Topographical Risk Factor Analysis of New Neurological Deficits Following Precentral Gyrus Resection

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BACKGROUND: Precentral gyrus resections (PGRs) have been regarded as excessively hazardous interventions because of the risk of postoperative major neurological complications.

OBJECTIVE: To evaluate the neurological deterioration that follows PGRs and to assess the topographical risk factors associated with these morbidities.

METHODS: We reviewed 33 consecutive patients who experienced pharmacologically intractable epilepsy and underwent PGR with intraoperative cortical stimulation and mapping while under awake anesthesia. The etiological diagnoses were brain neoplasm in 26 patients (78.8%), cortical lesion in 4 (12.1%), and no lesion in 3 (9.1%). The mean follow-up period was 62.6 months (range, 12-146 months). All topographical analyses of the resected quadrant area were performed based on postoperative magnetic resonance images.

RESULTS: After PGR, 22 patients (66.7%) experienced neurological worsening, including 5 permanent deficits (15.2%) and 17 transient deficits (51.5%). Permanent deficits included 2 instances of weakness, 1 dysarthria, 1 dysesthesia, and 1 fine-movement disturbance of the hand. While the neurological risk for anterior lower quadrant PGR was 20.0% (1/5), the risk for posterior upper quadrant PGR was 100.0% (10/10). The anterior upper and posterior lower quadrant PGR caused neurological deteriorations in 60.0% (6/10) and 62.5% (5/8) of the patients, respectively. In a multivariate analysis, PGR of the posterior and upper quadrant sections were significant risk factors for post-PGR neurological deteriorations (P = .022 and 0.030, respectively).

CONCLUSION: The posterior upper quadrant of the precentral gyrus was vulnerable to post-resective neurological impairment.

KEY WORDS: Neurological deterioration, Precentral gyrus, Resection, Topographical

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The precentral gyrus (PG), which is traditionally considered to be the same area as the primary motor cortex and Brodmann area 4, had been considered to be an unresectable region because of the potential for neurological morbidity.[1,2] In 1947, Pilcher et al[1] reported that the rate of permanent neurological deficits was 63% after even the partial sensorimotor cortical resections in 41 patients with jacksonian convulsions.

ABBREVIATIONS: ALQ, anterior lower quadrant; **AUQ,** anterior upper quadrant; **EEG,** electroencephalography; **PG,** precentral gyrus; **PGR,** precentral gyrus resection; **PLQ,** posterior lower quadrant; **PUQ,** posterior upper quadrant

Since then, recent advancements in magnetic resonance imaging (MRI), awake anesthesia, and intraoperative mapping and monitoring technologies have gradually enabled surgeons to remove the precentral gyrus with acceptable neurological morbidities.[3] In actuality, many authors have reported satisfactory results after the resection of the Rolandic area, including the precentral gyrus, for controlling medically intractable epilepsy or for removing tumorous lesions.[3-9] Based on previous reports, postoperative new neurological deficits were mostly transient or mild, and the rate of permanent neurological morbidities was acceptable, ranging from 0% to 50%.[3-9]

In many previous studies, however, there is uncertainty concerning the precise boundaries for

TABLE 1. Demographic Data and Surgical Results of the 33 Patients Who Underwent PGR[a]

