ORIGINAL CONTRIBUTION

Cerebellar Speech Representation

Lesion Topography in Dysarthria as Derived From Cerebellar Ischemia and Functional Magnetic Resonance Imaging

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Background: Lesion topography and the pathophysiological background of dysarthria due to focal cerebellar lesions have not yet been fully clarified.

Objectives: To investigate the lesion topography of dysarthria due to cerebellar ischemia and evaluate brainstem functions.

Design: Case studies.

Patients: Eighteen right-handed patients with sudden-onset dysarthria and cerebellar ischemia with and without brainstem involvement and 19 healthy, right-handed, monolingual, German-speaking volunteers.

Methods: In patients, we used multimodal electrophysiologic techniques to investigate brainstem functions. Functional magnetic resonance imaging (MRI) was performed in the 19 healthy volunteers. Activation tasks consisted of repetitive vertical silent movements of the tongue and lips at a self-paced rhythm.

Results: Cerebellar lesions and additional signs of brainstem involvement were observed in 11 patients with posterior inferior cerebellar artery, anterior inferior cerebellar artery, and superior cerebellar artery infarctions, respectively. In all other patients with isolated cerebellar infarction (n=7), only the superior cerebellar artery territory (6 right-sided, 1 left-sided) was affected, and the common lesion site was the rostral paravermal region of the anterior lobe. Functional MRI in healthy volunteers indicated that the cerebellar representation of the tongue and orofacial muscles corresponds to that of the area involved in patients with cerebellar dysarthria.

Conclusions: The results of this study demonstrate that articulatory movements of the tongue and orofacial muscles are involved in the activation of the rostral paravermal area of the anterior lobe. This location corresponds to the area involved in cerebellar ischemia in patients with dysarthria. Lesions in the upper paravermal area of the right cerebellar hemisphere, the site of coordination of articulatory movements of the tongue and orofacial muscles, may lead to the development of dysarthria that is unrelated to (often concomitant) brainstem infarctions.

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DYSARTHRIA IS a frequent sign in cerebral ischemia, ranging from 8%[1] to 12.4%[2] in large unselected stroke series. However, the lesion topography and the pathophysiological background of dysarthria due to focal cerebellar lesions have not yet been fully clarified. In the most extensive clinical study, which included 122 patients, most of whom had cerebellar tumors, Lechtenberg and Gilman[3] observed that impaired speech function was commonly associated with damage to the superior portion of the left cerebellar hemisphere. Subsequent studies demonstrated that dysarthria due to cerebellar stroke also occurred with right unilateral and bilateral paravermal lesions of the anterior rostral cerebellum in the territory of the superior cerebellar artery (SCA).[4-8] Other researchers observed involvement of the territories of the posterior inferior cerebellar artery (PICA)[9-12] and the anterior inferior cerebellar artery (AICA).[13] However, due to the vascularization, the brainstem is frequently affected in cerebellar infarction. The finding that brainstem lesions may also be responsible for the development of dysarthria[14-16] suggests that dysarthria might be due to the cerebellar infarct, brainstem involvement, or both. Dysarthria due to extracerebellar stroke is frequently associated with corticobulbar tract involvement of the tongue and orofacial muscles.[15,17-20] In the present study, we investigated the lesion topography of dysarthria due to cerebellar ischemia. Brainstem function was evaluated both clinically and with multimodal

electrophysiologic techniques. Functional magnetic resonance imaging (fMRI) was used to investigate the cerebellar representation of the tongue and orofacial muscles in healthy volunteers.

