Postoperative deficits and functional recovery following removal of tumors involving the dominant hemisphere supplementary motor area

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The supplementary motor area (SMA) is a region located within each cerebral hemisphere at the posterior mesial border of the frontal lobe adjacent to the falk. The functional significance of this area has been somewhat unclear, and information regarding its influence on motor output has largely been based on evoked responses to direct stimulation in primates and humans. In this series of patients with primary and metastatic tumors involving the dominant hemisphere SMA, a distinct pattern of postoperative deficits and recovery has emerged which emphasizes the role of this critical area in the initiation of motor activity, including speech. Based upon this analysis, ablation of this region after first identifying the primary motor cortex may be accomplished without risk of permanent loss of motor activity or speech function, despite the initial severe deficits.

KEY WORDS • supplementary motor area • motor cortex • speech • brain tumor

The supplementary motor area (SMA) is a segment of the premotor cortex located on the mesial aspect of the frontal lobe anterior to the leg region of the primary motor cortex. Although the precise function of the SMA is debated, it appears to play a role in the planning or initiation of motor activity, including speech function. There are few studies detailing the effects of surgically induced SMA lesions, and they often fail to clearly correlate clinical deficits with precise anatomical data.

In an attempt to further understand the function of the SMA, we retrospectively analyzed patients who had undergone removal of the dominant hemisphere SMA during tumor resection. This area was localized by using direct stimulation mapping of the primary motor cortex. Following ablation of the SMA, a myriad of reversible signs and symptoms related to movement and speech production developed. The role of the SMA in initiation of motor activity is described based on our experience, and a review of the literature is presented.

Clinical Material and Methods

Six patients with tumors of the dominant hemisphere were included in this analysis. All patients had seizure activity as a presenting symptom, and two had speech arrest associated with their seizures. Three patients were intact neurologically, whereas the other three demonstrated a mild right hemiparesis. One patient with a hemiparesis had a very mild expressive dysphasia. Two patients had previously undergone biopsy at another institution and were subsequently treated with irradiation and chemotherapy.

All patients had preoperative and postoperative evaluation by magnetic resonance (MR) imaging and/or computerized tomography (CT). Predetermined MR imaging landmarks were used to identify the Rolandic cortex, and the tumors were localized to the gyrus directly anterior to the primary motor cortex in the mesial posterior frontal lobe. Four patients had non-contrast-enhancing hypodense lesions on CT scans or gadolinium-enhanced MR images. Patchy enhancement was seen in one case and in another an enhancing cystic component formed part of the mass. In no case did postoperative imaging reveal an underlying etiology for the observed deficits, such as infarct or hemorrhage.

The technique mapping the motor cortex and subcortical motor fibers with the aid of direct stimulation, as used in our patients, has been described previously by Ojemann, et al., and Berger, et al., respectively. Briefly, a bipolar carbon-tipped electrode is used to
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cortex varies between 2 and 16 mA, depending upon
the anesthetic condition of the patient. Subcortical
stimulation is also performed during the resection to
identify the descending motor pathways. In all cases the
motor cortex was stimulated before and after resection
to confirm the integrity of the primary motor cortex.

Illustrative Cases

Case 2

This 32-year-old right-handed man had a 6-year his-
tory of intractable seizures. At another institution, the
patient had undergone CT-guided biopsy of a hypo-
dense nonenhancing posterior frontal lesion abutting
the precentral sulcus (Fig. 1). The pathology was a low-
grade astrocytoma.

Preoperatively, the patient had a mild right hemipa-
resis and normal speech. He subsequently underwent
an “asleep” craniotomy, and intraoperative stimulation
mapping was used to localize the motor cortex. A gross
total resection of the tumor was carried out, up to the
anterior pial bank of the motor cortex. The resection
cavity measured 4.0 cm deep, 3.0 cm lateral from the
falk, and 5.0 cm anteriorly from the motor strip. Sub-
cortical stimulation of the posterior inferior resection
margin resulted in leg motor activity, thus preventing
any further removal of tumor-infiltrated brain posterior
to the resection (Fig. 2).

