Transient Hemiglossal Denervation during Acute Internal Capsule Infarct in the Setting of Dysarthria—Clumsy Hand Syndrome

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While permanent hypoglossal denervation is well known to occur subsequent to lower motor neuron injury,1 hypoglossal palsy due to supranuclear infarction has rarely been reported.2-7 We report a case of transient reversible hemiglos- sal denervation edema on MR imaging, contralateral to acute internal capsule infarction in the clinical setting of DCHS. A neuroanatomic basis is discussed.

Case Report
A 73-year-old man with chronic hypertension presented with acute dysarthria and right arm weakness. Physical examination also revealed right facial droop and right arm incoordination. The tongue and uvula were midline without fasciculations or atrophy. The clinical diagnosis was DCHS. MR imaging performed at 1.5T revealed a focal area of restricted diffusion at the posterior limb of the left internal capsule, indicating acute infarction (Fig 1A). Additionally, the right hemitongue was markedly diffusely swollen and hyperintense on T2-weighted imaging with right-sided posterior prolapse into the oropharynx (Fig 1B). Fatty atrophy was not identified on T1-weighted images. The brain stem was normal.

Repeat MR imaging with gadolinium performed 2 days later to assess possible tongue mass revealed persistence of restricted diffusion in the posterior limb of the left internal capsule but with resolved right hemiglossal swelling and T2 hyperintensity and resolved posterior prolapse (Fig 2). There was no abnormal tongue enhancement or brain stem abnormality.

After 1 year, the patient had persistent ataxic right hemiparesis with spasticity. The tongue was midline with spasticity.

Discussion
Hypoglossal nerve dysfunction, often with tongue atrophy, is well known to occur secondary to lower motor neuron injury to the nerve or ganglion, with causes including brain stem infarction, nerve invasion or compression by tumor or intern- nal carotid artery dissection, and following injury during carotid endarterectomy or radical neck dissection surgery. MR imaging of acute hypoglossal denervation has been shown to produce edemalike hyperintensity on T2-weighted images, which may rapidly progress to fatty atrophy.1 Intramuscular T2 hyperintensity is commonly seen in the setting of lower motor neuron injury, may be detected as early as 24 hours following injury,8 and is thought to represent muscular edema from acute denervation due to fluid expansion of the extracel- lular space.9 Denervation has been shown to depolarize the resting potential of muscle cell membranes within 2 hours following injury due to inhibition of sodium-potassium pump function, thereby allowing increased membrane permeability of sodium and water.10-12 Muscle fiber is dependent on motor neuron integrity for maintenance of normal function and structure, mediated by sustaining action of electric impulse stimulation.11 As long as atrophy has not occurred, denerva- tion or partial denervation is reversible if neuromuscular con- nections are re-established, resulting in recovery of membrane potential.11

Corticolingual tract fibers (also referred to as corticohypoglos- sal tract or corticonuclear tract) extend from the precentral gyri through the corona radiata, internal capsules, and cerebral peduncles to the pontomedullary junction where some fibers decussate.6 Isolated dysarthria due to lacunar or cortical infarction is rare and is thought to be due to cortico- lingual tract dysfunction.2,7 DCHS has been reported to occur in 1.6% of patients with stroke and in 6% of those with lacunar infarcts.2 Commonly, supranuclear hypoglossal palsy is associated with ipsilesional arm weakness and supranuclear facial palsy, dysarthria, and dysphagia, presumably due to the close anatomic relationship of the respective motor pathways.6,7 While the hypoglossal nerve is a lower motor neuron, crossed and uncrossed upper motor neuron corticolingual fibers ex- tend from both hemispheres to the hypoglossal nuclei.5 Following unilateral infarction, TMS of the affected hemisphere fails to produce an ipsilateral or contralateral lingual response, whereas TMS of the unaffected hemisphere produces a bilat- eral lingual response, even in the setting of unilateral lingual paralysis.5 Also, unilateral infarction inconsistently results in lingual paralysis and is presumably dependent on the ability of the unaffected hemisphere to activate uncrossed corticolin-
Corticofacial tracts are primarily contralateral, without bilateral uncrossed fibers, so that corticofacial innervation most often results in facial paresis.7

Unilateral findings in this case support the existence of predominant contralateral innervation from the motor cortex to the hypoglossal nucleus. Lack of tongue deviation on physical examination in this case could be explained by transient or incomplete denervation of the tongue that was in the process of recovering, preserved crossed corticofacial projections during the acute phase of the infarct.5,6

Conclusions
Hemiglossal denervation is not always due to lower motor neuron injury, can occur with supranuclear infarction, and can be transient and reversible. Awareness of the existence of individually variable bilateral crossed and uncrossed corticofacial projections facilitates a neuroanatomic explanation for these findings. In the future, one could postulate a potential role for diffusion tensor fiber tractography and functional MR imaging in the assessment of normal and impaired corticofacial pathways and lingual motor response.

References
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Fig 2. Axial T2-weighted image obtained 2 days later shows resolved right hemiglossal swelling and edema and resolved posterior prolapse.