# Transient Neurological Deficit after Therapeutic Embolization of the Arteries Supplying the Medial Wall of the Hemisphere, Including the Supplementary Motor Area

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A 27-year-old, right-handed woman underwent intraoperative embolization for a left parasagittal arteriovenous malformation as part of a two stage plan that included subsequent surgical resection of the arteriovenous malformation. This report describes the neurological syndrome that developed after the first procedure. The relation of these deficits to localization of function within the supplementary motor area are correlated with recent advances in our understanding of this region. (*Neurosurgery* 18:353–356, 1986)

Key words: Anterior cerebral artery syndrome, Basal ganglia, Embolization, Primary motor cortex, Supplementary motor area

### INTRODUCTION

Many recent experimental studies of the supplementary motor area (SMA) have suggested that it has a major role in the cerebral control of motor planning (4, 27, 28, 33, 36). Cerebral blood flow studies in humans have demonstrated selective increase in regional cerebral blood flow in the SMA when complex motor acts are performed or when they are planned without movement (27, 28). Physiological studies in nonhuman primates have demonstrated that SMA neurons become active during the preparation for movement and during the execution of some movements (4, 5, 33, 36). Recent anatomical studies have shown that the SMA has dense somatotopic connections with motor cortex (14, 18, 19, 22, 23). In addition, recent studies have demonstrated that the SMA is the cortical motor area most directly influenced by the basal ganglia (31).

This case report describes the clinical manifestations resulting from an insult to the medial wall of the hemisphere, including the supplementary motor area. These deficits followed a discrete intraoperative embolization of an arteriovenous malformation (AVM) of the medial frontal lobe. The relation of these deficits to localization of function within the SMA will be discussed and correlated with recent advances in our understanding of this region. Pre- and post-embolization arteriograms and CT studies provided accurate topographic localization of the region being described.

## CASE REPORT

AJ is a 27-year-old right-handed woman with a 4-year history of seizures and severe headaches. Complete work-up had previously included a cerebral angiogram that revealed a large left midline hemispheric AVM. The patient had a progressive course of increasing difficulty with seizure control, intermittent weakness of her right arm and leg, and occasional difficulties with speech. The patient's most bothersome complaint, however, was the severe bitemporal throbbing headaches that were associated with these episodes of weakness and speech difficulty.

Results of cerebral angiography demonstrated that the AVM was supplied predominantly by distal branches of the left callosal marginal and the left anterior pericallosal arteries.

Operation was considered because of the worsening of her neurological condition, presumably secondary to steal phenomenon (21). The patient underwent a right parasaggital craniotomy to embolize the anterior vessels of the left hemispheric arteriovenous malformation. The operative exposure included a lateral retraction of the right medial frontal lobe so that the falx cerebri was exposed inferiorly to the superior sagittal sinus. A  $2 \times 5$  cm opening was made in the inferior aspect of the falx to allow access to the medial side of the left frontal lobe. The most distal branch of the left callosal margin artery as well as the main branch of the left pericallosal artery were identified. A cannula was inserted into each of these vessels which supplied the AVM and they were successfully embolized with isobutyl-2-cyanoacrylate (Bucrylate; Ethicon, Inc., Somerville, New Jersey). The embolic material was deposited into the nidus of the malformation under fluoroscopic control. There were no intraoperative complications, and there was no attempt at excision of this arteriovenous malformation. The postoperative plain films, computed tomographic (CT) scans, and angiograms illustrate the results of the procedure (Figs. 1 and 2).

Postoperatively the patient was found to have akinetic mutism that lasted appoximately 4 days. Physical examination on the day after operation showed the patient to be alert and awake, but incapable of spontaneous speech and movement. In addition, she had spasticity of the right lower extremity and a left gaze preference.

The patient's speech and motor patterns began to improve 4 days after operation. She began speaking in response to questions, but her speech was restricted to single words and showed no spontaneity. During this recovery period, the patient had no difficulty with repetition, speech comprehension, articulation, or naming. However on repeated examination, spontaneous speech remained sparse, and she became very frustrated even though her speech disturbance was apparently minimal. Purposeful movement of the upper extremities also first was noted on the 4th postoperative day. At this time, weakness of her right arm was noticed. Neurological examination demonstrated hypereflexia of the right leg, bilateral Babinski reflex, resolution of the gaze preference, and a normal sensory examination.