Parameters	Values[b]
Sex, n (%)	
Male	14 (42.4)
Female	19 (57.6)
Age	40.0 ± 14.5 (16-63)
Type of seizures, n (%)	
Simple partial seizures	10 (30.3)
Complex partial seizures (CPS)	7 (21.2)
CPS with secondary generalization	10 (30.3)
Generalized tonic-clonic seizures	3 (9.1)
Others	3 (9.1)
Duration of seizures, y	4.9 ± 6.3 (0.1-20.0)
Frequency of seizures, per mo	19.1 ± 58.4 (0.1-300.0)
No. of preoperative AEDs (%)	
1	11 (33.3)
2	11 (33.3)
≥3	11 (33.3)
Preoperative neurological deficits, n (%)	15 (45.5)
Preoperative MRI findings, n (%)	
Tumorous lesion	26 (78.8)
Cortical lesion	4 (12.1)
No lesion	3 (9.1)
Preresective subdural grid implantation, n (%)	6 (18.2)
Type of surgery, n (%)	
Lesionectomy	28 (84.8)
Corticectomy	5 (15.2)
Side of surgery, n (%)	
Right	10 (30.3)
Left	23 (69.7)
Resected area, n (%)	
Anterior upper quadrant	10 (30.3)
Posterior upper quadrant	10 (30.3)
Anterior lower quadrant	5 (15.2)
Posterior lower quadrant	8 (24.2)
Volume of resected area, cm^3	4.5 ± 3.3 (0.5-13.7)
Resection of postcentral gyrus, n (%)	9 (27.3)
Operation time, min	278.7 ± 64.2 (110-425)
Hospital stay, d	8.6 ± 5.5 (4-25)
Pathological examinations, n (%)	
Astrocytic tumors	12 (36.4)
Oligodendrocytic tumors	8 (24.2)
Other tumors	6 (18.2)
Cortical dysplasia	4 (12.1)
Others	3 (9.1)
Postoperative neurological deficits, n (%)	
Permanent	5 (15.2)
Transient	17 (51.5)
No	11 (33.3)
Seizure outcome at last f/u (Engel classification), n (%)	
I	18 (54.5)
II	3 (9.1)
III	7 (21.2)
IV	5 (15.2)
Clinical f/u duration, mo	62.6 ± 41.1 (12-146)

(Continues)

TABLE 1. *Continued*

[a]AED, antiepileptic drug; f/u, follow-up; MRI, magnetic resonance imaging; PGR, precentral gyrus resection.
[b]Values are expressed as number (%) or mean values ± standard deviation (range).

safe precentral gyrus resections (PGRs) and the concordant risk factors associated with post-PGR neurological worsening. Moreover, the postoperative neurological status of the patients who underwent a PGR was too variable, from no neurological symptoms to permanent hemiparesis. Therefore, this study was designed to describe postoperative new neurological impairments that follow PGR and to assess the risk factors for these deficits, focusing on the topographical resected area of the PG.

METHODS

Demographics

A total of 33 consecutive patients who underwent PGR for pharmacologically intractable epilepsy between 1995 and 2012 were retrospectively reviewed in this study. In all cases, the PG was limited anteriorly by the precentral sulcus, posteriorly by the central sulcus, medially by the cingulate sulcus, and laterally by the sylvian fissure.

Fourteen patients (42.4%) were male, and 19 patients (57.6%) were female. The mean patient age was 40.0 ± 14.5 years (range, 16-63 years). The mean length of seizure history and frequency were 4.9 ± 6.3 years (range, 0.1-20.0 years) and 19.1 ± 58.4/month (range, 0.1-300.0/month), respectively. Fifteen patients (45.5%) had neurological deficits preoperatively. Tumorous lesions were found in 26 patients (78.8%), and cortical lesions were found in 4 patients (12.1%) via preoperative MRI. Three patients (9.1%) showed no definite lesions on MRI. The radiological and clinical characteristics of all patients are summarized in Table 1. This study was approved by the institutional review board of the authors' institutions.

Surgical Procedures

Preoperative evaluation to localize an ictal onset zone included a routine history, neurological examination, interictal electroencephalography (EEG), ictal video-EEG monitoring (for at least 3 typical seizures), brain MRI, positron emission tomography, and interictal and ictal single-photon emission computed tomography. Subdural electrodes were implanted near the presumed ictal onset zone according to video-EEG monitoring results while under general anesthesia if no definite lesion was identified via preoperative MRI.

Surgical resection was performed on patients under awake anesthesia with intraoperative cortical mapping and monitoring. Detailed operating methods are described in a previous report.[3,10] Standard cortical mapping was performed by 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 ms/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.

TABLE 2. Clinical Findings of the 33 Patients Following PGR[a]

