PAPER

Body lateropulsion as an isolated or predominant symptom of a pontine infarction

Hyon-Ah Yi, Hyun-Ah Kim, Hyung Lee, Robert W Baloh

J Neurol Neurosurg Psychiatry 2007;**78**:372–374. doi: 10.1136/jnnp.2006.106237

See end of article for authors' affiliations

Correspondence to: Dr H Lee, Department of Neurology, Keimyung University School of Medicine, 194 Dongsan dong, Daegu 700–712, South Korea; hlee@dsmc.or.kr

Received 6 September 2006
Revised 26 October 2006
Accepted 2 November 2006
Published Online First 21 November 2006

Background: Lateropulsion of the body—that is, falling to one side—is a well-known clinical feature of stroke in the posterior circulation. Body lateropulsion as an isolated or predominant manifestation of a pontine stroke has not been reported previously.
Objective: To elucidate the possible mechanisms of patients presenting with body lateropulsion as an isolated or predominant symptom of an isolated pontine infarction.
Methods: Between May 2004 and February 2006, out of 134 patients admitted with an isolated pontine stroke, we identified 8 (6%) consecutive patients in the Keimyung University Stroke Registry who had body lateropulsion as the main presenting symptom.
Results: All lesions were localised to the paramedian tegmentum just ventral to the fourth ventricle. All except one showed a uniform pattern of body lateropulsion, in which the direction of falling was away from the side of an infarct. In two patients body lateropulsion was the sole clinical manifestation, whereas the other patients had other neurological signs. All but one patient had contraversive tilting of the subjective visual vertical (SVV). In all cases, the direction of SVV tilt corresponded to the direction of body lateropulsion. The mean net tilt angle was 6.1°.
Conclusions: Based on the known anatomy of ascending vestibular pathways, SVV tilting and MRI findings, it is concluded that body lateropulsion probably results from damage to the graviceptive pathway ascending through the paramedian pontine tegmentum.

Lateropulsion of the body,—that is, an irresistible falling to one side—is a well-known clinical feature of stroke in the posterior circulation.[1] In most cases there are other neurological symptoms or signs, which vary according to the structures involved. To the best of our knowledge, lateropulsion of the body as an isolated or predominant manifestation of a pontine stroke has not been described before. To elucidate the possible mechanisms of body lateropulsion with lesions of the pons, we analysed eight patients with small pontine infarcts who presented with lateropulsion as their main symptom. In all but one patient, subjective visual vertical (SVV) testing showed a pathological tilting opposite to the side of the lesion.

METHODS

Between May 2004 and February 2006, out of 134 patients admitted to the Keimyung University Dongsan Medical Center, Daegu, South Korea with an isolated pontine stroke we identified 8 (6%) consecutive patients who had body lateropulsion as the main presenting symptom. All had small isolated pontine infarcts. Although some patients with large infarcts had body lateropulsion, they also had long tract signs with hemi/tetra-weakness, sensory loss of the extremities or altered consciousness, and were excluded. Body lateropulsion was defined as a falling to one side without vertigo or weakness, in which the patient could not stand without assistance. An MRI was performed in all patients with a 3.0 T MR scanner (Signa VH/I, General Electric, Milwaukee, WI, USA), between 1 and 3 days after the stroke. MRI scanning with T1, T2, proton and diffusion-weighted images was done in the axial, sagittal and coronal planes with 6 mm thick sections. An MR angiogram (MRA) was also performed in all patients. Diagnostic tests were performed to determine risk factors of stroke for all patients. Vestibular function tests were performed by a computer-based electronystagmography (ENG) system (Nicolet Instrument Corporation, Madison, WI, USA), and included examination of oculomotor movements (saccade, smooth pursuit and optokinetic nystagmus), spontaneous and gaze evoked nystagmus, and caloric responses. Detailed testing techniques have been previously reported.[2] Testing of SVV tilting was done for all patients. The patients sat upright in a dark room with the head fixed using a head holder. A moulded neck rest covered the occiput and posterior neck, holding the head horizontal. In front of the patient at a distance of 120 cm was a dim light bar, 2 mm wide and 90 cm long. It could be rotated about its midpoint by means of an electronic motor and a remote control device. The patients adjusted the bar for parallel alignment with the perceived gravitational vertical. During each test, the patients performed 10 settings of the light bar with both eyes open. The average of the 10 settings was used as the measure of the SVV. The reference group consisted of 20 healthy age-matched volunteers who had no vertigo or nystagmus and had a normal brain MRI. ENG confirmed normal vestibular function in all members of the reference group. SVV tilting results with binocular viewing were considered abnormal when they were outside the average (2SD) values (more than 2°).

