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Role of the supplementary motor area in motor deficit following medial frontal lobe surgery

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Article abstract—*Objective:* Patients undergoing surgical resection of medial frontal lesions may present a transient postoperative deficit that remains largely unpredictable. The authors studied the role of the supplementary motor area (SMA) in the occurrence of this deficit using fMRI. *Methods:* Twenty-three patients underwent a preoperative fMRI before resection of medial frontal lesions. Tasks included self-paced flexion/extension of the left and right hand, successively. Preoperative fMRI data were compared with postoperative MRI data and with neurologic outcome. *Results:* Following surgery, 11 patients had a motor deficit from which all patients recovered within a few weeks or months. The deficit was similar across patients, consisting of a global reduction in spontaneous movements contralateral to the operated side with variable severity. SMA activation was observed in all patients. The deficit was observed when the area activated in the posterior part of the SMA (SMA proper) was resected. *Conclusions:* fMRI is able to identify the area at risk in the SMA proper whose resection is highly related to the occurrence of the motor deficit. The clinical characteristics of this deficit support the role of the SMA proper in the initiation and execution of the movement.

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Surgical ablation of tumors of the medial frontal lobe may result in immediate postoperative motor and speech deficits.[1-5] Different clinical presentations of these deficits have been reported previously but one of their main characteristics is a complete or almost complete recovery within several weeks or months.[1-5] Following surgery, the motor deficit remains unpredictable, however, although it is more frequent when

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the resection extends in caudal parts of the supplementary motor area (SMA).[2] Moreover, its pathophysiologic basis remains incompletely understood and several mechanisms may be advocated including edema, associated vascular lesions, surgical trauma, or true surgical removal of the SMA itself.

Comprehension of the structure and function of the motor areas of the medial frontal cortex has undergone important improvement in recent years.[6,7] In addition to the classic SMA, placed in area 6 of the medial frontal wall, several other motor areas have been described.[6,7] Several hypotheses have been proposed as to the functions of these areas, rostral regions (pre-SMA) being involved in higher order motor control, such as preparation or sequencing of movements, whereas caudal regions (SMA proper) are more closely related to the execution of movement.[8-17]

In the present study, 23 patients presenting with surgical lesions of the medial frontal lobe were studied using fMRI in order to determine whether the occurrence of the motor deficit was related to surgical lesion of the SMA. We hypothesized that the deficit would be related to the removal of the SMA proper, which is more closely linked to movement execution and initiation.[18] Anatomoclinical correlation would also allow making further inferences about the function of the SMA.

Patients and methods. *Patients.* Twenty-three patients referred for surgical treatment of lesions of the medial frontal lobe were retrospectively studied (11 women and 12 men; age range, 22 to 70 years, mean 43 years) (tables 1 and 2). All patients had a seizure history of 1 month to 36 years. Lesions were low-grade glioma (17 patients), anaplastic astrocytoma (5 patients), and cortical dysplasia (1 patient). Lesions were located in the frontal lobe (left hemisphere, 14 patients; right hemisphere, eight patients) or in both frontal and parietal lobes (right hemisphere, 1 patient). The pre- and postoperative motor deficit was assessed by a neurologist (M.V.) and neurosurgeons (H.D., P.C., L.C., S.C., C.A.V., A.L.B.) using a standardized motor scale.[19] The intensity of the motor deficit was rated as follows: 0 = no deficit; 1 = mild deficit (patient can use his or her limb almost normally; walking possible, impairment of fine movements of the upper limb); 2 = moderate deficit (movement possible with help of examiner); 3 = severe deficit (no spontaneous movement against gravity). Clinical examinations were performed the day of the fMRI, the day before surgery, immediately after surgery, several times a day during the following week, every month the first 3 months, every 3 months the first year, and every 6 to 12 months thereafter. Preoperative motor deficit was either mild (3 patients) or absent (20 patients) (see tables 1 and 2). No change in the clinical presentation was observed between the fMRI session and surgery in any patients. Speech functions were evaluated clinically with verbal comprehension, spontaneous speech, narrative tasks, verbal fluency, and repetition. All patients had steroid treatment during the 3 days immediately following surgery.

