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Postoperative speech disorder after medial frontal surgery

Role of the supplementary motor area

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Abstract—*Background:* Patients undergoing surgical resection of medial frontal lesions may present transient postoperative speech disorders that remain largely unpredictable. *Objective:* To relate the occurrence of this speech deficit to the specific surgical lesion of the supplementary motor area (SMA) involved during language tasks using fMRI. *Methods:* Twelve patients were studied using a verbal fluency task before resection of a low-grade glioma of the medial frontal lobe and compared with six healthy subjects. Pre- and postoperative MR variables including the hemispheric dominance for language, the extent of SMA removal, and the volume of resection were compared to the clinical outcome. *Results:* Following surgery, 6 of 12 patients presented speech disorders. The deficit was similar across patients, consisting of a global reduction in spontaneous speech, ranging from a complete mutism to a less severe speech reduction, which recovered within a few weeks or months. The occurrence of the deficit was related to the resection of the activation in the SMA of the dominant hemisphere for language ($p < 0.01$). Increased activation in the SMA of the healthy hemisphere on the preoperative fMRI was observed in patients with postoperative speech deficit. *Conclusions:* fMRI is able to identify the area at risk in the SMA, of which resection is related to the occurrence of characteristic transient postoperative speech disorders. Increased SMA activation in the healthy hemisphere suggested that a plastic change of SMA function occurred in these patients.

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Surgical resection of tumors of the medial frontal lobe may result in immediate postoperative motor and speech deficits.[1-8] The characteristics of the deficit vary in nature, severity, and evolution, but one of its main features is its complete or almost complete recovery within a few weeks or months.[1-8] It is thus of utmost importance for patients and the medical team to determine precisely not only the characteristics but also the cause of the deficit either to prevent this deficit or to inform the patient of the possibility

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Table 1 *Clinical characteristics of patients*

Patient no./age, y/sex	Side of frontal lesion	Handedness	Postoperative speech deficit	Speech recovery	Long-term follow-up	Motor deficit
Group 1						
1/45/M	Left	Right	Mutism	Begin after 3 d, complete at 8 mo	Normal at 1 y	Right hemiplegia, complete recovery at 4 mo
2/29/M	Left	Right	Reduced spontaneous speech	Begin after 3 d, complete at 6 mo	Normal at 1 y	Mild right hemiparesis, complete recovery at 2 mo
3/46/W	Left	Right	Mutism	Begin after 7 d, complete at 8 mo	Normal at 1 y	Moderate right hemiparesis, complete recovery at 3 mo
4/37/W	Left	Right	Reduced spontaneous speech	Begin after 4 d, complete at 6 mo	Normal at 1 y	Moderate right hemiparesis, complete recovery at 2 mo
5/22/W	Left	Right	Reduced spontaneous speech	Begin after 3 d, complete at 6 mo	Normal at 1 y	Moderate right hemiparesis, complete recovery at 2 mo
6/40/W	Right	Left	Mutism	Begin after 3 d, complete at 3 mo	Normal at 1 y	Moderate left hemiparesis, complete recovery at 3 mo
Group 2						
7/38/W	Left	Right	No	—	—	No
8/25/W	Right	Right	No	—	—	Moderate left hemiparesis, complete recovery at 3 mo
9/34/W	Left	Right	No	—	—	Right hemiplegia, complete recovery at 6 wk
10/45/M	Right	Right	No	—	—	Left hemiplegia, complete recovery at 2 mo
11/36/W	Left	Right	No	—	—	No
12/22/W	Right	Left	No	—	—	Moderate left hemiparesis, complete recovery at 3 mo

of its occurrence, being certain that it will recover within a short period of time.

