See the corresponding editorial in this issue, pp 4–6.

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Risk factor analysis of the development of new neurological deficits following supplementary motor area resection

Clinical article

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Object. Supplementary motor area (SMA) resection often induces postoperative contralateral hemiparesis or speech disturbance. This study was performed to assess the neurological impairments that often follow SMA resection and to assess the risk factors associated with these postoperative deficits.

Methods. The records for patients who had undergone SMA resection for pharmacologically intractable epilepsy between 1994 and 2010 were gleaned from an epilepsy surgery database and retrospectively reviewed in this study.

Results. Forty-three patients with pharmacologically intractable epilepsy underwent SMA resection with intraoperative cortical stimulation and mapping while under awake anesthesia. The mean patient age was 31.7 years (range 15–63 years), and the mean duration and frequency of seizures were 10.4 years (range 0.1–30 years) and 14.6 per month (range 0.1–150 per month), respectively. Pathological examination of the brain revealed cortical dysplasia in 18 patients (41.9%), tumors in 16 patients (37.2%), and other lesions in 9 patients (20.9%). The mean duration of the follow-up period was 84.0 months (range 24–169 months). After SMA resection, 23 patients (53.5%) experienced neurological deficits. Three patients (7.0%) experienced permanent deficits, and 20 (46.5%) experienced symptoms that were transient. All permanent deficits involved contralateral weakness, whereas the transient symptoms patients experienced were varied, including contralateral weaknesses in 15, apraxia in 1, sensory disturbances in 1, and dysphasia in 6. Thirteen patients recovered completely within 1 month. Univariate analysis revealed that resection of the SMA proper, a shorter lifetime seizure history (< 10 years), and resection of the cingulate gyrus in addition to the SMA were associated with the development of neurological deficits ($p = 0.078$, 0.069, and 0.023, respectively). Cingulate gyrus resection was the only risk factor identified on multivariate analysis ($p = 0.027$, OR 6.530, 95% CI 1.234–34.562).

Conclusions. Resection of the cingulate gyrus in addition to the SMA was significantly associated with the development of postoperative neurological impairment.
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Key Words • **supplementary motor area** • **cingulate gyrus** • **resection** • **neurological deficit** • **epilepsy**

The supplementary motor area, which is situated on the medial aspect of the superior frontal gyrus, is mainly associated with motor function and has reciprocal connections with multiple motor areas, including the primary motor cortex, prefrontal cortex, cingulate gyri, basal ganglia, and contralateral SMA.[12] In 1951, Penfield and Welch[16] identified postoperative neurological impairments following unilateral SMA resection, including transient contralateral hemiparesis, hemiplegia, and speech deficits. Since that first report, several other studies have confirmed these deficits and revealed that they are features of SMA syndrome.[1,2,4–7,9,10,17–20,23,26]

These postoperative motor and speech disorders generally resolve within several weeks or months without severe neurological sequelae.[1,11,19] Several compensatory mechanisms have been suggested as being responsible for recovery from SMA syndrome.[9,18] Thus, previous studies have focused more on the characteristics of SMA syndrome, which are mild and transient, as well as the mechanism by which they resolve.[18] However, according

Abbreviations used in this paper: EEG = electroencephalography; MCC = midcingulate cortex; pACC = perigenual anterior cingulate cortex; PCC = posterior cingulate cortex; RSC = retrosplenial cortex; SMA = supplementary motor area; VCA = vertical plane through anterior commissure.

to recent reports, not all patients who undergo SMA resection experience postoperative neurological deficits.[18,20] Patients with motor weakness or speech impairments may face greater rehabilitative difficulties when compared with patients who do not experience these deficits. In addition, few studies have reported on factors associated with postoperative neurological deficits commonly experienced after SMA resection or why SMA syndrome occurs in only a subset of patients.

Therefore, this study was performed to describe postoperative neurological impairments that follow SMA resection and to assess the risk factors for these deficits.