Fig. 1. Preoperative axial T1-weighted magnetic resonance
image in Case 2 showing a tumor located in the left posterior
mesial frontal lobe.

directly stimulate the exposed cortex to elicit the desired
motor movements. This is routinely done with the
patient awake under local anesthesia or under general
anesthesia (nonparalyzed). A constant-current genera-
tor is used to produce biphasic square-wave pulses with
a frequency of 60 Hz and a pulse duration of 1 ms per
phase. The current necessary to stimulate the motor

Fig. 2. Case 2. Left: Intraoperative brain map with the midline to the right and the anterior aspect
oriented superiorly. Numbers 1 through 6 (circled) represent mapped cortical motor activity with No. 5
representing the thigh and No. 6 representing the foot. Tumor boundaries determined by ultrasound are
indicated by the letters A, B, C, D, and E. Right: Postresection map showing subcortical motor areas for the
knee and foot (Nos. 7 and 8, respectively). The resection bed abuts the motor cortex (Nos. 1 through 5,
indicated by asterisks). Tumor boundaries determined by ultrasound are shown by the letters B, C, and D.
Immediately after the operation, the patient had moderate right upper-extremity paresis and profound lower-extremity paresis that progressed to apparent hemiplegia. An orofacial apraxia was noted that progressed to mutism 1 day postoperatively, although he remained alert and responded appropriately to verbal commands. Three days postoperatively, he began to mimic words and short phrases and, 2 days later, hesitant, spontaneous speech returned. At the time of discharge 12 days postoperatively, his speech was nearly normal with respect to fluency, production, and hesitancy. Five days postoperatively, he regained normal power in the right upper extremity. Right lower-extremity power returned more slowly, with only antigravity quadriceps and iliopsoas function at the time of discharge. Despite the return of normal power, spontaneous movement lagged behind. Even as some spontaneous movement returned, it had a markedly hesitant quality.

Three months postoperatively, the patient had regained normal fluent speech and motor power, without hesitancy or apraxia. Fifteen months following surgery, he is free of recurrent tumor, not receiving further treatment, and has no identifiable neurological deficit other than a weakness of right foot dorsiflexion that is not apparent when he walks.

Case 6

This 37-year-old right-handed woman presented with right upper-extremity focal motor seizures with intermittent speech arrest. Computerized tomography and MR imaging revealed a noncontrast-enhancing lesion in the left posterior-mesial frontal lobe (Fig. 3). The patient underwent an "asleep" craniotomy with motor mapping to identify the motor cortex. Gross total resection was performed, which included the epileptogenic tissue one gyrus anterior to the frontal tumor margin. Overall, the resection extended from the anterior pial bank of the motor cortex forward for a distance of 4 cm, inferiorly to the depth of the cingulate sulcus, and laterally to include the superior frontal gyrus (Fig. 4).

Immediately after the operation, the patient had a right hemiplegia and was mute but followed commands relating to the contralateral extremities. She was able to protrude her tongue symmetrically. One day postoperatively, she spoke in single words and short sentences; spontaneous speech returned 4 days postoperatively. The next day, some voluntary right-handed function returned and, by 8 days after surgery, antigravity arm movements and hip flexion were present. Spontaneous movements were sparse, despite the return of motor power, and were characterized by hesitancy and apraxia. Two weeks following surgery, full arm and hip movement had returned and, at 4 weeks, the patient became ambulatory. She had a residual right foot drop and decreased fine motor control of the right hand with subtle hesitancy and apraxia; however, spontaneous movement had improved. She also had some word-finding difficulties and hesitancy in initiating speech for several weeks after resumption of spontaneous speech.

Six months following surgery, neurological function was normal in all realms of motor and speech activity. The patient received focal radiation treatment for a low-grade, mixed oligoastrocytoma. She remains neurologically intact and free of tumor recurrence 30 months after surgery and radiation therapy.