With use of fMRI, transient motor deficits after medial frontal lobe resection have been related to the surgical removal of the posterior part of the supplementary motor area (SMA).[3] Although speech disturbances have been more frequently observed when the resection involved the hemisphere dominant for language,[1-4,6-8] whether a relation exists between the occurrence of language deficit and the lesion of the SMA has yet to be ascertained. FMRI has shown to be a powerful tool to study language functions. Compared with the Wada test, fMRI has been successfully used to determine the hemispheric dominance for language.[9-13] FMRI studies in healthy volunteers showed that the SMA was activated during various language tasks such as repetition[11,14] and silent verbal fluency[10,11,14] and that it was more frequently activated during productive tasks, such as semantic verbal fluency, than during receptive tasks, such as story listening.[11]

We used fMRI to determine whether the occurrence of speech deficit following surgical lesions of the medial frontal lobe was due to the specific lesion of the SMA. Similarly to motor deficit, it was predicted that speech deficit would be related to the resection of the SMA activated during language task. For that purpose, patients with surgical lesion of the medial frontal lobe were examined preoperatively using a verbal fluency task and compared with normal subjects. These observations should help identifying the area at risk for speech deficit before medial frontal lobe surgery to inform accurately the patient on the neurologic outcome and to plan the postoperative care.

Subjects and methods. *Subjects.* Patients. Twelve patients referred for surgical treatment of lesions of the medial frontal lobe were studied (nine women and three men; age range 22 to 46 years, mean 34.9 years) (table 1). All patients had a seizure history for 1 to 24 months. All lesions were low-grade gliomas. Lesions were located in the left frontal lobe in the eight right-handed patients and in the right frontal lobe in two right-handed patients and two left-handed patients. The pre- and postoperative neurologic examinations were performed by neurologists and neurosurgeons. Speech functions were assessed clinically with verbal comprehension, spontaneous speech, narrative tasks, verbal fluency, and repetition. Clinical examinations were performed on the day of the fMRI, the day before surgery, immediately after surgery, several times during the following week, after 1 and 3 months, every 3 months after 1 year, and then every 6 to 12 months. Preoperative motor or speech deficit was absent in all patients. No change in the clinical presentation was observed between the fMRI session and surgery in any patients. All patients had steroid treatment during 3 days immediately after surgery.

Table 2 *FMRI data for healthy subjects*

Subject no./age, y/sex	Handedness	LI_{Hem}*	LI_{sma}*	Activation volume left SMA, cm^3	Activation volume right SMA, cm^3	Maximum *T*-value of SMA activation in dominant hemisphere, *x, y, z*† (*T*-value)
1/30/M	Right	0.54	0.21	11.4	7.5	−3,12,57 (6.55)
2/34/W	Right	0.77	0.24	17.9	11.0	−3,18,54 (10.33)
3/53/M	Right	0.52	0.75	2.2	0.3	−3,−6,72 (4.51)
4/27/M	Right	0.34	0.09	11.6	9.6	−3,12,63 (10.02)
5/52/W	Right	0.67	0.11	12.3	9.8	−3,−9,60 (6.30)
6/29/W	Right	0.53	−0.22	2.9	4.6	−3,21,51 (5.57)
Mean ± SD	—	0.56 ± 0.15	0.20 ± 0.32	9.7 ± 6	7.1 ± 4	−3 ± 0, 8 ± 12, 60 ± 7

* LI_{Hem} and $LI_{sma} = ([L - R]/[L + R])$ with *L* being the number of activated voxels in the left and *R* in the right hemisphere (LI_{Hem}) or SMA (LI_{sma}).
† In Talairach space.

LI_{Hem} = hemispheric laterality index; LI_{sma} = laterality index in SMA; SMA = supplementary motor area.

Healthy volunteers. Six age-matched right-handed control subjects (three women and three men; age range 27 to 53 years, mean 37.5 years) (table 2) were compared with the patients. None of the volunteers had a history of neurologic or psychiatric disease or diagnosed disability.

Imaging. The MR protocol was carried out with a 1.5 T MR unit. The preoperative fMRI was performed between 3 and 150 days before surgery (mean 35 days) using blood oxygen level–dependent fMRI. The protocol included the following: 1) 20 axial gradient echo–echo planar images covering the whole frontal lobes (repetition time/echo time/flip angle: 5,000 ms/60 ms/90°, 5-mm slice thickness, no gap, in-plane resolution: 3.75 × 3.75 mm); and 2) axial inversion recovery three-dimensional T1-weighted images for anatomic localization. The postoperative MRI was performed between 1 and 370 days (mean 186 days) after surgery. The protocol included axial inversion recovery three-dimensional T1-weighted images and axial T2-weighted fast spin echo images.