Methods

Study Design

The institutional review board of the Seoul National University Hospital approved this study and exemption of informed consent because the study was retrospective. The records for patients who had undergone SMA resection for pharmacologically intractable epilepsy between 1994 and 2010 were gleaned from the epilepsy surgery database and retrospectively reviewed in this study. In all cases, the SMA was limited posteriorly by the precentral sulcus, inferiorly by the cingulate sulcus, anteriorly by the most rostral point of the genu of the corpus callosum, and laterally by the superior frontal sulcus according to previously described methods.[7,14,16] The rostral pre-SMA was defined as the anterior part of the SMA to the line vertical to the anterior commissure–posterior commissure plane and crossing the anterior commissure, or the VCA line. The caudal SMA proper was defined as the posterior part of the SMA to the VCA line, as per previous reports.[18,27]

Awake Surgery With Intraoperative Monitoring

Preoperative evaluation to localize an ictal onset zone included a routine history, neurological examination, interictal EEG, ictal video-EEG monitoring (for at least 3 typical seizures), brain MRI, PET, and interictal and ictal SPECT. While patients were under general anesthesia, subdural electrodes were implanted near the presumed ictal onset zone according to video-EEG monitoring results if no definite lesion was identified via preoperative MRI.

Resection was performed in patients under awake anesthesia by using intraoperative cortical mapping and monitoring. The detailed operative methods are described in our previous report.[8] Standard cortical mapping was performed using an Ojemann stimulator, which is a constant-current generator that produces a train of biphasic square wave pulses (at a rate of 60 Hz, 1 msec/phase) to minimize the possibility of inducing a seizure. To locate the primary motor cortex, stimuli were applied in 1-mA increments, starting at 1 mA up to a maximum of 10 mA. An epileptologist attended the entire process and monitored the patient during cortical stimulation and resection. Surgical removal continued until either the target epileptogenic zone was totally removed or the onset of unexpected neurological deficits occurred.

Postoperative Evaluation

Postoperative seizure outcome and the neurological status of each patient were assessed at regular clinical follow-ups, which occurred 1 month after surgery and subsequently every 2 or 3 months. Each assessment was completed by the same neurologist and neurosurgeon. Seizure outcome was assessed according to the Engel classification. Postoperative motor weakness was described according to a muscle strength grading score: Grade 0, no contraction; Grade 1, flicker or trace contraction; Grade 2, active movement with gravity eliminated; Grade 3, active movement through full range of motion against gravity; Grade 4, active movement against resistance; and Grade 5, normal strength against full resistance. Postoperative MRI was completed 3 months after surgery to measure the resected area. The resected area of the cingulate gyrus was defined as the area between the cingulate sulcus and the callosal sulcus of the corpus callosum, which was surgically removed and visualized on sagittal postoperative MR images.

Several demographic and clinical factors, including patient sex, age, seizure duration, seizure frequency, seizure type, presence of predisposing factors, side of surgery, surgery type, resected area, whether cingulate gyrus resection was performed, amount of time required for resection, histological type, and seizure outcome, were evaluated via risk factor analysis for the development of postoperative neurological deficits following SMA resection.

Statistical Analysis

All data are presented as the means ± standard deviation in addition to the range. Risk factors for postoperative neurological deficits were analyzed using logistic regression analysis. To reduce the risk of Type II errors due to modest sample size, variables were considered for multivariate analysis only if they were associated with a dependent variable in each analysis that was significant at a level of $p < 0.150$. Values of $p < 0.050$ were considered statistically significant. All statistical analyses were performed using SPSS (version 17.0.1, SPSS, Inc.).

Results

Forty-three patients with pharmacologically intractable epilepsy underwent SMA resection with intraoperative cortical stimulation and mapping while under awake anesthesia. Twenty-five patients were male and 18 were female. The mean patient age was 31.7 ± 11.2 years (range 15–63 years). The mean duration of the seizure history and seizure frequency were 10.4 ± 8.1 years (range 0.1–30.0 years) and 14.6 ± 29.0/month (range 0.1–150.0/month), respectively. Tumor-like lesions were found in 18 patients (41.9%) and cortical lesions were found in 9 (20.9%) via preoperative MRI. Sixteen patients (37.2%) showed no definite lesions on MRI. The radiological characteristics of all patients are summarized in Table 1.