METHODS

CEREBELLAR INFARCTION

Eighteen right-handed patients (according to the Edinburgh Inventory[21]) with sudden onset of dysarthria and cerebellar ischemia with and without brainstem involvement demonstrated by computed tomography (n=18) and MRI (n=14) were studied within the first week after the onset of symptoms. Brain MRIs were obtained to identify the acute brainstem infarction in each patient within the first 2 weeks according to a standard protocol: (1) T2-weighted echo planar imaging and echo planar imaging diffusion-weighted imaging within 24 hours after onset of symptoms (1.5-T Magnetom; Siemens Vision, Erlangen, Germany) (repetition time, 4000 milliseconds; echo time, 103 milliseconds; gradient strength, 1150 s/mm^2; scanning time per slice, 250 milliseconds); and (2) high-resolution T2-weighted imaging (slice thickness, 3 mm) in the axial and sagittal planes and T1-weighted imaging before and after intravenous administration of the contrast medium. Patients with a previous history of cerebral ischemia, transient ischemic attack, multiple infarctions, vascular encephalopathy, and space-occupying infarctions were excluded from the study. Dysarthria was diagnosed on the basis of auditory-perceptual presentation and confirmed by 2 experienced speech therapists. Speech function was assessed using a neurophonetic test battery (modified by Ziegler et al[22]). Articulation was evaluated meticulously on the basis of various samples, including spontaneous speech, repetition of sentences and words, reading of a short story, and rapid iteration of syllables (/pa/, /ta/, /ka/). The examination of laryngeal function included laryngoscopy, stroboscopy, and perceptual examination of voice quality, voice stability, pitch, and loudness. Sustained realization of vowels and fricatives and repetition of sentences of increasing length were used to obtain information on respiratory support.

Brainstem functions were investigated in the awake patient with brainstem auditory evoked potentials (BAEPs), blink reflex (BlinkR), masseter reflex (MassR), somatosensory evoked potentials (SEPs), and transcranial magnetic evoked potentials (MEPs) of the orofacial muscles and tongue (transcranial magnetic stimulation). All electrophysiologic examinations were performed according to International Federation of Clinical Neurophysiology recommendations.[23] Normal values have been published previously by our group (BAEPs,[24] BlinkR,[25] MassR,[25] SEPs,[26] and MEPs[15,17]).

fMRI STUDIES

Nineteen healthy, right-handed, monolingual, German-speaking volunteers participated in the present study (15 men and 4 women; age range, 24-45 years). Handedness was assessed with the Edinburgh Inventory.[21] Activation tasks included repetitive vertical silent movements of the tongue (/la/) and lips (/pa/) at a self-paced rhythm. All participants were instructed to move the tongue and lips as rapidly and accurately as possible in performing the activation tasks and to refrain from verbal thinking during rest periods. To minimize movement artifacts, the head of the participant was immobilized using a tightly adjusted vacuum head holder or adhesive band strips.

All studies were conducted with a 1.5-T tomograph (Magnetom; Siemens Vision). The fMRI data were acquired across the entire brain (24 slices, 5 mm thickness, no gap) by echo planar imaging (repetition time, 6000 milliseconds; echo time, 66 milliseconds; flip angle, 90°; matrix, 128×128; field of vision, 230 mm). The studies were performed in 8 successive groups alternately during rest and task administration. Postprocessing, including realignment of functional images, coregistration with anatomy, and statistical analysis were performed with Statistical Parametric Mapping (SPM96; Wellcome Department of Cognitive Neurology, London, England; available at http://www.fil.ion.ucl.ac.uk/spm/). The thresholds for activation were set at $P<.001$ ($z>3.09$) for voxel level and at $P<.05$ for cluster level (>9 voxels). T1-weighted images (repetition time, 576 milliseconds; echo time, 12 milliseconds; flip angle, 60°; field of vision, 230 mm) were acquired at the same anatomic level to obtain an anatomic reference.

Cerebellar activation was assessed only in participants who showed unequivocal bilateral activation of both primary motor cortices to ensure correct performance of the tongue and lip movement paradigms and to exclude patients with movement artifacts. All participants gave their informed written consent, and the study was approved by the local ethics committee (State Medical Council, Rhineland-Palatinate, Germany).