Preoperative fMRI examination. Imaging. The MR protocol was performed with a 1.5-T MR unit using blood oxygen level–dependent fMRI. The preoperative fMRI was performed between 3 and 150 days before surgery (mean, 45 days). The protocol included 1) 12 axial gradient echo–echo planar images covering the whole frontal lobes (repetition time/echo time/flip angle: 5,000/60 ms/90°, 5-mm slice thickness, no gap, in plane resolution: 3.75 mm × 3.75 mm); and 2) axial inversion recovery three-dimensional T1-weighted images for anatomic localization.

Tasks. Two different tasks were performed by the patients. The tasks consisted of self-paced flexion/extension of the fingers of the right and left side successively. The paradigm consisted of seven epochs of 30 seconds, alternating rest and activation (duration of each run, 3 minutes and 30 seconds).

fMRI analysis. Data were motion corrected, temporally filtered, and analyzed with a dedicated software (ACTIV, Service Hospitalier Frédéric Joliot, Orsay, France) written in Interactive Data Language (Research Systems International, Paris, France), using pixel-by-pixel autocorrelation and cross-correlation with a reference waveform of the MRI signal time course.[20,21] Activated clusters were defined as follows: minimum of three contiguous pixels, correlation coefficient >0.40, autocorrelation coefficient >0.10 ($p < 0.001$). Activated pixels were overlaid on axial anatomic images with a color scale representing the correlation coefficient.

Anatomic localization of the supplementary motor area. The anatomic localization of the SMA was performed on reformatted three-dimensional T1-weighted sections (Voxtool, General Electric Medical Systems, Milwaukee, WI) using the vertical lines passing at the level of the anterior (VCA line) and posterior commissures (VCP line) as landmarks.[22] The SMA (SMA proper) was bounded by the VCP caudally and VCA rostrally. The anterior part of the SMA (pre-SMA) was located rostral to the VCA line.

Postoperative MR examination. The postoperative MRI was performed between zero to 510 days (mean, 134 days) after surgery. The protocol included axial inversion recovery three-dimensional T1-weighted images and axial T2-weighted fast spin echo images.

Pre- and postoperative three-dimensional T1-weighted images were anatomically coregistered using automatic dedicated software.[23] The volume of tumor resection was determined using a semiautomatic segmentation software based on region growing. Analysis consisted in determining the overlap between the volume of tumor resection and the volume of activation in the SMA. This overlap corresponded to the proportion of SMA activation removed during the surgical procedure. The percentage of SMA activation removed during the surgical procedure was calculated as the ratio between the volumes of overlap and SMA activation. Pearson correlation factors (r) were calculated between the characteristics of the postoperative deficit and the volumetric measures.

Results. *Localization of fMRI activation.* Activation was observed in the contralateral primary sensorimotor cortex, the SMA, the posterior part of the premotor cortex, and the secondary parietal area. Significant activation was detected in the SMA in all 23 patients during simple fingers movement. In all patients, foci of signal intensity changes in the SMA were located caudally to the VCA line,

Table 1 *Clinical and surgical characteristics of patients with a motor deficit related to the resection of the supplementary motor area (SMA)*