No.	Sex/Age	Neurological Deficits	Recovery, d	Resected Area (AP)	Resected Area (UL)	Dx	Sz
1	F/19	Wrist and hand weakness (Gr 3)	21	P	U	AA	I
2	M/32	UE weakness (Gr 3)	12	P	U	CD	IV
3	M/34	UE and LE weakness (Gr 3), motor dysphasia	150	P	U	ODG	I
4	F/43	UE and LE weakness (Gr 4)	Permanent	A	U	Ast	IV
5	M/17	—	—	P	L	CD	I
6	M/24	Mild dysarthria, facial palsy	Permanent	P	L	CD	I
7	M/45	UE and LE weakness (Gr 2)	30	A	U	ODG	IV
8	M/34	—	—	P	L	No Bx	III
9	F/34	Motor dysphasia	180	A	U	AA	III
10	M/44	Motor dysphasia	3	A	L	AO	I
11	F/60	—	—	A	L	CA	I
12	M/16	—	—	A	L	Ast	I
13	F/31	Facial palsy	60	A	U	ODG	I
14	M/54	Motor dysphasia	300	P	L	AA	II
15	M/40	UE weakness (Gr 4), motor dysphasia	6	A	U	ODG	II
16	M/35	Motor dysphasia, facial palsy	180	P	U	AO	I
17	F/30	—	—	P	L	CD	I
18	F/51	Fine-movement disturbance of hand	Permanent	P	U	Ast	III
19	M/35	Hand hypesthesia and dysesthesia	Permanent	P	U	CA	I
20	M/58	—	—	A	L	GBM	I
21	M/48	UE weakness (Gr 4)	30	P	U	AO	IV
22	M/60	UE weakness (Gr 4)	90	A	U	GBM	III
23	F/41	Facial palsy	90	P	L	RG	I
24	F/19	Ankle weakness (Gr 3)	120	P	L	GG	III
25	M/48	—	—	A	U	AA	III
26	F/42	—	—	A	U	AA	I
27	F/36	—	—	A	L	AO	I
28	F/61	Dysarthria	7	P	L	GBM	III
29	M/25	Hand weakness (Gr 3)	90	P	U	Sch	I
30	F/20	—	—	A	U	Inf	II
31	M/63	UE weakness (Gr 3)	Permanent	P	U	Met	IV
32	F/59	Hand weakness (Gr 3)	60	P	U	GBM	I
33	M/61	—	—	A	U	Met	I

[a]AP, anteroposterior location of the resected area; UL, upper-lower location of the resected area; Dx, histopathological diagnosis; Sz, seizure outcome according to Engel classification; F, female; M, male; Gr, grade; UE, upper extremity; LE, lower extremity; PGR, precentral gyrus resection; A, anterior PGR; P, posterior PGR; U, upper PGR; L, lower PGR; AA, anaplastic astrocytoma; CD, cortical dysplasia; ODG, oligodendroglioma; Ast, astrocytoma; No Bx, no biopsy was performed; AO, anaplastic oligodendroglioma; CA, cavernous angioma; GBM, glioblastoma; RG, reactive gliosis; GG, ganglioglioma; Sch, schwannoma; Inf, CNS inflammation; Met, metastatic tumor.

An epileptologist attended the entire process and monitored the patient during cortical stimulation and resection. Resection continued until either the target epileptogenic zone was totally removed or the onset of unexpected neurological worsening occurred.

TABLE 3. Postoperative Neurological Risk Analysis According to the Topographical Resected Quadrant Area Based on the Postoperative Magnetic Resonance Image in the 33 Patients With Precentral Gyrus Resection

	Anterior, %	Posterior, %	Subtotal, %
Upper	60.0 (6/10)	100.0 (10/10)	80.0 (16/20)
Lower	20.0 (1/5)	62.5 (5/8)	46.2 (6/13)
Subtotal	46.7 (7/15)	83.3 (15/18)	66.7 (22/33)

Postoperative Evaluation and Topographical Analysis

Postoperative seizure outcomes and the neurological status of each patient were assessed at regular clinical follow-ups, which were scheduled at 1 month after the surgery and subsequently every 2 or 3 months. Each assessment was completed by the same neurologist and neurosurgeon. Seizure outcomes were assessed according to the Engel classification.

A postoperative MRI was completed 3 months after surgery to measure the resected area. In terms of the anterior-posterior location of the resected area, we defined anterior PGR as when the resected area did not reach the central sulcus (but only the precentral sulcus), and we defined posterior PGR as when the resected area reached the central sulcus. We used the extending line from the central sulcus at the most inferior part of the PG because the central sulcus by definition does not reach the sylvian fissure. In terms of the upper-lower location, the borders between upper and lower PGR was the extending line from the inferior frontal sulcus. Finally, we categorized the PGR into 4 quadrant resections: the anterior upper

TABLE 4. Risk Factors for Postoperative Neurological Deficits Following Precentral Gyrus Resection[a]