RESULTS

CEREBELLAR INFARCTION

Fourteen patients had cerebellar infarction in a single cerebellar arterial territory (SCA, n=8; PICA, n=6). In 4 additional patients, the infarction affected 2 territories (PICA and AICA, n=3; PICA and SCA, n=1). The infarction area was unilateral in 16 patients (13 right-sided, 3 left-sided), and involvement of both cerebellar hemispheres was identified in 2 cases.

Additional brainstem lesions detected by clinical, electrophysiologic, and MRI findings were present in 11 patients (**Table**; patients 8-18). These findings were associated with PICA, AICA, and SCA infarctions. The SCA territory (6 right-sided, 1 left-sided) was affected in all patients with isolated cerebellar infarction (n=7) (Table). The rostral paravermal region of the anterior lobe was the most common lesion site of isolated cerebellar infarctions (**Figure 1** and **Figure 2**).

No difference was detected between dysarthria in the patient group with isolated cerebellar infarction (Table, patients 1-7) and the group with cerebellar infarction and brainstem involvement (Table, patients 8-18); in both groups, dysarthria was characterized by reduced articulatory precision, resulting in slurred pronunciation of single consonants. Articulatory deficits were inconsistently present during speech, giving rise to the assumption of irregular articulatory breakdown.[27] Articulatory movements and speech rate were mildly slowed at a mean syllable repetition rate of 4.3 syllables per second (normal, 6 syllables per second). Phonatory disturbances included fluctuations in pitch and loudness, although speech was not scanning or explosive in any patient. The degree of dysarthria ranged from mild to moderate; no patient had unintelligible speech. The voice was normal in most patients.

fMRI STUDIES

Fourteen of 19 participants included in the study showed bilateral activation of the primary motor cortex during silent tongue movements, with additional bilateral acti-

Clinical Findings of 18 Study Patients

Patient No.	Imaging	Localizaton	Clinical Findings	BlinkR	MassR	BAEP	SEP	MEPVII	MEPXII
			Cerebellar Damage Only (n = 7)						
1	MRI	R SCA	Stance and gait ataxia	N	N	N	N	N	N
2	MRI	R SCA	R upper limb ataxia	N	N	N	N	N	N
3	MRI	R SCA	Limb, stance, and gait ataxia	N	N	N	N	N	N
4	MRI	R SCA	R upper limb ataxia	N	N	N	N	N	N
5	CCT	L SCA	Limb, stance, and gait ataxia	N	N	Art	N	N	N
6	CCT	R SCA	Limb, stance, and gait ataxia	N	N	N	N	N	N
7	CCT	R SCA	Limb, stance, and gait ataxia	N	N	N	N	N	N
			Cerebellar Plus Additional Brainstem Lesions (n = 11)						
8	CCT	L SCA	Vertical gaze palsy, L hemihypesthesia, R hemiparesis, limb, stance, and gait ataxia	N	Bi*	N	N	N	N
9	MRI	R PICA	Limb, stance, and gait ataxia	R-R2*	R*	Art	N	N	N
10	MRI	L PICA	R III paresis, dysphagia, limb, stance, and gait ataxia	N	N	N	N	N	N
11	MRI	R PICA	Ataxia, ocular tilt	N	N	N	N	N	N
12	MRI	BI (R SCA/L PICA)	L gaze palsy, up-beat nystagmus, L VII-palsy, limb, stance, and gait ataxia	L-R1/R2*	Bi*	N	N	N	N
13	MRI	R PICA/AICA	Limb, stance, and gait ataxia	R-R1*	N	L*	R*	N	N
14	MRI	R PICA	R Horner, R facial hemihypesthesia, R hemiparesis, limb, stance, and gait ataxia	R-R2†	Bi*	Bi*	N	N	N
15	MRI	R PICA/AICA	R Horner, R VI-paresis, up-beat nystagmus, R VII-palsy, limb, stance, and gait ataxia	L-R1†	N	L*	N	N	N
16	MRI	BI PICA	L Horner, skew deviation, limb, stance, and gait ataxia	L-R2*	L*	Bi*	R*	N	N
17	MRI	R PICA	Spontaneous nystagmus, R facial hemihypesthesia, limb, stance, and gait ataxia	R-R2†	R*	N	R†	N	N
18	MRI	R PICA/AICA	Skew deviation, limb, stance, and gait ataxia	L-R2*	L*	Bi*	N	N	N