fMRI tasks. All subjects were tested in their native language (French). An auditory-cued semantic fluency task was performed. Stimuli were recorded on a digital audio tape and presented using standard headphones customized for fMRI experiments and inserted in a noise-protecting helmet that provided isolation from scanner noise. Subjects had to generate mentally as many words as possible in a semantic category (fruits, vegetables, pieces of furniture, body parts, animal, sports). The paradigm was block-designed, alternating rest and activation, and consisted of seven epochs of 30 seconds (duration of each run: 3 minutes 30 seconds).

fMRI analysis. Statistical analysis of preoperative images was performed in MATLAB (Mathworks, Natick, MA) with SPM99 (Wellcome Department of Cognitive Neurology, London, UK) using the general linear model.[15] For each subject, images were corrected for subject motion and transformed stereotactically to Talairach coordinates using the standard template of the Montreal Neurologic Institute.[16] The resulting images were smoothed with a Gaussian spatial filter to a final smoothness of 5 mm. The data were then analyzed statistically using a two-temporal basis functions model.[17] Overall signal differences between runs were also modeled. A temporal cutoff of 240 seconds was applied to filter subject-specific low-frequency drift related mostly to subject biologic rhythms. Statistical parametric maps were calculated for the contrast of verbal fluency vs rest. We used a voxel-wise threshold of $p < 0.01$ with a corrected $p < 0.05$ for cluster extent (corrected for multiple comparison). Postoperative anatomic MR images were also transformed stereotactically to Talairach coordinates, allowing coregistration with preoperative images.

fMRI quantification of hemispheric dominance. An index of hemispheric dominance for language was computed on the basis of fMRI data as $LI_{Hem} = ([L - R]/[L + R])$ with *L* and *R* being the number of activated voxels in the left and right hemispheres, respectively.[9,18] LI_{Hem} varied from −1 to +1; a positive index corresponded to left-predominant activation, whereas a negative index corresponded to right-predominant activation. Activation ranging from −0.20 to 0.20 were considered symmetric.

For the SMA, a specific laterality index (LI_{sma}) was calculated following the same principle. The anatomic boundaries of the SMA were determined as follows. The SMA was located in the medial aspect of the superior frontal gyrus. The anterior limit of the SMA was the genu of the corpus callosum, the posterior limit the primary motor cortex for the foot, the superior limit the edge of the medial cortex, and the inferior limit the cingulate sulcus.[19,20]

To determine the relationships between the language deficit and the lesion of the SMA, the following parameters were studied: topography and volume of the tumor and the surgical lesion, extent caudal to the VCA line (a vertical line passing through the anterior commissure, perpendicular to the anterior commissure–posterior commissure line), and overlap between the resected volume and the area activated in the SMA during the fluency task (percentage of activation volume removed, coordinates of the maximum *T*-value). The resection volumes were determined using a semiautomatic segmentation software based on region growing (J.F. Mangin; SFHJ-CEA, Orsay, France).

Comparisons were performed using *r* correlation coefficient for comparison between two quantitative data sets (volumes of tumor, activation and resection, laterality indexes, recovery onset, duration of deficit), χ^2 with Yates' correction for comparison between qualitative data (occurrence and severity of deficit, side of lesion, resection of T_{max} value, extent caudal to VCA line), and Mann–Whitney *U*-test for comparison between qualitative and quantitative data.

Results. *Control subjects.* FMRI activation. In all subjects, activation was observed in the frontal lobes, including the inferior and middle frontal gyri, in the anterior insular cortex, and in posterior temporoparietal regions (see table 2).

In all subjects, the left hemisphere was dominant for language. The mean hemispheric laterality index (LI_{Hem}) was 0.56 ± 0.15.

Activation in the SMA was bilateral in all subjects. It was predominant in the left hemisphere in three subjects, in the right hemisphere in two, and symmetric in one. The mean laterality index of the SMA (LI_{sma} ± SD) was 0.20 ± 0.32. The mean Talairach coordinates (± SD; mm) of maximum *T*-values in the left hemisphere were as follows: $x = -3 \pm 0$; $y = 8 \pm 12$; $z = 60 \pm 7$.

Patients. Two groups of patients were defined: patients with postoperative speech disorders (Group 1) and patients without postoperative speech disorders (Group 2).