	Univariate	Multivariate	
Factors	*P* Value	*P* Value	OR (95% CI)
Patient sex (male)	1.000	NI	
Patient age (≥40 y)	.721	NI	
Sz duration (≥5 y)	.703	NI	
Sz frequency (≥2/wk)	.721	NI	
Sz type	.864	NI	
Preoperative ND	.246	.715	
Side of surgery (left side)	.696	NI	
Type of surgery	.304	NI	
Intraoperative Sz	1.000	NI	
Posterior quadrant PGRs	.061	**.022**	**14.445 (1.458-143.130)**
Upper quadrant PGRs	.065	**.030**	**12.401 (1.275-120.629)**
Resected volume (>3 cm^3)	.712	NI	
Postcentral gyrus resection	.438	NI	
Operation time (≥270 min)	.721	NI	
Extent of resection	1.000	NI	
Intraoperative neurological worsening	.643	NI	
Histological diagnosis (tumors)	.147	.344	
Unfavorable seizure outcome (Engel classification III and IV)	.249	.378	

[a]OR, odds ratio; CI, confidence interval; Sz, seizure; NI, not included; ND, neurological deficits; PGR, precentral gyrus resection.

quadrant (AUQ) PGR, the posterior upper quadrant (PUQ) PGR, the anterior lower quadrant (ALQ) PGR, and the posterior lower quadrant (PLQ) PGR.

Several demographic and clinical factors including patient sex, age, seizure duration, seizure frequency, seizure type, presence of preoperative neurological deficits, side of surgery, surgery type, topographical resected area, resected volume, amount of time required for surgical resection, extent of resection, intraoperative neurological worsening, histological type, and seizure outcomes were evaluated via risk factor analysis for the development of postoperative neurological deficits following PGR.

Statistical Analysis

All data are presented as the mean value ± standard deviation in addition to the range. The risk factors for developing postoperative neurological deficits were analyzed by 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 the $P < .25$ level. P values of $<.05$ were considered statistically significant. All statistical analyses were performed using SPSS (version 20.0., 2011; SPSS, Chicago, Illinois).

RESULTS

Surgical Outcome

Resective surgeries included 28 lesionectomies (84.8%) and 5 corticectomies (15.2%). Anterior PGR was performed in 15 patients (45.5%) and posterior PGR in 18 (54.5%). In terms of the upper and lower orientation, 20 patients (60.6%) underwent upper quadrant PGRs and 13 patients (30.4%) lower quadrant PGRs. Finally, AUQ, PUQ, ALQ, and PLQ PGR was performed in 10 (30.3%), 10 (30.3%), 5 (15.2%), and 8 patients (24.2%), respectively. Additional postcentral gyrus resection was performed in 9 patients (27.3%). Histopathological examinations revealed 12 astrocytic tumors, 8 oligodendrocytic tumors, 6 other tumors, 4 cortical dysplasias, and 3 other lesions.

At the last follow-up evaluation for seizure outcome, 18 patients (54.5%) were found to have achieved Engel class I (seizure-free), 3 patients (9.1%) achieved class II (rare seizures), and 7 patients (21.2%) achieved class III (worthwhile improvement). The seizure control rate was 84.8% (Engel class I, II, and III). The mean time elapsed from surgery to final clinical follow-up was 62.6 ± 41.1 months (range, 12-146 months). The surgical results are also summarized in Table 1.

Postoperative Neurological Deficits

Of the 33 patients with PGR, 22 patients (66.7%) experienced new and postoperative neurological deficits (Table 2). Five of these patients experienced permanent deficits (15.2%), and 17 experienced transient deficits (51.5%). The permanent deficits were composed of 2 contralateral instances of weakness (patients 4 and 31 in Table 2), 1 fine-movement disturbance of the hand (patient 18 in Table 2), 1 sensory disturbance including hypesthesia and dysesthesia (patient 19 in Table 2), and 1 mild dysarthria (patient 6 in Table 2). Of the 5 patients, 3 patients (patients 18, 19, and 31 in Table 2) underwent resection of the PUQ, especially posterior and middle third part, of the PG, 1 patient (patient 4 in Table 2) underwent resection of the AUQ of the PG, and 1 patient (patient 6 in Table 2) had resection of the PLQ of the PG. At the last follow-up, 4 patients (patients 4, 6, 18, and 19 in Table 2) were living a normal social life.

