A topodiagnostic investigation on body lateropulsion in medullary infarcts

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CME

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Abstract—Body lateropulsion may occur without signs of vestibular dysfunction and vestibular nucleus involvement. The authors examined 10 such patients with three-dimensional brainstem mapping. Body lateropulsion without limb ataxia reflected an impairment of vestibulospinal postural control caused by a lesion of the descending lateral vestibulospinal tract, whereas body lateropulsion with limb ataxia was probably the consequence of impaired or absent proprioceptive information caused by a lesion of the ascending dorsal spinocerebellar tract.

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Body lateropulsion (i.e., falling to one side) is a common, disabling symptom in patients with lateral medullary infarctions (LMIs). It is usually associated with additional clinical signs, such as ipsilesional Horner syndrome, ipsilesional limb ataxia, ipsilesional sensory disturbances in the face, contralesional impairment of pain and temperature sensation in the arm and leg, hoarseness, dysarthria, and dysphagia.[1] LMIs often involve caudal parts of the vestibular nucleus causing vestibular signs and symptoms, such as vertigo, head and body lateropulsion, ocular motor disturbances (e.g., skew deviation, ocular torsion, and horizontal rotatory spontaneous nystagmus), and perceptual deficits (e.g., tilts of the subjective visual vertical).[1,2] However, body lateropulsion may also occur with LMI sparing the vestibular nucleus (i.e., by involvement of the descending lateral vestibulospinal tract [LVST] or the ascending dorsal spinocerebellar tract [DSCT]). Lesions of both may be followed by ipsilesional body lateropulsion, either as a consequence of impaired vestibulospinal posture control (LVST lesions) or impaired or absent proprioceptive information (DSCT lesions). Whether lesions of the former, the latter, or both are crucial to cause body lateropulsion remains uncertain. Previous reports on single patients with isolated lateropulsion caused by cerebrovascular diseases of the medulla,[3,4] the cerebellum,[5-7] or the midbrain[8] are unable to answer this question. Based on MRI with biplane T2- and echo-planar diffusion-weighted imaging (EPI-DWI) with slice orientation parallel and perpendicular to slices of the stereotactic anatomic atlas of Schaltenbrand and Wahren,[9] we tried to identify the structures crucial for the occurrence of body lateropulsion.

Methods. During a 4-year period, we prospectively recruited 258 patients with acute signs and symptoms indicating vertebrobasilar ischemia. All patients were examined within 24 hours after onset of symptoms, and all had DWI MRI within 48 hours after onset of symptoms, when they were symptomatic. High-resolution T1- and T2-weighted MRI was done as soon as the patients could tolerate this longer-lasting examination (median, 6.5 days after onset of symptoms). All patients gave their informed consent to these investigations, which were approved by the local ethics committee.

Clinical files were reviewed for the occurrence of body lateropulsion as the only or predominant clinical sign. Patients with signs of vestibular dysfunction (i.e., vertigo, nausea, vomiting, gaze-evoked or spontaneous nystagmus, unilateral hyporesponsiveness of the lateral semicircular canal by caloric testing, or skew deviation) were excluded from further analysis. We also excluded patients with earlier brainstem or cerebellar infarctions or those taking central sedative drugs.

MRI acquisition and postprocessing. MRI was done with a 1.5-T superconducting system (Magnetom Vision, Siemens, Erlangen, Germany). For EPI-DWI (repetition time [TR], 4,000 ms; echo time [TE], 103 ms), we separately applied diffusion gradients in three spatial axes ($b = 1{,}164$ s/mm^3, 128 matrix; 250 ms per slice; 20 slices; thickness, 3 mm; eight measurements). Axial and sagittal high-resolution T2-weighted imaging (TR, 3,810 ms; TE, 90 ms; 256 matrix; slice thickness, 3 mm) and T1-weighted imaging (TR, 600 ms; TE, 14 ms; 256 matrix; slice thickness, 3 mm) before and after IV gadolinium was done as soon as the patients could tolerate this longer-lasting MRI (median, 6.5 days after onset of symptoms). All MRI examinations were done with slice orientation parallel (sagittal sections) and perpendicular (axial sections) to the sagittal brainstem cuts of the stereotactic anatomic atlas of Schaltenbrand and Wahren.[9]