Patient no. /age, y*/sex	Histology of the lesion	Location of the lesion	Preoperative motor deficit*	Volume of surgical resection, cm^3	Percentage of resection of the area activated in the SMA ipsilateral to the tumor	Postoperative motor deficit	Motor recovery	Long-term follow-up	Speech disorders
1/33/M	Low-grade astrocytoma	L frontal	No	10	41.7	Immediate moderate right hemiparesia with RU predominance	Begin after 4 d, complete at 3 mo	Normal at 1 y	—
2/55/M	Cortical dysplasia	R frontal	No	7.2	42.2	Immediate mild left hemiparesia with LU predominance	Begin after 2 d, complete at 2 mo	Normal at 1 y	—
3/31/M	Low-grade oligo-dendroglioma	L frontal	No	20.2	44.5	Immediate mild right hemiparesia with RU predominance	Begin after 3 d, complete at 8 d	Normal at 1 y	Mutism 3 d
4/25/F	Low-grade astrocytoma	L frontal	No	15.0	53.9	Immediate right hemiplegia	Begin after 3 d, complete at 3 mo	Normal at 1 y	Mutism 4 d, reduced spontaneous speech 3 mo
5/37/F	Low-grade oligo-dendroglioma	L frontal	No	14.0	100	Immediate moderate right hemiparesia with RU predominance	Begin after 5 d, complete at 2 mo	Normal at 1 y	Reduced spontaneous speech 4 d
6/57/M	Low-grade oligo-dendroglioma	R frontal	Mild left hemiparesia with LL predominance	11.5	97.7	Immediate left hemiplegia	Begin after 7 d, complete at 3 wk	Normal at 1 y	—
7/68/F	Anaplastic astrocytoma	L frontal	No	13.3	53.2	Immediate moderate right hemiparesia with RU predominance	Begin after 3 wk, complete at 3 mo	Underutilization of the RU at 1 y	Yes–no answers 8 d, reduced spontaneous speech persistent after 1 y
8/41/F	Low-grade astrocytoma	R frontal	Mild spastic monoparesia of distal LL	54.2	88.9	Immediate left hemiplegia	Begin after 1 mo, partial at 3 mo	Mild spastic monoparesia of distal LL at 1 y	—
9/66/F	Anaplastic astrocytoma	L frontal	No	7.1	50.1	Immediate right hemiplegia, right grasping	Begin after 6 wk, partial at 3 mo with RL spasticity	Underutilization of the RU and spasticity of the RL, grasping of the RU at 6 mo	Aphasia 3 mo, progressive recovery until death at 6 mo
10/28/M	Low-grade astrocytoma	R frontal	No	62.6	33.3	Immediate left hemiplegia, left grasping	Begin after 7 d, complete at 3 mo	Grasping, bimanual coordination impairment at 1 y	—
11/34/M	Low-grade oligo-dendroglioma	R frontal	No	17.3	9.1 Secondary extradural hematoma	Delayed left hemiplegia after 8 h, left grasping	Begin after 10 d, complete at 2 mo	Grasping, bimanual coordination impairment at 1 y	—

All patients were right-handed.

* The day before surgery.

RU = right upper limb; LU = left upper limb; RL = right lower limb; LL = left lower limb.

Table 2 *Clinical and surgical characteristics of patients without motor deficit related to the resection of the supplementary motor area*

Patient no./ age, y/sex	Handedness	Histology of the lesion	Location of the lesion	Volume of surgical resection, cm^3	Speech disorders
12/55/M	L	Anaplastic astrocytoma	R frontoparietal	53	—
13/38/M	R	Low-grade oligodendroglioma	L frontal	17.1	Reduced spontaneous speech 3 mo
14/40/F	R	Low-grade astrocytoma	L frontal	8.7	Mutism 2 d, reduced spontaneous speech 6 mo
15/32/F	R	Low-grade oligodendroglioma	L frontal	10.0	Mutism 12 h, reduced spontaneous speech 2 d
16/45/F	R	Anaplastic astrocytoma	L frontal	7.0	Reduced spontaneous speech 3 d
17/70/F	R	Anaplastic astrocytoma	L frontal	0.9	—
18/22/M	R	Low-grade oligodendroglioma	L frontal	10.1	—
19/48/M	R	Low-grade astrocytoma	R frontal	29.0	—
20/48/M	R	Low-grade oligodendroglioma	R frontal	7.0	—
21/33/M	R	Low-grade astrocytoma	L frontal	23.4	—
22/43/F	R	Low-grade oligodendroglioma	R frontal	3.5	—
23/36/F	L	Low-grade oligodendroglioma	L frontal	56.2	—

RU = right upper limb; LU = left upper limb; RL = right lower limb; LL = left lower limb.

in the SMA proper. Only five patients had additional activation in the pre-SMA. In the SMA, activation was either contralateral to the moving hand (18 patients), or bilateral (5 patients).