Results

A schematic diagram of the tumor location and extent of resection in these six patients is shown in Figure 5. Pathological examination revealed four patients with low-grade astrocytoma, one with anaplastic astrocytoma, and one with metastatic breast adenocarcinoma. A gross total or nearly total (90% to 99%) resection was accomplished in all six patients, as documented by the postoperative scans.

Five patients with primary glial tumors became mute within 24 hours of resection without disturbance of sensorium or comprehension. All patients were able to protrude the tongue on command during the postoperative period. Partial speech function returned within 3 to 5 days following surgery, with spontaneous fluent speech present at 4 to 12 days postoperatively. All patients continued to display hesitation and initiation difficulties, which persisted after resumption of spontaneous speech although the severity abated with time. In three patients, transient word-finding difficulty and self-corrected verbal perseveration was documented in addition to a transient dysnomia in two patients and dysgraphia in one individual. All five patients, who were followed at least 1 year after surgery, had no
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Fig. 4. Case 6. Left: Intraoperative brain map oriented with the anterior aspect superiorly and the midline to the right. Motor responses are indicated by Nos. 1 through 6 (circled), No. 4 representing leg and hip movement. The anterior tumor margin (A) was determined by ultrasound. Epileptogenic activity was recorded from the subdural strip electrode (large arrow) shown anterior and medial to A. Right: Postresection map with the resection bed abutting the motor cortex posteriorly (asterisks) and the falk (F) medially. The epileptogenic cortex was resected anterior to the tumor (arrow).

residual deficits. No speech or cognitive deficits were observed in the one patient who underwent resection of a metastasis underlying the SMA without removal of surrounding tissue. Additional findings common to patients with speech deficits were a blunted affect and overt frustration that cleared when normal speech returned.

Motor deficits were more variable in type and course of recovery. Of the five patients with primary tumors, two displayed a contralateral hemiplegia postoperatively and one patient had mixed upper-extremity paresis and lower-extremity paralysis. Two patients, one with normal motor power and the other with a mild diffuse contralateral paresis, displayed severe “neglect”

Fig. 5. Schematic representation of supplementary motor area (SMA) tumor resections in posterior-frontal coronal (a) and sagittal (b) planes in the six patients presented. The SMA is represented by the shaded area.
of the contralateral extremities. One of these was initially thought to be paralyzed but upon more provocative motor testing was found to have antigravity strength in the upper extremity. He was also observed to use the affected extremity with great facility during an automatic bimanual task but could not repeat the movement upon request.

Return of motor function began 1 week postoperatively and either returned to preoperative status or was nearly fully functional within 4 to 8 weeks postoperatively. Despite regaining motor power, there was marked restriction of spontaneous movement which gradually returned over a period of several weeks. Qualitative dysfunction of movement, manifest as hesitancy and apraxia, became apparent at this time. Six months following surgery, all patients had at least regained baseline function with no significant qualitative movement abnormalities. One patient with a mild hemiparesis preoperatively regained normal power except for a mild foot drop that was barely evident with ambulation. The patient whose metastatic lesion was resected without disruption of the SMA cortex had transient worsening of leg weakness but no change in spontaneity or evidence of hesitancy or apraxia.

Postoperatively, all patients exhibited a slight degree of dyspraxia and reduced fine-motor control that was not grossly evident by 1 year following surgery. Two patients with preoperative normal motor function developed transient hyperreflexia with increased tone after surgery. A patient with preoperative paresis developed transient hypertonicity. One patient had decreased contralateral gaze function which resolved shortly after surgery. Both patients who exhibited a contralateral neglect postoperatively progressed from an inability to recognize the right side of the body to profound akinesia prior to resolution of the problem. In addition to deficits concerning initiation-related functions, several patients were unable to inhibit certain inappropriate speech and motor functions. Two patients displayed transient grasping reflexes of the contralateral hand, and one patient exhibited motor perseveration of the upper extremity. These three patients also displayed verbal perseveration.

