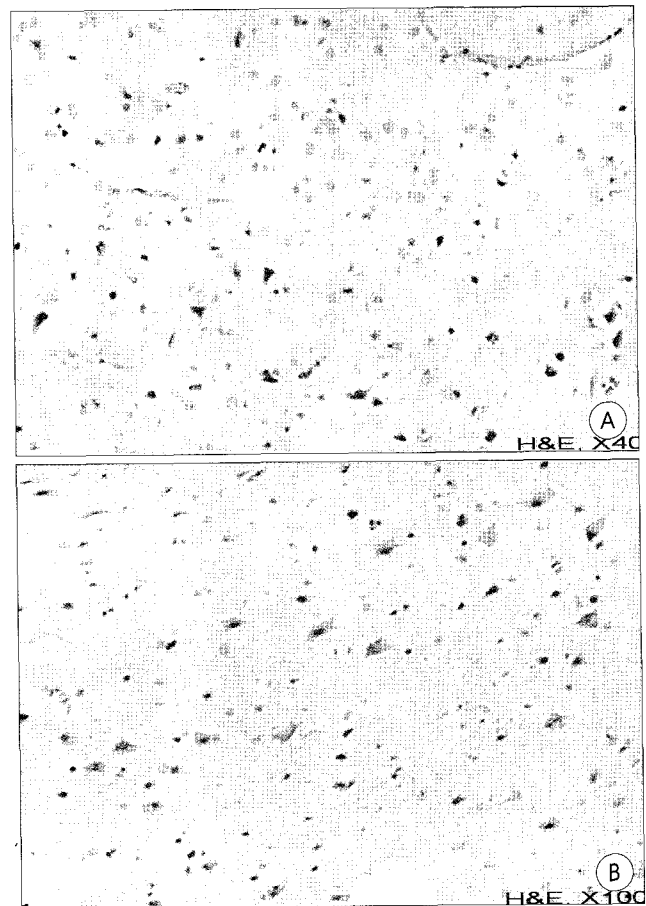


Fig. 6. Histopathologic findings revealing dyslamination of pyramidal neurons, focal collection of small dysplastic neurons, and mild neocortical gliosis (H&E, A : $\times 40$ and B : $\times 100$).

surgery is limited, hence unresectable, when the epileptogenic zone reaches a functionally important cortex, like the sensorimotor and speech area. Significant neurological deficits like hemiparesis and aphasia could appear immediately after resection of the SSMA, and those are soon resolved relatively well, but fine motor and speech function in complex tasks or/at high speed may be impaired¹⁹, and a deterioration in response maintenance and inhibition could remain. Therefore, the precise identification of the epileptogenic zone is essential in epilepsy surgery for patients with intractable clinical SSMA seizure. By means of scalp EEG, it is almost impossible to differentiate whether the seizures arise from the SSMA or spread to the SSMA from adjacent or other areas¹⁸. A subdural grid or strip electrodes, rather than depth electrodes, are useful to delineate ictal onset zone and the location of the SSMA¹⁵. A recent study by subdural EEG recording clearly documented that clinical SSMA seizures often arise from the cortical area close to the SSMA, and thus that the lesions aside from the SSMA potentially cause SSMA seizures^{3,7}. In order to avoid functional deficits with epilepsy surgery in patients with clinical SSMA seizure as much as possible, the present results provide us with clinically useful information when considering the surgical indication and planning presurgical evaluation. For precise localization of the epileptogenic zone, the authors used imaging study including MRI, SPECT, PET. In the MRIs, eight patients (47.0%) had structural abnormality. Except for visible lesions such as gyral disorganization in 3D-surface rendering in three patients, SPECT and PET could not give information on the epileptogenic zone. David studied 25 patients with SSMA seizures and showed that 12 patients (48%) of 25 patients had abnormal MRI findings⁶. For precise localization of the epileptogenic zone, invasive studies including depth electrode insertion and/or subdural grid implantation were performed. In spite of the development of post-operative complications including subdural hematoma or infection, these invasive studies must be done to locate the precise epileptogenic zone². We experienced post-operative CSF leakage only in one case. EEGs with subdural grid electrodes could record ictal EEG onset from the SSMA in three patients, from the frontal lobe and SSMA in three patients, from the frontal lobe in ten patients, and from the parietal area in one patient. Based on these data of multimodal diagnostic methods, the epileptogenic zone was resected.

It has been studied that the most important factor in predicting postoperative seizure outcome is the observation of visible lesions in neuroimaging studies, especially in MRI¹¹. In our study, rather than the observation of the visible lesions in neuroimaging studies, a recording of the ictal EEG onset for the localization of the epileptogenic zone and resection of the ictal EEG onset zone was the most important factor in the complete

control of the seizures. Postoperatively, mild hemiparesis developed in four patients and apraxia was noticed in one patient. These complications, however, were relieved back to normal in 3 days to 3 weeks. During the period of recovery, nearly normal motor power could be demonstrated despite a continuing lack of spontaneous movement and a hesitancy in performing motor commands. This has been referred to as an underutilization or motor neglect of the contralateral limbs^{9,10}.

Previous studies reported that extra-temporal resection, including lesional and non-lesional resection, showed seizure free results in 45.1% of patients, improvement in 35.2%, and unchanged results in 19.8%, which compared with seizure free results in 67.9%, and improvement in 24% of patients receiving temporal resection⁸. In our study, 65% were seizure free, 29% improved, and 6% unchanged.

Conclusion

Surgical strategies in patients with SSMA seizure should be directed toward the identification of the epileptogenic zone underlying the signs and symptoms reflected in the seizures propagated by SSMA circuitry. A successful outcome is most likely when the epileptogenic zone is completely resected, with the preservation of the eloquent area. In order to define the epileptogenic zone in patients with SSMA seizures, it is essential to use multimodal diagnostic methods, and variable meticulous surgery are necessary.

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Commentary

It is a very interesting and well-written paper. The authors reported their experience of 17 surgically treated patients. Their result looks very nice, obtaining nearly 94% good outcome. They suggest that MR visible lesion is not a good indicator of the better outcome, but that good delineation of the ictal onset zone would be.

I think this paper needs to be more clarified.

1. Their inclusion criteria should be more specified. They mentioned that they included patients having SSMA seizures who underwent surgical treatment. As we are very well aware of, it is very difficult to make a diagnosis of SSMA seizure. My concern is how they differentiate hypermotor seizure from SSMA seizures. It would be really good if they could describe the diagnostic difficulties in their paper, such as how many persons participated in decision of the seizure type, and how much their opinion is splitted.

2. I would be more happy to have exclusion criteria as well.

3. Their major subject is patients having SSMA seizures not SSMA epilepsy. The seizure type of the SSMA epilepsy is really different from that of distant area from SSMA, or isn't? This paper would be a better one if they could provide more detailed description of the intracranial ictal recording from the area far from the SSMA. I am very interested how a seizure starting far from SSMA area could generate SSMA seizure semiology. It has to involve SSMA. A controversial timing issue stands out at this point. If the seizure spread is late to SSMA, probably it could not generate SSMA seizure.

4. Histopathologic exam needs to be more clarified, because migration disorder is a very broad term.

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