Speech and movement patterns gradually improved over the next 3 weeks. Speech, nonfluent, slow, and deliberate on

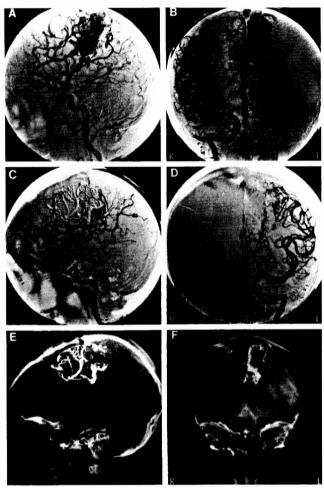


FIG. 1. A and B, left common carotid, lateral view, (A) and right common carotid, frontal view, (B), injections demonstrate a left parasagittal frontal and frontoparietal AVM predominantly supplied by the middle and posterior internal frontal and paracentral arteries. C and D, cerebral arteriograms performed after intraoperative embolization with tantalum-impregnated Bucrylate show significant obliteration of the AVM. A deeply situated residual AVM is still evident. E and F, plain films illustrate the actual distribution of the tantalum-impregnated Bucrylate occluding the arterial feeders and nidus of the AVM.

day four, rapidly improved to its preoperative state. The weakness of her right arm, the spasticity of her right leg, and her gaze preference completely resolved. Weakness persisted in her right leg. Although the patient could function well when the right or left hand was used independently, she was not able to coordinate motor functions when using both hands simultaneously. Simple tasks involving bilateral manual coordination, such as buttoning her shirt or transferring a cup from one hand to another were performed very poorly. Simultaneous pronation and supination of her forearms was very difficult, whereas this alternating movement pattern was executed normally when each arm was tested independently. This bimanual disturbance of alternating movements did not improve during subsequent examinations.

# DISCUSSION

Intraoperative embolization has become an integral part of the management of AVMs. A major complication of this procedure is infarction of potentially normal neural tissue in and around the AVM (21, 35). Because embolization occludes the major feeding arteries of the AVM and the smaller distal branches that supply more distal neural tissue, there may be neurological deficit from infarction of neural elements. This case report emphasizes the transient neurological deficits, particularly difficulties with speech and the lasting difficulty with bimanual movements, that resulted from embolization of an AVM that involved the left supplementary motor area.

The neurological findings resulting from tumors and infarcts affecting the medial aspect of the frontal lobe, including the SMA, have been previously reported (1, 6, 10, 11, 13, 17, 25, 26, 30, 32, 39), but often go unrecognized in common neurological practice (12). This cortical region is commonly involved with lesions, such as parasagittal meningiomas, anterior cerebral artery vasospasm, and infarctions. Most lesions in this area produce what has been termed the anterior cerebral artery syndrome. This syndrome consists of contralateral paralysis of the lower extremity, forced grasping of the ipsilateral hand, a transient, but profound, expressive aphasia, and an ideomotor apraxia of the left arm regardless of the side of the lesion (30). However, the correlation of these neurological signs with anatomical structures has been difficult because branches of the anterior cerebral artery supply major anatomic structures, including the anterior 4/5 of the corpus callosum, the anterior limb of the internal capsule, the SMA, and the medial aspect of the sensorimotor cortex. Many manifestations of this motor syndrome have frequently been attributed to lateral hemispheric and callosal structures. However, this report, together with other recent reports (1, 11, 15, 29), indicates that the anterior cerebral artery syndrome may be largely the result of damage to the SMA.