**Discussion**

**SMA Anatomy and Function**

The territory of the SMA was initially defined by stereotypical behavior elicited during direct electrical stimulation of the cortical surface. Penfield and Welch were the first of several investigators to elaborate these effects, which included complex postural movements, arrest of voluntary movement and speech, sensory phenomenon, and autonomic responses. The anatomical boundaries of the SMA, determined by stimulation mapping, are leg motor representation posteriorly and the cingulate sulcus inferiorly. The lateral and anterior borders are less precise but probably do not extend beyond the superior frontal gyrus either laterally or more than 5 cm anteriorly.

Current opinions regarding the functions of the SMA revolve around its role in the planning and initiation of motor activity, including speech output. Measurements of regional cerebral blood flow (rCBF), which demonstrate bilateral SMA activity during motor tasks (including speech) and activity isolated to the SMA during mental execution of motor tasks, support this role. Anatomical studies show connections between the SMA and all components of the motor system, including the following: bilateral motor cortex, cingulate gyrus, contralateral SMA (which may be the basis for return of function after unilateral lesions), and caudate and spinal cord with basal ganglia input via the thalamus. Unlike the primary motor cortex, no consistent somatotopic organization or cytoarchitectonic specialization exists in the SMA in humans. This lack of defined structure and diffuse interconnection with the motor system appears well suited to the SMA’s putative role in integration and initiation of motor function.

**SMA Ablation in Humans**

Beginning with Penfield and his coworkers, there have been scattered reports in the literature of transient motor and speech deficits after SMA ablation. Most of these studies fail to elaborate surgical, anatomical, or clinical details. Laplane, et al., described stereotactic SMA resections of epileptogenic foci in three patients who displayed global akinesia and speech arrest postoperatively. There was a marked transient reduction in spontaneous speech output and contralateral motor activity which was believed to represent motor neglect. Longer-term sequelae, noted in all three patients, included abnormalities in alternating movements of the hands, with slight motor perseveration in two patients and a grasp reflex in one.

**The SMA Syndrome**

In our patients, following total or nearly total resection of the dominant hemisphere SMA, a characteristic syndrome of reversible contralateral weakness and/or neglect and mutism (without cognitive impairment) emerges. The initial paralysis/paresis or neglect of the contralateral extremities (profound enough in one case to be misinterpreted as paralysis) returns, variably, within weeks to baseline motor power but gives way to a longer period of grossly impaired initiation dysfunction exhibited by a paucity of spontaneous movement and hesitancy in activating volitional movement as well as apraxia. A similar pattern of recovery is demonstrated in speech function. Almost all motor deficits in our patients can be interpreted as varying degrees of severity of initiation dysfunction. However, it is possible that the SMA has other roles in motor function aside from initiation. Three patients were noted to have either transient grasping reflexes or motor perseveration and to display transient verbal perseveration. These deficits may demonstrate the impairment of an additional proposed SMA function, namely, inhibition of
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inappropriate motor activity. The results of a longer-term analysis of targeted arm movements in patients with SMA resections, including some patients in the present study, showed bilateral impairment of movement speed and strategy rather than initiation (ME Anderson, personal communication, 1990).

The role of surgical “shock” as the sole explanation for the deficits we describe seems unlikely for several reasons. The recovery of speech and motor function follow very similar courses, although the resection sites are very distant from primary motor speech areas. The patient with a metastasis resected from the SMA did not display any postoperative evidence of initiation or speech dysfunction although surgical traction was undoubtedly transmitted to the adjacent motor cortex. The integrity of the primary motor cortex was documented by both cortical and, in some cases, subcortical stimulation at the end of each resection and by routine postoperative CT scans to rule out hemorrhage or infarction. Surgical shock may be in part responsible for the transient weakness seen in some patients but it is unlikely to explain changes of initiation function documented long after surgical edema would be present. The variable motor deficits, other than impaired initiation, may reflect resection of lateral premotor or anterior prefrontal areas that were necessary, as dictated by individual tumor topography.

