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Figure. (A) T2-weighted MRI at initial presentation shows an extensive brainstem low signal abnormality consistent with demyelination. Follow-up image 8 months later (B) shows a small residual area of increased T2 signal at the “sneeze center” only (arrow). Images are displayed according to anatomic, rather than radiologic, convention (dorsal aspect uppermost). (C) Diagram demonstrates the location of the lesion in the “sneeze center” in the rostral dorsolateral medulla (right) and the approximate positions of major brainstem nuclei and tracts (left). ICP = inferior cerebellar peduncle, ML = medial lemniscus, N = nucleus ambiguus, P = pyramid, S = solitary nucleus and tract, SL = spinal lemniscus, T = trigeminal tract and nucleus (adjacent), VN = vestibular nuclei, X = dorsal vagal nucleus, XII = hypoglossal nucleus.

Localization of the “sneeze center”

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The existence of distinct sneeze-evoking centers in the brainstem has been demonstrated in cats, represented bilaterally along the ventromedial spinal trigeminal nuclei and in the adjacent pontomedullary lateral reticular formation.¹ In humans, unilateral damage to the sneeze center in cases of lateral medullary syndrome has resulted in inability to sneeze.² The case presented here demonstrates the localization of the sneeze center in humans with more precision.

A 23-year-old woman presented with 10 days of progressive numbness of the face and arms, diplopia, oscillopsia, and dysphagia. She reported an inability to sneeze or yawn and meals were followed by 5 minutes of hiccups. She had a history of venous thrombosis and a family history of systemic lupus erythematosus (SLE). General examination was remarkable for malar rash. On neurologic examination there was upbeat nystagmus but normal range of eye movement. Left facial sensation was reduced. Facial move-

ment and hearing were normal. Palatal sensation was intact but movement was absent. Cough, swallowing, and tongue movements were impaired. Touch and pin sensation were reduced in the arms but there were no other long-tract signs. MRI revealed evidence of extensive brainstem demyelination, but no other abnormalities (figure). The diagnosis of SLE was supported by positive ANA and anti-Ro antibodies. CSF was acellular; oligoclonal bands were not present. She was treated with IV methylprednisolone, followed by oral prednisone. Eight months later her only symptom was persistent inability to sneeze. Nasal irritation resulted in a strong desire to sneeze and she was able to mimic a sneeze voluntarily, but without relief of symptoms. The examination was otherwise normal. Repeat MRI (see the figure) demonstrated a small residual abnormality in the rostral dorsolateral medulla only, where the “sneeze center” has been predicted.^{1,2}

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