Abbreviations: AICA, anterior inferior cerebellar artery; Art, artifact; BAEP, brainstem auditory evoked potential; Bi, bilateral; BlinkR, blink reflex; CT, computer tomography; L, left; MassR, masseter reflex; MEP, magnetic evoked potential; MRI, magnetic resonance imaging; N, normal; PICA, posterior inferior cerebellar artery; R, right; SCA, superior cerebellar artery; SEP, somatosensory evoked potential.

*Latency >2.5-fold of the mean of controls.

†Absent potential.

vation of the rostral paravermal region of the anterior lobe in 11 patients (**Figure 3**). No activation was found in 1 participant, whereas activation was not assessable due to movement artifacts in the posterior fossa of 2 additional participants.

Silent lip movements evoked bilateral cerebellar activation of the motor cortex in 12 patients. Bilateral cerebellar activation in the paravermal region of the anterior lobe was found in 8 of these patients (**Figure 4**). In another patient, no activation was observed, and activation was not assessable due to movement artifacts in the posterior fossa of 2 patients.

COMMENT

CEREBELLAR INFARCTIONS

Dysarthria due to cerebellar infarction has been described for all vascular territories of the cerebellum. In infarctions restricted to the PICA territory, dysarthria was found in 0% to 39% of cases.[8,10-12,28,29] Dysarthria in the AICA territory has been reported in 4 of 13 patients[13]; all 4 of these patients also had brainstem involvement. Infarctions in the SCA territory led to dysarthria in 56%,[6] 67%,[30] and 75%[31] of patients. Brainstem involvement is known to occur in most cerebellar infarctions due to the pattern of vascularization.[6,13,32] In view of the finding that not only cerebellar lesions but also brainstem lesions may represent a cause of dysarthria,[14,15,33] it is impossible to determine whether dysarthria in patients with combined lesions is due to cerebellar infarction, brainstem involvement, or both. The varying frequency of dysarthria reported by previous studies may be accounted for by the small patient sample, the occurrence of brainstem involvement, and the lack of a standardized procedure in the evaluation of dysarthria.

In the present study, the use of multimodal electrophysiologic investigations enabled the assessment of brainstem functions of the tegmental areas of the medulla oblongata (BlinkR-R2, BAEPs, SEPs), the pons (BlinkR-R1, MassR, BAEPs, SEPs), midbrain (MassR, BAEPs, SEPs), and the ventral parts of the brainstem (MEP VII, MEP XII).[15,25,26] In the present series, clinical