Patients with speech deficit (Group 1). *Neurologic findings.* Six of 12 patients (Patients 1 to 6) had transient speech disorders presenting as a global reduction in spontaneous speech ranging from a complete mutism (three patients) to a less severe speech reduction (three patients) (see table 1). In all patients, comprehension was normal. No paraphasia or dysnomia was observed. The recovery began during the first week and was com-

Table 3 *FMRI data for patients*

Subject no.	LI_{Hem}*	LI_{sma}*	Activation volume SMA in left hemisphere, cm^3	Activation volume SMA in right hemisphere, cm^3	Volume of tumor, cm^3	Volume of surgical resection, cm^3; extends caudal to VCA line, Y/N	Percentage of resection of activated area in SMA of dominant hemisphere	Maximum *T*-value of SMA activation in dominant hemisphere, *x,y,z*† (*T*-value); resection, Y/N
Group 1								
1	0.25	−0.53	3.7	12.1	51.7	51.6 Y	62.5	−6,−12,60 (6.15) Y
2	0.71	−0.48	2.2	6.2	59.4	40.0 Y	37.8	−3,3,60 (4.66) Y
3	0.22	−0.27	4.7	8.2	45.6	45.9 Y	35.2	−3,0,66 (6.76) N
4	0.29	−0.55	3.8	13.2	37.5	19.5 Y	16.1	−3,−3,45 (4.08) Y
5	0.42	0.02	15.0	14.3	50.4	46.0 Y	16.1	−3,6,60 (8.84) N
6	−0.46	−0.24	4.0	6.5	15.9	40.0 Y	93.4	3,15,57 (5.02) Y
Mean ± SD	0.24 ± 0.38	−0.34 ± 0.22	5.6 ± 4.7	10.1 ± 3.5	43.4 ± 15.3	40.5 ± 11.2	43.5 ± 29.8	−3 ± 3, 2 ± 9, 58 ± 7
Group 2								
7	0.57	0.21	15.8	10.3	100.2	80.6 N	7.1	−3,9,60 (8.56) N
8	0.85	0.61	9.5	2.3	28.8	24.3 Y	0	−3,12,60 (8.34) N
9	0.31	0.06	2.8	2.5	81.3	14.4 Y	0	−9,21,63 (4.07) N
10	0.67	0.24	7.6	4.6	14.3	6.8 Y	0	−6,−3,72 (5.68) N
11	0.42	0.14	2.2	1.7	122.1	55.9 N	0	−6,−12,63 (4.60) N
12	−0.42	−0.31	6.5	12.2	10.1	6.9 Y	8.6	6,15,42 (5.82) N
Mean ± SD	0.40 ± 0.44	0.16 ± 0.30	7.4 ± 5.0	5.6 ± 4.5	69.3 ± 46.2	30.5 ± 31.3	2.6 ± 4.1	−4 ± 5, 7 ± 12, 60 ± 10

* LI_{Hem} and $LI_{sma} = ([L - R]/[L + R])$ with L being the number of activated voxels in the left and R in the right hemisphere (LI_{Hem}) or SMA (LI_{sma}).
† In Talairach space.

LI_{Hem} = hemispheric laterality index; LI_{sma} = laterality index in SMA; SMA = supplementary motor area; VCA = vertical line passing through anterior commissure, perpendicular to anterior–posterior commissure line.

plete or almost complete within 8 months. At 1 year, spontaneous speech was normal, although two patients (Patients 2 and 3) had mild word-finding difficulties.

All patients had a transient motor deficit, contralateral to the resection, which occurred immediately after surgery. The severity ranged from hemiparesis (five patients) to hemiplegia (one patient). In all patients, motor recovery began during the first week after surgery and was complete within 3 months.

Hemispheric laterality index. The five right-handed subjects were left hemisphere–dominant with an LI_{Hem} ranging from 0.22 to 0.71 (mean 0.38) (table 3). The left-handed patient was right hemisphere–dominant, with an LI_{Hem} of −0.46.

Activation in the SMA. Activation in the SMA was bilateral in all patients (see table 3; figure 1). It was predominant in the right hemisphere in five patients and symmetric in one. The mean LI_{sma} was −0.34 ± 0.22. In Talairach space, the mean coordinates of the maximum *T*-values (± SD; mm) in the dominant hemisphere for language were as follows: $x = -3 \pm 3$; $y = 2 \pm 9$; $z = 58 \pm 7$.