RESULTS

Demography and risk factors

Table 1 summarises the demography and clinical characteristics of the patients. There were eight patients (five men and three women), ranging in age from 57 to 83 years. Risk factors included hypertension in six, diabetic mellitus in two and current smoking in two patients. One patient had a history of myocardiac infarction. None had a history of stroke or gait disturbance. Follow-up periods ranged from 2 to 9 (mean 4.6) months.

Abbreviations: ENG, electronystagmography; INO, internuclear opthalmoplegia; MRA, magnetic resonance angiogram; RPT, rostral pontine segmentum; SVV, subjective visual vertical

Table 1 Characteristics of patients with body lateropulsion

Patients/sex/age (years)	Symptoms at onset	Signs	SVV tilting	MRI	Follow up	Sequelae
1/F/82	Falling	Lt axial lateropulsion	5.9°, Lt	Rt MPT	5 months	None
2/M/83	Falling, visual blurring, diplopia, dizziness	Rt axial lateropulsion, one (Lt) and half (Rt) syndrome, downbeat nystagmus	3.7°, Rt	Lt M/CPT	5 months	Dizziness, LR weakness (Lt)
3/F/71	Falling, dizziness, dysarthria	Lt/backward axial lateropulsion LR weakness (Lt), facial palsy (Lt), facial sensory loss (Lt)	6.5°, Lt	Lt CPT	9 months	None
4/M/59	Falling, diplopia, numbness on face (Lt)	Lt axial lateropulsion, Rt INO	5.5°, Lt	Rt RPT	2 months	Dizziness, MR weakness (Rt)
5/M/77	Falling, diplopia, dizziness	Rt axial lateropulsion, Lt INO, mild limb dysmetria	8.7°, Rt	Lt RPT	2 months	None
6/F/57	Falling, diplopia, perioral numbness (Lt)	Rt axial lateropulsion, Lt INO	3.9°, Rt	Lt RPT	3 months	None
7/M/72	Falling	Lt axial lateropulsion	7.8°, Lt	Rt RPT	2 months	None
8/M/68	Falling, diplopia, dizziness	Lt axial lateropulsion, Rt INO	6.7°, Lt	Rt RPT	9 months	None

CPT, caudal pontine tegmentum; F, female; INO, internuclear opthalmoplegia; LR, lateral rectus; Lt, left; M, male; MPT, middle pontine tegmentum; MR, medial rectus; RPT, rostral pontine tegmentum; Rt, right; SVV, subjective visual vertical.

Clinical findings

The most common and striking symptom at onset was prominent gait ataxia with falling to one side, which was present in all (n = 8) patients. The patients could not stand or walk without assistance. The falls were irresistible and occurred without warning. All except one patient (No 3) showed a uniform pattern of body lateropulsion, in which the direction of falling was away from the side of an infarct. Patient 3 fell backwards and on towards the side of the infarct. None of the patients showed lateropulsion of closed eyes (ocular lateropulsion) or saccadic lateropulsion,—that is, overshoot or undershoot of saccades. In two patients (Nos 1 and 7) body lateropulsion was the sole clinical manifestation, whereas the other patients had other neurological signs (most commonly an internuclear opthalmoplegia (table 1)). All patients were treated with antiplatelet agents. All patients had complete regression of body lateropulsion within 2 weeks after the stroke. The patients were followed for 2–9 months. Only two patients (Nos 2 and 4) complained of non-specific dizziness with mild unsteadiness. During the follow-up period, none of the patients had recurrent strokes.