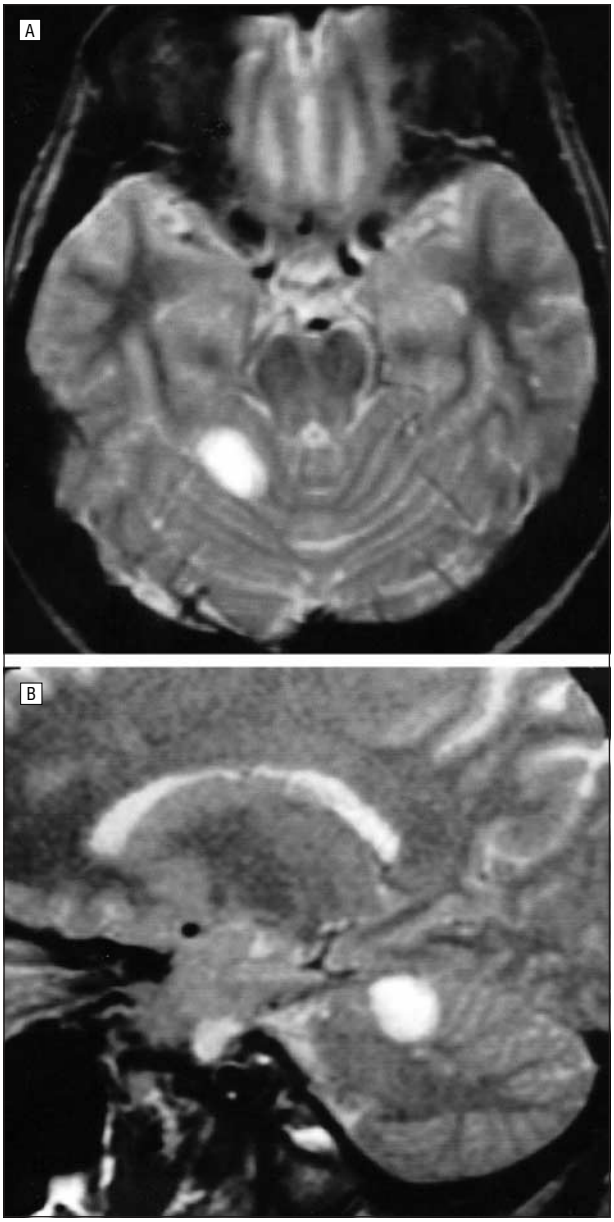

Figure 1. Magnetic resonance image of a patient seen for dysarthria–clumsy hand syndrome with an infarction in the right superior cerebellar artery territory. A, axial view; B, sagittal view.

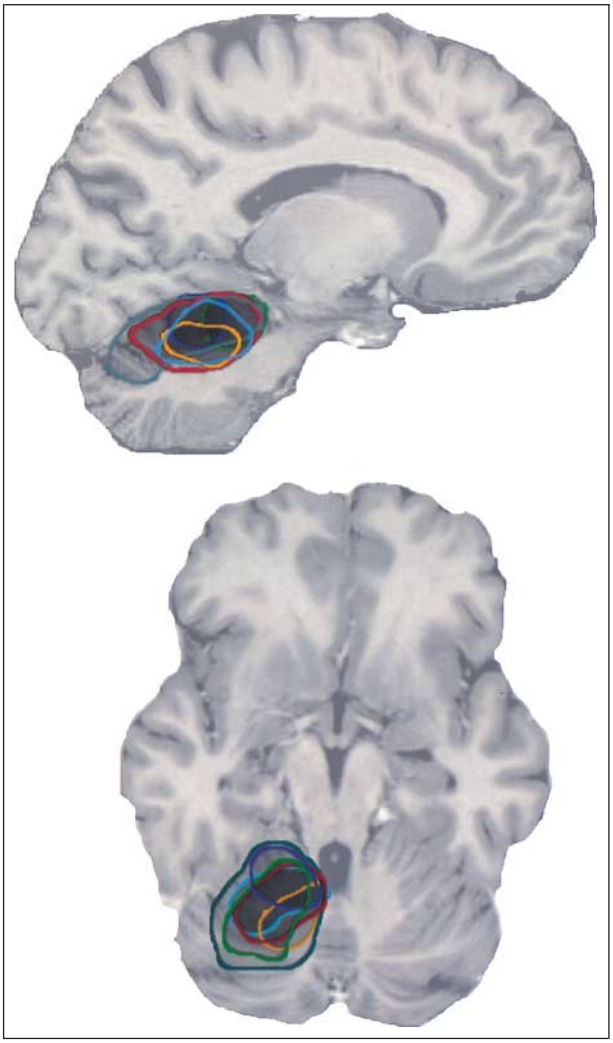

Figure 2. Superposition of the outlines of the isolated cerebellar infarctions (n=7). All infarctions were mirrored to the right side in the axial plane for better comparison.

findings, neuroimaging, and multimodal electrophysiologic studies identified brainstem involvement in 11 patients with cerebellar infarction (Table). Electrophysiologically, the most dorsally located pathways, mediating the BlinkR, MassR, and BAEPs, were most frequently affected, whereas pathways with a more ventral location, such as the medial lemniscus and the pyramidal tract, were rarely (SEPs) or not at all (MEPs) involved. This lesion pattern agrees with results of pathoanatomic studies, demonstrating predominantly tegmental brainstem involvement in cerebellar infarctions.[34]