This patient displayed a striking profile of the anterior cerebral artery syndrome after selective embolization of branches of the left callosal marginal and pericallosal arteries (Fig. 1C, D). The lesion included most of the SMA and some

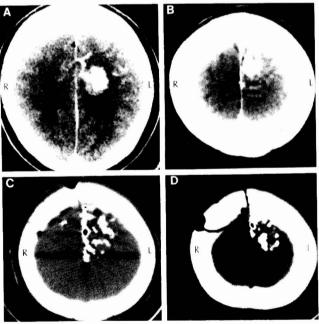


FIG. 2. A and B, intravenously enhanced computed tomographic scans demonstrate the cortical and subcortical location of the left parasagittal AVM. C and D, plain computed tomographic study defines the extent of the embolized AVM.

of the "leg representation" of primary motor cortex. Lateral hemispheric structures and regions supplied by Heubner's artery were not involved. Neurological deficit from retraction of the right medial hemisphere cannot be excluded; however, this was not thought to have played a major role in this patient.

Preoperatively, the language disturbance in this patient consisted of multiple episodes of speech arrest attributed to either focal seizures (24) or local steal phenomena (21). These language disturbances consisted of mutism lasting for 4 days, initial severe impairment of spontaneous speech, rapid recovery over 2 to 4 weeks from well-articulated but slow speech to nearly normal speech, and a tendency to become very frustrated when using complex language. Throughout this period, comprehension of simple verbal and written material was normal.

Rubens states that the aphasia associated with the anterior cerebral syndromes can be distinguished from classic Broca's aphasia and attributes the speech disturbance to damage of the SMA (30). The disorder of speech observed in SMA lesions and in the anterior cerebral artery syndrome is transient mutism with rapid improvement of the expressive aphasia. The speech disturbance associated with the anterior cerebral syndrome has been attributed to the undercutting of Broca's area by a white matter lesion in the distribution of Heubner's artery (30). However, even though there are similarities between the speech deficits associated with anterior cerebral artery syndromes and Broca's aphasia, patients with Broca's aphasia frequently have difficulty with articulation and are seldom mute. The present case supports the concept that a type of expressive aphasia does occur as a result of lesions distant from Heubner's artery and the perisylvian speech areas

Immediately postoperatively the patient had a transient period of marked increase in tone of the right lower extremity while tone remained normal in the right upper extremity. This phenomenon of spasticity in this context has been the subject of many experimental studies and is beyond the scope of the present discussion (7–9, 34, 37, 38). The spasticity of the right leg may have resulted from involvement of the "leg representation" of both the primary motor area and SMA (Fig. 1C, D). The residual right leg weakness can be explained by infarction of the leg representation of primary motor cortex. The absence of right arm spasticity may be related to the sparing of the "arm representation" of primary motor cortex, which is supplied by the middle cerebral artery (Fig. 1C, D).

This patient also demonstrated global akinesia for 4 days, transient weakness of the right upper extremity for 2 weeks, permanent weakness of the right lower extremity, and a residual difficulty with complex simultaneous movements of both hands. The transient motor disturbances have been frequently reported after SMA lesions and anterior cerebral artery syndromes (5, 10, 11, 15, 25, 29). The bimanual deficit persisted even though there was no weakness demonstrated in the upper extremities. A similar movement disorder was demonstrated experimentally in monkeys after SMA ablations (3) and in humans (15). The bimanual difficulty with coordination is a feature unique to SMA lesions and may be related to extensive bilateral connections of the SMA with the motor cortex (18, 19, 22, 23) and the spinal cord (2, 20) (Schell G, Strick PL: Unpublished observations).

The concept that the SMA is the cortical area most influenced by the basal ganglia has significant functional implications relating to the study of motor control in humans (31). Marsden has suggested that the major function of the basal

ganglia is to run a sequence of motor programs to achieve a motor plan (16). Similarly, cerebral blood flow studies in man have demonstrated selected increase in regional blood flow in the SMA in man during movement sequence tasks (27, 28). The anatomical relationship between the basal ganglia and the SMA (31) suggests that certain aspects of movement disorders associated with basal ganglia disease may be mediated by the SMA. The marked akinesia and associated mutism observed in this patient after a lesion involving the SMA might be compared with the akinesia of Parkinson's disease. Further observations on how lesions of the SMA might affect the movement disorders related to basal ganglion disease could add further understanding of the pathophysiology of these motor systems.

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