Tumor volume and surgical resection. All lesions affected the hemisphere dominant for language (see table 3). Mean tumor volume was 43.4 ± 15.3 cm^3. Mean resection volume was 40.5 ± 11.2 cm^3. The resections were extended caudal to the VCA line in all patients. The resection of the area activated in the SMA ranged from 16.1 to 93.4% (mean 43.5%). The maximum *T*-values were located within the resected area in four patients and outside this area in two.

Patients without speech deficit (Group 2). *Neurologic findings.* Six of 12 patients had no speech disorder following surgery (Patients 7 to 12) (see table 1). Among these patients, four patients had a transient motor deficit contralateral to the resection. The severity ranged from moderate hemiparesis (two patients) to hemiplegia (two patients). Except for their frequency, motor deficits had the same clinical characteristics as in Group 1.

Hemispheric laterality index. The five right-handed subjects were left hemisphere–dominant with LI_{Hem} ranging from 0.31 to 0.85 (mean 0.56) (see table 3). The left-handed patient was right hemisphere–dominant, with an LI_{Hem} of −0.42.

Activation in the SMA. Activation in the SMA was bilateral in all patients (see table 3; figure 2). It was predominant in the left hemisphere in three patients, symmetric in two, and in the right hemisphere in one. The mean LI_{sma} was 0.16 ± 0.30. In Talairach space, the mean coordinates of the maximum *T*-values (± SD; mm) in the dominant hemisphere for language were as follows: $x = -4 \pm 5$; $y = 7 \pm 12$; $z = 60 \pm 10$.

Tumor volume and surgical resection. The lesions affected the hemisphere dominant for language in four patients and the nondominant in two (see table 3). Mean tumor volume was 69.3 ± 46.2 cm^3. Mean resection volume was 30.5 ± 31.3 cm^3. The resections extended caudal to the VCA line in four patients and remained rostral in two. In the dominant hemisphere, the resection of the area activated in the SMA ranged from 0 to 8.6% (mean 2.6%). In the nondominant hemisphere, the resection of the SMA ranged from 24.9 to 25.1% (mean 25.0%) (Patients 8 and 10; see figure 2). The maximum *T*-value was never resected in the dominant hemisphere. In the nondominant hemisphere, the maximum *T*-value was removed in one patient.

Relationships between clinical, functional, and surgical parameters. The occurrence of postoperative speech disorders was not correlated with the hemispheric dominance for language, the side and volume of the tumor, or the volume and the extent caudal to the VCA line of the surgical resection (see table 3). There was a relationship between the occurrence of the deficit and the percentage of resection of SMA activation in the dominant hemisphere for language ($p < 0.01$). Among all patients, the resection of the T_{max} value of the activation in the SMA was present in only four of six patients with speech disorders ($p < 0.07$). A negative correlation was present between the LI_{sma} and the percentage of SMA resection in the dominant hemisphere ($r = -0.58$; $p < 0.05$). SMA activation in the healthy hemisphere was larger in patients with