Neurologic findings. Neurologic findings are summarized in tables 1 and 2. Thirteen of 23 patients had no motor deficit following surgery (Patients 12 through 23). Patient 12 had a mild spastic hemiparesia of the left lower limb before surgery that remained unchanged after surgery. The 11 remaining patients had a postoperative motor deficit (Patients 1 to 11), which appeared after surgery. The deficit was always contralateral to surgery. Mild hypotonia and normal and symmetric stretch reflexes were observed. The severity and duration of the deficit varied among patients. In six patients, the deficit was complete and no movement could be evidenced even with repeated strong commands. The clinical presentation of the deficit was similar to a proportional contralateral hemiplegia, except for the normal reflexes. The five remaining patients had a general reduction in spontaneous contralateral movements and the muscular strength was partly reduced. The deficit predominated in the upper limb in five patients. A transient grasping reflex was observed in three patients.

Recovery began after 2 days to 6 weeks. Eight patients recovered completely within 1 week to 3 months. In these patients, the muscular strength and tonus recovered rapidly within the first 2 weeks. Their reflexes remained normal. Spontaneous movements, absent or greatly diminished at onset, gradually reappeared. During the recovery period, although there was a decrease in spontaneous movements, movements could be elicited on the side contralateral to surgery with strong verbal commands. At 1 year, six of these eight patients had normal neurologic findings. Patient 6 had a mild preoperative hemiparesia that was probably due to mass effect, as it disappeared following recovery of the additional postoperative motor deficit. The two remaining patients had slight persistent motor disabilities. At 12 months, Patient 7 had a persistent underutilization of the right upper limb without any disorder in bimanual coordination tasks. At 12 months, Patient 10 had an impairment of bimanual coordination and a grasping of the left hand.

One patient (Patient 11) had a similar clinical presentation except for the evolution of the symptoms. In this patient, the deficit appeared progressively after 8 hours, as a consequence of an extradural hematoma, which required emergency surgery. After removal of the hematoma, the patient progressively recovered within 2 months.

In the two remaining patients (Patients 8 and 9), the evolution of the deficit was different, with additional signs evocative of a lesion of the corticospinal system. Patient 8 had a preoperative spastic paresia of the left lower limb. Immediately after surgery, complete absence of movements was observed with hypotonia. The hypertonia reappeared rapidly within the first week. Motor recovery began after 1 month and lasted 2 months. At 1 year, neurologic findings were similar to the preoperative state. Patient 9 had a complete postoperative hemiplegia with hypotonia. A pyramidal syndrome contra-