SVV tilting test

SVV tilting tests were performed 1–3 days after the onset of stroke. All patients had pathological tilts of the SVV from the true vertical, in which all except one, SVV tilted to the side opposite to the infarct. Ipsiversive tilts of SVV were found in only one patient (No 3) with a caudal pontine infarct. In all cases, the direction of SVV tilt corresponded to the direction of body lateropulsion. The mean net tilt angle was 6.1°, with a range of 3.7–8.7°.

MRI and MRA findings

MRIs were performed 1–3 days after the onset of stroke. Figure 1 presents the MRI findings. All patients had tiny infarcts located in the paramedian tegmental area just ventral to the fourth ventricle. Five patients (Nos 4, 5, 6, 7 and 8) had an infarct in the

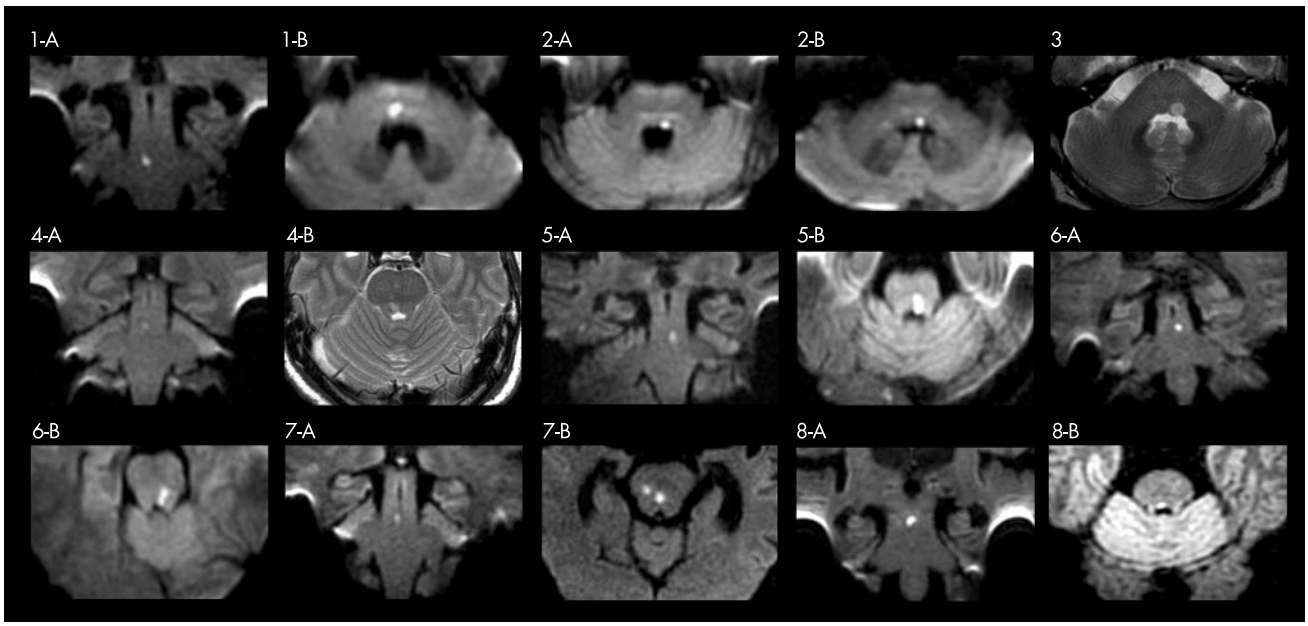

Figure 1 MRIs of patients with an isolated or predominant body lateropulsion disclose lesions restricted to the just ventral portion of the fourth ventricle in the pontine tegmentum.

rostral, one patient (No 1) in the middle, one patient (No 3) in the caudal, and the remaining patient (No 2) in the middle and caudal level of the pons. MRA showed a normal basilar artery in five patients (Nos 1, 2, 4, 7 and 8). Two patients (Nos 3 and 6) had severe stenosis of the proximal vertebral artery and one had stenosis of the proximal basilar artery (No 5).