Surgical Outcome

Twenty-four patients (55.8%) underwent subdural grid implantation to localize the ictal onset zone before resection. The resective surgeries included 18 lesionectomies (41.9%), 13 corticectomies (30.2%), and 12 frontal lobectomies (27.9%). Resection of the SMA proper was performed

TABLE 1: Clinicoradiological characteristics of 43 patients who underwent SMA resection*

Parameter	Value
patient sex (%)	
M	25 (58.1)
F	18 (41.9)
mean age in yrs (range)	31.7 ± 11.2 (15–63)
type of Sz (%)	
simple partial Sz	7 (16.3)
CPSs	6 (14.0)
CPSs w/ secondary generalization	15 (34.9)
generalized tonic-clonic Sz	14 (32.6)
other	1 (2.3)
mean duration of Szs in yrs (range)	10.4 ± 8.1 (0.1–30.0)
mean frequency of Szs/mo (range)	14.6 ± 29.0 (0.1–150.0)
predisposing factors for Sz (%)	
trauma	9 (20.9)
CNS infection	2 (4.7)
febrile convulsion	4 (9.3)
developmental anomaly	2 (4.7)
no	26 (60.5)
no. of preop AEDs (%)	
1	11 (25.6)
2	18 (41.9)
≥3	14 (32.6)
preop MRI findings (%)	
tumor-like lesion	18 (41.9)
cortical lesion	9 (20.9)
no lesion	16 (37.2)
pre-resective subdural grid implantation (%)	24 (55.8)

* Values indicate the number of patients, unless indicated otherwise. Mean values are presented ± SD. Abbreviations: AED = antiepileptic drug; CPS = complex partial seizure; Sz = seizure.

TABLE 2: Diagnostic and surgical results of 43 patients who underwent SMA resection*

Parameter	Value
type of surgery (%)	
lesionectomy	18 (41.9)
corticectomy	13 (30.2)
frontal lobectomy	12 (27.9)
side of surgery (%)	
rt	19 (44.2)
lt	24 (55.8)
resected area (%)	
SMA proper	17 (39.5)
pre-SMA	11 (25.6)
SMA proper & pre-SMA	15 (34.9)
cingulate gyrus resection (%)	15 (34.9)
mean op time in mins (range)	300.0 ± 70.3 (130–425)
mean hospital stay in days (range)	7.2 ± 3.8 (3–24)
pathological exam (%)	
astrocytic tumor	4 (9.3)
oligodendrocytic tumor	10 (23.3)
other tumor	2 (4.7)
cortical dysplasia	18 (41.9)
other	9 (20.9)
postop neurological deficit (%)	
permanent	3 (7.0)
transient	20 (46.5)
no	20 (46.5)
Engel class at last FU (%)	
I	21 (48.8)
II	9 (20.9)
III	6 (14.0)
IV	7 (16.3)
mean clinical FU duration in mos (range)	84.0 ± 49.0 (24–169)

* Values indicate the number of patients, unless indicated otherwise. Mean values are presented ± SD. Abbreviation: FU = follow-up.

in 17 patients (39.5%), pre-SMA resection was performed in 11 patients (25.6%), and resection of both areas was performed in 15 patients (34.9%). Additional cingulate gyrus resection was performed in 15 patients (34.9%). Histopathological examinations revealed 4 astrocytic tumors, 10 oligodendrocytic tumors, 2 other tumors, 18 cortical dysplasias, and 9 other lesions.

At the last follow-up evaluation for seizure outcome, 21 patients (48.8%) had Engel Class I (seizure free), 9 patients (20.9%) had Class II (rare seizures), and 6 (14.0%) had Class III (worthwhile improvement). The seizure control rate was 83.7% (Engel Classes I–III). The mean time elapsed from surgery to the final clinical follow-up was 84.0 ± 49.0 months (range 24–169 months). The surgical results are summarized in Table 2.