Transient deficits included contralateral motor weakness in 10 patients, facial palsy in 3 patients, motor dysphasia in 6 patients, and dysarthria in 1 patient. Of the 17 patients with transient deficits, 12 patients (70.6%) fully recovered within 3 months. The mean recovery time was 84.1 ± 81.2 days (range, 3-300 days).

Topographical Analyses

All topographical risk factor analyses were performed according to the location of the resected quadrant area in the

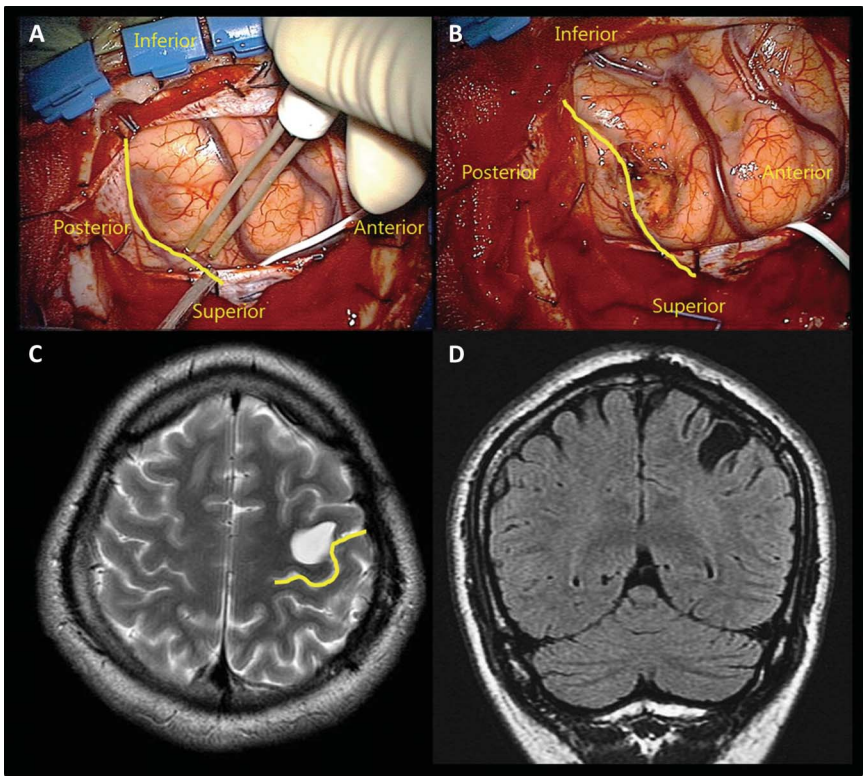

FIGURE 1. *Intraoperative photographs and postoperative brain magnetic resonance images (MRI). The patient (patient 29 in Table 2) showed the tumorous lesion in the left precentral gyrus (A) and underwent the lesionectomy and resection of the posterior and middle third precentral gyrus (B). The yellow lines indicate the left central sulcus. Postoperative axial (C) and coronal (D) MRIs depict the resected area. The patient experienced grade 3 right hand weakness for 3 months following surgery.*

PG (Table 3). Of the 15 patients with the anterior quadrant PGRs, 7 patients (46.7%) experienced neurological impairments and 1 patient (6.7%) experienced permanent ones. Of the 18 patients with posterior quadrant PGRs, 15 patients (83.3%) experienced neurological impairments and 4 patients (22.2%) experienced permanent ones. In terms of the upper-lower location of the resected area, of the 20 patients with upper quadrant PGRs, 16 patients (80.0%) experienced neurological deficits and 4 patients (20.0%) permanent ones, and of the 13 patients with the lower quadrant PGRs, 6 patients (46.2%) experienced neurological deficits and 1 patient (7.7%) experienced permanent ones.

All 10 patients who underwent resection of the PUQ PG experienced postresective neurological deficits (100.0%); of the 10 patients, 3 patients (30.0%) had permanent deficits. On the contrary, of the 5 patients with the ALQ PGR, only 1 patient (20.0%) experienced transient motor dysphasia for 3 days after surgery. The neurological risks for the AUQ PGR group (n = 10) and the PLQ PGR group (n = 8) were 60.0% (6/10) and 62.5% (5/8), respectively. Concerning only permanent neurological impairments, the risk for the PUQ PGR was 30.0% (3/10), while that of the other quadrant PGRs was 8.7% (2/23).

