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 of the other characteristics 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. Previous data suggest that cerebellar injuries could also cause it, but this has not been documented before. We report a case of visual tilt illusion probably associated with an isolated cerebellar lesion, studied with T2-weighted MRI.

A 56 year old man with hypertension and hypercholesterolemia had a sudden attack of continuous vertigo not related to cephalic movements, with nausea and vomiting and deviation to the left while walking. When it disappeared, 48 hours later, he complained that he saw objects as if they were tilted to his right by 30° and they should be rotated antit-clockwise—that is, to his left—as if to be perceived as vertical. He had a slight head and body tilt to his left that worsened when he was asked to close his eyes and stand upright. There was no skew deviation or other ocular motor disorders. Fundal photographs were not taken, so that ocular torsion could not be assessed. There were no other alterations on neurological examination. Two weeks later the patient was asymptomatic. Brain CT and MRI showed a right hemispheric cerebellar lesion, suggesting an ischaemic infarction (figure). No brainstem or cortical alterations were found.

Visual tilt illusion has been described in unilateral peripheral vestibular lesions; in brainstem injuries, typically in the Wallenberg syndrome, in other medullary and mesencephalic lesions, and in thalamic and parietoinsular cortex disorders. The most frequent conditions associated with visual tilt illusion are vascular lesions. As far as we know, there are no reports on documented cerebellar injuries associated with the illusion.

Physiologically, the vestibular pathways make contact with the ocular motor system, the spinal cord, and the vestibular cortex, contributing to the stabilization of posture and perception of verticality and self-motion. 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”. 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. The vestibular tone in the frontal or “roll” plane allows a correct perception, 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. 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. 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. 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 structure related to the control of vertical and torsional head and eye position. Both are connected by the medial longitudinal fasciculus, which crosses the midline in the pons. Visual vertical tilt is, then, ipsiversive to peripheral or pontomedullary lesions and contraversive to pontomesencephalonic lesions and, in both cases, is usually associated with other tilt effects; in most rostral lesions it may be either ipsiversive or contraversive and is usually isolated. The role of the vestibular cerebellar structures with respect to the control of subjective verticality is not well known at the moment. Our patient’s clinical findings suggest that 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 a central tendency 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. Our patient showed a right hemispheric cerebellar ischaemic lesion, in a territory dependent on the posteriorinferior cerebellar artery (PICA), with no mass effect and no brainstem or other alterations on MRI. His perceptual and postural tilt was contraversive to the lesion. It is possible that an additional subtle medullary lesion in the distribution of the PICA, not evident with clinical and imaging studies, produced the tilt effects described in this case, because the major infratentorial arteries supply both brainstem and cerebellum and it is very difficult to differentiate the effects of cerebellar and brainstem lesions. 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 should be other 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 hypothesized role for the cerebellar structures in the control of perception of verticality, and may contribute to a better knowledge of the pathophysiolog and the topographic diagnosis of the central vestibular syndromes.
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