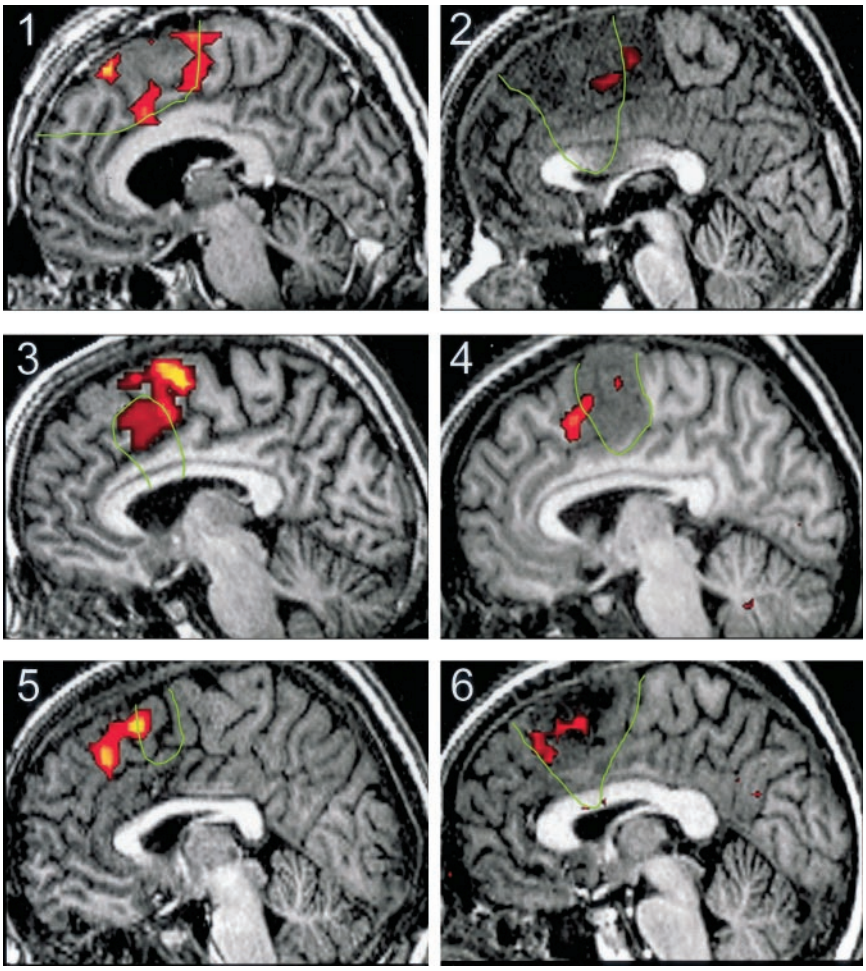

Figure 1. Group 1 patients (Patients 1 to 6) presented transient postoperative speech impairment. For each patient, fMRI activation map and resection margins (green line) are displayed on the preoperative midsagittal MR image of the lesioned hemisphere (left hemisphere in Patients 1 to 5 and right hemisphere in Patient 6). All lesioned hemispheres were dominant for language. Percentage of resection of the area activated in the supplementary motor area ranged from 16.1 to 93.4% (mean 43.5 ± 29.8%) (table 3).

than without postoperative speech disorders, although the difference did not reach significance, whereas activation was similar in the damaged SMA. There was no relation between SMA activation and tumor or resection volumes. In Group 1 patients, the onset of recovery and the severity and duration of the deficit were not related to tumoral and resection volumes or to the characteristics of SMA activation (percentage of resection of SMA activation in the dominant hemisphere, activation volumes, and lateralization). Postoperative motor deficits were observed only when the resection extended in the posterior part of the SMA, caudal to the VCA line ($p < 0.02$).

Discussion. The current results confirm that speech deficit that followed medial frontal lobe surgery was similar across patients. It consisted in a global speech reduction, ranging from complete mutism to a less severe speech reduction. This deficit recovered in all patients within 3 to 8 months. Based on an fMRI verbal fluency task, it was shown that speech disorders occurred after resection of at least 16% of the area activated in the SMA of the dominant hemisphere. Increased activation in the SMA contralateral to the tumor was observed, suggesting that a reorganization of SMA function occurred in the presence of brain tumor.

The SMA is located in the medial aspect of Brodmann area 6 on the medial wall of the frontal lobe. 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 (also called SMA proper), the pre-SMA, and cingulate motor areas buried in the cingulate sulcus.[20,21] Anatomically, the limit between the SMA and the pre-SMA is usually defined by the VCA line.[20,21] The pre-SMA receives its afferents from associative frontal and parietal areas. The SMA is mainly connected with the primary motor cortex, caudal premotor areas, and primary and secondary sensory areas.[20,21] This anatomic organization supports functional differences. PET studies showed activation in the SMA near or caudal to the VCA line during simple language tasks like word repetition,[22]. On the contrary, more complex verbal tasks like silent word generation tasks[23] or word production in new conditional demands[24] activated the pre-SMA. A recent fMRI study showed activation in the pre-SMA during free, paced, and semantic word generation and activation near the VCA line during simple word repetition.[14] Thus, there is a rostrocaudal shift in SMA activation related to the external constraint during silent speech tasks, with the SMA implicated in the control of vocal sound production[25] and the pre-SMA recruited for more complex verbal demands. In control subjects and patients without deficit, we observed activation during silent semantic verbal fluency in the pre-SMA (mean anteroposterior [*y*] Talairach coordinate: +8 mm). However, in patients with postop-