DISCUSSION

Our patients presented with body lateropulsion due to a small pontine infarction. Body lateropulsion as the main clinical manifestation of posterior circulation stroke is rare. We are aware of only five previously reported cases of lateral medullary infarction,[3–5] infarction of the superior and inferior cerebellar peduncle,[6] and midbrain infarction involving the red nucleus.[7] There are no prior reports of isolated body lateropulsion due to a pontine stroke.

All patients had a lesion(s) located just ventral to the fourth ventricle in the pontine tegmentum. The anatomic structure most likely responsible for the sudden falling was the ascending graviceptive pathway (resulting in an imbalance in the roll plane). The direction of SVV tilt corresponded to the direction of body lateropulsion consistent with a lesion of the ascending graviceptive pathway. Graviceptive input from the otoliths converges with that from the vertical semicircular canals at the level of the vestibular nuclei to subserve vestibular function in the pitch and roll plane. These signals are passed on to the oculomotor nuclei and cortical centres for perception of verticality. Although we did not measure ocular torsion in our patients, previous studies have shown a high correlation between ocular torsion and the SVV tilt.[1 8]

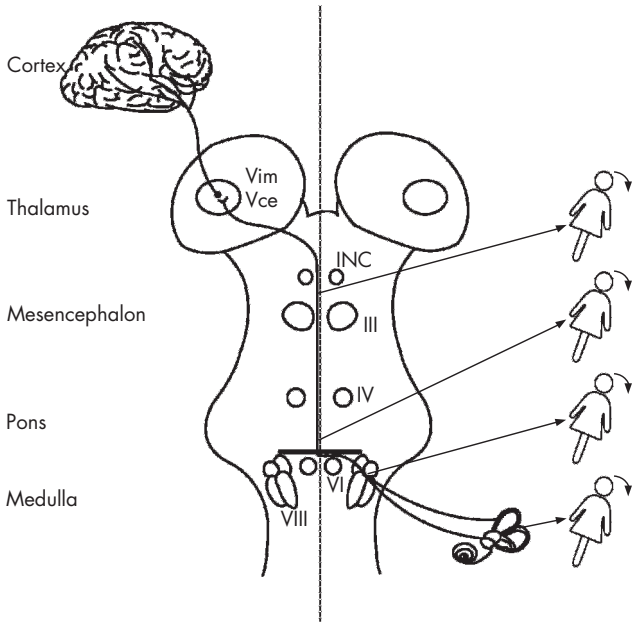

Figure 2 Hypothetical explanation of body lateropulsion due to a lesion of the ascending graviceptive pathways. Graviceptive inputs (dashed lines) from the otoliths converge with those from the vertical semicircular canals at the level of the vestibular nuclei and cross the midline at the pontine level, just above the level of the vestibular nuclei (horizontal thick bar). After crossing, the graviceptive pathways ascend the brainstem through the paramedian tegmentum. Finally, the graviceptive pathways reach the multiple cortical centres through the thalamus. Curved arrows on the right side indicate the direction of the tilts of the SVV. Note that brainstem lesions just rostral to the vestibular nuclei cause contraversive tilts and falls, whereas all lesions caudal to this level cause ipsiversive tilts and falls. INC, interstitial nucleus of Cajal; Vce nucleus ventrocaudalis externus; Vim, nucleus ventrooralis intermedius; III, oculomotor nucleus; IV, trochlear nucleus; VI, abducens nucleus; VIII, vestibular nucleus.

