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treatment and plasmapheresis are effective in preserving sensory nerve potentials and motor function.

JU-ShIN LOU
ROBERT SNYDER
ROBERT C GRIGGS
Department of Neurology, University of Rochester Medical Center, Rochester, NY, USA.

Correspondence to: Dr Jau-Shin Lou, University of Rochester Medical Center, Department of Neurology, Box 675, 601 Elmwood Avenue, Rochester, NY 14642, USA.


Contraversive visual tilt illusion associated with a cerebellar infarction

Visual tilt illusion consists of an abnormal perception of the environment, which seems to be rotated at a variable angle without any change in gaze. It is sometimes associated with the symptoms of the objects. It is sometimes associated with other postural and ocular tilt effects. It can be secondary to disturbances in the peripheral or central vestibular pathways.1 Previous reports suggest that cerebellar injuries could also cause this effect.2 This has not been documented before. We report a case of visual tilt illusion probably associated with an isolated cerebellar lesion, studied with CT and MRI.

A 56 year old man with hypertension and hypercholesterolaemia had a sudden attack of continuous vertigo not related to cephalic motion. He had an inclination of the internal representation of the gravitational vector to his left and he tried to adjust both visual objects and posture to what he perceived as being vertical. It would have been interesting to assess whether there was ocular torsion, to define his clinical setting more exactly, but it makes no difference to interpretation as ocular torsion can be associated or not with perceptual or other tilt effects.1 Our patient showed a right hemispheric cerebellar ischaemic lesion, in a territory dependent on the posteroinferior cerebellar artery (PICA), with no mass effect and no brainstem or other alterations on MRI. The postural and postural tilt was contraversive to the lesion. It is possible that an additional subcortical lesion in the distribution of the PICA, not evident with clinical and imaging studies, produced the tilt effects in this case, because the motor infranuclear arteriopathy supplying both brainstem and cerebellum and it is very difficult to differentiate the effects of cerebellar and brainstem lesions.1 But the tilt should then be ipsiversive, not contraversive, to the hypothetical lesion. Therefore it is not likely that an associated medullary ischaemia could cause the tilt effects in our patient. A mesencephalic injury could cause this clinical picture but there were no other upper brainstem symptoms and MRI was normal at this level. A supratentorial disorder is unlikely because there were no MRI alterations and there were associated postural tilt effects. In this patient, we think that cerebellar dysfunction could be responsible for the tilt effects.

The present report confirms a previously hypothesised role for the cerebellar structures in the control of perception of verticality and may contribute to a better knowledge of the pathophysiology and the topographic diagnosis of the central vestibular syndromes.

M BARON
JM GOBERNADO
MJ MAJUAN
M LOUSA
Servicio de Neurología, Hospital Ramón y Cajal, Universidad de Alcalá de Henares, Madrid, España.

Correspondence to: Dr M Baron, Servicio de Neurología, Hospital Ramón y Cajal, Ctra de Colmenar, Km 9.100, 28034 Madrid, Spain.


Low striatal D2 receptor binding as assessed by ['H]IBZM SPECT in patients with writer’s cramp

Writer’s cramp is a form of idiopathic focal task specific dystonia. In accordance with other studies on idiopathic and symptomatic dystonia, Tempel and Perlmutter suggested the presence of an abnormal striatothalamocortical drive in writer’s cramp.1 In view of the physicaliy, the vestibular pathways make contact with the ocular motor system, the spinal cord, and the vestibular cortex, contributing to the stabilisation of posture and perception of verticality and self-motion.5,6 The tonic bilateral vestibular input builds up the actual central vestibular tone in the three major planes: horizontal or “yaw”, sagittal or “pitch”, and frontal or “roll”.7,8 It seems that central pathways that mediate vestibular function in either of the three planes travel independently of each other, so that a specific lesion could cause a disorder restricted to one of them.9 The vestibular tone in the frontal or “roll” plane allows a correct perceptual, ocular, and postural alignment to the “gravitational vertical”; an imbalance in this tone causes a lateral tilt with alteration in perception of verticality, head and body posture, misalignment of the visual axes, or ocular torsion.4–6 Patients perceive the surroundings and their body as if they were tilted in the opposite direction to what the CNS erroneously computes as being vertical and try to adjust the visual objects and posture to it. Dietrich and Brandt showed that an alteration in the perceived verticality is not just the sensory consequence of the rotation of the eyes, as they can appear separately and are not proportional in degree.7 Furthermore, it is possible that not all the effects of tilt occur in one patient, and the perceptual disorder itself is the most sensitive sign of a vestibular tone imbalance in the frontal plane.1,9 Brainstem structures that mediate the vestibular tone in the “roll” plane include the vestibular nuclei and the interstitial nucleus of Cajal—perhaps the most rostral portion both brainstem and cerebellum and it is likely that an associated medullary ischaemia could cause the tilt effects in our patient. A mesencephalic injury could cause this clinical picture but there were no other upper brainstem symptoms and MRI was normal at this level. A supratentorial disorder is unlikely because there were no MRI alterations and there were associated postural tilt effects. In this patient, we think that cerebellar dysfunction could be responsible for the tilt effects.

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M BARON
JM GOBERNADO
MJ MAJUAN
M LOUSA
Servicio de Neurología, Hospital Ramón y Cajal, Universidad de Alcalá de Henares, Madrid, España.

Correspondence to: Dr M Baron, Servicio de Neurología, Hospital Ramón y Cajal, Ctra de Colmenar, Km 9.100, 28034 Madrid, Spain.

We did not find a correlation between severity of dystonia and [123I]IBZM. A dysfunction of such thalamostriatal interneurons is also consistent with the increased density of striatal cholinergic interneurons in dystonia after perinatal anaphylactic injury and with the well known efficacy of anticholinergic therapy in dystonia.

Letters to the Editor

Our results suggest that the striatal dopaminergic system is involved in writer’s cramp and that an increase in the density of striatal [123I]IBZM binding is related to dystonia. We found no evidence to support the hypothesis that the symptoms of dystonia are caused by an increased density of [123I]IBZM binding in patients with writer’s cramp. The results raise some questions. Firstly, there was a bilateral reduction of available striatal D2 receptors, whereas the symptoms were unilateral and there was no asymmetry between the hemispheres.

**References**