Seven of 18 patients in this study had an isolated cerebellar infarction. Dysarthria can be attributed to the cerebellar infarction in these patients only. The SCA territory was affected in all 7 cases, and the most common lesion site in these patients was the rostral paravermal region of the anterior lobe. This finding is in accordance with results of previous MRI studies (n=1,[7] n=4,[8] and n=1[35]), reporting the location of an infarction in the left (n=1) or right (n=4) cerebellum and bilaterally (n=1). The detection of right-sided preponderance corresponds to the right-left ratio found in the patients of this study (6:1). All of our patients with unilateral right-sided SCA ischemia were right-handed. Reversed cerebellar lateralization of speech associated with left-handedness, as suggested by Lechtenberg and Gilman,[3] does not explain dysarthria in our patients. The presence of unilateral cerebellar lesions therefore seems to be sufficient to lead to dysarthria.

Speech impairment in our patients was similar to that described by Ackermann et al,[8] with the most frequently observed features of irregularly distributed articulatory deficits, slurred pronunciation of single consonants, and slowed speech tempo as the most common features. The number of patients with infarction restricted to half of the cerebellum is too small to enable identification of differences in the individual auditory-perceptual presentation.

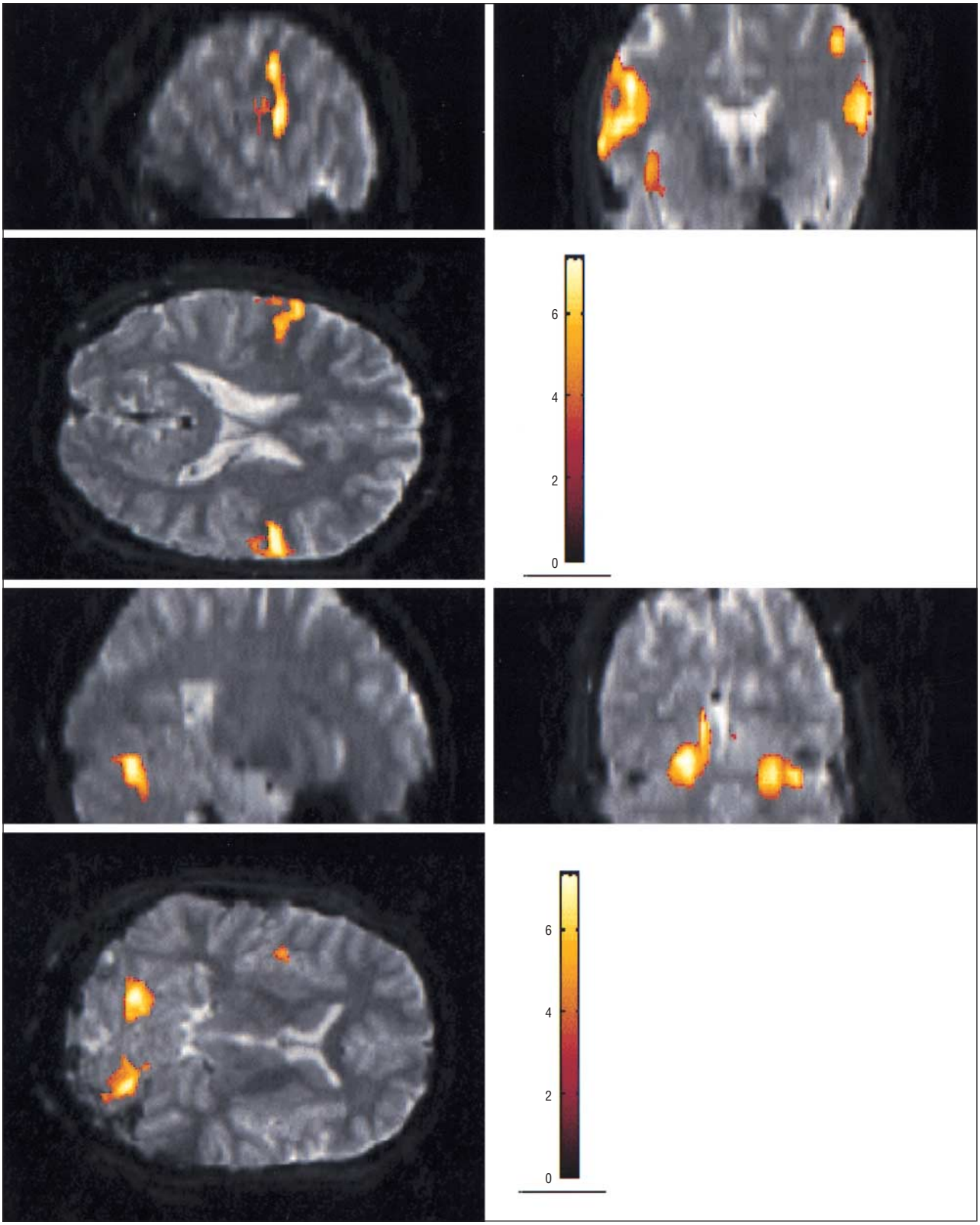

Figure 3. Functional magnetic resonance image during repetitive silent vertical tongue movements of a representative patient.

fMRI

Although the somatotopic representation of different body parts in the human primary motor cortex by different techniques (direct electrical stimulation of the motor cortex and fMRI[36,37]; positron emission tomography[38,39]; transcranial magnetic stimulation and fMRI[40]; magnetoencephalography, transcranial magnetic stimulation, and fMRI[41]; magnetoencephalography, positron emission tomography, and fMRI[42]) is well established, motor so-

