Letter to the Editor

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Letter by Terao et al Regarding Article, "Damage to the Left Precentral Gyrus Is Associated With Apraxia of Speech in Acute Stroke"

To the Editor:

We read with great interest the article by Itabashi et al[1] recently published in *Stroke*. Using voxel-based lesion-symptom mapping analysis on magnetic resonance imagings of a large number of patients, the authors clearly demonstrated that a restricted area in the left motor cortex is associated with the presentation of apraxia of speech, notably even with that without aphasia. We have previously reported that lesions in almost the same region in the left motor cortex can cause aphemia, but without overt signs of aphasia, suggesting this motor region to be essential for speech articulation.[2] Some authors associated the same region with the foreign accent syndrome. Together, it is plausible that this motor area plays an important role in motor speech production.[1]

Located in the posterior aspect of the motor cortex, the region may reside in primary motor area (area 4) directly related to motor output, more precisely in the face motor representation (face-M1), rather than in lower motor areas usually implicated in tongue and pharyngeal movements. Indeed, in many of the reports, facial palsy accompanies the impairment in speech motor production. In humans and primates, regions within the primary motor cortex where intracortical microstimulation evokes movements at the angle of the mouth are known to play important roles not only in trained orofacial movements but also in the control of tongue and jaw movements necessary for speech production. Thus, this region may serve as a bottle-neck for speech motor output.[2] Consistently, single pulse transcranial magnetic stimulation over the presumed face-M1, either of the dominant or nondominant hemisphere or both, can delay or even temporarily abolish vocalization by reducing speech motor output.[3] Thus, theoretically, lesions either in the right or in the left face-M1 can cause disruption of commands for speech motor output, in line with the bilateral motor cortical innervation to the face, pharynx, and laryngeal muscles.

In view of this, an important aspect of the motor cortical region is its laterality. Although Itabashi et al only studied isolated nonlacunar infarcts in the left middle cerebral artery territory, most cases of aphemia were caused by left hemispheric lesions.[2] How does this laterality arise? Lotze et al reported that articulating /ka/ produces a largely asymmetrical activation centered around the lip area of the motor cortex in the left hemisphere and the entire tongue motor representation in the right hemisphere.[4] During the production of more complex polysyllables, however, activation gets more focused to a region near the left face-M1. Therefore, although utterance of simple syllables engages motor regions of both hemispheres, complex articulation may involve the left hemisphere predominantly. Additionally, cortico-cortical fibers connecting face-M1 with the lower premotor areas including Broca's area may also be important for complex articulatory control, but even here, face-M1 may serve as the final output stage for speech.[2] Although apraxia of speech is defined in cognitive terms as impairment in the translation of phonological representations into specifications for articulation, analysis of the precise motor impairment required for phonological articulation may provide further insights into the different degrees of hemispheric lateralization.[5] Studying patients with infarction in the right middle cerebral artery territory would also help to clarify the issue.

Disclosures

None.

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(*Stroke*. 2016;47:e74. DOI: 10.1161/STROKEAHA.116.012755.)

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Stroke. 2016;47:e74; originally published online March 10, 2016;
doi: 10.1161/STROKEAHA.116.012755
Stroke is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231

Print ISSN: 0039-2499. Online ISSN: 1524-4628