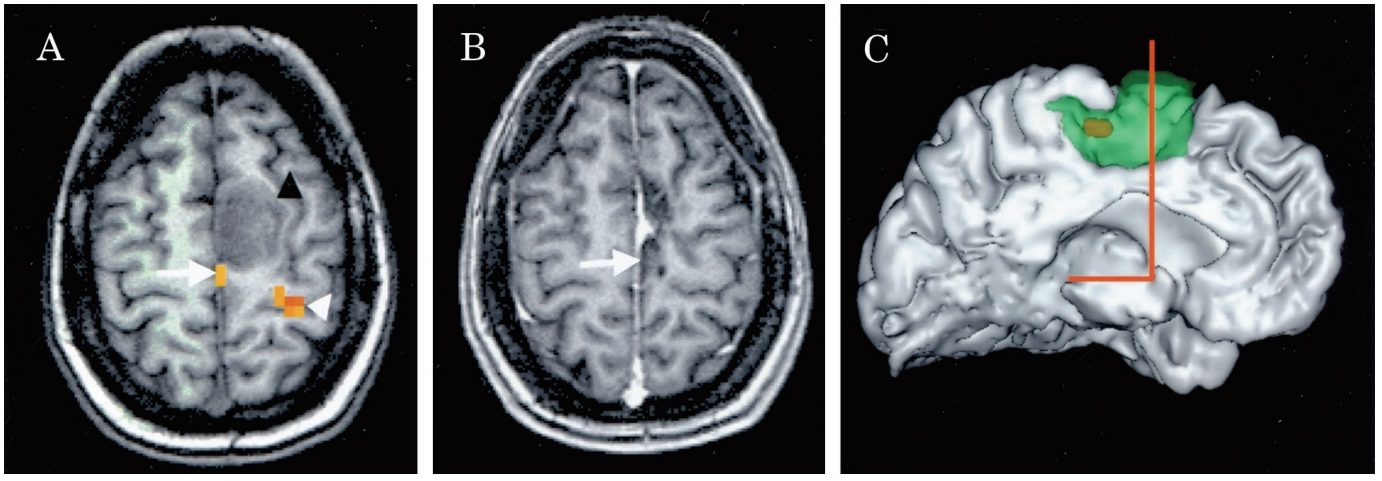

Figure 1. A 37-year-old woman (Patient 5) presented a low-grade oligodendroglioma of the medial part of the left frontal lobe (black arrowhead in A). (A) Preoperative fMRI during flexion/extension of the fingers of the right hand showing activation in the left primary sensorimotor area (white arrowhead) and the left supplementary motor area (SMA) proper (arrow). (B) Coregistered postoperative T1-weighted axial section showing that the area activated preoperatively in the SMA proper (arrow) was removed during surgery (star). (C) Three-dimensional surface rendering (ANATOMIST, CEA, Orsay) showing the area activated in the SMA (red) and the surgical resection (green). The vertical line passing at the level of the anterior commissure (VCA) corresponds to the vertical line perpendicular to the anterior commissure–posterior commissure line (horizontal line). The SMA was completely resected. This patient had a postoperative contralateral deficit from which she recovered in 2 months.

lateral to the lesion side with hyperreflexia and a Babinski sign gradually appeared during the first week following surgery. Motor recovery began 6 weeks after surgery and remained partial at 3 months with a persistent involuntary grasping of the upper limb and spasticity of the lower limb. Then, the patient's state remained unchanged with a spastic involvement of the lower limb and an obvious underutilization of the upper limb until death, which occurred 6 months later.

Speech disorders were also observed in nine patients. They were associated with a motor deficit in five patients and isolated in four patients (see tables 1 and 2). The deficit consisted of a global decrease in speech output ranging from a complete mutism to a partial reduction of spontaneous speech. In all patients but one, comprehension was normal. When speech was possible, utterances were correct but limited to "yes" or "no" or to short and simple sentences. No paraphasia and no grammatical errors were observed. At this time, repetition and verbal fluency were possible but difficult, as if "words could not come out." All patients recovered from these deficits; recovery was complete in seven patients and partial in two patients. Patients recovered from mutism within 12 hours to 4 days, whereas spontaneous speech reduction lasted from 1 week to 6 months. At 1 year, Patient 7 still had a slight reduction in spontaneous speech. Patient 9 had a different clinical presentation. In this patient, comprehension and repetition were impaired. Word substitution and fluency