Postoperative Neurological Deficits

Of the 43 patients with SMA resection, 23 patients (53.5%) experienced new postoperative neurological deficits (Table 3). Three of these patients (7.0%) experienced permanent deficits, and 20 had transient deficits (46.5%). Permanent deficits corresponded to contralateral Grade 4 motor weakness. One patient (Case 5) underwent resection of the most posterior part of the superior, middle, and inferior frontal gyri and experienced permanent Grade 4 hand weakness. A second patient (Case 12) demonstrated Grade 4 leg weakness after resection of the SMA proper. Sudden hand weakness occurred during resection of the most posterior area of the SMA in the third patient (Case 25); therefore, the procedure was stopped immediately. The patient in this case experienced permanent Grade 4 upper- and lower-extremity weakness. Transient deficits included contralateral motor weakness in 15 patients, sensory disturbances in 1 patient, dyspraxia in 1 patient, and motor dysphasia or dysarthria in 6 patients. Three patients experienced both motor weakness and dysphasia, and 1 patient experienced both weakness and dyspraxia. Dyspraxia was characterized by impairment of initiation

TABLE 3: Clinical findings in 43 patients who underwent SMA resection*

Case No.	Age (yrs), Sex	Neurological Deficit	Recovery (days)	Resected Area	Cingulate Gyrus Resection	Dx	Engel Class
1	19, F	—	—	SMA proper	no	CH	II
2	57, F	motor dysphasia	30	SMA proper	yes	CA	I
3	39, M	hand weakness (Gr 4)	270	SMA proper	yes	AO	I
4	31, M	—	—	SMA proper	no	CD	II
5	36, M	hand weakness (Gr 4)	permanent	whole SMA†	yes	CD	I
6	34, M	motor dysphasia, UE & LE weakness (Gr 4)	150	whole SMA	yes	ODG	I
7	15, F	UE & LE weakness (Gr 4)	42	whole SMA	yes	CD	I
8	20, M	—	—	pre-SMA	yes	CS	II
9	25, M	—	—	SMA proper	no	CD	IV
10	27, F	UE weakness (Gr 3)	3	SMA proper	yes	CC	III
11	31, M	—	—	pre-SMA	no	CS	I
12	43, F	LE weakness (Gr 4)	permanent	SMA proper	no	Ast	IV
13	43, F	UE & LE weakness (Gr 4)	60	whole SMA	yes	ODG	I
14	24, M	—	—	whole SMA	yes	CD	II
15	24, M	UE & LE weakness (Gr 4)	14	SMA proper	no	GGO	I
16	26, M	UE & LE weakness (Gr 3)	3	whole SMA	no	CD	I
17	28, F	motor dysphasia	30	Pre-SMA	no	CD	III
18	23, M	—	—	whole SMA	no	CD	IV
19	22, F	—	—	pre-SMA	no	AVM	I
20	23, F	UE & LE weakness (Gr 4)	5	SMA proper	yes	CI	I
21	28, M	motor dysphasia	30	whole SMA	no	CD	III
22	17, F	UE weakness (Gr 3)	30	pre-SMA	no	CD	II
23	23, M	—	—	whole SMA	yes	CD	II
24	45, M	UE & LE weakness (Gr 2)	30	whole SMA	no	ODG	IV
25	33, F	UE & LE weakness (Gr 4)	permanent	SMA proper‡	no	GBL	III
26	28, F	—	—	pre-SMA	no	CM	IV
27	19, M	—	—	pre-SMA	no	CD	I
28	37, F	—	—	pre-SMA	no	CD	I
29	24, M	—	—	SMA proper	no	ODG	I
30	40, M	motor dysphasia, UE weakness (Gr 4)	6	SMA proper	yes	ODG	II
31	32, F	—	—	SMA proper	no	ODG	I
32	51, F	—	—	pre-SMA	no	CD	I
33	30, M	UE weakness (Gr 3)	60	whole SMA	no	CD	III
34	40, M	hand sensory disturbance	1500	whole SMA	yes	ODG	I
35	30, F	UE & LE weakness (Gr 4), motor dyspraxia	8	whole SMA	yes	CD	I
36	19, M	—	—	SMA proper	no	RG	IV
37	16, M	—	—	whole SMA	no	CD	II
38	38, M	LE weakness (Gr 4)	42	SMA proper	no	AA	III
39	63, M	motor dysphasia, ankle weakness (Gr 4)	7	SMA proper	no	AO	IV
40	30, M	LE weakness (Gr 3)	1	pre-SMA	yes	CD	I
41	52, M	—	—	whole SMA	no	ODG	II
42	37, F	—	—	SMA proper	no	EVN	I
43	41, F	—	—	pre-SMA	no	AA	I