Risk Factor Analyses

The associations between postoperative neurological deficits and several clinicoanatomic factors were evaluated (Table 4). Univariate analysis found that the posterior quadrant PGRs and upper quadrant PGRs were related to postoperative neurological deficits; this result was not significant. However, in the multivariate analysis, the posterior quadrant PGRs (P = .022; odds ratio = 14.445; 95% confidence interval, 1.458-143.130) and the upper quadrant PGRs (P = .030; odds ratio = 12.401; 95% confidence interval, 1.275-120.629) were the only 2 significant risk factors for postoperative neurological deficits following PGR.

In addition, the risk factors for permanent post-PGR neurological deficits were assessed. The PUQ PGR (P = .149), left side resection (P = .291), and additional postcentral gyrus resection (P = .111) were associated with permanent deficits; however, this was not significant.

Illustrative Case

A 25-year-old man (patient 29 in Table 2) with pharmacologically intractable seizures was observed at our hospital. Although he was treated with valproate, levetiracetam, and lamotrigine separately and in combination, he experienced several seizure attacks a day for 9 years. His seizures initially involved eyeball deviation and clonic movements of the right arm and evolution into generalized tonic-clonic seizures within a few minutes. He showed a right upper extremity weakness (grade 4) before surgery. Preoperative MRI scan found a tumorous lesion located in the left PG.

Intraoperative findings found a corresponding mass occupying the posterior and middle third part, that is the PUQ of the PG (Figure 1A), and the gross total removal of the mass was performed under awake anesthesia (Figure 1B). After surgery, he experienced aggravation of the right arm weakness (grade 3), and he fully recovered 3 months after the surgery. Postoperative MRI visualized the resected area in the PUQ PG (Figures 1C and 1D). Postoperatively, he remained seizure-free without using any antiepileptic drugs, and he had no neurological symptoms for 26 months.

DISCUSSION

Since resective surgery with intraoperative mapping and neurological monitoring under awake anesthesia has been developed, eloquent cortical resections, including PGR, are no longer impossible procedures.[3,10] Many previous reports, however, could not demonstrate why some patients experienced postoperative neurological sequelae, whereas others did not following PGR, and did not propose how neurosurgeons could avoid the postoperative neurological complications of PGR. In this study, the authors tried to evaluate the topographical risk factors for post-PGR neurological sequelae and to delineate more vulnerable or resistant areas in the PG and their relationships to postresective neurological worsening. Taking these risk factors into account before and during surgical resection may lead to improved clinical care.

Resection of the posterior part of the PG was the most notable risk factor for developing postoperative neurological impairments following PGR. To our knowledge, no study has focused on the relationship between the anterior-posterior location of the PGR and the neurological outcome. The present study provided limited but significant evidence that the posterior portion of the PG is more vulnerable to postresective neurological morbidities than the anterior part. The evidence of this phenomenon can be found in any traditional guide to cortical anatomy, including a Brodmann map.[2] Of the Brodmann areas, the primary motor cortex corresponds to Brodmann area 4, which is located in the posterior part of the PG (Figure 2). In particular, the width of Brodmann area 4 is wider at the upper part of the PG and narrower at the lower part, and the shape of area 4 is an inverted triangle.[2] In other words, the core of primary motor function may be located in the posterior part of the PG, whereas the anterior part of the PG, especially, the ALQ of the gyrus, may be a relatively less functional area, and, thus, may be more resistant to possible postresective neurological deficits. The present data demonstrate clinical evidence for the topographical validity of Brodmann area 4.