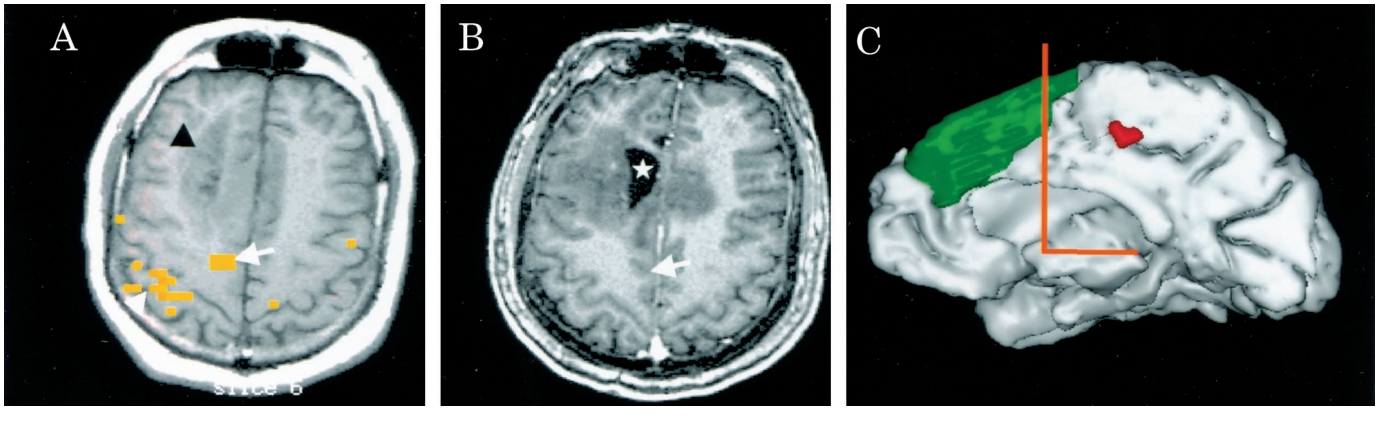

Figure 2. A 48-year-old man (Patient 19) presented a low-grade astrocytoma of the right frontal lobe (black arrowhead in A). (A) Preoperative fMRI during flexion/extension of the fingers of the left hand showing activation in the right primary sensorimotor area (white arrowhead) and the left supplementary motor area (SMA) (arrow). (B) Coregistered postoperative T1-weighted axial section showing that the area activated preoperatively in the SMA proper (arrow) was spared after surgery (star). (C) Three-dimensional surface rendering (ANATOMIST, CEA, Orsay) showing the area activated in the SMA (red) and the surgical resection (green). Postoperative neurologic findings were normal.

difficulties were also observed. Spontaneous speech and fluency remained reduced for 3 months and then recovered slowly until the patient's death.

Comparison of the clinical outcome and the extent of supplementary motor area resection. The volumes of surgical resections range from 0.9 to 62.6 cm^3 (mean, 19.9 cm^3). The percentage of the area activated in the SMA proper removed during surgery ranges from 0 to 100%. In all patients who presented a postoperative deficit (Patients 1 to 11), the activated part of the SMA was at least partially removed during surgery (figure 1). In patients 0 to 10, the resection involved at least one-third of the activated part of the SMA (33.3 to 100%; mean, 60.6%) (see table 1). In Patient 11, only 9.1% of the area activated in the SMA was resected. This patient had a postoperative extradural hematoma that probably contributed to the occurrence of the postoperative motor deficit. In all patients without any additional postoperative motor deficit compared with the preoperative clinical status (Patients 12 to 23), the area activated in the SMA was preserved during surgery (figure 2). In Patient 12, the postoperative hemiparesia was unchanged compared with the preoperative examination and was related to the extension of the tumor into the primary motor cortex. In all patients, there was a strong positive relationship between the resection of the SMA activation and the occurrence of the postoperative deficit ($r = 0.83$; $p < 0.01$). On the opposite, the volume of surgical resection was not correlated with the occurrence of the postoperative deficit ($r = 0.04$; $p > 0.05$). No statistical relationship was found between the volume of resection and recovery onset ($r = 0{,}15$; $p > 0.05$) or duration ($r = 0{,}26$; $p > 0.05$). Similarly, no statistical relationship was found between the percentage of SMA resection and either recovery onset ($r = 0{,}12$; $p > 0.05$) or duration ($r = -0.17$; $p > 0.05$).