* AA = anaplastic astrocytoma; AO = anaplastic oligodendroglioma; Ast = astrocytoma; AVM = arteriovenous malformation; CA = cavernous angioma; CC = cortical contusion; CD = cortical dysplasia; CH = cortical heterotopia; CI = cortical ischemia; CM = cerebromalacia; CS = cortical scar; Dx = histopathological diagnosis; EVN = extraventricular neurocytoma; GBL = glioblastoma; GGO = ganglioglioma; Gr = grade; LE = lower extremity; ODG = oligodendroglioma; RG = reactive gliosis; UE = upper extremity; whole SMA = whole SMA, including SMA proper and pre-SMA.

† The patient underwent resection of the most posterior part of the superior, middle, and inferior frontal gyri.

‡ The patient experienced development of hand weakness during surgery, and the resection stopped.

and slowing of skilled and complex movements such as putting on and removing clothing. Of the 20 patients with transient deficits, 13 patients (65%) fully recovered within 1 month of surgery. The mean recovery time was 116 ± 332 days (range 1–1500 days).

All 3 patients with permanent hemiparesis underwent resection of the most posterior part of the SMA proper, which is just adjacent to the precentral sulcus. Resection of the most posterior area of the SMA just anterior to the precentral sulcus was performed in 9 patients. Of these 9 patients, 7 (77.8%) experienced postoperative motor weakness. Of the remaining 34 patients, 16 (47%) experienced postoperative neurological deficits ($p = 0.142$). Moreover, the permanent neurological risk in the patients with the most posterior SMA resection was significantly higher compared with that in the remaining patients ($p = 0.007$). However, cingulate gyrus resection and pathological diagnosis were not associated with permanent neurological deficits ($p = 1.000$ and 0.545, respectively).

The risk of postoperative neurological deficits was higher in the patients with SMA proper resection (20 [62.5%] of 32) than in those with only pre-SMA resection (3 [27.3%] of 11; $p = 0.078$). We analyzed the association between postoperative neurological deficits and resected area, which was stratified by concurrent cingulate gyrus resection (Fig. 1). Overall, cingulate gyrus resection was significantly related to postoperative neurological deficits (12 [80.0%] of 15 vs 11 [39.3%] of 28; $p = 0.023$). Resection of the SMA proper along with additional removal of the cingulate gyrus was most strongly related to postoperative neurological deficits as compared with any other type of resection (11 [84.6%] of 13; $p = 0.009$). Resection of the pre-SMA without removal of the cingulate gyrus was the safest; however, this finding did not reach statistical significance (2 [22.2%] of 9; $p = 0.059$). Stratified analysis revealed that resection of the SMA proper and the cingulate gyrus was related to postoperative neurological impairments.

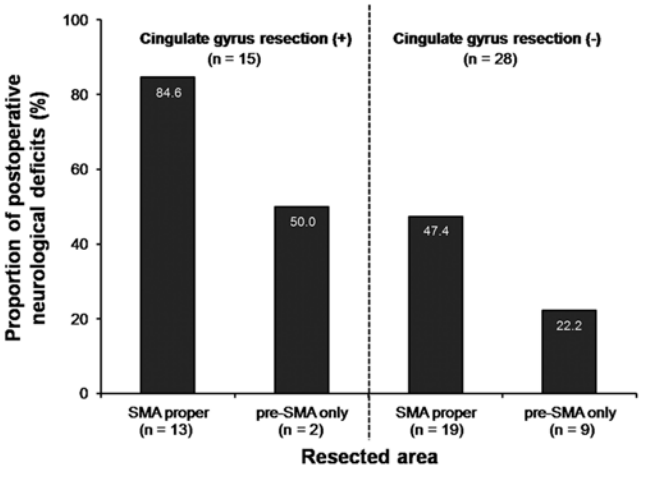

Fig. 1. Analysis of the association between the development of new postoperative neurological deficits and the resected area stratified by additional cingulate gyrus resection in patients who underwent SMA resection. Stratified analysis revealed that cingulate gyrus resection together with resection of the SMA proper was associated with postoperative neurological deficits.