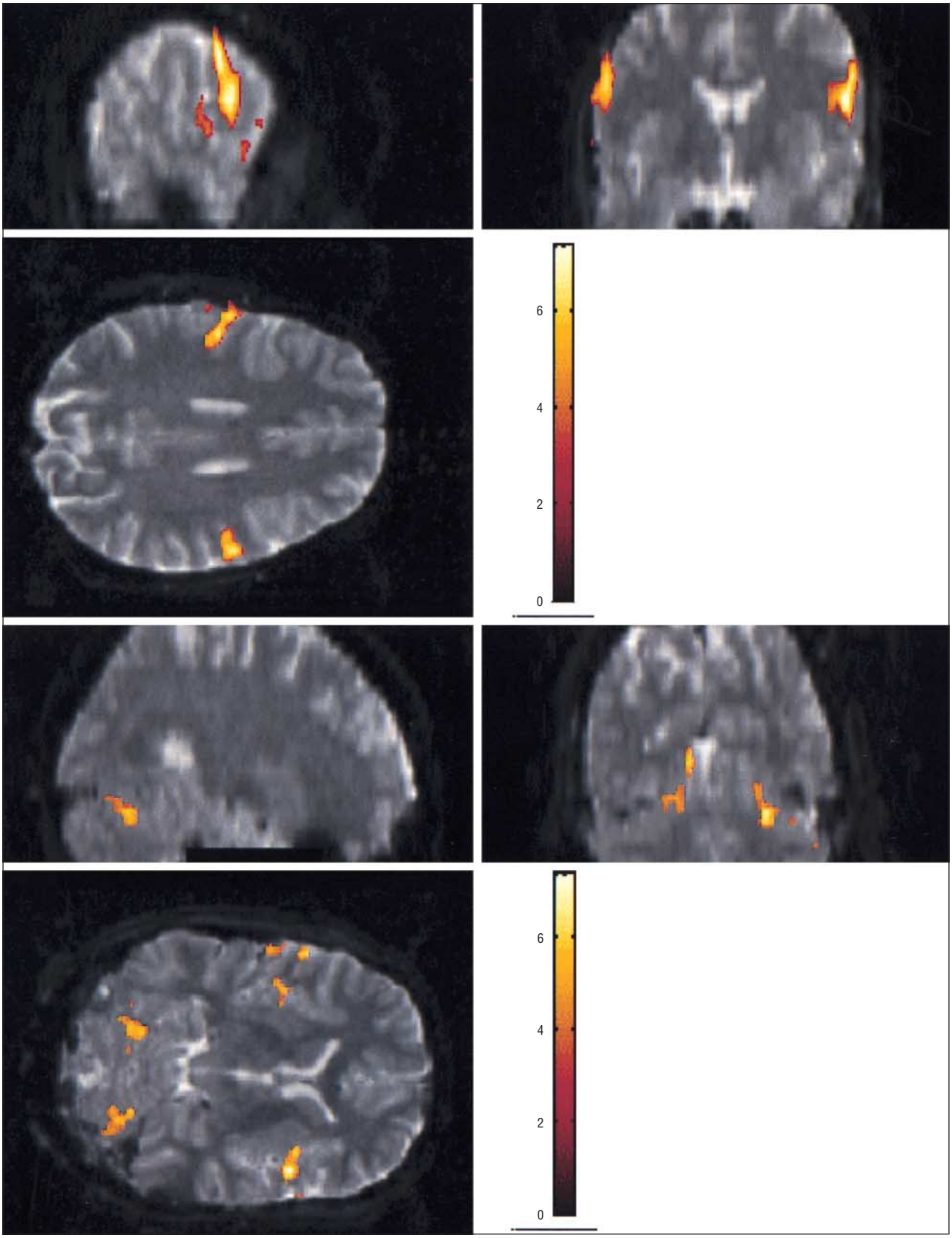

Figure 4. Functional magnetic resonance image during repetitive silent lip movements of a representative patient.

matotopy of the cerebellum is less well documented. In animal models, Hampson et al[43] and Snider and Eldred[44] first described somatotopic representations of motor responses in the cerebellar anterior lobe. In contrast, more recent microelectrode[45] and horseradish peroxidase studies[46] in rats suggest that cerebellar representa-

tions of body parts are broken up into smaller, discontinuous patches that show multiple, overlapping representations. Conversely, fMRI motor studies in humans have shown distinct and nonoverlapping somatotopic activation for finger, hand, and foot movements in the anterior lobe[47,48] and in other parts of the cerebellum, such as the prepyramidal fissure of the posterior lobe, the vermis, and pyramis.[49] Positron emission tomography studies have demonstrated an increase in regional cerebral blood flow in the ipsilateral, anterior, and superior cerebellum during finger movements and tactile finger stimulation[50] and in the superior vermis during elbow movements.[38] We observed an increased signal intensity following tongue and lip movements at a paravermal position in more medial parts of the anterior lobe. This paradigm was selected because the tongue and to a lesser degree the orofacial muscles are important articulators.[19,20] However, some additional jaw movement might have been included, whereas laryngeal function was not present due to the silent speech performance. This matches the fMRI-based somatotopic activation pattern for hand and foot movements.[47] Thus, the activation pattern of repetitive movements of articulatory muscles is characterized by the same topography as that derived from cerebellar infarctions that lead to dysarthria. Cerebellar activation was bilateral in our healthy volunteers and does not explain the right-sided preponderance of cerebellar infarctions associated with dysarthria. However, in a recent positron emission tomography study, including 12 right-handed people, repetition of nouns led to bilateral rostral paravermal activation, which was significant for the right side only.[51]

In conclusion, the results of our study demonstrate that articulatory movements of the tongue and orofacial muscles activate not only the caudal motor cortex but also the rostral paravermal area of the anterior lobe. This location corresponds to the area involved in purely cerebellar ischemia in patients with dysarthria. We conclude that lesions in the upper paravermal region of the right cerebellar hemisphere, the site of coordination of articulatory movements of the tongue and orofacial muscles, may lead to the development of dysarthria that is unrelated to (often concomitant) brainstem infarctions.

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