Discussion. Activation in the SMA was observed in all patients performing a simple movement of the hand. This activation was located caudally to the VCA line (SMA proper). Eleven of 23 patients had a motor deficit following surgery. The deficit was transient and the recovery was complete (eight patients) or almost complete (three patients) within a few weeks or months. The occurrence of the deficit was highly related to the resection of the area activated in the SMA contralateral to the deficit. These results demonstrate the role of the SMA proper in the occurrence of the motor deficit observed following surgical resection of the medial frontal lobe.

The SMA corresponds to the medial aspect of area 6 on the medial wall of the frontal lobe.[24] The SMA is entirely located in the superior frontal gyrus. The anatomic limits of the SMA are the primary motor cortex for the foot posteriorly, and the cingulate sulcus inferiorly.[6,7] The SMA is limited anteriorly by the genu of the corpus callosum and superiorly by the edge of the medial cortex.[6,7] Based on anatomic and physiologic evidence, the motor fields of the medial wall of the frontal cortex are formed by several distinct areas, including the SMA proper, the pre-SMA, and cingulate motor areas buried in the cingulate sulcus. Anatomically, the limit between the SMA proper and the pre-SMA is usually defined by the VCA line (a vertical line passing through the anterior commissure, perpendicular to the anterior commissure–posterior commissure line).[6,7] The pre-SMA and the SMA proper receive precentral and postcentral afferents. However, whereas the pre-SMA receives its afferents from associative frontal and parietal areas, the SMA proper is connected mainly with the primary motor cortex, caudal premotor areas, and primary and secondary sensory areas. Only the SMA proper sends direct corticospinal efferences and projects to the primary motor cortex.[6] The SMA proper but not the pre-SMA has a somatotopic organization.[6,7]

This anatomic organization supports functional differences.[6,7] Functional imaging studies have shown that activation in the SMA is task related. Thus, the pre-SMA has an important role in supramotor activities such as the selection, the preparation, and the sequencing of movements[8-13,15-17] whereas the SMA proper is more closely related to movement execution.[8,13,17] During simple movement tasks, activation limited to the SMA proper is commonly observed,[6,25] in line with our data. However, contrary to previous PET or fMRI studies that failed to show activation in the SMA for such simple self-paced movements,[8-10,12] all patients in the present study had significant signal changes in the SMA proper. This may be due to increased sensitivity of more recent MRI or PET scanners. Recent imaging studies using either technique have reported consistent activation in the SMA in subjects performing similar simple self-paced hand movement.[26,27] In line with this hypothesis is the fact that recent PET studies have reported activation in the SMA during simple motor tasks[26] whereas previous ones failed.[10] Furthermore, fMRI may be more sensitive than PET studies.[28] Thus, these data suggest that the SMA proper is involved in the execution of often-used self-paced movements. The posterior part of the SMA also appears to be involved in the initiation of internally generated movements. In monkeys, lesions of the posterior part of the SMA proper impair self-initiated movements. In humans, the SMA proper is more active when movements are executed than when they are only imagined.[14] Lastly, in patients with PD, impaired activation in the SMA proper may account for the difficulties these patients have in initiating movements or akinesia.[29]

The present results demonstrate a strong correlation between the occurrence of the deficit following surgery of the medial frontal wall and the resection of the area activated in the SMA proper during performance of simple finger movement. On the opposite, when the resection respected the SMA proper, the postoperative outcome was favorable in all patients. In Patient 11, the resection involved only 9.1% of the cortex activated in the SMA proper. The postoperative deficit was delayed several hours after the surgical procedure and was probably related to a secondary extradural hematoma. The presence of a

correlation between the occurrence of the deficit and the resection of the SMA proper also strengthens the fact that the deficit is due to the lesion of the SMA proper itself and is not the result of reversible surgical trauma, or secondary ischemia in adjacent cortical areas. This is in line with the clinical characteristics of the deficit. First, the occurrence of the postoperative deficit was independent of the volume of surgical resection. Second, the deficit was similar among patients. Third, it appeared immediately after the resection, earlier than would be expected for deficits resulting from postoperative edema or venous thrombosis. Fourth, the time for recovery was also longer than expected for edema or venous thrombosis. Lastly, follow-up MR scans showed no evidence of ischemia in adjacent cortical areas. However, we cannot exclude the possibility that edema may have contributed to a lesser extent to the severity of the deficit.