Risk Factors for Postoperative Neurological Deficits

Associations between postoperative neurological deficits and several clinicoanatomical factors were evaluated (Table 4). Univariate analysis revealed that cingulate gyrus resection, shorter lifetime history of seizure (< 10 years), and SMA proper resection ($p = 0.023$, 0.069, 0.078, respectively) were associated with postoperative neurological impairments after SMA resection. However, cingulate gyrus resection ($p = 0.027$, OR 6.530, 95% CI 1.234–34.562) was the only significant risk factor for postoperative neurological deficits identified via multivariate analysis.

Illustrative Case

A 30-year-old woman (Case 35) with pharmacologically intractable seizures was seen at our hospital. Although she was treated with valproate, carbamazepine, and topiramate, both separately and in combination, she experienced several seizure attacks per day for 23 years. Her seizures initially involved extension of her left arm and evolution into generalized tonic-clonic seizures within a few minutes. Ictal video-EEG findings suggested a frontotemporal seizure focus; however, no specific lesion was found on 3.0-T MRI, and no metabolic abnormality was detected on PET.

Subdural grids were implanted for localization of an ictal onset zone (Fig. 2A). Invasive monitoring revealed that the seizure activity initiated at both the right SMA and the cingulate gyrus. Seven days after the first craniotomy, she underwent corticectomy of the right SMA and cingulate gyrus while under local anesthesia (Fig. 2B). She experienced Grade 4 weakness of her left side and mild motor dyspraxia; however, she was fully recovered 8 days after the second surgery. Postoperative MRI visualized the resected area (Fig. 2C–E). Postoperatively, she remained seizure free without using any antiepileptic drugs, and she had no neurological symptoms for 4 years.

Discussion

Not all patients who undergo SMA resection experience postoperative neurological impairments such as SMA syndrome. Authors of initial studies reported that the SMA syndrome occurred in every patient who underwent SMA resection.[11,19] However, the overall incidence rate of SMA syndrome is variable, ranging from 23% to 100% according to recent series.[5,7,18,20] In the present study, only 53.5% of patients experienced postoperative neurological deficits. In other words, while some patients who undergo SMA resection recover well without any neurological complications, others experience motor weakness or language problems lasting as long as several months. Moreover, these neurological conditions greatly impact a patient's quality of life. Therefore, it is essential to explore the risk factors associated with neurological deficits that follow SMA resection. Controlling these risk factors before and during resection may lead to improved clinical care.

Resection of the cingulate gyrus was the most notable risk factor for postoperative neurological impairments

TABLE 4: Risk factors for postoperative neurological deficits following SMA resection*

Factor	Univariate Analysis, p Value	Multivariate Analysis	
		p Value	OR (95% CI)
patient sex	1.000	NI	
patient age (>30 yrs)	**0.131**	**0.264**	**2.278 (0.537–9.666)**
Szs duration (<10 yrs)	**0.069**	**0.108**	**3.391 (0.765–15.041)**
Sz frequency (<1/wk)	0.223	NI	
Sz type	0.758	NI	
predisposing factor	1.000	NI	
side of surgery	1.000	NI	
type of surgery	0.213	NI	
SMA proper resection	**0.078**	**0.204**	**2.893 (0.561–14.922)**
pre-SMA resection	0.756	NI	
cingulate gyrus resection	**0.023**	**0.027**	**6.530 (1.234–34.562)**
op time (>300 mins)	0.354	NI	
histological diagnosis (tumors)	0.206	NI	
unfavorable Sz outcome (Engel Classes III & IV)	0.203	NI	

* Boldface indicates statistical significance. Abbreviation: NI = not included.

following SMA resection. The cingulate gyrus is a major medial cortical structure that overlies the corpus callosum from the lamina terminalis rostrally to the splenium at its caudal extent.[15,21] Traditionally, the cingulate gyrus has been associated with limbic and emotion pathways.[24] However, recent neurophysiological and neuroanatomical studies of humans and animals have dramatically changed our understanding of cingulate function.[15] Specifically, the cingulate cortex has been recognized to consist of 4 divisions, each with a unique function: the perigenual anterior cingulate cortex (pACC), the midcingulate cortex (MCC), the posterior cingulate cortex (PCC), and the retrosplenial cortex (RSC).[24] Among these cortices, the MCC constitutes approximately one-third of the total cingulate cortex beneath the SMA, and it functions to 1) select an appropriate motor response based on its motivational significance, and 2) carry out that response via direct connections to the spinal cord, limbic cortices, and primary or supplementary motor areas.[13,21] The other cortices—the pACC, PCC, and RSC—are associated with emotional, visuospatial, or memory functions rather than motor functions.[3,21,22,24] Resected cingulate cortical areas corresponded to the MCC beneath the SMA in this study. Moreover, the 12 patients who underwent resection of both the SMA and the cingulate gyrus experienced postoperative motor weakness or apraxia. The resected area of the cingulate gyrus may thus be responsible for motor functions, because resecting this area produces motor impairments.