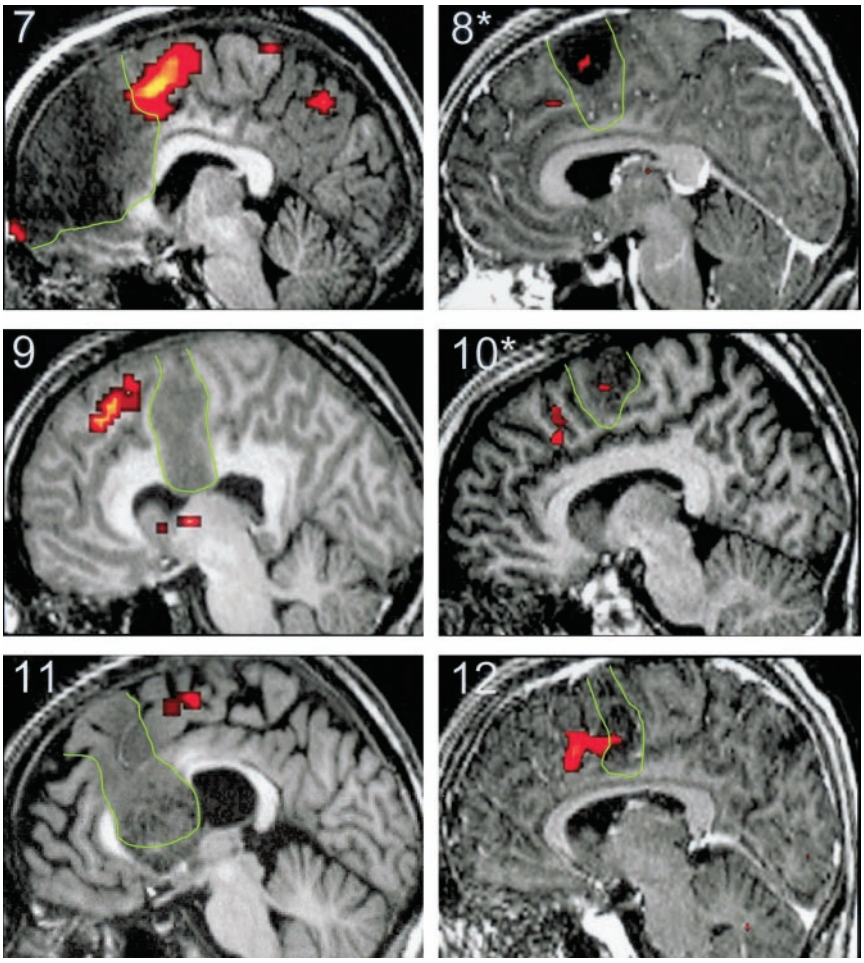

Figure 2. Group 2 patients (Patients 7 to 12) had no postoperative speech disorder. For each patient, fMRI activation map and resection margins (green line) are displayed on the preoperative midsagittal MR image of the lesioned hemisphere (left hemisphere in Patients 7, 9, and 11, and right hemisphere in Patients 8, 10, and 12). In Patients 7, 9, 11, and 12, the lesioned hemisphere was dominant for language, and the percentage of resection of the area activated in the supplementary motor area (SMA) ranged from 0 to 8.6% (mean 2.6 ± 4.1%). In Patients 8 and 10*, the lesioned hemisphere was not dominant for language, and the percentage of resection of the area activated in the nondominant SMA was 24.9 and 25.1%.*

erative speech deficit, the mean coordinate was more posterior, located only 2 mm rostral to the VCA line. This relative posterior shift was probably secondary to mass effect due to the tumor, which resulted in a posterior displacement of the activation, although the difference was not significant because of large intersubject variation.