In all but one patient, the direction of body lateropulsion was to the side opposite the lesion. The single patient (No 3) with ipsilesional body lateropulsion had a small infarct in the caudal pontine tegmentum. Although it is well known that graviceptive brainstem pathways from the vestibular nuclei cross the midline at the pontine level, the exact location of crossing is still unclear. Dieterich and Brandt[8] concluded that the crossing occurs in the rostral pons whereas our data suggest that the crossing is more caudal, just above the level of the vestibular nuclei (figure 2).

Although the body lateropulsion was the predominant finding, other neurological signs were seen in all but two patients (table 1). Most common was a unilateral internuclear opthalmoplegia (INO) from the involvement of the medial longitudinal fasciculus located just adjacent to the graviceptive vestibular pathways.[9] In these patients with INO, skew deviation might have contributed to the patients' body lateropulsion since the skew deviation associated with INO also resulted from the vestibular dysfunction in the roll plane of the vestibulo-ocular reflex (ie, disruption of the otolith-ocular connections).

The tegmental area of the pons is supplied by branches from the basilar, anterior–inferior cerebellar and superior cerebellar arteries.[10] Considering the small size of the lesion on brain MRI, an occlusion of a small penetrating branch of one of these arteries is the likely mechanisms for the strokes in most of our patients.

We have previously reported body lateropulsion as the sole manifestation of lateral medullary infarction.[3] Together, these reports highlight the importance of acute, severe gait ataxia with lateropulsion as a sole manifestation of vertebrobasilar ischaemic stroke. Clinicians should be aware of the possibility of a pontine stroke in patients with body lateropulsion even if other classic brainstem signs are absent.

Authors' affiliations
Hyun-Ah Kim, Hyung Lee, Department of Neurology, Keimyung University School of Medicine, Daegu, South Korea
Hyon-Ah Yi, School of Medicine and Institute for Medical Science, Keimyung University School of Medicine, Daegu, South Korea
Hyung Lee, Brain Research Institute, Keimyung University School of Medicine, Daegu, South Korea
Robert W Baloh, Department of Neurology, UCLA School of Medicine, Los Angeles, California, USA; Division of Surgery (Head and Neck), UCLA School of Medicine, Los Angeles, California, USA

This work was supported by a grant for promotion of research in 2006, from the Keimyung University Dongsan Medical Center.

Competing interests: None.

REFERENCES

1 **Dieterich M**, Brandt T. Wallenberg syndrome: lateropulsion, cyclorotation, and subjective visual vertical in thirty-six patients. *Ann Neurol* 1992;**31**:399–408.
2 **Lee H**, Sohn SI, Jung DK, Sudden deafness and anterior inferior cerebellar artery infarction, *et al. Stroke* 2002;**33**:2807–12.
3 **Lee H**, Sohn CH. Axial lateropulsion as a sole manifestation of lateral medullary infarction: a clinical variant related to rostral-dorsolateral lesion. *Neurol Res* 2002;**24**:773–4.
4 **Kim SH**, Cho J, Cho JH, *et al*. Isolated lateropulsion by a lesion of the dorsal spinocerebellar tract. *Cerebrovasc Dis* 2004;**18**:344–5.
5 **Maeda K**, Saikyo M, Mukose A, Lateropulsion due to a lesion of the dorsal spinocerebellar tract, *et al. Intern Med* 2005;**44**:1295–7.
6 **Bertholon P**, Michel D, Convers P. Isolated body lateropulsion caused by a lesion of the cerebellar peduncles. *J Neurol Neurosurg Psychiatry* 1996;**60**:356–7.
7 **Felice KJ**, Keilson GR, Schwartz WJ. ''Rubral'' gait ataxia. *Neurology* 1990;**40**:1004–5.
8 **Dieterich M**, Brandt T. Ocular torsion and tilt of subjective visual vertical are sensitive brainstem signs. *Ann Neurol* 1993;**33**:292–9.
9 **Kim JS**. Internuclear ophthalmoplegia as an isolated or predominant symptom of brainstem infarction. *Neurology* 2004;**62**:1491–6.
10 **Parent A**. *Carpenter's human neuroanatomy*, 9th edn. Baltimore: Williams & Wilkins, 1996:93–128.