In our opinion, cingulate gyrus resection has not been studied adequately. Tate et al.[21] reported on 90 resections of cingulate gyrus gliomas, the largest surgical series to date. In their study, a high rate of postoperative morbidity, such as SMA syndrome, was induced by resection of the MCC or PCC when compared with resection of the pACC. The authors demonstrated that these morbidities occurred only in cases with SMA resection as a surgical trajectory. Therefore, postoperative neurological impairments associated with cingulate cortical resection may be induced by SMA damage. This phenomenon was also found in a recent series described by von Lehe et al.[25] In their 22 resective surgeries for cingulate gyrus epilepsy, extracingulate resection, including the SMA, induced SMA syndrome. The aforementioned studies[21,25] and the present study demonstrate that resection of both the SMA and the cingulate gyrus induces postoperative neurological deficits. Consequently, SMA resection, when accompanied by resection of the cingulate cortex below it, is likely to cause postoperative neurological deficits, although the exact mechanism of this and the connections between these 2 areas require further study.

In the present study, 3 patients (7.0%) experienced permanent Grade 4 motor weakness. All of these patients underwent resection of the most posterior part of the SMA without definite cortical damage or resection of the precentral gyrus. Moreover, these patients were prone to postoperative motor impairments. Presumably, any unnoticed white matter injury of the primary motor fibers, such as the corticospinal tract, could cause permanent motor weakness. Peraud et al.[17] and Kasasbeh et al.[7] showed that the distance between the resected area and the precentral gyrus was significantly related to both transient and permanent neurological impairments. Moreover, the anterior-posterior extent of the resected SMA was the most frequently reported risk factor of SMA syndrome in previous reports.[5,23,26] Resection of a longer anterior-posterior extent of the SMA may have caused white matter injuries that went unnoticed, while sparing the primary motor area of any damage. However, cingulate gyrus resection was not associated with permanent neurological risk after SMA resection. Consequently, cingulate gyrus resection with SMA resection was the significant risk factor for SMA syndrome, which induces mostly transient motor weakness or language problems.

