Acute upside down reversal of vision in vertebrobasilar ischaemia

Acute upside down reversal of vision is an uncommon and little known phenomenon consisting of transient complete 180 degree inversion of the visual image. The pathogenesis and the anatomical sites of this dysfunction are unknown. Lesions involving cortical areas, mainly the parieto-occipital region, or the vertebrobasilar cerebellar system have occasionally been documented.1,2

We observed two patients who experienced this bizarre visual illusion, both revealing features of vertebrobasilar ischaemia.

Patient 1, a 69 year old woman, was admitted because two weeks earlier she had experienced sudden malaise, sweating, nausea, vomiting, right occipital headache, followed by a 180 degree vertical inversion of the visual image, lasting about 20 minutes. Two similar episodes had occurred the day before admission. On admission the patient was alert, cooperative and well-oriented. The neurological examination was normal. In particular, no neuroophthalmological abnormalities were found on clinical examination. Blood parameters, urine, chest radiograph and ECG proved normal. Cerebral CT and MRI (figure) showed an ischaemic-like lesion, 2 cm diameter, in the right cerebellar hemisphere in the territory of the medial branch of the posterior inferior cerebellar artery (PICA), without mass effect. Moderate periventricular white matter abnormalities coexisted. Four vessel cerebral angiography revealed a right vertebral artery stenosis (50%) and two small arteriovenous malformations on the course of the right ascending cervical artery; a decreased flow in the basilar artery was noted. Ticlopidine 250mg daily was given and the patient was discharged. No further attacks or other neurological disturbances occurred during the next two years.

Case 2, a 52 year old woman, with a 40 year history of bilateral chronic otitis with residual deafness, had recent recurrent episodes of sudden sweating, nausea, occipital headache, dizziness, sometimes followed by a transient loss of consciousness. The whole episode usually lasted about 30–40 minutes. Frequently, at the height of dizziness, the patient experienced an 180 degrees vertical visual inversion of images. These episodes occurred monthly. On admission, the neurological examination was normal. Rare, isolated, left-sided jerks of horizontal nystagmus, sometimes revealed by ENG. A 20 mmHg difference between right and left brachial arterial pressure (right > left) was noted. Ultrasound vascular investigations (Doppler cortico-vertebral echotomography and cerebral transcranial Doppler) revealed a left subclavian artery stenosis with a steal syndrome. Cerebral SPECT, CT and MRI proved normal. BAER was unavailable due to the peripheral nature of the audiometric test. Cerebral angiography was refused. Fluorantine 10mg daily was given and the patient was discharged with a warning to avoid strenuous physical activities, especially those involving upper limbs and neck. No further episodes were reported in the subsequent six months.

These two women presented episodes of vertically inverted vision—upside down phenomena—associated with clinical signs and symptoms of vertebrobasilar insufficiency. Both reported transient visual inversion of 180 degrees, which was bilateral, of sudden onset and lacking subjective impression of movement (or of inversion).

In the first patient, neuroimaging revealed a right hemispheric cerebellar infarction. In the second, a vertebrobasilar failure due to a left subclavian stenosis was detected. The pathogenetic mechanism underlying upside down visual inversion is unknown. Since the visual images enter the retina inverted, it may be assumed that the upside down phenomenon is associated with a significant failure of the mechanisms mediating reinversion, even though the anatomical structures involved are unknown. In earlier observations,3 parietal and/or occipital lesions were sometimes associated with cortical origin of the dysfunction, probably affecting the integrative control of spatial vision. More recent cases,4,5 documented with neuroimaging techniques, revealed an association with vestibular/cerebellar lesions, that is, vertebrobasilar TIA’s, Wallenberg’s syndrome and also cerebellar infarct in two cases.6 In our patients, the relationship between the asymmetric visual inversion and the signs and symptoms of vertebrobasilar insufficiency, without evidence of cerebral damage, supports the idea that a transient inactivation of infratentorial structures may cause this visual phenomenon. Besides the integrity of the visual system, space visual perception needs a flow of extraretinal information, mediated by the vestibular and cerebellar systems.7 It has been suggested that damage to such structures may cause tilt and complete inversion of the visual space.8 The upside down phenomenon may occur following dysfunctions at various levels of these vestibulo-cerebellar-ocular system mediating the stabilization of the visual function so that cortical involvement is not indispensable.

References


Subcortical environmental reduplication: SPECT findings in a patient with a right thalamocapsular haemorrhage

Recently Nighoghossian et al1 reported the case of a patient with a previous history of a left fronto-basal haemorrhage, who developed environmental reduplication following an infarction of the retrolenticular portion of the right internal capsule. SPECT revealed right fronto-parietal cortical hypoperfusion. A similar dislocated misidentification syndrome was described previously in a patient with a right thalamic haemorrhage, but its functional correlate using SPECT was not studied.2 We describe the neuroimaging and cognitive functioning of a case of environmental reduplication associated with a right thalamocapsular haemorrhage.

A 71 year old ambidextrous man suddenly developed a left-sided weakness and mild dysarthria. He had had hypertension but no history of previous cerebrovascular events. Neurological examination revealed a dense left hemiplegia, and a left sensory loss affecting all modalities, were present, full, and there was no evidence of visual or auditory extinction on double simultaneous stimulation. He showed left hemispatial neglect on drawing, and on a letter cancellation task he only crossed targets on the right side of the paper. He did not deny his left hemiplegia, but he had a tendency to attribute it to previous "chest problems". He reported a feeling of nonlocalization of his paralysed left arm, and also said that he had three left legs and a strange left arm crossed over his chest. The patient said that he could walk almost normally and repeatedly tried to walk unaided despite his left hemiplegia. He was alert and oriented to time and person, but not to place. While he...
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A Stracciari, M Guarino, G Ciucci and P Pazzaglia

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