Two neuroradiologists and one neurologist independently identified the area of infarction. We used DWI-MRI to prove the acuity of the ischemic lesion and high-resolution T2-weighted MRI to outline the extension of this lesion. Using Unix and NT workstations and Photoshop (Adobe Systems, San Jose, CA) and Photo-Paint (Corel, Ottawa, Canada) software, the individual slices were normalized and projected into the corresponding slices of the anatomic atlas. Zero point was set at the pontomesencephalic junction, and the number of the given level indicates the distance from zero in millimeters in the craniocaudal direction. Axial slices were used for normalizing the individual slices in plane according to their T2- and T1-weighted brainstem outlines. Sagittal or coronal slices were used for normalizing in the z-axis by determining the best fitting of the anatomic plates with anatomic landmarks like the fourth ventricle or cranial nerve exit zones in projection to the anatomic plates. Given a functional right-left symmetry of the brainstem, all right-sided lesions were flipped to the left side for an easier comparison. We used the projections of the lesions into the corresponding slices of the anatomic atlas to determine the anatomic structures involved. We used a voxel-based model of the human brainstem for the statistical comparisons.[10]

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Table *Clinical findings in patients with body lateropulsion and MRI-documented lateral medulalry infarctions*

	Ipsilesional				Contra-lesional
Patient no.	Ataxia of limbs	Impaired facial sensation	Horner syndrome	Dysphagia, hoarseness vocal cord paralysis	Impaired pain and thermal sense over the half of the body
1		+			
2		+			
3				+	+
4		+	+		+
5			+	+	+
6	+			+	
7	+	+			
8	+	+		+	
9	+	+		+	
10	+		+		+

None had vertigo, nausea, vomiting, gaze evoked or spontaneous nystagmus, skew deviation, or vestibular hyporesponsiveness on caloric testing.

Results. None of our 258 patients had axial lateropulsion as the only clinical sign of the brainstem dysfunction. We identified 10 patients, all with acute unilateral LMI, who had body lateropulsion with several additional signs (table). Using a two-sample analysis (Mann–Whitney), we compared these 10 patients with a group of another 14 patients from the same group of 258 patients, who had brainstem infarctions at the same level but without lateropulsion and without clinical signs of vestibular dysfunction. In patients with body lateropulsion, the lesion area was close but just rostral to the dorsal column decussation and midway between the end of the DSCT and the beginning of the lower cerebellar peduncle (significance, ~0.005; figure 1). In a second analysis, we compared patients with body lateropulsion without limb ataxia (Patients 1 through 5) and those with limb ataxia (Patients 6 through 10). The lesions of patients without limb ataxia centered more dorsomedially than those with limb ataxia, who centered more ventrolaterally (figure 2). This difference was not significant because the number of patients in each group was too small.

Discussion. Body lateropulsion as the only clinical sign of a brainstem infarction is rare. We are aware of only five previously reported patients with different kinds of lesions: medullary hemorrhage,[3] LMI,[4] infarction of the flocculonodular region,[5] infarctions of the inferior and superior cerebellar peduncles,[6] and midbrain infarction in the region of the red nucleus.[7] We were unable to identify a single patient with isolated body lateropulsion. Body lateropulsion in the absence of any sign of vestibular dysfunction was present in only 10 patients. Infarctions in these patients involved an area close but just rostral to the dorsal column decussation and midway between the end of the dorsal spinocerebellar tract and the beginning of the inferior cerebellar peduncle. Lesions of this location may affect LVST and DSCT. However, infarctions of patients without limb ataxia centered more dorsomedially, likely involving the LVST. Patients with limb ataxia had more ventrolaterally centered infarctions involving the "border" between the ventral and the dorsal spinocerebellar tract, which might affect the DSCT. Although this difference was not significant, which might be attributed to the small number of patients, our findings suggest an LVST lesion as the cause of lateropulsion in patients with LMI without limb ataxia.

In the absence of clinical signs of vestibular dysfunction, body lateropulsion without limb ataxia may be attributed to an impaired vestibulospinal posture control because the sites of the responsible lesions