In this study we evaluated the risk factors for postoper-

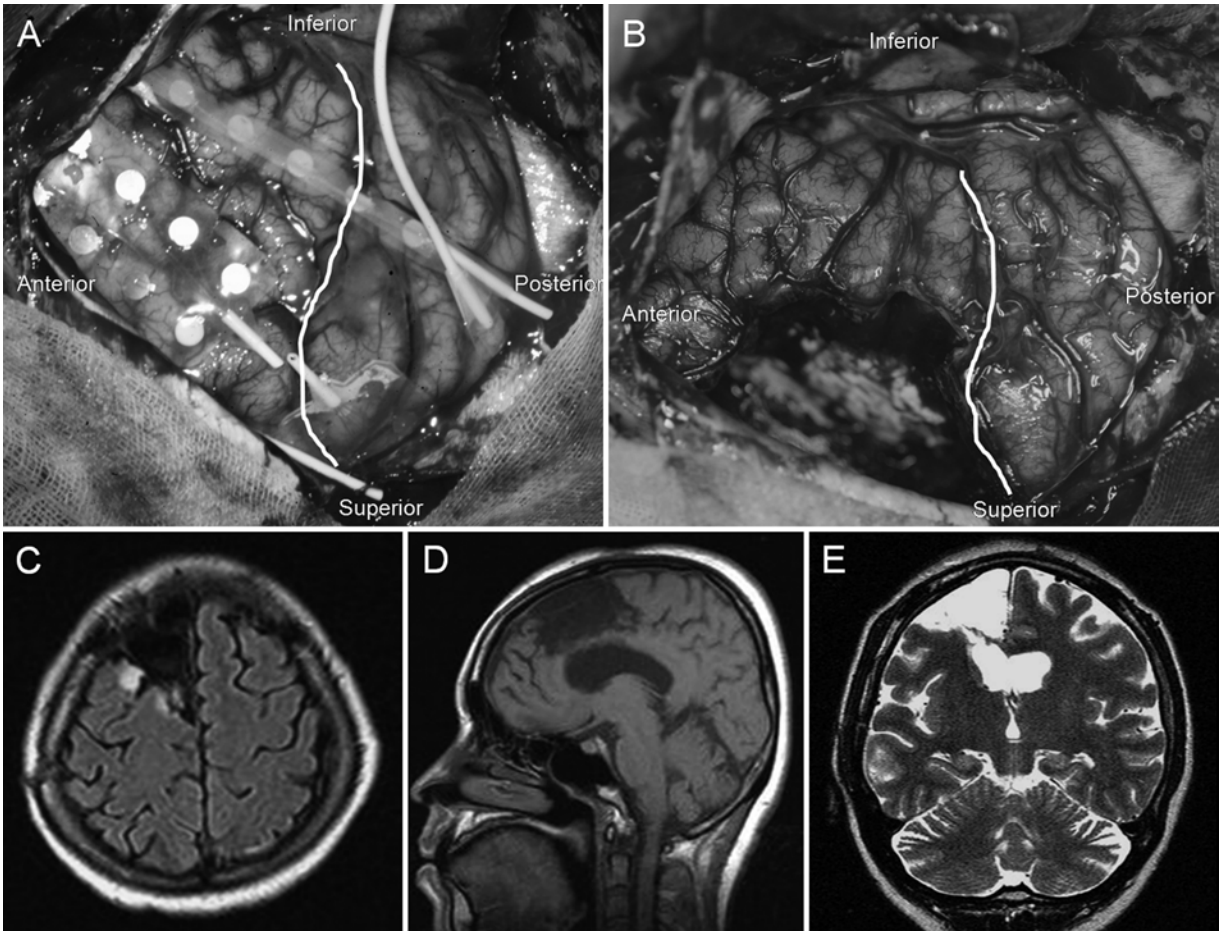

Fig. 2. Case 35. Intraoperative photographs and postoperative brain MR images. The patient underwent implantation of a subdural grid to localize the ictal onset zone **(A)** as well as resection of both the right SMA and right cingulate gyrus **(B)**. *White lines* indicate the right precentral sulcus. Postoperative axial **(C)**, sagittal **(D)**, and coronal **(E)** MR images depicting the resected area. The patient experienced mild left-sided weakness and motor apraxia for 8 days following the surgery.

ative neurological deficits after SMA resection. Moreover, the significance of SMA resection accompanied by cingulate gyrus resection was discussed for the first time in the literature. However, even this large analysis is not without limitations. The inclusion of heterogeneous disease groups and the retrospective nature of the study may have affected the results directly or indirectly. In particular, the heterogeneous pathology of this study was a significant limitation. Further analyses including more patients are necessary to determine the relationship between resection of the most posterior aspect of the SMA and permanent neurological deficits. Potentially unnoticed white matter injuries should be assessed using diffusion tensor imaging. In addition, topographical analysis of the cingulate gyrus is necessary to define the differences between the motor functions of the ventral and dorsal cingulate.

Conclusions

After SMA resection, 47% of patients experienced transient neurological symptoms, and 7% experienced permanent deficits. Cingulate gyrus resection that was accompanied by SMA resection was significantly associated with postoperative transient neurological impairments.

Disclosure

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Author contributions to the study and manuscript preparation include the following. Conception and design: Chung, YH Kim, Lee, Han, CY Kim. Acquisition of data: YH Kim. Analysis and interpretation of data: Chung, YH Kim, CH Kim, JS Kim. Drafting the article: YH Kim. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Chung. Statistical analysis: YH Kim, CH Kim, JS Kim. Administrative/technical/material support: Chung, CH Kim, Lee, Han, CY Kim. Study supervision: Chung, YH Kim, Han, CY Kim.

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