The clinical characteristics of the deficit observed in patients with surgical removal of the medial frontal cortex provide further evidence in favor of the role of the SMA proper in movement initiation and execution. The postoperative motor deficit had similar clinical presentation among patients. Patients had a global reduction in spontaneous movements contralateral to the operated side with variable severity ranging from a complete absence to a milder reduction in movements predominating in the upper or lower limb. When no movements were observed, the deficit was similar to that observed following a corticospinal lesion except for the normal reflexes. This is in line with previous reports of patients with resection of the medial frontal lobe, describing the deficit as an hemiplegia.[1,2,5] In less severely affected patients, strong verbal commands could elicit movements of the affected half of the body. This is in line with the observation that the SMA is preferentially involved in the generation of internally driven movements rather than sensory-guided movements.[13] When the deficit predominated in the upper limb, the patients would rather use the opposite arm. This was particularly striking when the deficit involved the dominant side. A discrepancy was observed between movement reduction and the actual strength of the affected limb that could be obtained with insistence of the examiner. Thus, the deficit seemed to be more related to an akinesia or motor neglect rather than to a hemiparesia, as previously described.[3]

The evolution of the deficit was also one of its main characteristics.[1-5] The deficit appeared immediately after the resection. It was transient in all patients but one (Patient 9). Recovery began within the first days or weeks in all cases but one. At 3 months, the recovery was complete without deficit of the muscular strength. Once the recovery began, the muscular strength returned to normal rapidly. Long-term follow-up was performed over at least 1 year in all patients, except in Patient 9, who died of the extension of the anaplastic glioma. In six patients (Patients 1 to 6), the recovery was complete, even in bimanual coordination tasks. Patient 8 recovered preoperative clinical status, with a residual spastic monoparesia of the lower limb due to neoplastic infiltration of the posterior part of the putamen and the internal capsule. In the two remaining patients, long-term follow-up demonstrated a persistent underutilization of the dominant arm (Patient 7) or an impairment in bimanual coordination with a grasping reflex of the affected hand (Patient 10), as already reported.[1-5] In Patient 9, after an initial recovery period, a spastic deficit gradually appeared. Follow-up MRI at 3 months showed that this deficit was due to the extension of the malignant tumor in the periventricular white matter toward the pyramidal tract. A possible explanation is that the deficit was initially due to the SMA resection, and then by the expression of a corticospinal tract lesion.

Nine patients also presented speech disorders with (Patients 3 to 5, 7, 9) or without (Patients 13 to 16) motor deficit. All speech disorders were observed after resection of a lesion of the left hemisphere among right-handed patients. Speech disturbances are commonly reported after lesion of the SMA in the dominant hemisphere[1-4] although disturbances have also been reported in lesions of the nondominant hemisphere.[1-3] The clinical presentation was a global reduction in spontaneous speech ranging from a complete mutism to a less severe speech reduction. Comprehension remained normal in all patients but one. Neither paraphasia nor dysnomia was reported. Recovery of the initial mutism was fast, within several days, whereas recovery of the spontaneous speech reduction was progressive over several months. At 1-year follow-up, only one patient had a persistent reduction in speech fluency. These results are similar to those reported previously after resection of the SMA.[1-4] Unlike the other patients, Patient 9 presented word substitutions, comprehension (errors in verbal commands), and verbal fluency and repetition difficulties. As for the motor deficit, this different clinical presentation of the speech disorders was likely due to the rapid extension of the malignant tumor toward the frontoinsular regions.

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