Clinical and electrophysiologic studies in patients also support a role of the SMA in speech. Electrical stimulation performed rostral to the supplementary motor representation of the face, which is located at the VCA line level,[20] resulted in vocalization and speech arrest or slowing of speech.[26,27] Ictal epileptic speech arrest and vocalizations have also been reported in patients with SMA lesions.[28-30] Speech deficits have been observed after infarction in the territory of the anterior cerebral artery[31-33] and surgical resection of the medial part of the frontal lobe.[1-4,6-8]

The current data provide strong evidence that speech disorders associated with medial frontal lobe surgery are due to the selective resection of the SMA in the hemisphere dominant for language activated during language tasks. In patients with speech deficit, resection was at least superior to 16% of the activated area. Speech disorders were not related to tumor and resection volumes per se. The severity and the course of the speech deficit remained unpredictable on the basis of anatomic and functional imaging data. These observations are entirely in keeping with a previous work, which has related the occurrence of motor deficit after medial frontal lobe surgery to the selective resection of the post-SMA using a simple motor task.[3] These results also argue strongly against a role of postoperative edema or venous thrombosis in the occurrence of the deficit. Although we cannot exclude the possibility that edema may have contributed to a lesser extent to the severity of the deficit, the deficit appeared earlier and the time for recovery was longer than would be expected for edema or venous thrombosis. Last, follow-up MR scans showed no evidence of ischemia in adjacent cortical areas.

In agreement with the current results, the clinical presentation was similar across patients and consisted in a global reduction in spontaneous speech. During the first week, the presentation varied from a complete mutism to a less severe speech reduction. Articulation was preserved.[1-4,7,8,31-34] Comprehension remained normal. No paraphasia or dysnomia was reported. Thus, the deficit involved the production of speech and spared the receptive aspects of language. Recovery of the initial mutism was fast, within several days, whereas recovery of spontaneous speech reduction was more progressive over several months.[2,4,7,8] The initial mutism and the delay and speed of recovery were variable. This characteristic

presentation is consistent with the fact that it corresponds to a specific speech disorder related to limited lesion of the SMA. These speech disturbances have been initially called transcortical motor aphasia.[35,36] In a previous study, residual speech impairment following recovery was influenced by the length, the complexity, and the meaning of words, arguing for a role of the SMA in short-term articulatory buffering in speech production.[34] Speech disorders were also commonly associated with motor deficits. This association is congruent with the presence of functional areas in the SMA dedicated either to the production of speech or to motor control.[1-4,6-8]

In agreement with previous studies, speech disturbances were observed after lesion in the SMA in the dominant hemisphere,[1-4,6-8,31-33] although such deficits have also been reported after lesion in the nondominant hemisphere.[2,4] FMRI production tasks such as verbal fluency or word generation, which activate predominantly frontal areas, have proven to be reliable alternatives to the Wada test to lateralize language dominance.[9,10,12,13,18,37-41] With the verbal fluency task, Wada laterality indexes (LI) were correlated with fMRI LI in the entire frontal lobe, the medial frontal gyrus, and insula but less so or not with LI in the SMA.[9,10,18,38] Based on the LI_{Hem}, all right-handed patients had a classic left hemisphere dominance and the two left-handed patients had a right hemisphere dominance. No symmetric pattern was found. The verbal fluency task is adequate to study frontal activation, although it is not as valid for temporal lobes, for which other tasks such as receptive[18] or read–response naming tasks[12,13] may be more reliable. The higher prevalence of women as patients than control subjects should not have any significant influence on the LI_{Hem}, which was not gender dependent in a silent verbal fluency task.[42] LI_{sma} decrease observed in patients with postoperative speech deficit was due to a recruitment of the SMA in the healthy hemisphere. Although the small number of patients studied did not allow us to demonstrate a significant recruitment, these abnormal LI_{sma} values argue in favor of a preoperative dysfunction of the SMA ipsilateral to the tumor. Such a reorganization of brain function has also been reported for language in patients with epilepsy using fMRI[43,44] and with poststroke aphasia[45] and brain tumors using PET.[46] Changes from dominant to nondominant healthy hemisphere for language in LI_{sma} values suggest that the SMA in the hemisphere contralateral to the tumor is recruited to compensate for the deficient SMA This plasticity is supported by the existence of a strong anatomic and functional connectivity between the two SMA.[47] Such a recruitment of the healthy hemisphere was not sufficient to prevent the occurrence of the deficit and was not related to the onset of recovery, the severity, or the duration of the deficit.

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