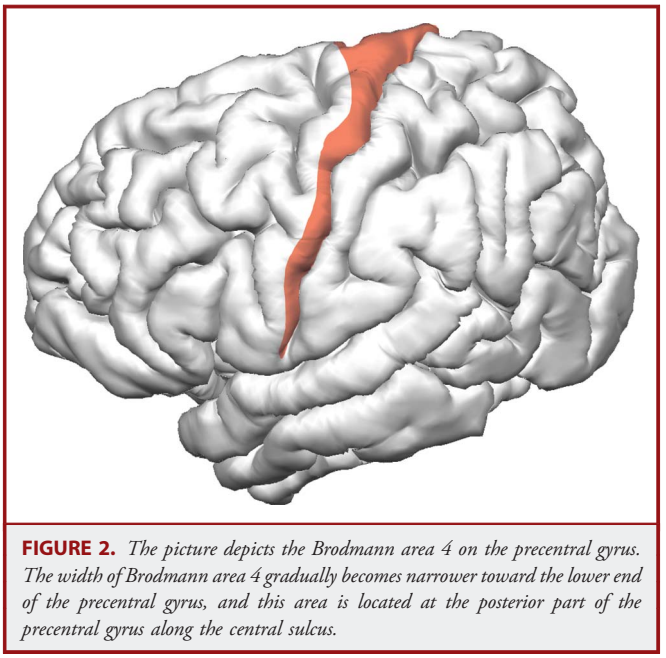

FIGURE 2. *The picture depicts the Brodmann area 4 on the precentral gyrus. The width of Brodmann area 4 gradually becomes narrower toward the lower end of the precentral gyrus, and this area is located at the posterior part of the precentral gyrus along the central sulcus.*

In addition, the lower part of the PG was the safest area in terms of postresective neurological morbidities (compared with the upper parts) in this study. As mentioned above, the width of Brodmann area 4 gradually becomes narrower toward the lower end of the PG, and this area is located at the posterior part of the PG along the central sulcus (Figure 2). This inverted triangle shape of area 4 coincides with the results of this study. Of the 4 quadrants of the PG, the ALQ was the safest for resection and the PUQ was the most vulnerable area, which can be explained by the shape and location of Brodmann area 4. The fact that unilateral excision of the lower part of the PG, which corresponds to the face and tongue motor area, was not associated with significant long-term neurological sequelae has previously been well known.[3,5] This may be due to the bilateral cortical representation of the face and tongue area.[3,5] This bilateral cortical projection may cause the lower postresective neurological risk of the lower part of the PG.

Another notable finding of the present study was the delineation of the most vulnerable area for postresective permanent neurological deficit, which is the posterior and middle-third area of the PG. This area corresponds to the hand motor area, according to common homunculi representations of the motor cortex. Previous studies have shown that a resection of the proximal limb region was better tolerated than the distal limb, including the hand and fingers.[4,6,11] The present results are compatible with those of previous reports.

Limitations

This study evaluated the topographical risk factors for developing postoperative neurological deficits after PGR, and it

demonstrated for the first time in the literature that the PUQ of the PG are vulnerable to postresective neurological deficits. However, even this relatively 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 addition, the present study considered only the cortical resected area of the PG but did not take into account the depth of the resection reaching the underlying white matter or the medial area of the PG controlling the leg motor. Further analyses including more cases are necessary to determine the association between the depth of the resection and neurological outcome.

CONCLUSION

After PGR, 52% of patients experienced transient neurological impairments, and 15% experienced permanent deficits. The PUQ of the PG was more vulnerable to postresective neurological deficits than the anterior and lower parts.

Disclosure

The authors have no personal, financial, or institutional interest in any of the drugs, materials, or devices described in this article.

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COMMENT

The authors present their results on surgery of lesions in the vicinity of the precentral gyrus (PCG). They assess whether different portions of the PCG exhibit varying sensitivity to surgical resection. Although the data set they use is necessarily small, their analysis is careful and comprehensive, allowing them to derive a "sensitivity map" of the PCG. They divide the PCG into 4 quadrants—anterior/posterior and superior/inferior. They conclude, based on the patient cohort studied, that operating on the anterior-inferior quadrant is least likely to result in permanent neurological morbidity, whereas the posterior-superior quadrant is associated with the highest likelihood of permanent deficits.

The results of this analysis stand to reason. The inferior portion of the PCG, devoted to motor function of the face, has long been known to be associated with a motor deficit that is mild and usually recovers in time, whereas the motor representation of the hand or foot, represented in the more superior aspects of the PCG, is a much more unforgiving region. The current study extends these findings, and provides surgeons with a more granular sensitivity map to be used in surgical planning. There are several caveats, however. One should keep in mind that the observations made in this study were based on a small group of patients, whose lesions may have exhibited various degrees of infiltration, and may have had variable effects on the risks of resection. The risk of resection of a cavernous angioma, for example, may be entirely different from the risk of resecting an astrocytoma. In addition, the lesions were of differing depths and likely involved differential risks based upon these, and other nonregional factors. A retrospective analysis like this one should always be examined with such caveats in mind.

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