The SMA and Speech Function

The association between the SMA and speech has been known for some time, but its functional role has been unclear. Speech modulation occurs with stimulation of or seizures within, either the dominant or nondominant SMA. During speech, rCBF data have shown simultaneous activation of both SMA’s, with a slight predominance of flow on the left. In the present study, speech deficits primarily reflected disruption of initiation, with mutism followed by nonsynchronous then hesitant speech. However, some deficits, such as word-finding difficulty and dysnomia may represent aphasic changes. Penfield and Roberts considered that “motor” speech was represented bilaterally but “aphasic” changes could only be elicited from the dominant hemisphere SMA. The role of the nondominant SMA in speech production is controversial, with examples of speech dysfunction reported from either hemisphere after SMA lesions. In the experience of one of us (G.A.O.), only rarely are nondominant SMA resections associated with speech dysfunction. Additionally, the extent of resection may influence the severity and character of speech deficits. In the present study, normal speech function was retained in only one case, which involved a limited SMA resection of a metastasis.

Conclusions

With the use of intraoperative cortical and subcortical mapping to identify primary motor cortex and descending motor fibers, large tumor resections of the dominant hemisphere SMA can be performed safely but may be accompanied by a dramatic, yet reversible, syndrome of transient speech and motor deficits termed the “SMA syndrome.” Although many aspects of SMA function remain controversial, awareness of the SMA syndrome may serve to warn patients of severe yet transient postoperative neurological dysfunction after extensive SMA resections. These deficits are related to one of the SMA’s putative functions, namely motor initiation, including speech function. Continued work with brain mapping and ablation studies may help to answer some of the unresolved questions regarding SMA function.

References

17. Laplane D, Talairach J, Meininger V, et al: Clinical consequences of corticotomy involving the supplemen-
18. Larsen B, Skaughe E, Lassen NA: Variations in regional
cortical blood flow in the right and left hemispheres during
Corticospinal neurones of the supplementary motor area
with hemispatial and limb motor neglect. Brain 109:
293–305, 1986
motor area seizures: clinical and electroencephalographic
findings. Neurology 38:1075–1082, 1988
localization in left, dominant hemisphere. An electrical
stimulation mapping investigation in 117 patients. J Neurosurg
71:316–326, 1989
23. Orgogozo JM, Larsen B: Activation of the supplementary
motor area during voluntary movement in man suggests
it works as a supramotor area. Science 206:847–850,
1979
supplementary motor area seizures. Neurology 34:
110–111, 1984
25. Penfield W, Jasper HH: Epilepsy and the Functional
Co, 1954
27. Penfield W, Welch K: The supplementary motor area of
the cerebral cortex: a clinical and experimental study.
Arch Neurol Psychiatry 66:289–317, 1951
motor area and other cortical areas in organization of
voluntary movements in man. J Neurophysiol 43:
118–136, 1980
blood flow changes in cortex and basal ganglia during
voluntary movements in normal human volunteers. J
Neurophysiol 48:467–480, 1982
30. Rubens AB: Aphasia with infarction in the territory of
31. Sandis F: The cyto-myeloarchitecture of the human
frontal lobe and its relation to phylogenetic differentiation
32. Schell GR, Strick PL: The origin of thalamic inputs to
the arcuate premotor and supplementary motor areas. J
Neurosci 4:539–560, 1984
33. Talairach J, Bancaud J: The supplementary motor area
in man (anatomofunctional findings by stereo-electro-
encephalography in epilepsy). Int J Neurol 5:330–347,
1966
34. Van Buren JM, Fedio P: Functional representation on the
medial aspect of the frontal lobes in man. J Neurosurg
44:275–289, 1976
35. Verhaellie M, Heilman KM: Response preparation and
response inhibition after lesions of the medial frontal
and the supplementary motor area. Arch Neurol 43:
787–792, 1986
37. Wiesendanger M: Recent developments in studies of the
supplementary motor area of primates. Rev Physiol
38. Wiesendanger M, Wiesendanger R: The supplementary
motor area in the light of recent investigations. Exp Brain

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