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Anomia produced by direct cortical stimulation of the pre-supplementary motor area in a patient undergoing preoperative language mapping

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ABSTRACT – There is sparse data on the analysis of supplementary motor area in language function using direct cortical stimulation of the supplementary motor area. Here, we report a patient who experienced isolated anomia during stimulation of the anterior supplementary motor area and discuss the role of the supplementary motor area in speech production. The role of the pre-supplementary motor area in word selection, observed in fMRI studies, can be confirmed by direct cortical stimulation.

Key words: anomia, cortical stimulation, supplementary motor area, language mapping, speech production, MRI

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The role of the supplementary motor area (SMA) in speech function has been investigated mostly using functional MRI studies. Results indicate a functional compartmentalisation of SMA into pre-SMA, which is associated with higher-level processing and initiation of language, and the more caudally located SMA-proper, involved in speech articulation (Alario *et al*., 2006).

However, there is sparse data on the analysis of SMA in language function using direct cortical stimulation of the SMA. Here, we report a patient who experienced isolated anomia during stimulation of the anterior SMA and discuss the role of SMA in speech production.

Case study

We report a 44-year-old, right-handed female clerk who underwent presurgical EEG-video monitoring for drug-resistant focal epilepsy due to cortical dysplasia of the left mesial frontal cortex. Seizures had started at the age of 5 and occurred up to seven times a

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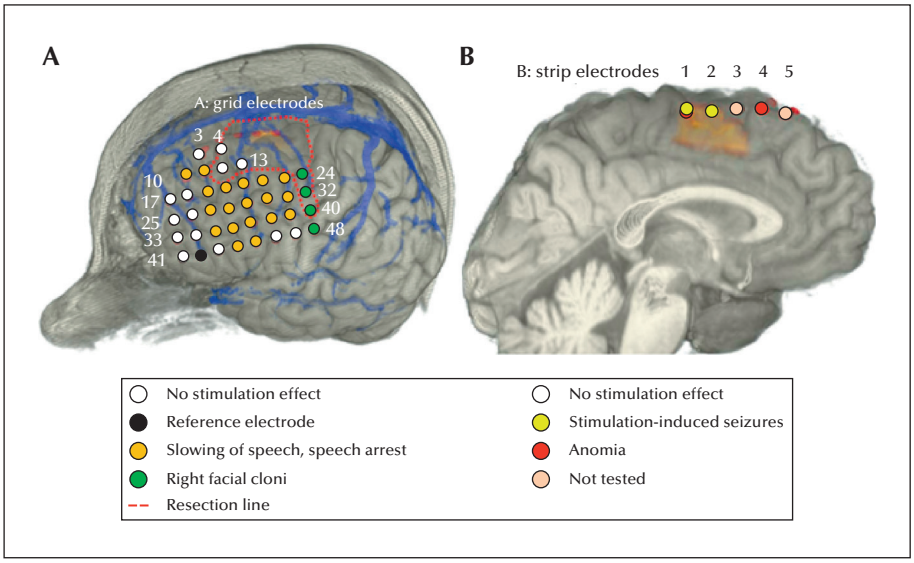

Figure 1. Subdural grip (A) and strip (B) electrodes over the lateral frontal lobe. The cortical dysplasia is marked in orange and the reference electrode (A41) in black. Electrical stimulation induced anomia at electrode (B4) marked in red, orofacial cloni at electrodes marked in green, and speech arrest at electrodes marked in orange. Seizures were elicited by stimulation of electrodes B1 and B2, marked in yellow (B). The resection line is indicated in red.

week. There was no history of febrile seizures. Clinically, seizure semiology consisted of bilateral ocular paraesthesia and blepharospasm, followed by bilateral tonic posture and, at times, cloni of the right face and arm.

Subdural grid and strip electrodes were implanted over the left mesial and lateral frontal cortex to delineate the epileptogenic zone and identify eloquent cortex (*figures 1 and 2*). Interictal activity, which consisted of sharp waves and spikes, was mainly recorded at electrodes B1, B2, A32 and A39. Seizure onset was seen over electrodes B1-3, A32, A39 and A40. Electrode-MRI coregistration was performed using the AMIRA software; first, electrodes were segmented from postoperative CT images and were then coregistered to preoperative MRI scans.

Cortical stimulation was performed with bipolar stimuli of 300-μs duration at a frequency of 50 Hz and stimulus train duration of 10 s with adjacent electrodes, as well as in a referential manner with electrode A41, distant from the epileptogenic zone, chosen as reference electrode.

Only stimulation of electrode B4 elicited pure anomia at an impulse amplitude of 9 mA. The patient was unable to name objects, but could pronounce a carrier phrase, read, and count. To differentiate between speech arrest resulting from a motor inhibitory response or motor activation of orofacial muscles and true motor aphasia, the patient was asked to name objects together with a carrier phrase ("This is a. . ."). In the case of a motor response, neither the carrier phrase nor the object name will be articulated correctly. Motor aphasia will not impair the carrier phrase, but inhibit naming (Ojemann and Mateer, 1979). There were no afterdischarges.

Stimulation of electrodes A10, A11, A19-23, and A 27-31 produced speech arrest at 7 mA with local afterdischarges; stimulation of electrodes A24, A32 and A40 yielded a tonic motor response in the right orofacial muscles at 5 mA, also with local afterdischarges. Stimulation of electrodes B1 and B2 elicited generalized convulsions. Therefore, electrodes B3 and B5 were not stimulated.