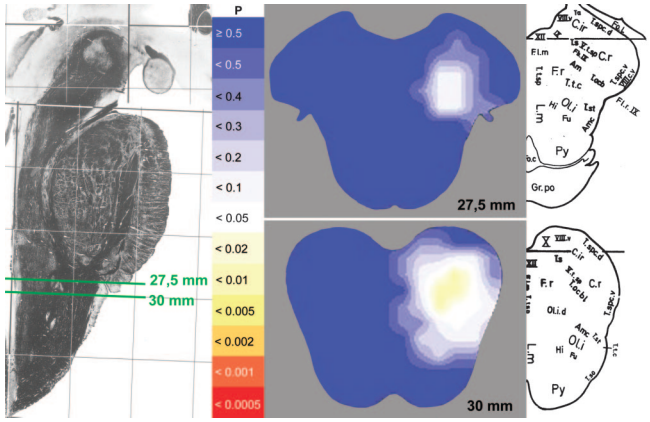

Figure 1. Comparison of the lesions of 10 patients with body lateropulsion without signs of vestibular dysfunction with the lesions of 14 patients without body lateropulsion and unilateral medullary infarctions at the same level (two-sample analysis; Mann–Whitney). The crucial structure for the occurrence of body lateropulsions was an area just rostral to the dorsal column decussation and midway between the end of the dorsal spinocerebellar tract and the beginning of the inferior cerebellar peduncle (significance, ~0.01). As shown in the left, the slices were 27.5 and 30 mm below the pontomesencephalic junction and were taken from Schaltenbrand and Wahren's Atlas for Stereotaxy of the Human Brain.[9] *Am and Amc = amiculum of the inferior olive; C.ir = juxtarestiform body; C.r = restiform body; Fb.IX = hypoglossal nerve fibers; Fb.iol = intraolivary fibers; Fo.c = foramen cecum; Fo.L = foramen of Luschka; F.l.m = medial longitudinal fasciculus; Fl.r.IX = rootlets of the glossopharyngeal nerve; F.r = tegmental reticular formation; Fu = fundus of the inferior olive; Gr.po = pontine grey matter; Hi = hilus of the inferior olive; L.m = medial lemniscus; Ol.i = inferior olive; Py = medullary pyramid; Ta = acoustic tuberculum; T.obc and T.ocbl = olivocerebellar tract; T.s = solitary tract; T.so = spinoolivary tract; T.spc.d = dorsal spinocerebellar tract; T.spc.v = ventral spinocerebellar tract; T.st = spinothalamic tract; T.t.c = central tegmental tract; T.tsp = tectospinal tract; V.t.sp = spinal trigeminal nucleus; VIII.c.v = ventral cochlear nucleus; VIIIv = vestibular nucleus; IX = glossopharyngeal nucleus; X = nuclei of vagus; and XII = hypoglossal nucleus.*

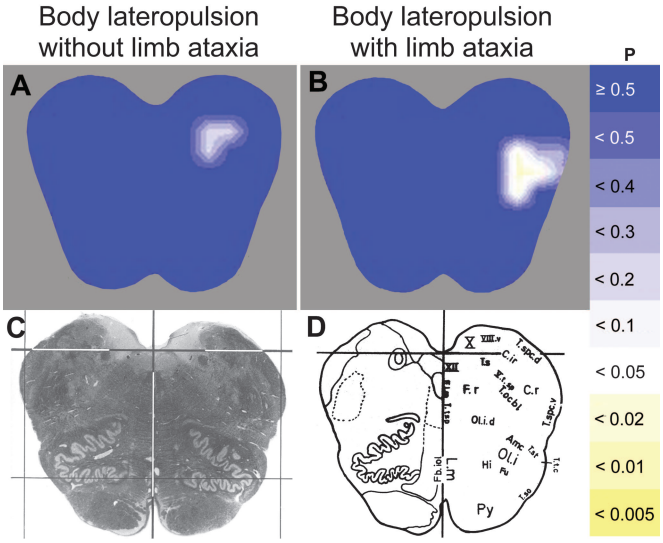

Figure 2. Comparison of the lesions of five patients with body lateropulsion without limb ataxia (A) and five patients with body lateropulsion and limb ataxia (B). The lesions of patients without limb ataxia centered more dorsomedially than those with limb ataxia, who centered more ventrolaterally. (This difference was not significant because the number of patients in each group was small.) The anatomy of this slice is shown in C and D, which were taken from Schaltenbrand and Wahren's Atlas for Stereotaxy of the Human Brain.[8] *Abbreviations are the same as in figure 1.*

involved parts of the LVST. Ipsilesional body lateropulsion with limb ataxia more likely reflects impaired or absent proprioceptive information because lesions in these patients more likely involved the DSCT. In most patients with LMI (Wallenberg syndrome), however, impaired vestibulospinal posture control and impaired proprioception may contribute to the occurrence of body lateropulsion because these infarctions usually involve the DSCT and LVST.

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