Resection of the epileptogenic zone was performed in consideration of the stimulation results and detection of the epileptogenic zone (see resection line in *figure 1A* and postoperative MRI in *figure 2*). Primary motor areas were spared during the resection which therefore did not cover the entire cortical dysplasia. In particular, the anterior part of the cortical dysplasia remained.

Postoperatively, the patient suffered from mild impairment of speech initiation that completely resolved within two weeks. There were no motor deficits. Seizure frequency was reduced to twice a week.

Discussion

We identified isolated anomia upon direct cortical stimulation of the SMA in the mesial aspect of the superior frontal gyrus. Few studies report on stimulation in this region, most likely due to the difficulty to place electrodes in the mesial aspect of the frontal

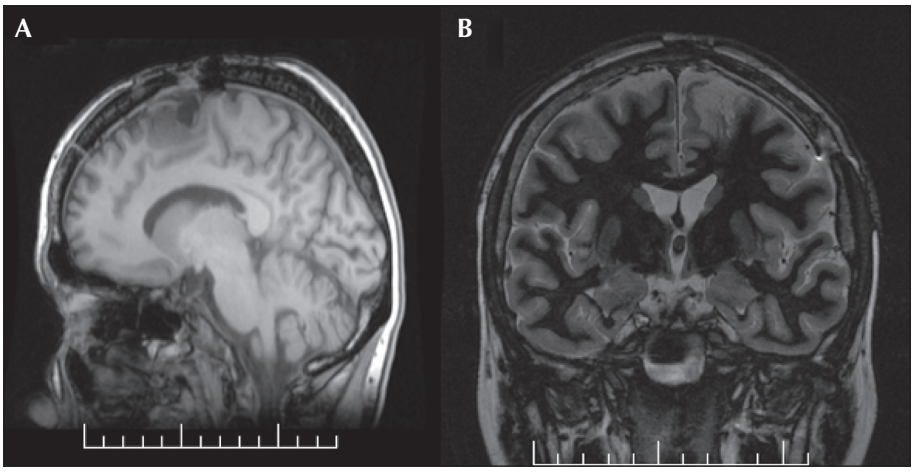

Figure 2. Postoperative MRI on sagittal (A) and frontal (B) slices.

lobe. Previous studies applying direct cortical stimulation to SMA elicited "vocalisation", "complete speech arrest", and "speech slowing or hesitancy" (Fried *et al*., 1991; Lim *et al*., 1996). Stimulation-induced speech disorders were mostly accompanied by orofacial cloni, interfering with testing of speech. Thus, a clear distinction between speech deficit due to orofacial motor response and pure aphasia is hindered. In our case, however, speech arrest due to inhibitory motor effects or motor activation of facial muscles and comprehension deficit could be ruled out by the testing protocol used.

This finding reflects the functional subdivision of SMA previously seen in an fMRI study (Alario *et al*., 2006); anomia is due to an isolated deficit in lexical selection, without affecting motor speech functions. This explains why our patient was able to talk and read during stimulation, but was unable to name objects.

The anatomical region termed "SMA" is composed of two functionally distinct compartments: the rostrally located pre-SMA is activated by cognitive and planning tasks, whereas the caudal SMA-proper is associated with motor execution (Alario *et al*., 2006). Although originally conceived for motor tasks, this functional compartmentalisation of SMA also seems to apply to language tasks. Thus, pre-SMA was activated during word selection, an associative task, whereas SMA-proper was activated during word articulation, a pure motor action. Unfortunately, we could not apply direct cortical stimulation to SMA-proper owing to the high risk of seizure induction by stimulation. Therefore, we could not assess the function of SMA-proper with direct cortical stimulation.

The boundary line between pre-SMA and SMA-proper is generally set as the vertical through the anterior commissure (Zilles *et al*., 1996). This functional subdivision into an associative pre-SMA and an executive SMA-proper is reflected by the connectivity profile both regions display. SMA-proper displays stronger connections to motor and premotor regions involved in executive functions, whereas pre-SMA is more strongly connected to the superior frontal cortex (Johansen-Berg *et al*., 2004). In our case, electrode B4 is situated within the dorsal part of pre-SMA. Our stimulation result of an associative dysfunction within pre-SMA matches these findings. We did not detect any hints of a functional reorganisation of language areas, which might have been assumed regarding the early seizure onset age and the presence of focal cortical dysplasia.

After mesial frontal resections, patients may experience speech disorders ranging from reduced spontaneous speech to complete mutism (Krainik *et al*., 2003). Postoperative speech deficit occurred after resection of at least 16% of SMA volume activated in the language-dominant hemisphere during verbal fluency tasks (Krainik *et al*., 2003). However, it did not correlate with the volume of tissue resection outside SMA, thus highlighting the role of SMA in speech. Speech deficits after mesial frontal resections usually remain transient and recover within weeks or months, as in our patient (Peraud *et al*., 2002).

To conclude, we were able to confirm the role of pre-SMA in word selection by direct cortical stimulation. This finding is congruent with a previous fMRI study detecting language initiation over pre-SMA. Postoperative recovery of language function is most likely due to the fact that SMA plays an accessory, instead of a central, role within language networks.

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